Mike W. Ross • Sue J. Dyson

Diagnosis and Management of

LAMENESS IN THE HORSE

[SAUNDERS]

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Abbreviations

AAEP	American Association of Equine Practitioners
ALARA	As low as reasonably achievable
ALDDFT	Accessory ligament of the deep digital flexor tendon
ANA	Antinuclear antibodies
AST	Aspartate aminotransferase
AVA	Arteriovenous anastomosis
BAP	Bone-specific alkaline phosphatase
BID	Twice daily
BSP	Bone sialoprotein
CdCr	Caudocranial
CdL-CrMO	Caudolateral-craniomedial oblique
Ci	Curie
CK	Creatine kinase
CNS	Central nervous system
COMP	Cartilage oligomeric matrix protein
COX	Cyclooxygenase
CrCd	Craniocaudal
CRGP	Calcitonin gene related peptide
CS	Chondroitin sulfate
CSA	Cross-sectional area
CSF	Cerebrospinal fluid
CT	Computed tomography
CTX	Collagen C telopeptides
CVM	Cervical vertebral malformation
DDFT	Deep digital flexor tendon
DFTS	Digital flexor tendon sheath
DIP joint	Distal interphalangeal joint
DL-PaMO	Dorsolateral-palmaromedial oblique
DL-PlMO	Dorsolateral-plantaromedial oblique
DM-PaLO	Dorsomedial-palmarolateral oblique
DM-PlLO	Dorsomedial-plantarolateral oblique
DMSO	Dimethylsulfoxide
DPa	Dorsopalmar
DPl	Dorsoplantar
DPr-PaDiO	Dorsoproximal-palmarodistal oblique
EDM	Equine degenerative myeloencephalopathy
EHVI	Equine herpes virus I
EIPH	Exercise-induced pulmonary hemorrhage
EL	Exercise level
EM	Electron microscopy
EMG	Electromyography
EPM	Equine protozoal myelitis
ER	Exertional rhabdomyolysis
EVA	Equine viral arteritis
FAS	Fiber alignment score
FEI	Federation Equestre Internationale
FFD	Focus film distance
GAG	Glycosaminoglycan
GBE	Glycogen binding enzyme
GBq	Giga becquerel
GRF	Ground reaction force
GSH	Glutathione peroxidase
HDP	Hydroxymethane diphosphonate
H & E	Hematoxylin and eosin
HU	Hounsfield unit
HyPP	Hyperkalemic periodic paralysis
ICR	Instant center of rotation
IM	Intramuscularly
IRU	Increased radiopharmaceutical uptake
IV	Intravenously
KS	Keratin sulfate
LDH	Lactate dehydrogenase
LF	Left forelimb
LH	Left hindlimb
LH	Luteinizing hormone
LM	Lateromedial
MAC	Minimum alveolar concentration
MBq	Mega becquerel
mCi	Millicurie
McII	Second metacarpal bone
McIII	Third metacarpal bone
McIV	Fourth metacarpal bone
MCP joint	Metacarpophalangeal joint
MDP	Methylene diphosphonate
MIC	Minimum inhibitory concentration
MMP	Metalloproteinase
MRI	Magnetic resonance imaging
MSD	Minimum sagittal diameter
MtII	Second metatarsal bone
MtIII	Third metatarsal bone
MtIV	Fourth metatarsal bone
MTP joint	Metatarsophalangeal joint
NKA	Neurokinin A
NPY	Neuropeptide Y
NSAID	Non-steroidal anti-inflammatory drug
OAAM	Occipitoatlantoaxial malformation
OC	Osteocalcin
PaPr-PaDiO	Palmaroproximal-palmarodistal oblique
PAS	Periodic acid—Schiff
PASM	Periodic acid—silver methanamine
PCR	Polymerase chain reaction
PET	Photon emission tomography
PHI	Peptide histidine isoleucine
PIP joint	Proximal interphalangeal joint
PMMA	Polymethylmethacrylate
PSB	Proximal sesamoid bone
PSGAG	Polysulfated glycosaminoglycans
PSSM	Polysaccharide storage myopathy
QH	Quarter Horse
RER	Recurrent exertional rhabdomyolysis
RF	Right forelimb
RH	Right hindlimb
RVI	Rubeola virus immunomodulator
SDFT	Superficial digital flexor tendon
SID	Once daily
SL	Suspensory ligament
SLE	Systemic lupus erythematosus
SP	Substance P
SPECT	Single photon emission computed tomography
STB	Standardbred
T1-T18	First to eighteenth thoracic vertebrae
TB	Thoroughbred
TENS	Transcutaneous electrical stimulation
TGF-β	Transforming growth factor β
TNF	Tumor necrosis factor
TS	Type or echo score
VIP	Vasoactive intestinal peptide
WBL	Warmblood

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FOREWORD

It has been said that "no one drinks from the same water when dipping from a fast river." Nothing is more true or better describes the fast flow of information that has come from veterinary medicine and surgery. I remember when I was a senior veterinary student watching a highly recognized equine specialist fire a horse with a large chip fracture of the distal, lateral radius. The horse was rested for sure and returned to training and did win. Yes, the old fracture became a large spur.

Any swelling of the fetlock joint was considered an osselet and the horse was treated either by blister or firing and blister. This was done until Dr. Ed Churchill found it best to remove the apex fracture from the top of the proximal sesamoid bone. Dr. Churchill's contribution inspired others such as Dr. Jacques Jenny and Dr. Charles Raker to perform surgery on horses with other fractures of the fetlock joint. Drs. Jenny and Raker were the first to remove a base fracture from the proximal sesamoid bone, to remove chip fractures from the carpus, and to reattach a slab fracture of the third carpal bone. These three men added a large clear stream to the veterinary river that many of us have used from the late '50s to the present. Although Dr. Jenny was the first to use a lag screw to repair a stress fracture of the third metacarpal bone and many advocate this form of management, I still find that deep firing well above and below the fracture site is more satisfactory. In horses that are fired, there is no pain from the screw and the screw does not have to be removed. Dr. M.B. Teigland used this method, deep firing, to treat many good racehorses with stress fractures with excellent success. It is important to remember that methods that have worked well for many years are not necessarily worthless or antiquated. Many times a great river comes from many small but important tributaries.

Other tributaries that helped swell the river were from the work of Dr. John Wheat on the subject of treating horses with tendonitis. He pointed out the necessity of preventing edema, which can lead to fibrosis of the injured tendon. Dr. James Rooney helped us better understand how lameness occurred, and Dr. Norman Rantanen and Dr. Ron Genovese were first to give us information on how to use ultrasound to better diagnose tendon and ligament injuries. Dr. Virginia Reef enlarged this tributary and made the use of the ultrasound more sophisticated in lameness diagnosis. Excellent radiologists such as Dr. Charles Reid at New Bolton Center and others have provided us with better knowledge when using our x-ray machines. The stream continued with better quality radiographs, xeroradiographs, scintigraphy, and recently, magnetic resonance imaging. These modalities have made us all better diagnosticians.

When it comes to treating lame horses, we have to remember the flow that came from Dr. Larry Bramlage and Dr. David Nunamaker. When arthroscopic surgery was first done by Dr. Steve Selway in Florida and by Dr. Wayne McIlwraith in Colorado, many thought that it should never be used on the horse. Both men forged a very wide tributary, one that has helped us all become more effective surgeons. After arthroscopic surgery, horses suffer less and many return faster to competition. Fear of something new is only normal and being skeptical is expected, since most of us have tried something new that just did not work. With any fast-moving river there will always be something picked up that is superfluous, but it will soon settle out.

The better one gets as a lameness diagnostician, the better he or she will be in selecting the best treatment. The many authors who have contributed to this book, *Diagnosis and Management of Lameness in the Horse,* have helped to clean false debris from the ever-increasing flow of veterinary knowledge and should be congratulated and thanked for sharing their knowledge and experiences with us. We will all be indebted to them after procuring and reading this book.

Both Dr. Mike Ross and Dr. Sue Dyson were well acquainted with me early in their careers during internship (Sue) and surgical residency (Mike) programs at New Bolton Center, University of Pennsylvania, at which I taught for 32 years. They both know very well that one does not become skilled in lameness diagnosis and management overnight. They both became quickly aware that lameness diagnosis in the gaited American Saddler or the Dressage horse was considerably different than in the racing Standardbred or Thoroughbred. For these reasons I am especially pleased that they have added a new dimension by providing actual videos on the CD showing the abnormal gaits of lame horses. This will be great for everyone.

I am confident that many will drink and benefit from the text, *Diagnosis and Management of Lameness in the Horse,* which should both widen and deepen the veterinary river.

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FOREWORD

The history of veterinary lameness diagnosis and treatment has gone through three stages.

The first stage could be termed the personal stage; there was principally anecdotal observation occasionally supported by post-mortem evidence. Much of this was limited to first- or second-hand accounts. The focus was on workhorses, with some information about cavalry mounts. Occasionally a gifted veterinarian was prescient and insightful. People like Horace Hayes and Dollar come to mind. They were very experienced horsemen and careful observers, with an intuitive insight into lameness. Unfortunately, intuition is not easily taught or passed on. The European veterinary literature was frequently dominated by pathologists, and the clinical findings were underemphasized.

The second stage, beginning in the 1960s, was characterized by the organization of lameness and a more scientific approach to diagnosis. Included was the use of diagnostic nerve blocks, radiography, and later, ultrasonography and scintigraphy. By this time the focus had shifted from the workhorse to the light horse for pleasure and competition. This was typified by the work of J. Hickman in the United Kingdom and O.R. Adams in the United States. There was also good work and valuable observations by individuals or groups. This was reported in the veterinary literature and in various proceedings, some even as letters to the editor. Unfortunately, some of these are not easily discovered through literature searches. There was also the stimulating writing of J. Rooney to explain lameness from a biomechanical perspective. This has been followed by the work of Clayton, Denoix, Barrey, and others. Much of the work pertaining to conformation and its effect on performance has never found its way into the veterinary literature and remains in hippological sources.

As so often happens in science, just when it appears that we are almost confident of our basic understanding, new observations question our assumptions. The past decade has seen a burst of new documented clinical investigation and anatomical knowledge. Some of the accepted and codified lameness beliefs are now in question. This includes nerve blocks. Unfortunately, rather than simplifying the subject, these careful observations have introduced complexities and uncertainties into what was previously regarded as a straightforward diagnostic procedure. As Dr. Sue Dyson was largely responsible for this, it is only apposite that she should make amends by way of contributing to this book. The same goes for Dr. Bowker.

The third phase is one of synthesis. It combines the latest technology and science with an understanding that the lame horse must be viewed from an athletic perspective. This often depends on the demands of specific sports. This book, *Diagnosis and Management of Lameness in the Horse* by Ross and Dyson, is the prototype treatise for this phase. An important premise of this book considers "lameness" in its most sophisticated form, to subsume all of the musculoskeletal and neurological dysfunctions that have an impact on maximal athletic performance. This is much more than is obvious by just trotting the horse to evince gait asymmetry. There is an understanding that it is frequent to have two or more "lamenesses" in an individual and that their etiology, effects on specific movements, and prognosis are interrelated and require an understanding of the competitive implications to best manage the horse. Thus the chapters by veterinarians experienced with different types of horses are an integral part of this book. The adjunctive approaches of acupuncture, manipulative procedures, physical, and herbal medicine are covered. It is hoped that the inclusion of these in a book with an impeccable scientific background will encourage practitioners to seek education in these modalities rather than be contemptuous prior to investigation. The Editors do not make the easy error of neglecting the clinical examination to focus principally on glamorous technology. All this culminates in maximum benefit to the veterinarian who must try to solve real-world problems. Hopefully the horse will also benefit.

The incorporation of a CD-ROM with moving pictures is so obvious it is surprising it has not been done before. Verbal description of a visual (and to a much lesser extent, auditory) phenomenon is a poor substitute. This is especially so since there is not yet even a general agreement of how to describe the alterations of movement associated with lameness. Another vital but often perplexing area is communication with the horse's connections. For example, many high-level dressage riders are acutely perceptive and analytical. Understanding is greatly facilitated by the veterinarian having a background in the field. One of the difficult problems is to decide whether a complaint about the horse's performance results from a "lameness"; is a training or riding problem, a shoeing problem, or a tack (saddle, bitting, hobbles, boots) problem; results from the horse's character and physiology; is an inherent biomechanical limitation for this individual; or even exists at all. There is a human tendency to discount what we cannot directly perceive. If the veterinarian cannot see any lameness or deficit in movement, then there is an inclination to assign the problem to being in the rider's head," or "the horse is just not good enough." Now, the latter are real possibilities. How does one decide? There are many strategies: a course of non-steroidal anti-inflammatory analgesic drugs, treating suspected locations, blocking suspicious areas, incorporating acupuncture and chiropractic evaluation and treatment, or changing the training, the rider, the going, the shoeing, the tack, and so on. However, if the veterinarian is also an educated and a sensitive rider, who can get on the horse and interpret the feelings in biomechanical and diagnostic criteria, then you have someone like Sue Dyson. She is the rare combination of a high-level rider and horsewoman *and* a talented veterinarian. I can speak of Sue's abilities because we have been friends and colleagues for over 20 years. I admire her for her veterinary and equestrian abilities, which include a rigorously honest intellectual approach with a tough dedication. Dr. Mike Ross I know through his many publications and lectures and the high standard of his academic work at a leading university and his pursuit of being in the forefront. His particular interest in Standardbred racehorses and methodical approach have provided a new perspective to lameness in these horses. This complementary combination of talent and experience make Mike and Sue ideal co-Editors.

Notwithstanding Churchill's comment about being divided by a common language, here is a successful collaboration between Editors on both sides of the pond. The incorporation of leading American, Australian, and European veterinarians as co-authors adds additional perspective. The nature of this book promises regular updates to include forthcoming knowledge. One hopes that the Editors' schedules will permit this.

Daniel Marks, VMD
August 2002
Santa Fe, New Mexico

PREFACE

We come from different equine, veterinary, and cultural backgrounds, but we share a passion for lameness diagnosis and have individually developed very similar diagnostic approaches, believing that there is both art and science to lameness diagnosis. In the past 20 years we have seen huge advances in our knowledge of conditions that cause lameness through careful clinical observation combined with the more stringent use of diagnostic analgesia. It was with consideration of a strong belief in the values of clinical examination and diagnostic analgesia that our list of contributors was formulated. We have experienced advances in diagnostic imaging with the development of ultrasonography, scintigraphy, diagnostic arthroscopy, and more recently, computed tomography and magnetic resonance imaging (MRI). Doubtless, while we continue to look, we will continue to find new causes of lameness and this text will rapidly become outdated. There have even been significant developments from the time of manuscript submission to completion of production of the book, especially in the field of MRI. It is nonetheless our aim to provide information as up to date as possible, based on our own experiences and those of other experienced veterinarians worldwide.

When we embarked on this project, we felt strongly that the emphasis on different lameness conditions in other texts did not reflect our own personal experiences and it did not reflect the differences seen in horses used for a variety of sports disciplines. We set out to provide a comprehensive, thought-provoking text, viewed from a global perspective, sometimes challenging long-held dogma. We wanted to provide a balanced approach, reflecting as much as possible our own personal experiences based on careful observation while also including the opinions of other respected lameness diagnosticians in areas where we believed we did not have sufficient experience or expertise.

We selected the term *management of lameness* carefully, recognizing that our treatment abilities are limited and currently fall behind our diagnostic capabilities. We have tried to be as broad minded as possible, including many aspects of alternative therapy. We recognize that many of these treatment modalities have not undergone scientific scrutiny, and they do not necessarily form part of our own therapeutic armamentarium. Although we are not implying endorsement, we felt that the experiences of others should be chronicled.

We were fortunate to enlist a broad cross-section of coauthors, with widely ranging experiences, but regret that several notable veterinarians were unable to accept because of other commitments. We challenged all authors to provide practical information, reflecting not only the current literature but also their own experiences. However, when our experiences were strongly at variance with an author, we have added editorial comments accordingly.

The book is divided into five major sections, first presenting an in-depth diagnostic approach, then dealing with lameness conditions based on anatomical location. Different disease entities are then considered in depth, followed by a broad review of different treatment modalities. The final section addresses different types of horses and the injuries to which they are particularly prone, providing a unique insight into different diagnostic and therapeutic approaches. It also provides a background to each sport, which is so important in understanding why particular problems occur and how they may be manifested. Inevitably, this approach creates some duplication of information, but often there are subtle and fascinating differences among authors based on the type of horse with which they deal and where in the world they practice. To encompass all this, of necessity we sacrificed didactic chapters on anatomy, and we have not included a comprehensive review of all standard radiographic views and surgical procedures. We believe that other dedicated texts are better suited to provide this information.

We tried to be as generous as possible with page allocation to authors but found that we had hugely outstripped the total page allocation for the book. To try to reduce the text, we introduced many abbreviations, which some readers will find difficult. Therefore we have included a list of these abbreviations for easy reference. Space limitations made it necessary to limit the number of figures, which we regret. Ideally, we would have liked to provide examples of all conditions, but this was impractical. Good and useful examples have been chosen.

Although the title of the book is *Diagnosis and Management of Lameness in the Horse,* we have tried to address all potential musculoskeletal and neurological causes of gait abnormalities or changes in performance of sport horses. We have addressed the common problem of what to do when a diagnosis cannot be made.

We believe strongly that accurate diagnosis of lameness relies heavily on clinical experience and the development of an eye for the horse, both stationary and in movement. Lameness diagnosis cannot be taught in the classroom but must be learned from many hours of observation of normal and lame horses and tutelage. We have therefore included a CD-ROM, showing short video clips of a variety of lameness conditions, which we hope are representative examples and will assist an inexperienced clinician in developing an eye for lameness.

When we embarked on this project, we were both keen to learn from other peoples' experiences and to broaden our own horizons, and we have not been disappointed. We have learned a huge amount, both from each other and from contributing authors. We remain constantly excited by the challenges of lameness diagnosis and management, since many horses do not entirely fit the textbook picture. Nonetheless, we are also sometimes frustrated that a diagnosis eludes us. We hope that by continued careful observation and a logical, comprehensive diagnostic approach, in the future our knowledge will further expand to understand causes of lameness and altered performance better and to provide further information for the next edition of *Diagnosis and Management of Lameness in the Horse.*

Sue J. Dyson
Mike W. Ross
Suffolk, United Kingdom, and
Kennett Square,
Pennsylvania
2002

ACKNOWLEDGMENTS

We owe a huge debt of gratitude to our co-authors, since the scope of this book far exceeds our combined knowledge and experience. Writing a book chapter brings no financial rewards and the time involved always far exceeds expectations. While it is invidious to single out specific authors, we are particularly grateful to Ron Genovese, who contributed large sections on ultrasonographic examination and soft tissue injuries and also gave constant encouragement through the long gestation period of the book. We were tremendously excited when our colleague, co-author, and friend Fabio Torre agreed to help with our cover. His original paintings reflect the unique approach we have taken to review our beloved subject. Fabio's illustrations, based on one of the earliest analytical assessments of gait ever to be made by Muybridge, encourage us to both look and see.

We have received tremendous support from Karena Bean and Sally Whitefield, secretaries at the Animal Health Trust, who deciphered many editorial hieroglyphics and were responsible for cheerfully and tirelessly re-typing the majority of manuscripts. We thank Joyce Underwood, Vivian Stacy, and Karyn Van Norman from New Bolton Center for their help and support. We especially thank Jan Butler and John Wilkinson, who have produced a large number of the photographic illustrations, and Alex Baker for her excellent artwork.

The book would not have been possible without key people from WB Saunders Company: Stephanie Donley, Ray Kersey, Arlene Chappelle, Denise LeMelledo, Judy Ahlers, Julia Dummitt, David Saracco, and Bruce Robison.

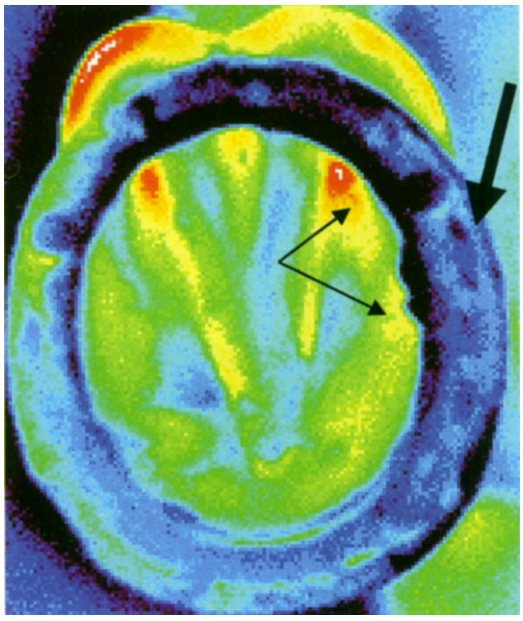
We have both been influenced and tutored by notable people in our careers. Sue's early teaching in equine orthopaedics came from John Hickman at the University of Cambridge, who was later a consultant at the Animal Health Trust. John Ayliffe, an equine practitioner and lameness enthusiast, encouraged Sue to go to the United States, where she spent a year at New Bolton Center and a year in practice. This was initially made possible through a Thouron Scholarship, one of the principal aims of which is to foster Anglo-American relationships. This book, with Sue and Mike as co-Editors, is a true fulfillment of that goal. Charlie Reid at the University of Pennsylvania stimulated an interest in diagnostic imaging, while Bill Moyer, Dan Marks, and Midge Leitch were all hugely influential in the development of an approach to lameness diagnosis. A little later Ron Genovese shared his knowledge, experience, and enthusiasm for ultrasonography. Colleagues, past and present at the Animal Health Trust, notably Svend Kold, Ian Wright, Chris Whitton, and Michael Schramme, have provided stimulus and support. The interns, by testing the hypothesis that by teaching you learn, have been a constant inspiration to improve diagnostic capabilities, to advance knowledge, and to distill clarity of thought. McGinty, Kinvarra, and Otterburn were internationally famous horses in their own right. As horses produced by Sue, they were great teachers in the art and science of equestrianism and in the diagnosis and management of lameness.

For Mike, John "Jack" Lowe of Cornell University, whose support and encouragement were unequalled, instilled an early interest and passion for lameness. Jack allowed him the freedom of thought and provided a solid foundation of clinical investigation and experience on which to build. He had excellent instructors, colleagues, and friends at the New York State College of Veterinary Medicine, Cornell University as a student and intern, and from New Bolton Center, University of Pennsylvania as a surgery resident, lecturer, and faculty member, but special thanks go to Norm Ducharme, Dean Richardson, and David Freeman. Loren Evans, mentor, lameness diagnostician, horseman, surgeon, and friend, had a unique influence, instilling confidence and imparting knowledge but most importantly in showing him the value of careful clinical examination and observation. Mike owes tremendous gratitude to Howard "Gene" Gill, Ben Martin, Jim Palmer, Paul Nolan, and Ron Gurfein for their loyalty and guidance, and the Standardbred racehorse, which allowed him to "cut his teeth" and taught him so much. Mike has been motivated by the many dedicated interns and residents and referring veterinarians, whose ideas, energy, and enthusiasm for his work throughout the years have truly been inspirational.

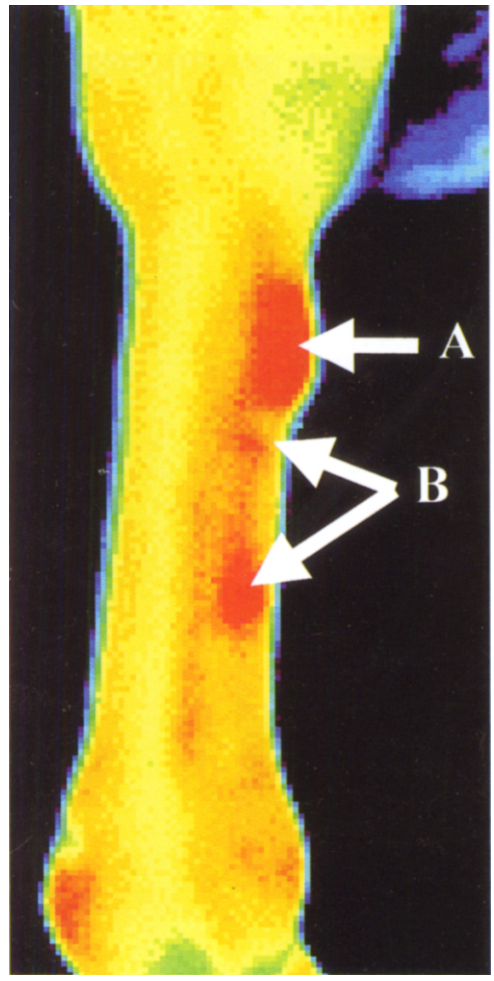
We must not forget the horse. Watching, palpating, listening to, and trying to understand the horse has taught us so much.

Finally we owe an enormous thank you for the love, friendship and support of our partners, John and Debbie, themselves veterinarians. They have been extraordinarily patient and understanding throughout this huge project, have provided constructive criticism, have listened to our frustrations, and have shared our enthusiasm and passion for the book. Mike gives special thanks to Stone for his patience and love, and the inspiration only a son can endow, his daughter Kennedy, and his parents.

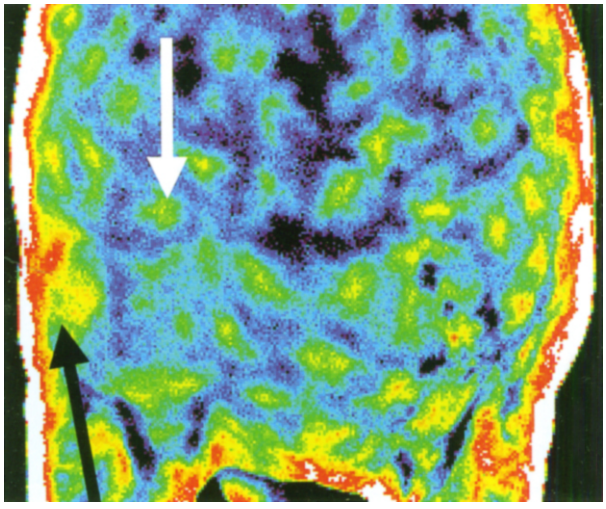
Sue Dyson and Mike Ross



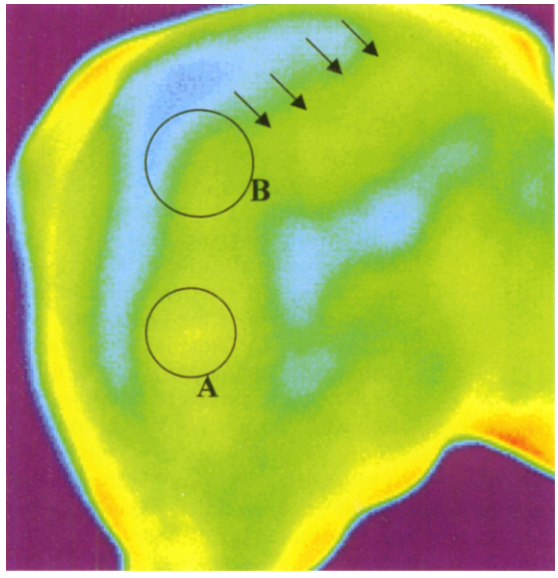
Color Plate 1 Thermal image of the sole of a horse's foot showing asymmetrical heat in the sole of the hoof (*small arrows*). The shoe on that side (*bold arrow*) is colder, indicating less friction on the shoe, probably as a result of avoiding the injured sole.



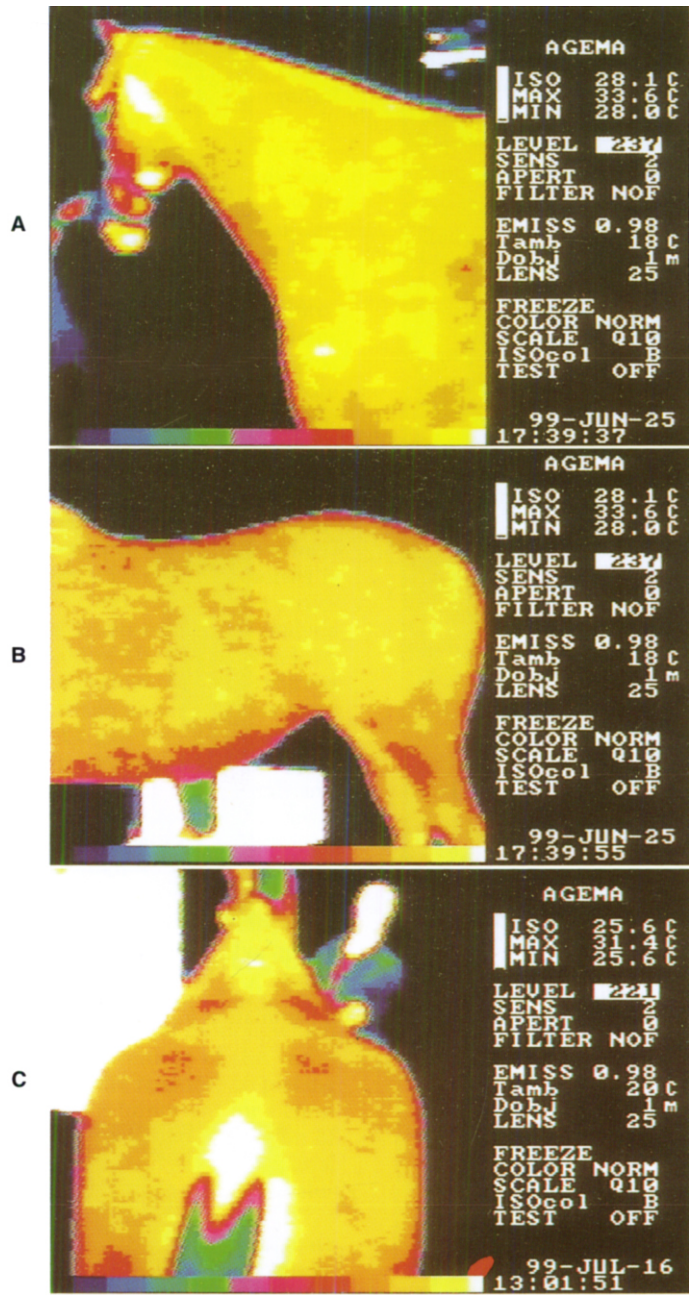
Color Plate 2 Thermal image of the left medial metacarpal region showing an active splint (*A*) that is causing secondary inflammation in the suspensory ligament distal to the injury (*B*).



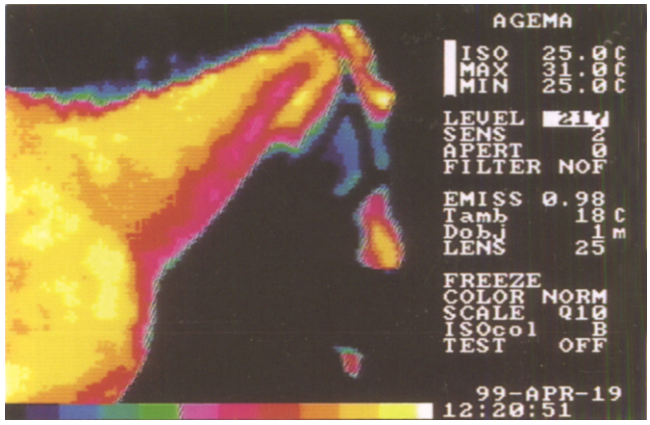
Color Plate 3 Thermal image, front view, of a horse's chest showing increased heat over the lateral aspect of the right shoulder (*black arrow*) and decreased heat over the right bicipital tendon (*white arrow*). Ultrasonography showed increased fluid in the intertubercular (bicipital) bursa. The decreased heat over the muscle was presumed to be due to lack of use because of pain.



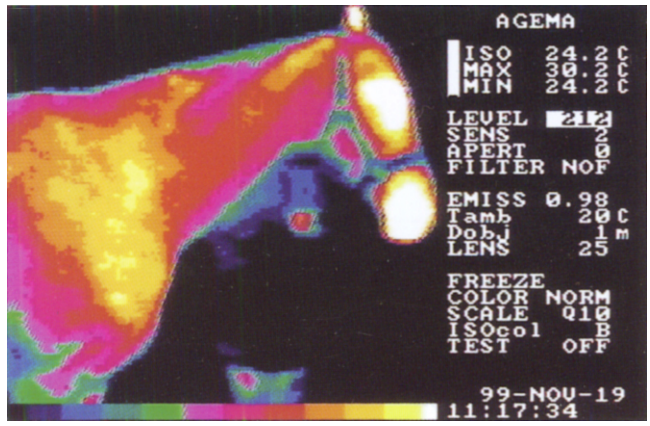
Color Plate 4 Thermal image of the lateral view of the right hip region showing increased heat over the third trochanter (*A*) and greater trochanter (*B*). In addition, the body of the gluteal muscle has increased heat (*small arrows*). This condition was diagnosed as a gluteal muscle strain.



Color Plate 5 Thermographs show the appearance of normal horses. The color scale represents 5.6° C, using 10 colors; that is, each color represents about a 0.5° C change in temperature. The color bar under the image shows the colors used; those to the right-hand side are the hottest and those to the left are the coldest. White is above the top of the scale and black below the scale. **A,** Lateral view of the head, neck, and shoulder. **B,** Lateral view of the thorax, abdomen, and hindquarters. **C,** Oblique dorsal view from behind the horse looking toward its head and neck. Temperature variation is 1° C over the neck, trunk, and hindquarters. A warm midline dorsal stripe along the back extends from the withers to the base of the tail, with symmetrical muscle temperature on either side.

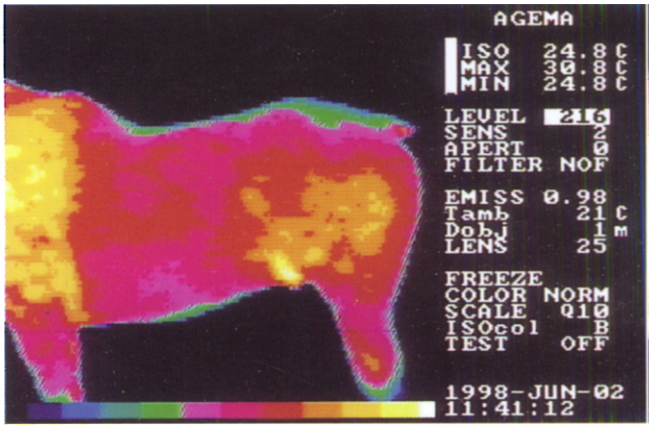


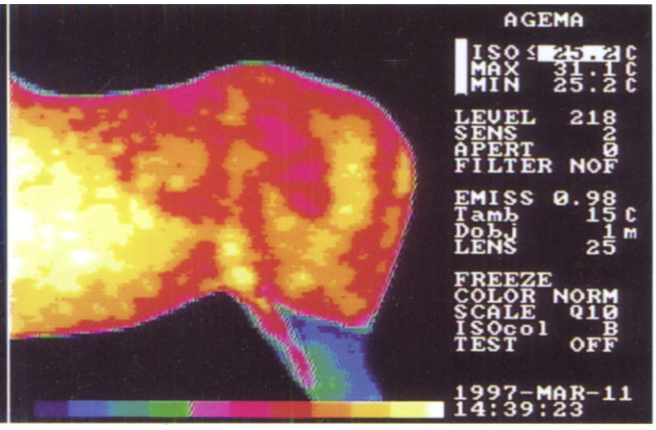
Color Plate 6 Lateral view of the neck of horse with reduced mobility in the occipito-atlantal and atlanto-axial joints. A cool line runs obliquely from the region of the atlanto-axial joint caudally to the base of the neck and is 1.5° C cooler than the surrounding muscle, indicating an area of sympathetic dystonia.



Color Plate 7 Lateral view of the neck of horse with reduced mobility in the neck. Note the significant cooling from the occiput back to the level of the sixth and seventh cervical vertebrae, indicating a problem involving all joints in the neck.

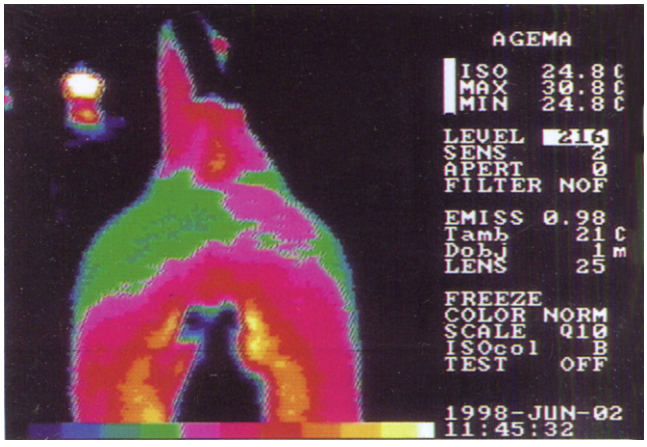
A



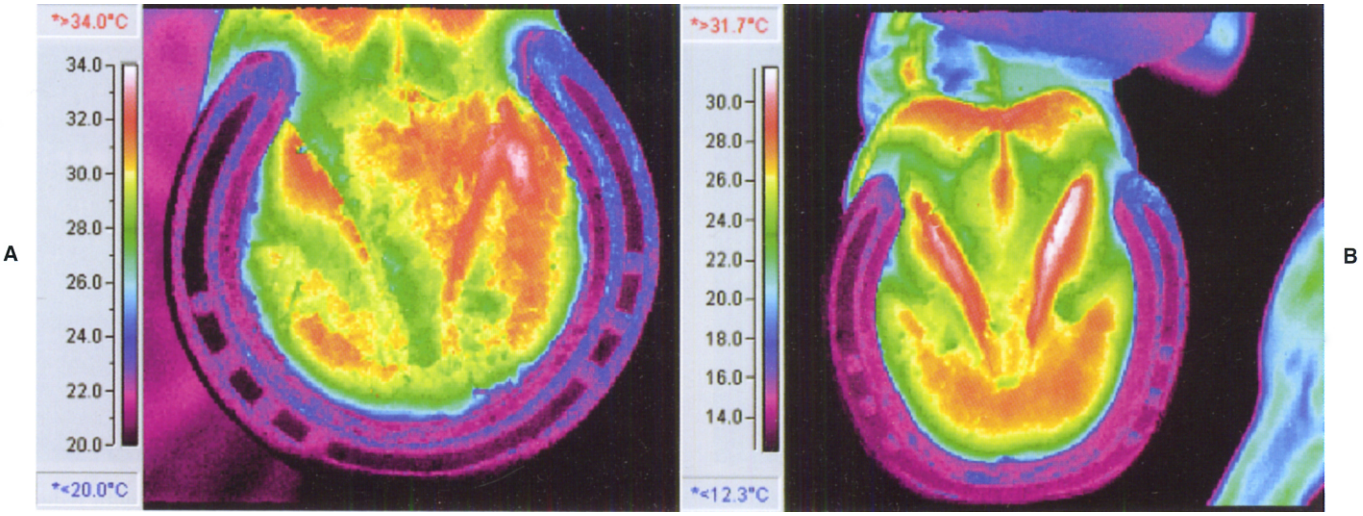


B

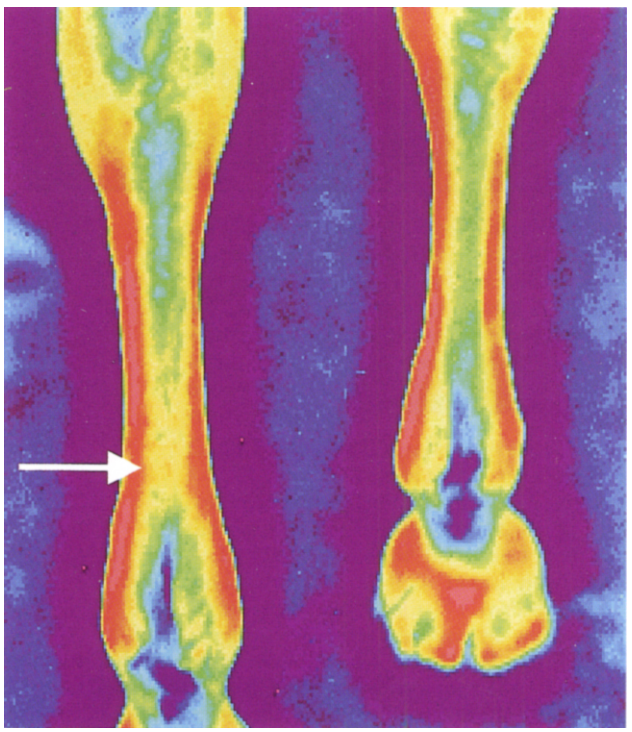
Color Plate 8 Lateral views of the thoracolumbar area of two horses, both showing significant cooling in the musculature of the dorsal spine from the saddle region caudally. **A,** The vertical cranial boundary to the zone of cooling indicates that the injured area is in the region of the cervicothoracic junction, but the shoulder muscles overlying this area receive innervation from the lower neck, partly masking the muscles supplied by the upper thoracic area. **B,** The typical appearance of cooling in the muscle resulting from an injury to the region of the twelfth thoracic vertebra, with the cranial border of the region running obliquely down and back. This horse has been clipped, but long hair left on the hind limb shows as an area 5° C cooler than the surface temperature of the thorax of the horse.



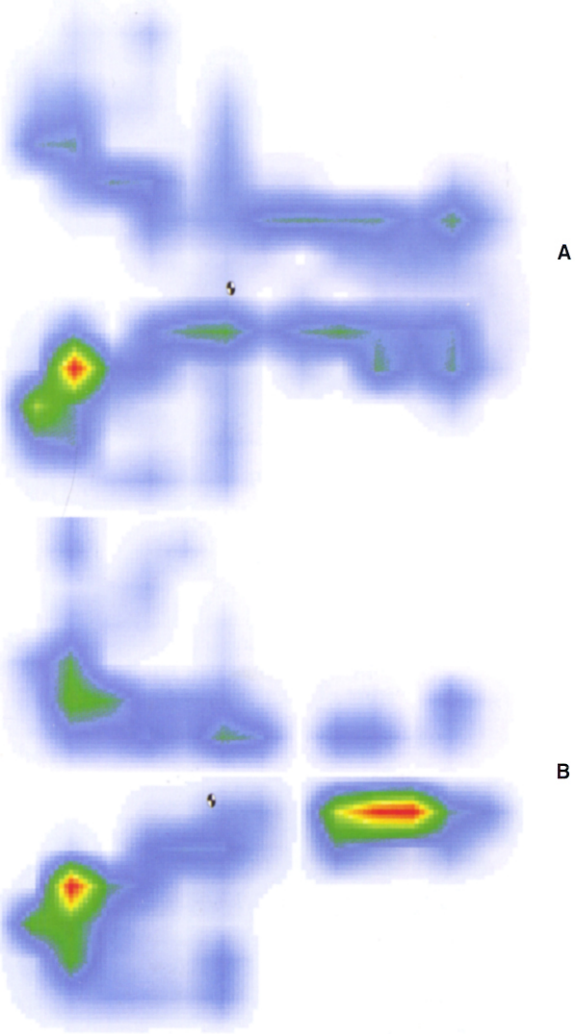
Color Plate 9 Dorsal view of horse showing abnormal heat patterns of the thoracolumbar spine. Note the complete loss of the normal central stripe, with significant cooling indicating the presence of reduced mobility of the entire thoracolumbar spine and pelvis. The asymmetrical nature of the heat pattern indicates the horse is likely to move with an asymmetrical gait, resulting from increased muscle tone on the left-hand side of the body.



Color Plate 10 Examples of different thermographic foot patterns (solar views). **A,** This hoof has a medial corn, manifested as a focal hot spot (*white*) within an area of increased temperature. **B,** This hoof has subacute laminitis, with a pattern of increased heat in the region of the tip of the distal phalanx.



Color Plate 11 Palmar thermographic image and subsequent transverse (on the left) and longitudinal ultrasonographic images of an advanced event horse 10 days after successfully completing a Three Day Event. The horse was having a routine examination, and no clinical localizing signs were evident in the tendon. The thermogram demonstrates a focal hot spot over the left distal superficial digital flexor tendon (*arrow*), and the ultrasonographic images reveal a hypoechoic core lesion in the same region.



Color Plate 12 Computerized saddle pressure analysis images. Cranial is to the left, and left is to the bottom. **A,** This image shows a poorly fitting saddle, with a focal pressure point in the left wither region. **B,** This image demonstrates failure of a gel pad to alleviate the pressure point and the development of an additional pressure point caudally.

SECTION • 1
The Lameness Examination

CHAPTER • 1

Lameness Examination: Historical Perspective

Mike W. Ross

If your horse is lame in his shoulder, take off his shoes…Young and inexperienced practitioners are quite too apt to commit the error of overlooking the examination of the foot, looking upon it as a matter of secondary importance, and attending to it as a routine and formal affair only.

A. Liautard, 1888[1]

As the twenty-first century begins, the extent of change in the diagnosis of lameness in the horse depends on the individual's clinical and ideological perspective. A veritable explosion of new imaging capabilities has advanced the current understanding of many musculoskeletal abnormalities. Yet, to accurately assess clinical relevance, the clinician must possess a feel for the horse, developed only by careful clinical examination, a procedure that has changed little in hundreds of years. Successful detection of equine lameness does not so much require knowledge of science as it does art. Inasmuch as *art* is defined as "skilled workmanship, craft, or studied action,"[2] the lameness examination demands artistic experience acquired by years of clinical practice and working and learning from experienced practitioners. From Liautard's advice more than 100 years ago to that of modern lameness diagnosticians, the change in the basic skills of lameness diagnosis may be small.

Development of the artistic skills needed to become a true lameness diagnostician requires a thorough, somewhat methodical approach, much like that of a crime scene detective. I often refer to the lameness diagnostician as a lameness detective, and although this statement may lack sophistication, in reality, how boring the task would be if the horse could talk. To make a horse talk to you through careful palpation and observation is the essence of the lameness examination yet the most difficult to teach. Great lameness diagnosticians likely possess this ability to read or feel the horse and skilled, workmanship-like qualities to appreciate the art in lameness diagnosis. Some, with the added ability to share this knowledge effectively, have influenced clinicians more than others simply by writing about those experiences. In the mid- to late 1900s, Adams had the most profound influence by his teachings and writings. His former students and friends acknowledge his artistic talent, gained primarily from a ground-up approach to the lame horse, and his profound interest in corrective shoeing. More important, Adams' original lameness notes became his classic textbook.[3] For most clinicians, *Lameness in Horses* represented the "lameness Bible," an excellent resource of information on equine lameness. Adams himself revised the textbook several times; most recently, his respected colleague, Ted S. Stashak, has continued in Adams' footsteps. This important work served as the foundation for the fundamentals of equine lameness.

Adams was influenced greatly by the work of Dollar and Lacroix. Adams' original notes contain many drawings similar to those originally published in Dollar's *A Handbook of Horseshoeing*,[4] a wonderful collection of drawings and excellent descriptions of shoeing, conformation, and lameness of the equine foot. Adams references the work of Lacroix[5] in the late 1800s. In fact, until Adams' treatise on lameness, scant information existed about equine lameness. The information available in the American literature during most of the 1900s consisted of only sporadic case reports or case series in the *Journal of the American Veterinary Medical Association*. A potential explanation may lie in the importance of the World Wars or other important social events in the early to mid-1900s. Experience in the cavalry also may have influenced later writings in the 1900s.

Peters'[6] work detailing lameness in the Thoroughbred racehorse emphasized the importance of lameness in the racetrack practice and the most common cause of poor performance. Many problems he observed in 1939 still exist, although treatment options have expanded considerably. Early important writings included manuscripts by Churchill[7] and Wheat and Rhode[8] on surgical removal of proximal sesamoid bone fractures (the Churchill approach), Forsell[9] on surgical management of navicular bursitis and tendonitis, and Lundvall[10] and later Delahanty[11] debating the subject of the existence or nonexistence of fibular fractures. An early reference of note was the surgical textbook by Frank.[12] Originally written in 1939, with several subsequent editions, this influential and often quoted textbook contained information about numerous musculoskeletal problems and often sensational examples of common and rare abnormalities.

In the late 1800s, several informative, interesting, and entertaining textbooks about equine lameness were written, primarily by European authors. Most publications contained wonderful descriptions of lame horses, and many emphasized shoeing techniques, a mainstay in management of the lame horse both then and now. The writings of Percivall[13] and Gamgee[14] are particularly informative. Although a definitive reason was not provided, Gamgee observed that 42% of horses in the United Kingdom were lame, whereas only 9% of horses in Paris were lame. Disorders of the foot, many of which increased in frequency with age, were most common, and marked remodeling of the distal phalanx was seen in horses undergoing postmortem examination.[14] In addition to the time-honored management technique of shoeing the lame horse, conformation and its relationship to lameness also were emphasized. In *How to Judge a Horse,* Bach[15] emphasized balance, body part length and angulation, and distal extremity conformational faults. In a chapter entitled "Horse-Docturing in the Nineteenth Century," Dunlop and Williams[16] emphasized the contribution of Mayhew, described as artist, activist, and veterinary surgeon. Mayhew described and illustrated many common abnormalities of the locomotor system recognized at that time, including splints, spavin, curb, tendon sprains, and thoroughpin, most of which are still recognized today.[16] Of interest, Mayhew was credited for trying "experimental" injections into inflamed areas, an obviously important

treatment modality practiced today.[16] Detailed descriptions of laminitis, navicular disease, and other common conditions of the equine foot were provided.[16] Dunlop and Williams,[17] in their treatise on the history of veterinary medicine, also detailed the transition from farriery to veterinary medicine that occurred in the 1700s, although the close association and harmonious working relationships between blacksmiths and equine diagnosticians remain integral parts of a successful lameness management team today. In fact, "the term veterinarian came into use when colleges were established in different parts of Europe for improving, or rather for creating the art of treating disease in the lower animals."[17] The first veterinary school was founded in France in 1761, and soon veterinary schools were formed in the United Kingdom.[17]

Although an exhaustive historical review might be interesting, this brief review highlights critical issues central to modern lameness diagnosis. First, the basics have not changed for hundreds of years and will likely not change in the foreseeable future. Second, with the exception of Adams' work, few comprehensive reports on lameness diagnosis were written in the 1900s. The modern lameness detective likely has learned most from experience working with accomplished lameness diagnosticians and by word of mouth. Third, many of the most knowledgeable colleagues have not published writings but have made their contributions in day-to-day teachings in academic settings, private practice, and small gatherings at national meetings.

Since 1955 the annual convention of the American Association of Equine Practitioners has played a special role in the dissemination of information and ideas about lameness. Early meetings included a handful of practitioners, gathering and discussing equine medicine and surgery, sometimes late into the night. Much current lameness experience can be traced to these early meetings and practitioners such as Adams, Peters, Frank, Farquharson, Churchill, Goddall, Gabel, and Delahanty. Loren Evans and Howard "Gene" Gill influenced the molding of many modern lameness detectives, including me. Emphasizing the value of acquiring horse sense and spending time palpating and "learning" the horse, Gill often quotes Will Rogers, "…the outside of a horse is good for the inside of a man."

In the United Kingdom the British Equine Veterinary Association was established in 1961, providing a similar formula for dissemination of information through its annual congress and regular day meetings. The establishment of the *Equine Veterinary Journal* in 1968 provided a high-quality, refereed journal. The standard for the journal was set by the first editor, John Hickman, an astute observer of lame horses and an influence on many practitioners.

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CHAPTER • 2

Lameness in Horses: Basic Facts before Starting

Mike W. Ross

DEFINITION

Lameness is therefore not so much an original evil, a disease per se, as it is a symptom and manifestation of some antecedent vital physical lesion, either isolated or complicated, affecting one or several parts of the locomotive apparatus.—A. Liautard, 1888[1]

The clinical manifestations of lameness in the horse are well known, but an exact definition is difficult. The word *lame* is an adjective, meaning "crippled or physically disabled, as a person or animal,…in the foot or leg so as to limp or walk with difficulty."[2] A medical dictionary defines *lameness* as "incapable of normal locomotion, deviation from the normal

treatment modality practiced today.[16] Detailed descriptions of laminitis, navicular disease, and other common conditions of the equine foot were provided.[16] Dunlop and Williams,[17] in their treatise on the history of veterinary medicine, also detailed the transition from farriery to veterinary medicine that occurred in the 1700s, although the close association and harmonious working relationships between blacksmiths and equine diagnosticians remain integral parts of a successful lameness management team today. In fact, "the term veterinarian came into use when colleges were established in different parts of Europe for improving, or rather for creating the art of treating disease in the lower animals."[17] The first veterinary school was founded in France in 1761, and soon veterinary schools were formed in the United Kingdom.[17]

Although an exhaustive historical review might be interesting, this brief review highlights critical issues central to modern lameness diagnosis. First, the basics have not changed for hundreds of years and will likely not change in the foreseeable future. Second, with the exception of Adams' work, few comprehensive reports on lameness diagnosis were written in the 1900s. The modern lameness detective likely has learned most from experience working with accomplished lameness diagnosticians and by word of mouth. Third, many of the most knowledgeable colleagues have not published writings but have made their contributions in day-to-day teachings in academic settings, private practice, and small gatherings at national meetings.

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gait."[3] The noun *lameness* can be but infrequently is used interchangeably with *claudication*, described as "limping or lameness."[3]

Lameness is simply a clinical sign, a manifestation of the signs of inflammation, including pain, or a mechanical defect, that results in a gait abnormality characterized by limping. The definition is simple, but recognition, localization, characterization, and management are complex.

LOCALIZATION OF PAIN

In certain conditions, characteristic gait abnormalities allow immediate and straightforward recognition and localization of the problem. Sweeny, fibrotic myopathy, upward fixation of the patella, stringhalt, shivers, and radial nerve paresis are examples. However, similar gait deficits exist for a variety of lameness problems, complicating recognition and localization. A fundamental concept in lameness diagnosis is the application of diagnostic analgesic techniques to localize the source of pain causing lameness. The sequence of properly determining the lame leg (recognition) and then abolishing the clinical sign of lameness by use of diagnostic analgesia (localization), only to have lameness return when the local anesthetic effects abate, is essential for accurate diagnosis. With experience and under certain circumstances, this step in lameness diagnosis can be omitted. The degree of lameness, certain gait characteristics, and palpation findings allow the clinician to strongly suspect a certain diagnosis. The next step may be diagnostic imaging. Trial and error also occasionally work and in some instances may be the preferred approach. However, because pathognomonic signs are rare, proficiency in diagnostic analgesic techniques is mandatory for the lameness diagnostician.

BASELINE AND INDUCED LAMENESS

Baseline, or primary, lameness is the gait abnormality recognized when the animal is examined at a walk or trot in hand, before flexion or manipulative tests are used. The clinician usually recognizes this abnormality by watching the horse on a firm or hard surface, while it is being trotted in a straight line. Diagnostic analgesia is used to abolish this lameness. Changing the surface or nature of the exercise by lunging, or circling the horse at a trot in hand, potentially changes the baseline lameness. The surface and exercise (gait and speed) must be consistent. In some horses, no observable lameness is present at a walk or trot in hand. Lameness may be evident when the horse is ridden, and this lameness becomes the baseline lameness.

Flexion tests and other forms of manipulation are used to exacerbate baseline lameness or to induce lameness. An induced lameness is one that is observed after flexion or manipulative tests, but induced lameness may not be the same as the baseline lameness. Manipulative tests are expected to, and often do, exacerbate the primary lameness. However, flexion and manipulative tests can cause development of additional lameness, unrelated to the primary or baseline lameness, and test results must be interpreted carefully.

COEXISTENT LAMENESS

Horses often have several sites of pain, although one usually is most obvious and the cause of baseline lameness. In many horses, secondary or compensatory (sometimes referred to as *complimentary*) lameness develops in predictable sites or limbs. Concomitant bilateral forelimb or hindlimb lameness is common, but horses often demonstrate more prominent clinical signs in one limb or the other. In horses with palmar heel pain, initially pronounced single forelimb lameness that is abolished by palmar digital analgesia may be present, with subsequent recognition of contralateral forelimb lameness. In racehorses, bilateral lameness, such as in the carpi or metacarpophalangeal or metatarsophalangeal joints, is common. The clinician should carefully examine the contralateral limb. Predictable compensatory or secondary lameness often exists in the ipsilateral or contralateral forelimb, when primary lameness is present in the hindlimb, or vice versa. In a Thoroughbred (TB) racehorse with left forelimb lameness, compensatory problems in the right forelimb and left hindlimb are not uncommon, because these limbs presumably are succumbing to excessive loads, while protecting the primary source of pain. In a trotter, diagonal lameness often occurs (primary lameness in the left hindlimb and compensatory lameness in the right forelimb), whereas in pacers, ipsilateral lameness is most common (primary right forelimb and compensatory right hindlimb). When several limbs are involved, identification of the primary or major source of pain is important. If forelimb and hindlimb lameness exist simultaneously, diagnostic analgesic techniques should begin in the hindlimb (see Chapter 10). A common secondary lameness abnormality, proximal suspensory desmitis, can develop in the compensating forelimb or hindlimb.

LAMENESS DISTRIBUTION

Among all types of horses, forelimb lameness is more common than hindlimb lameness. A horse's center of gravity or balance, while dictated to a certain extent by conformation (see Chapter 4), is not located in the center of the horse but is closer to the forelimbs than the hindlimbs. Thus the forelimb/hindlimb (F/H) weight (load) distribution ratio is approximately 60%:40% (Fig. 2-1). Higher loads are expected on the individual forelimbs (30% each), predisposing the horse to greater injury.

At certain times during the stride cycle of gaits such as the canter (three-beat gait) and gallop (four-beat gait), a single forelimb is weight bearing, which predisposes the limb to injury. The weight of a rider may shift F/H load distribution to 70%:30% (Fig. 2-2). Two-beat gaits, such as the pace and trot, allow more equal load sharing between forelimbs and hindlimbs because a forelimb and hindlimb (ideally, if the gait is balanced perfectly) hit the ground simultaneously. In pacers and trotters the proportion of forelimb lameness is less. The added load of pulling a sulky, cart, or any heavy load increases the likelihood of hindlimb lameness in Standardbreds (STBs), other harness breeds, and draft horses (Fig. 2-3). The F/H distribution of lameness in STB racehorses is 55%:45%. Sporting activities such as dressage and jumping also may shift lameness distribution to the hindlimbs because collection (working off the hindlimbs) and propulsion needed by horses to perform these activities may predispose to hindlimb lameness.

In the forelimb, up to 95% of lameness problems occur at the level of or distal to the carpus.[4] The distal parts of the limb always should be excluded as a potential source of lameness before the upper limb is addressed, although many owners believe otherwise, and may try to mislead an inexperienced practitioner. The foot should be suspected first. Pain in the foot is one of the most common causes of forelimb lameness in all types of horses, except draft breed horses, in which the foot is the most common site of pain in the hindlimb.

Hindlimb Lameness

Hindlimb lameness should not be underplayed, although its recognition is more difficult. In the forelimb lameness–prone TB racehorse, many hindlimb lameness problems are overlooked. However, a rider or jockey often may suspect a hindlimb problem when the lameness actually exists in the forelimb.

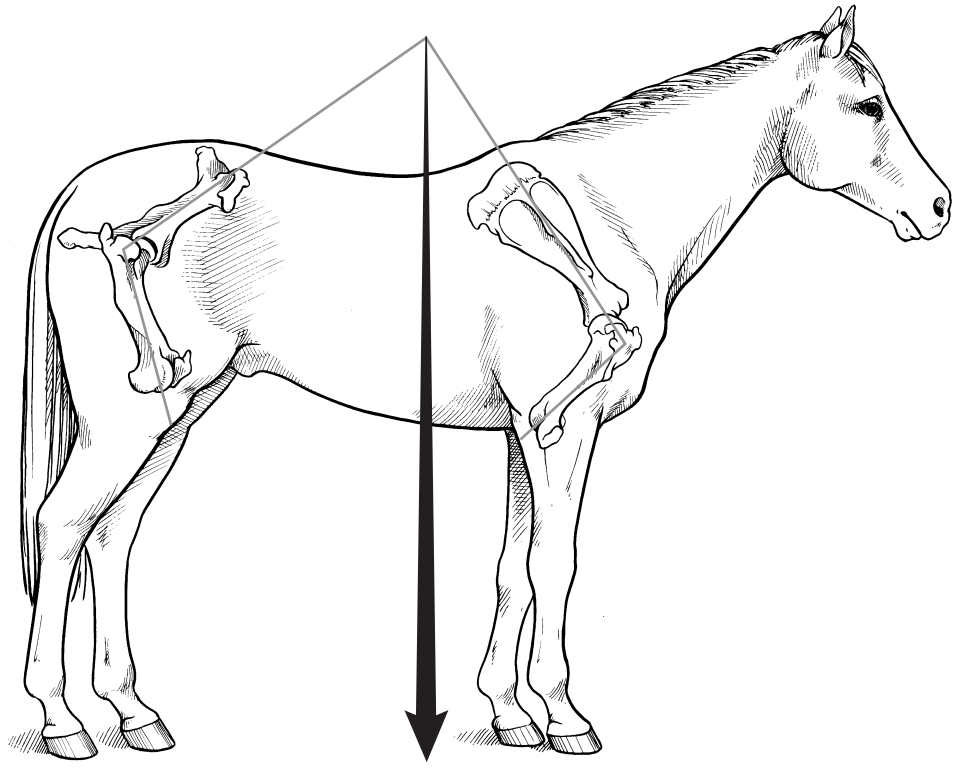

Fig. 2-1 The center of balance (gravity) of the horse is located closer to the forelimbs, which accounts for the load distribution difference between the forelimbs and hindlimbs. Conformation, namely the angles of the shoulder and rump, and weight of the head and neck and gait can change this load distribution.

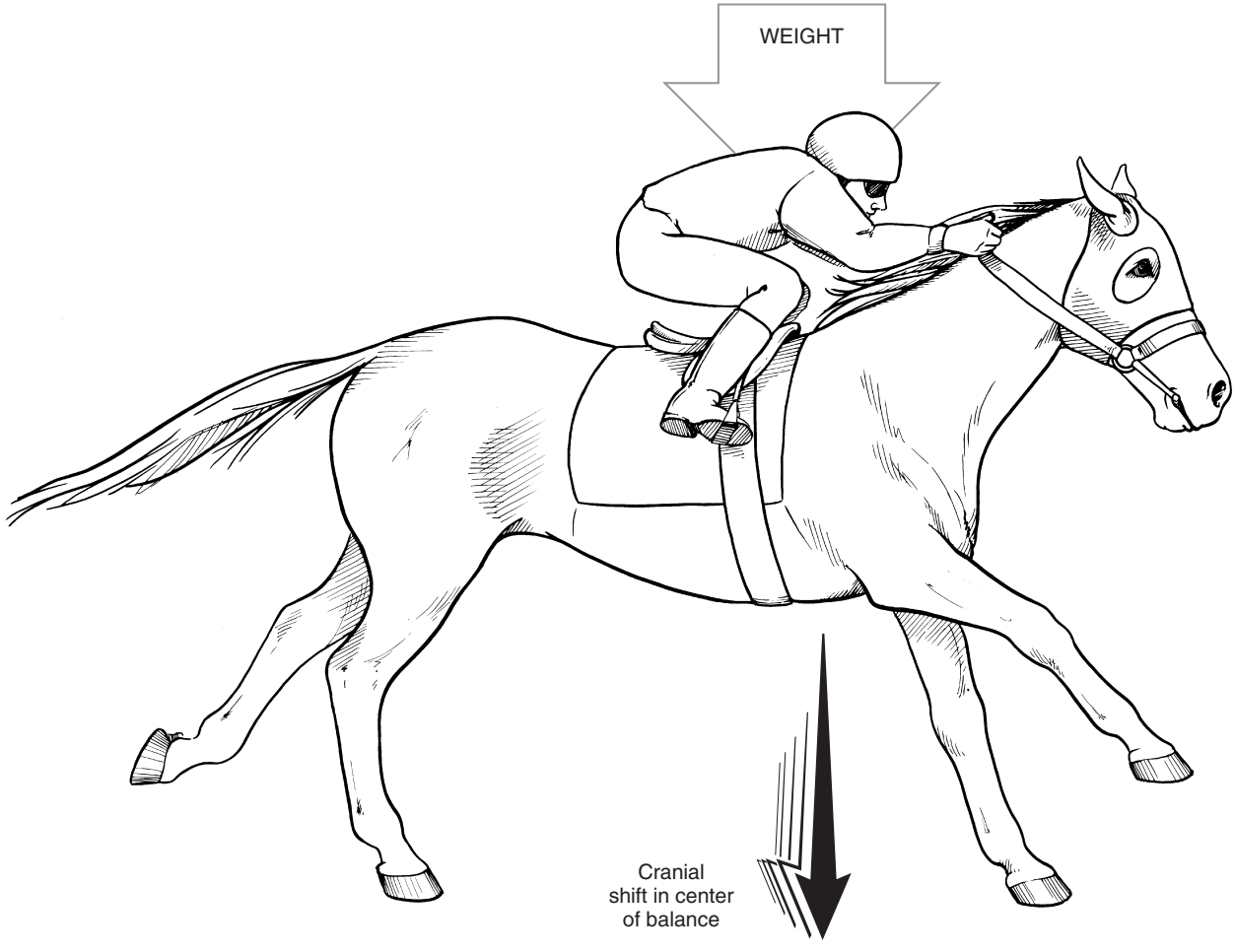

Fig. 2-2 Gaits such as the canter and gallop (depicted) and the added weight of a rider, as shown here in a Thoroughbred racehorse, increase load on the forelimbs by shifting the center of balance, thus increasing the likelihood of forelimb lameness.

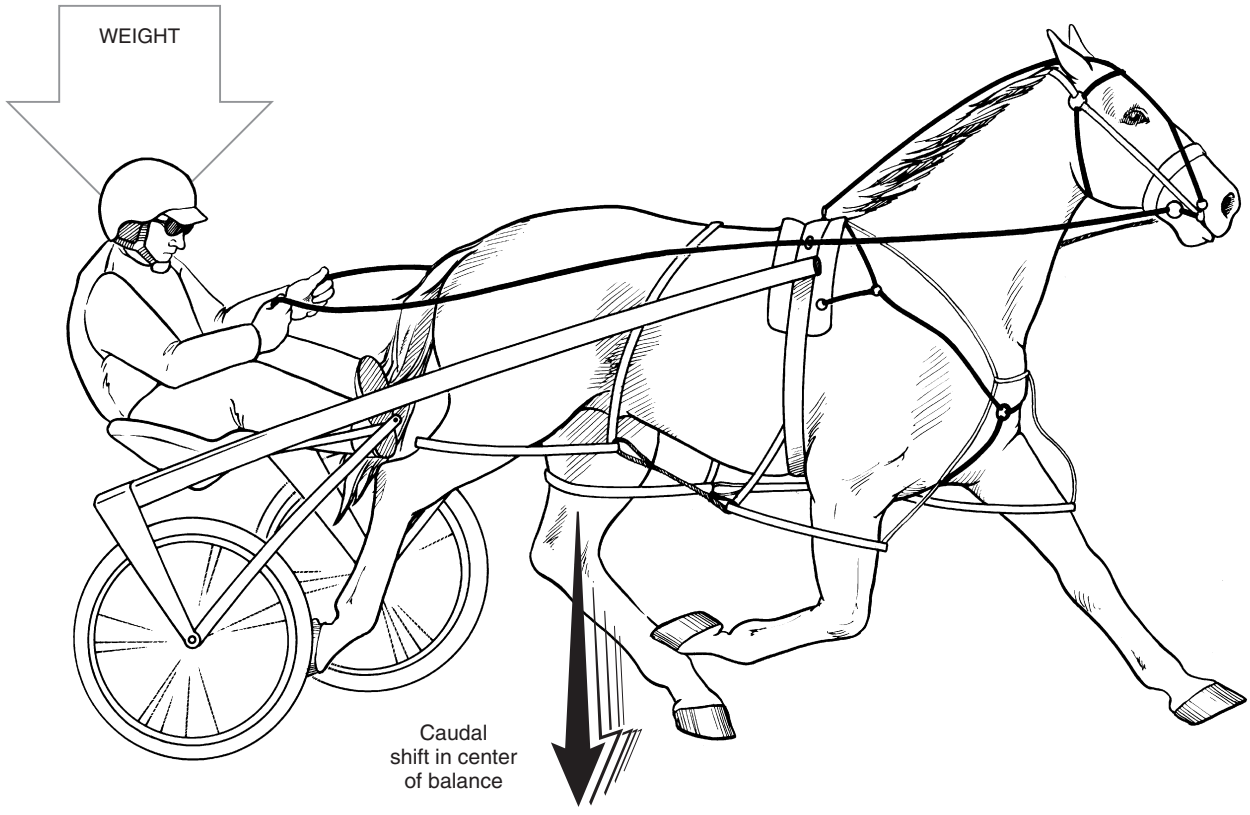

Fig. 2-3 In a Standardbred racehorse (pacer depicted), the hindlimbs share added load compared with a Thoroughbred racehorse because of a caudal shift in the center of balance. The type of harness with the overcheck bit, the added weight of the sulky and driver, and the necessity of pulling a load increase the likelihood of hindlimb lameness in this breed.

A practitioner should consider carefully the distribution of sites of hindlimb lameness. Historically, the hock has been regarded as the major source of problems, and although it is an important source of hindlimb lameness, other sites also are important. For instance, in the STB racehorse the metatarsophalangeal joint is a major source of lameness that historically has been overlooked.[5,6] Stress or nonadaptive bone remodeling of the distal third metatarsal bone (MtIII) cannot be seen radiographically in early stages and requires careful diagnostic analgesic techniques to achieve localization. Scintigraphic examination is mandatory for definitive diagnosis.[7] Lameness of the metatarsophalangeal joint in STBs is almost as common as that of the hock, but without careful examination and the use of diagnostic analgesia, hock lameness is suspected in many such horses and they are treated for it. The combined use of diagnostic analgesia, ultrasonography, and scintigraphy has increased clinical knowledge of the broad spectrum of lameness conditions in the hindlimbs.

In the draft horse, lameness in the hindlimb most commonly develops in the foot. Lameness in this area reflects the work performed by these horses, and innate characteristics of the draft horse foot, which predispose the foot to conditions such as laminitis. In jumping and dressage horses, problems in the fetlock region such as osteoarthritis and tenosynovitis are common and reflect the stress imposed by these disciplines. Although owners, trainers, and veterinarians often suspect an upper hindlimb lameness, gait characteristics of lower limb lameness problems often are similar. Only use of diagnostic analgesia allows an accurate diagnosis.

RELATIONSHIP OF LAMENESS AND CONFORMATION

Conformation of the distal extremities, and to a lesser extent the overall body, plays a major role in the development of forelimb and hindlimb lameness (see Chapter 4). When a practitioner examines a weanling or yearling with poor conformation, predicting the time and the exact way the lameness will occur may be difficult, but many well-recognized conformational faults can lead directly to lameness problems. Conformation faults of the carpus, such as carpus varus or valgus, back-at-the-knee, and offset knees, can be important factors in carpal and lower forelimb lameness. In the hindlimbs, excessively straight hindlimbs ("straight behind"), sickle hocked, and in-at-the-hock conformation can lead directly to predictable lameness conditions. Although exceptions do exist, in the case of poor conformation, predictable lameness conditions consistently develop in poorly conformed horses. Evaluation of conformation is therefore an essential part of a lameness examination.

POOR PERFORMANCE

Convincing trainers and owners may be difficult, but the leading cause of poor performance in racehorses is lameness, and lameness is the most prevalent health problem among all horses.[8-11] In a recent study, 50% of U.S. operations with three or more horses had one or more lame horses, and 5% of the

horses could be expected to be lame.[10] In another study, 74% of racehorses evaluated for poor racing performance had substantial musculoskeletal abnormalities contributing to poor performance. Lameness examination was emphasized as a most important aspect of comprehensive performance evaluation.[12,13] Others have emphasized the importance of lameness in epidemiological studies evaluating wastage in TB racehorses.[14,15] The same is likely true among all sport horses, particularly those competing at upper levels, although comprehensive studies have not been performed. Obvious lameness need not be demonstrated for performance to be compromised in horses, especially those competing at high speeds or upper levels. The possibility of achieving maximal performance in horses with substantial lameness is a common misconception. Notwithstanding the ignorance of many in the horse industry, the ability of many horses with obvious lameness to compete is a tribute to the mental and physical toughness. For example, bilateral forelimb or hindlimb lameness is common in STBs, but in some instances goes unrecognized if the condition is of similar severity. Unilateral lameness of this magnitude would be recognized easily, but because the lameness is bilateral, horses still race, albeit at a lower level. Bilateral third carpal slab fractures and sagittal fractures of the proximal phalanx have been diagnosed in horses examined for poor racing performance but, if seen unilaterally, would have caused pronounced lameness.

GAIT DEFICITS NOT CAUSED BY LAMENESS

Gait abnormalities can exist with or without the presence of clinically apparent lameness. Deficits such as stringhalt, mild intermittent upward fixation of the patella, and shivers (see Chapter 49) can be present without obvious lameness and may complicate diagnosis of a completely different primary source of lameness. Horses with neurological disease may have gait deficits that are considered the result of painful lameness conditions (see Chapter 11). Horses with lower motor neuron diseases, such as equine protozoal myelitis, may have lameness associated with muscle atrophy or unexplained low-grade lameness associated with the disease. Concomitant lameness conditions can and do occur in these horses. Recurrent exertional rhabdomyolysis can cause stiffness and in some instances lameness, or it can cause poor racing performance, all of which can be misinterpreted as lameness (see Chapter 84).

UNEXPLAINED LAMENESS

A diagnosis is made for most, but not, all lame horses through careful clinical examination and ancillary imaging modalities. Even with advanced imaging techniques a solution is not always found (see Chapter 12), but hopefully future innovations in clinical examination and imaging will result in the continued expansion of the science of lameness diagnosis.

COMPONENTS OF THE LAMENESS EXAMINATION AND LAMENESS STRATEGY

Lameness Examination

Lameness examinations should be performed in an orderly, step-by-step way, but many factors may change or abbreviate the examination (Box 2-1). Owner financial constraints may not allow performance of certain diagnostic tests and may curtail the time necessary to complete the entire examination. Drug testing of competing racehorses or show horses may limit a practitioner's ability to perform diagnostic analgesic techniques and restrict management options. Clients do not always understand the need for diagnostic analgesia. Education about the value of this technique, and the difficulties of interpretation of the results of diagnostic imaging without it, is vital.

Box • 2-1

Components of the Lameness Examination

History—anamnesis
Examination from a distance—conformation, symmetry, posture
Palpation
  Hoof tester examination
Physical examination—other ancillary testing
Movement
  Baseline
  Additional movement
Selected examinations—manipulation, flexion, direct pressure, wedge
Diagnostic analgesia
Imaging
Diagnosis
  Certain, presumptive, open
Management
Follow-up examination

Abbreviated lameness examinations often are performed in horses that exhibit severe lameness compatible with a fracture. Typical or obvious clinical signs may accompany severe lameness, and in many instances, prolonged or extensive lameness examination is contraindicated. If incomplete fractures are suspected, diagnostic analgesic techniques may be dangerous and should be performed only in certain situations. A clinician may proceed directly to conventional or advanced imaging techniques before completing the initial steps of the conventional lameness examination.

Many other factors affect the ability to complete a comprehensive evaluation. Time constraints (usually of the veterinarian) often are cited, although shortcuts, if taken, usually create future problems. Omission of a diagnostic block or failure to perform detailed palpation often leads to misdiagnosis. Omission of a brief physical examination, including assessment of the horse's temperature, can lead to embarrassing situations.

The footing available on which to complete the lameness examination can be problematic. A dry, flat, hard surface or space for lunging or riding may be unavailable. The horse's temperament may preclude adequate movement and often limits the practitioner's ability to perform diagnostic analgesia. Many ill-tempered horses are referred for advanced imaging techniques, such as scintigraphic examination, because diagnostic analgesic techniques are dangerous to the veterinarian and handler.

Lameness Etiquette

Owners frequently request an opinion from more than one veterinarian. Therefore professional, ethical conduct is important, with practitioner acknowledgment that horses can appear very different every day and response to diagnostic analgesia is not always consistent. For example, differences of opinion concerning radiographic interpretation can exist. A good working relationship with the client or agent is essential but should extend also to the farrier and any paraprofessionals involved in the management of the horse, even when opinions differ.

Prognosis Assessment

Assessment of prognosis for performance is important, but because few published data relating to many sports disciplines are available, clinicians often must rely on personal experience based on an understanding of the sport. Owners and trainers should consider prognosis carefully when making decisions to pursue therapeutic options, particularly when a long layoff period is required. I prefer to define prognosis as the "chance the horse will return to its previous level of competition." However, this may not be a fair or reasonable definition.

Retrospective studies can be used to evaluate prognosis after surgical or conservative management for various conditions in racehorses. Objective data such as numbers of race starts, race times, earnings per start, and time from treatment to first race start can be assessed. The criterion, earnings per start, is important because it establishes racing class or level of competition. However, in most retrospective studies, earnings per start decrease after treatment. The question is whether practitioners can accurately state that the horse will drop in class after treatment and whether this drop in class is the result of the injury, treatment, or aging of the horse an additional year before returning to racing. Use of this information is not easy.

Most owners and trainers have a different view of prognosis than the veterinarian. In considering the prognosis for a horse undergoing arthroscopic surgical removal of a small osteochondral fracture of the carpus, most owners emphasize the surgery rather than the original injury. Clinicians must explain thoroughly the magnitude of the injury and related damage and discuss prognosis, using terminology that clearly indicates that the extent of injury is the factor that determines prognosis.

Expecting a racehorse to return to its previous racing class may be an unrealistic expectation or at least a very strict definition for success. In a recent retrospective study of postoperative racing performance of STBs treated for carpal chip fractures, 74% of horses made at least one race start after surgery.[16] Median earnings per start significantly decreased, but the median race mark (best winning time) also significantly decreased, indicating horses made less money but ran faster after surgery.[16] These results must be compared with a normal population of STBs as the horses age, without considering injury, because STB racing performance is not standard over time.[17] Average earnings per start is highest in 2-year-old horses and decreases exponentially until retirement.[17] A population of horses undergoing any long-term layoff that requires recommencement of racing the following year can be expected naturally (unrelated to the original injury) to have lower earnings per start, regardless of whether the injury occurred or treatment was given. Therefore retrospective studies may underestimate prognosis associated with injuries and management choices.

Success criteria and outcome assessment must be standardized to compare treatment results and define prognosis. The current standard for racehorses is comparison of performance for five starts before and after injury and treatment. This criterion is strict; a practitioner first may prefer to predict the chance of the horse returning to racing and then assess the chance that the horse will perform at or near its previous level.

Other statistical methods have been used to evaluate racing performance in TBs using a regression model accounting for variables such as track surface, race distance, and age.[18-20] To date, this performance analysis has been restricted to evaluation of horses after upper respiratory tract surgery but probably will be applied to horses with musculoskeletal injuries.

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CHAPTER • 3

Anamnesis (History)

Mike W. Ross

The importance of a detailed clinical history, the anamnesis, cannot be overemphasized. Information is divided into two categories, basic facts necessary for every patient and additional information from questions tailored to the specific patient. The veterinarian must understand the breed, use, and level of competition of each horse, because prognosis varies greatly among different types of sport horses. Firsthand experience of the particular type of sport horse being examined is useful but is not essential. Clinicians must understand the language associated with the particular sporting event, and this may be a challenge. For some sporting events, understanding the clinical history and the ability to ask the right questions is like speaking a different language. A veterinarian unfamiliar with the sporting activity should briefly review the type of activities performed and the array of potential lameness problems encountered with them. In some instances the veterinarian may lose credibility when talking to trainers or riders, particularly those involved in upper-level competition, if they perceive unfamiliarity.

The veterinarian must understand the difference between subjective and objective information in the clinical history. Objective information is gained from the horse, and subjective information is perceived by the rider or owner. Information about a horse's performance such as "the horse is bearing out," "the horse is on the right line," "the horse is lugging in," "the horse has just started to refuse fences," or "the horse no longer takes the right lead" is valuable objective information. Common examples of information perceived by the owner or rider include "the horse feels off behind," "the horse is stiff behind," or "the horse is lame behind" and it "feels up high." Such information generally is useful and indicates a change in the horse's gait, but only an experienced rider or trainer can discriminate accurately between forelimb and hindlimb lameness at any gait. Erroneous information obtained from the rider can complicate communication during lameness examination, particularly if the individual is strong willed and seemingly authoritative; this situation occurs if riders or trainers insist they are correct and the veterinarian disagrees. In my experience, many horses considered to have hindlimb lameness by a rider actually are lame in front, but convincing a disbelieving trainer is difficult. Similarly, lameness perceived as "up high" in most horses originates from the lower part of the limb. The veterinarian must understand that everyone is trying resolve the problem, but sometimes diplomacy is needed for successful communication. The veterinarian must be forthright and objective to determine the current source of lameness, even if the determination contradicts well-intentioned but strong-willed trainers.

Clinical history is important but should not override clinical findings. In racehorses that perform at high speed, physical examination generally supports the finding that a horse bears away from the source of pain. During counterclockwise racing or training and with left forelimb lameness, a Thoroughbred (TB) will lug out (away from the inside of the track) and a Standardbred (STB) will be on the "left line" (bearing out; the driver must pull harder on the left line). Some horses, however, especially STBs with medial right forelimb pain, bear out particularly in the turns, presumably because the source of pain is medial or on the compression side of the limb.

The veterinarian must seek out as much information as possible, particularly if the problem is complex or not readily apparent. Videotapes are useful, particularly if the gait deficit, behavioral problem, or any other circumstances necessary to elicit the suspected lameness cannot be duplicated during the examination. Paraprofessionals working with the horse provide useful information, but not everyone may agree about the source of the problem, and in some instances diplomacy is key to negotiating among concerned individuals.

CLINICAL HISTORY: BASIC INFORMATION

Signalment

Age

The age, sex, breed, and use of the horse are basic vital facts (Box 3-1). Flexural deformities, physitis, other manifestations of osteochondrosis, and angular limb deformities are age-related problems. Infectious arthritis (hematological origin), lateral luxation of the patella, and rupture of the common digital extensor tendon are conditions usually unique to foals. Emphasis on training skeletally immature, 2- and 3-year-old racehorses causes predictable soft tissue and bone changes, often resulting in stress-related injury. Liautard observed more than 100 years ago: "When an undeveloped colt, whose stamina is not yet established and constitution not yet confirmed, with tendons and ligaments relatively tender and weak, and bones scarcely out of the gristle, is unwisely condemned to hard labor, it is irrational to expect any other results than lesions of one or another portion of the abused apparatus of locomotion. They will be fortunate if they escape a fate still worse, and become sufferers from nothing worse than mere lameness."[1] This statement aptly summarizes the situation then and now. The high value of races for 2- and 3-year-olds results in high-intensity training as an early 2-year-old, which may result in injury such as nonadaptive remodeling of the third carpal bone (C3), precluding racing at a young age.

Some problems are unique to older horses (Box 3-2). Overall, osteoarthritis and other degenerative conditions such as navicular disease are most common but certainly not unique to the geriatric patient. These problems worsen with advancing age, particularly if several limbs are involved. In former racehorses, progressive osteoarthritis is of particular concern; this condition most commonly affects the carpal and metacarpophalangeal joints (Fig. 3-1). Occasionally in older horses, severe, progressive osteoarthritis of the carpometacarpal joint occurs without any history of carpal lameness (Fig. 3-2). In some horses, angular deformities develop at the carpometacarpal joint. Primary osteoarthritis of this joint is rare in young horses, even in racehorses with middle carpal joint abnormalities, unless C3 slab fracture or infectious arthritis occurs. Osteoarthritis of the coxofemoral joint is rare

Box • 3-1

Anamnesis: Basic and Specific Information

Basic Information

Signalment: age, sex, breed, use
Current lameness: what is the problem?
- History of trauma
- Duration of lameness
- Deterioration or improvement of lameness
  - Circumstances when lameness worsens or improves
- Effects of exercise: worsening or improvement in lameness
- Management changes
  - Changes in shoeing and related issues
  - Changes in training or performance intensity
  - Changes in surface
  - Changes in diet and health
  - Changes in housing
- Current medication and response; response to rest

Past lameness problems

Specific Information

Type of sporting activity
- Level of competition: current and future

Additional sources
- Videotapes
- Images
- Records
- Discussions with others

Box • 3-2

Summary of Lameness Conditions of the Geriatric Horse

Chronic, progressive osteoarthritis
- Proximal, distal interphalangeal joints
- Metacarpophalangeal joint
- Carpometacarpal joint*
- Coxofemoral joint*
- Femorotibial joints
- Tarsus
- Progressive osteoarthritis—previous injury (usually retired racehorses)

Navicular disease
Unexplained, severe soft tissue injuries*
- Superficial digital flexor tendonitis
- Flexural deformity
- Suspensory desmitis

Fractures during anesthetic recovery

*Some of these conditions are unique to the older horse and often are unexplainable.

in horses with the exception of young horses with osteochondrosis, but it does occur in older horses.

An unusual group of soft tissue injuries of unknown origin occurs in older horses. Superficial digital flexor tendonitis and suspensory desmitis generally are considered

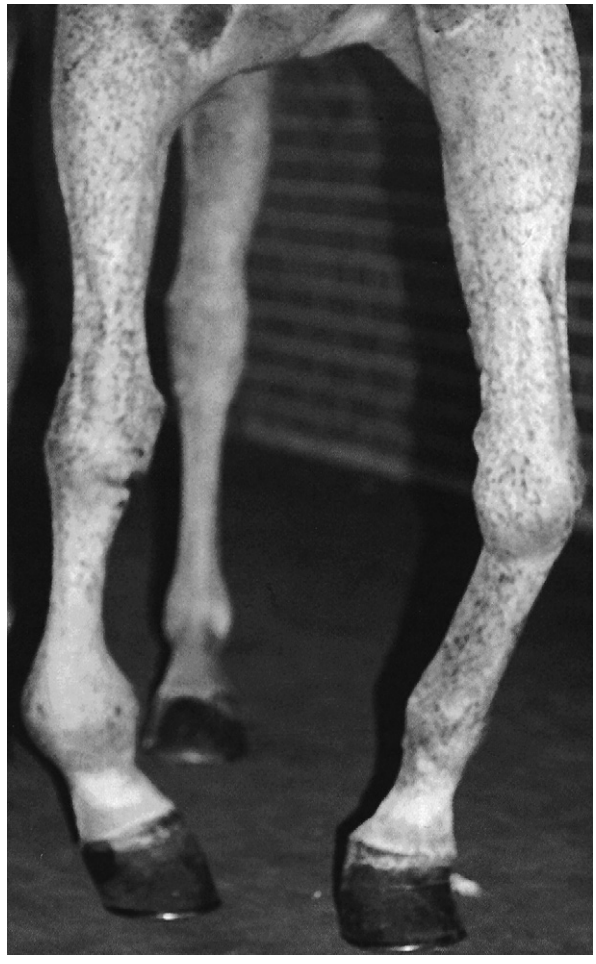

Fig. 3-1 An aged Thoroughbred broodmare with severe forelimb deformity caused by primary severe osteoarthritis of the right metacarpophalangeal joint and secondary or compensatory (chronic overload) carpus varus in the left forelimb.

overuse injuries and usually occur in upper-level performance horses or racehorses. However, severe tendonitis and desmitis do occur, often suddenly and without provocation, in older (teenage) horses. Horses usually are turned out at pasture when initial lameness is observed. In some patients, superficial digital flexor tendonitis is severe and progressive, leading to later flexural deformity because of adhesions. Suspensory desmitis may be unilateral or bilateral, may involve the forelimbs or hindlimbs, and is most common in the older broodmares.

Older horses, particularly older broodmares, are at greater risk than younger horses to fracture long bones during recovery from general anesthesia.[2] Between 1988 and 1994, 9 of 14 horses with catastrophic fractures or dislocations that developed during recovery from general anesthesia were older than 10 years of age.

Age prominently affects prognosis. A common premise in considering lameness in foals is that young horses have time to outgrow the problem. Maturation will aid in angular limb deformities, some forms of osteochondrosis, and distal phalanx and diaphyseal fractures. However, fractures of important physes such as the proximal tibia may result in progressive angular deformities or disparity in limb length, limiting future prognosis. Early surgical management of flexural deformity of the distal interphalangeal (DIP) joint before 6 to 8 months of age optimizes future soundness and the possibil-

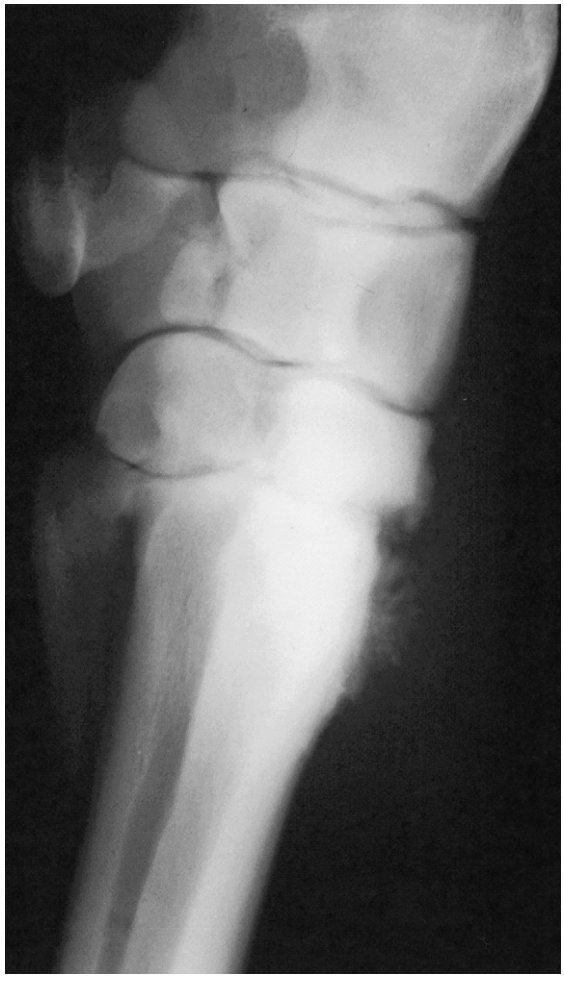

Fig. 3-2 Dorsolateral-palmaromedial oblique radiograph of the carpus of an aged horse showing unusual, severe osteoarthritis of the carpometacarpal joint.

ities for normal hoof conformation. In one study the reported success rate was 80%.[3] If surgical management is done later in life or when deformity is severe, the prognosis decreases substantially. The prognosis for survival of foals treated for infectious arthritis is reasonable, but only 31% of TB foals and 36% of STB foals started one or more races, indicating the prognosis for future racing performance is poor, because articular healing even in young foals is not possible.[4]

In middle-aged (12 to 18 years of age), upper-level performance horses, prognosis is difficult to assess, particularly in horses with several problems. Level of competition, rather than age, may be the most important factor, and often performance level declines.

Sex

Most lameness conditions affect stallions, geldings, and mares with similar frequency. Sex-specific conditions are unusual but do exist. The most important consideration, however, regarding the sex of the patient is future breeding potential or lack thereof in the case of geldings. In many types of horses, and specifically in racehorses, decisions about future performance or racing potential often are important when considering management options and financial aspects. This factor is particularly important when life-or-death decisions must be made after catastrophic injury (see Chapter 13). Frank discussions about the prognosis for return to the current sporting activity or level of performance often are necessary, and the clinician should consider reproductive capability of the horse. Owners are more likely to refrain from racing females and elect treatment for geldings and, in some instances, stallions. Future stallion prospects usually must prove race or performance success, thereby putting pressure on trainers to continue horses in training or racing.

Behavioral abnormalities associated with the estrous cycle in fillies or mares are well recognized and may cause performance problems confused or misinterpreted as lameness (refusing fences, going off stride, striking) (see Chapter 50). An ill-defined behavioral problem in middle-aged nonracehorse mares could explain sudden performance problems often associated with or misinterpreted as lameness.[5] Recurrent exertional rhabdomyolysis (RER) is more common in female TB racehorses[6] and event horses.[7] An association between sex and RER in STBs may exist and be more common in fillies administered anabolic steroids.

Obscure or unexplained hindlimb lameness has been attributed, rightly or wrongly, to retained testicles. The origin of lameness in these horses is difficult to prove without removing the retained testicle, and anecdotal reports suggest that hindlimb lameness has resolved after castration in some horses. The origin of pain in an abdominal cryptorchid is difficult to explain and questionable. The source of pain may be easier to understand in a horse with a testicle located within the inguinal canal. Activity of the external and internal abdominal oblique muscles and tension on the spermatic cord are possible explanations.

Breed and Use

Most lameness conditions affect all breeds of horses. Although breed has considerable influence on sporting activity, sporting activity or use primarily has the greatest impact on lameness distribution (see Chapters 116 through 130).

Current Lameness

Determination of the Problem

Accurate information is necessary to determine precisely the horse's current problem. Obtaining reliable information may be difficult if the horse has been purchased recently. Additional objective information may be necessary to assess the effect of lameness on the horse's performance. Evaluation of the horse's race record may indicate when the problem began and if it is ongoing or new. The groom, rider (if not the owner), assistant trainer, blacksmith, and other paraprofessionals may have other pertinent information. Horses with poor performance usually are lame, although respiratory problems, rhabdomyolysis, shoeing, tack or equipment, and other medical problems can contribute.

The horse's past history is important in determining the cause of the current problem, particularly in racehorses training or racing with existing low-grade osteoarthritis that develop new overload injury to supporting limbs (secondary or compensatory lameness). Existing problems such as osteoarthritis worsen insidiously but may reach critical levels, causing sudden, severe unexpected lameness. Osteoarthritis of the metacarpophalangeal joint may exist for months in racehorses without causing obvious lameness, although in many horses joint effusion ultimately leads to treatment ("maintenance injections"). The horse suddenly may be much lamer after racing or training, and the trainer may assume the cause is different. Because intra-articular analgesia may only partially relieve lameness, persuading the trainer that the problem is still the fetlock may be difficult. Horses can endure extensive cartilage damage in any joint for many months, but at some point they reach a threshold level beyond which they cannot tolerate the pain.

History of Trauma

Many lameness problems develop during or shortly after a traumatic incident, but unfortunately many owners presume trauma played a role, even though no one witnessed the

alleged incident. A common but often erroneous assumption when examining a lame foal is that the dam stepped on it, but usually infection is the cause. Conditions such as osteochondrosis dissecans of the shoulder or stifle often are expressed after a traumatic incident, and yet most lame weanlings or yearlings are assumed to be lame because of trauma, not a developmental problem.

Duration

The veterinarian must understand the duration of the current lameness problem and determine whether a preexisting chronic, low-grade lameness exists and if now sudden exacerbation of this problem has occurred, or a completely unrelated new problem has developed.

Worsening of Condition

The veterinarian must establish if the horse's current problem is worsening or improving, under which conditions or circumstances the lameness deteriorates or improves, and if the horse responds to treatment such as shoeing or management changes. Most lameness problems worsen with time, particularly if training or performance continues despite owner or trainer recognition. Racehorses with stress-related bone injury often are noticeably lame after work but become sound relatively quickly, within 1 to 3 days. A minimal number of other clinical signs are present, particularly because the most common bones (tibia, humerus) are difficult to palpate and buried by soft tissue. This cycle of lame-sound-work-lame is an important part of the history.

Improvement of lameness with rest is important from historical and therapeutic perspectives. Lameness in most horses with severe articular damage, usually from severe osteoarthritis, does not improve substantially with rest. Severe osteoarthritis most commonly appears in the fetlock, femorotibial, and tarsocrural joints. Horses with fractures or mild to moderate soft tissue injuries generally improve with rest.

Warming into Lameness

Warming into lameness means the horse's lameness worsens during the exercise period. *Warming out of lameness* means the lameness improves. This concept is important. Lameness associated with stress or incomplete fractures, soft tissue injuries (tendonitis and suspensory desmitis), splints, curb, and foot soreness worsens with exercise. In racehorses a worsening lameness appears as progressive bearing in or out during training or racing. In riding horses, this may be progressive stumbling, problems taking leads, progressive asymmetry in diagonals, or refusing to jump later fences. Horses with osteoarthritis may be stiff and obviously lame at a walk, but lameness may improve with work. In western performance horses, osteoarthritis of the proximal and distal interphalangeal joints, and in some horses navicular syndrome, cause lameness with this characteristic. The most dramatic example is distal hock joint pain, particularly in racehorses. Horses may be noticeably lame at a walk and trot, warm out of the lameness to the point of racing successfully, and then show pronounced lameness after the race.

One frequent statement at the racetrack is that the horse throws the lameness away at speed. This decrease occurs with some lameness conditions, such as distal hock joint pain, but two other factors are important. A horse may be able to race with lameness but not be able to perform at peak, particularly if lameness is bilateral. Horses often can race with bilateral conditions and show minimal signs of lameness, but performance is reduced. Lameness at the gallop may be impossible to perceive, and even at the fast trot or pace, most persons have difficulty seeing lameness. The same limitation occurs in observing a dressage or jumping horse at the canter. The veterinarian may gain some information by observing that a horse is reluctant to take either the left or right lead, but lameness is difficult if not impossible to detect at the canter. Unless slow-motion video analysis is available, the horse appears to be able to "throw lameness away," but lameness *is* present but difficult to see. In this situation, horses do not warm out of lameness but simply cope with the pain while racing. Horses in this situation are at risk of developing compensatory problems.

Older horses with osteoarthritis may have difficulty in getting up and later may warm out of the lameness. Horses of any age with pelvic fractures or severe lameness may have difficulty in rising.

Recent Management Changes

Many lameness conditions start after a change in management. Changes in shoeing, training or performance intensity, surface, housing, and diet or other medical issues can have a profound effect on the musculoskeletal system. Changes in ownership often dictate changes in exercise intensity and certainly in owner expectations. The veterinarian must be careful in questioning and responding to questions if a horse has been purchased recently, especially if a colleague performed a prepurchase examination. Clinicians should avoid implying that a condition may have been preexisting or missed.

Shoeing. The veterinarian should determine when the horse was last shod and whether the shoeing strategy was changed. Nail bind often causes acute progressive lameness related temporally to shoe application. Abscesses that result from a "close nail" may take several days to cause lameness.

Foot balance is critical and, in some horses, changing foot angles results in lameness. A substantial increase or decrease in heel angle in a horse with chronic laminitis may exacerbate lameness. In horses with palmar heel pain, raising the heel angle may produce an obvious improvement in clinical signs briefly, whereas in horses with subchondral pain of the distal phalanx, raising the heel may worsen clinical signs. In racehorses with "sore feet" resulting from soft tissue and bone pain, changing shoes may result in improvement, related in part to temporary reduction in weight bearing in the painful area of the foot.

Temporary lameness often occurs in horses with recently trimmed but unshod hooves, particularly if the horses' hooves are trimmed aggressively or the ground is unusually hard for that time of year. The veterinarian must remember that a horse with recently trimmed hooves often shows bilateral forelimb lameness when trotted on flat or uneven hard surfaces, regardless of the primary cause of the current lameness. The horse should be reassessed on a soft surface.

Attempts to make both front feet symmetrical may create substantial lameness immediately after trimming. Horses may cope well with different size and shaped front feet, but when radical trimming is performed, they may develop severe lameness.

The veterinarian must determine whether any recent or past changes in shoeing either improved or worsened lameness. Lameness in a STB trotter with foot pain may improve by changing from conventional shoes, such as half-round or flat steel shoes in the front, to the "flip-flop" shoe.

The farrier often first notices the common problem that a horse is reluctance to pick up the hindlimbs. In some horses, this problem is purely behavioral, whereas in others it is a real sign of pain. This history most often is associated with conditions such as osteoarthritis of one or more joints but also may be a sign of pelvic or sacroiliac pain. In Warmbloods, draft breeds, and draft-cross horses, reluctance to pick up a hindlimb may be an early sign of shivers.

Training or performance intensity. Lameness that worsens in response to recent increase in training intensity may be related to stress-related subchondral or cortical bone injury. Stress fractures, bucked shins, or nonadaptive stress-related injuries of subchondral bone occur typically during defined periods of training and often after brief periods of rest. When horses in active race training are given time off, even

brief periods such as 7 to 21 days, bone undergoes detraining, leaving it subject to stress-related injury. If training resumes at the pre-rest level or is accelerated, stress fractures or bucked shins often develop. In 3-year-old TBs, stress fractures of the humerus often occur within 4 to 8 weeks after returning to training. Bucked shins often develop in 2-year-old horses after a brief rest period for an unrelated medical condition.

Surface. Most lameness conditions worsen if the horse performs on a harder surface. In show horses such as the Arabian or half-Arabian breeds, foot lameness often results when horses are warmed up or shown on harder surfaces. An association exists between fracture development and hard racing surfaces. A dramatic change in any racing surface may lead to unexpected episodic lameness in racehorses. On breeding farms, anecdotal evidence suggests drought conditions causing harder than normal pastures lead to a higher prevalence of osteochondrosis or distal phalanx fractures.

Lameness that is most pronounced on hard surfaces is often seen with conditions of the foot. Lameness that worsens on softer surfaces, however, may be associated with soft tissue injuries such as proximal suspensory desmitis. Uneven surfaces may exacerbate lameness and other gait abnormalities. Horses prone to stumbling on uneven surfaces may have palmar heel pain, proximal suspensory desmitis, or neurological disease. Horses with bilateral lameness may be lame in one leg going in a particular direction on a banked surface (such as a racetrack) and lame in the opposite leg going the other way. Bilateral lameness may be confused with other causes of poor performance because of inconsistencies in gait. Lameness may worsen or improve when a horse goes uphill or downhill.

Diet and health. Changes in diet or dietary factors may lead to or exacerbate existing lameness conditions. Dietary factors, especially dietary excesses or deficiencies, are important in the many manifestations of developmental orthopedic disease (see Chapter 57). Sudden changes in diet, such as those associated with turning horses out on lush pastures or consumption of large quantities of grain (grain overload), may cause laminitis or exacerbate existing chronic laminitis. Overweight horses normally consuming a high-grain diet may be prone to laminitis or gastrointestinal tract disturbances that lead to laminitis.

Lameness may be associated with, or result from, other medical conditions. Obvious associations exist in foals between conditions such as infectious arthritis and physitis with umbilical, gastrointestinal, or respiratory tract infections. Immune-mediated synovitis also occurs in older foals with chronic infections (see Chapter 67). In adult horses, infectious arthritis generally develops after intra-articular injections or penetrating wounds but may result from hematological spread of bacteria. Occasionally, horses develop distal extremity edema and lameness after vaccination, presumably caused by vasculitis or other immune-related mechanisms. Similar signs appear in horses with purpura hemorrhagica or viral illnesses such as equine viral arteritis.

Housing. Many lameness conditions develop while a horse is turned out, or as the result of turnout, often as the result of trauma such as kick wounds or fence-related injuries. Sudden changes in weather may excite horses, particularly those turned out at pasture. During thunderstorms horses may run into fences or buildings and suffer unexpected injuries. One such problem is suprascapular nerve injury. Minimizing problems with turnout requires the use of well-groomed and well-maintained pastures or paddocks with individual paddocks to reduce horse to horse interactions.

Dramatic housing changes have a substantial impact on the development of lameness. Shipping to and from sales, foaling, and weaning are associated with soft tissue injuries, puncture or kick wounds, and other injuries.

Current Medication Changes and Response

The veterinarian must establish if the horse currently is receiving medication or was administered medication recently and the response to treatment. Response to medication or a management change is important information in formulating a treatment plan. For example, recent improvement with rest and the administration of nonsteroidal anti-inflammatory drugs indicates more of the same treatment may be reasonable. The veterinarian must establish dosages of medication because a horse may not respond to phenylbutazone because of under-dosage.

Many owners and trainers do not understand that although intra-articular analgesia relieves lameness, intra-articular medication may not, thus causing doubt about the diagnosis. This characteristic commonly appears in horses with subchondral bone pain and is useful in diagnosis. Horses with early osteoarthritis and negative or equivocal radiographic signs, or those with short, incomplete fractures, often do not respond to intra-articular medication. Negative radiographic findings are a good sign because dramatic radiographic evidence of subchondral lucency or fracture reduces the prognosis considerably. However, convincing the trainer of the validity of the diagnosis may be difficult.

The amount and quality of rest are important. Many acquired conditions, such as osteoarthritis, or degenerative conditions, such as navicular syndrome, take many months and usually years to develop. Therefore expectation that a horse will show marked improvement with a brief rest period is unreasonable. Lameness in many horses with severe osteoarthritis may not improve substantially, even with prolonged rest. In horses with early osteoarthritis in which pain occurs primarily in subchondral bone and for which radiographic findings are negative or equivocal, rest or controlled exercise for 3 to 6 months may be necessary. The same regimen applies for horses with navicular syndrome, fractures, and many soft tissue injuries.

Quality of rest is equally important. Did the horse receive absolute box stall rest with handwalking, or was it lunged or turned out in a paddock or field with other horses? Was a brief rest period followed by an attempt to ride or train the horse? Those associated with the horse often consider this type of intermittent rest *complete* rest, but many conditions remain chronically active. Without adequate rest, re-injury follows temporary improvement and early healing.

Past Lameness History

Obtaining the horse's entire lameness history may not be necessary or possible, but the veterinarian should gather as much information as is practically available. Prognosis for many injuries is affected adversely by recurrence, and often management options differ in these situations. Recurrence may prompt more aggressive therapy, considerations for referral, or perhaps surgical evaluation if the problem involves a joint. If a reliable diagnosis was made previously, retreatment for the past problem may be a reasonable or preferred management approach, particularly between races or competitions. If a horse has responded previously to intra-articular medication, re-injection may be reasonable. However, in many horses with progressive osteoarthritis, results of additional therapy often are diminished. The veterinarian should not assume that the failed response to intra-articular medication means the problem lies elsewhere because medication does not affect subchondral bone pain in early or late osteoarthritis.

Recent history is important. Small, innocuous-looking wounds over the third metacarpal bone or third metatarsal bone, radius, tibia may be associated with bone trauma with delayed-onset severe lameness. Incomplete or spiral fractures may develop from small cortical defects (Fig. 3-3).

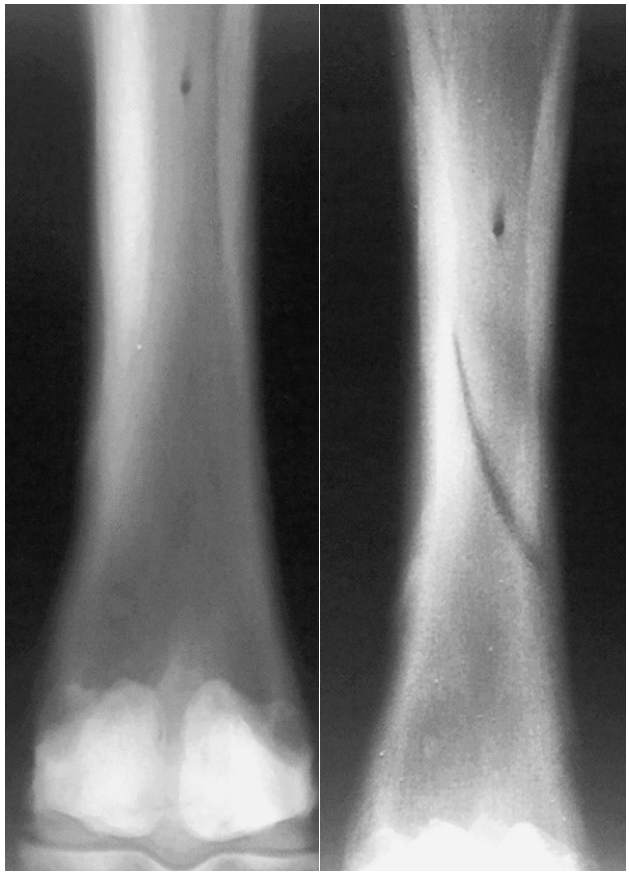

Fig. 3-3 Dorsopalmar radiographs of left third metacarpal bone in an 18-year-old Thoroughbred gelding showing initial (**A**) and 2-week follow-up (**B**) views. The initial view was taken after the horse was found to have a small skin wound in the region but was sound. Acute, severe lameness developed 9 days after initial injury, and the follow-up radiograph shows a long oblique fracture of the third metacarpal bone.

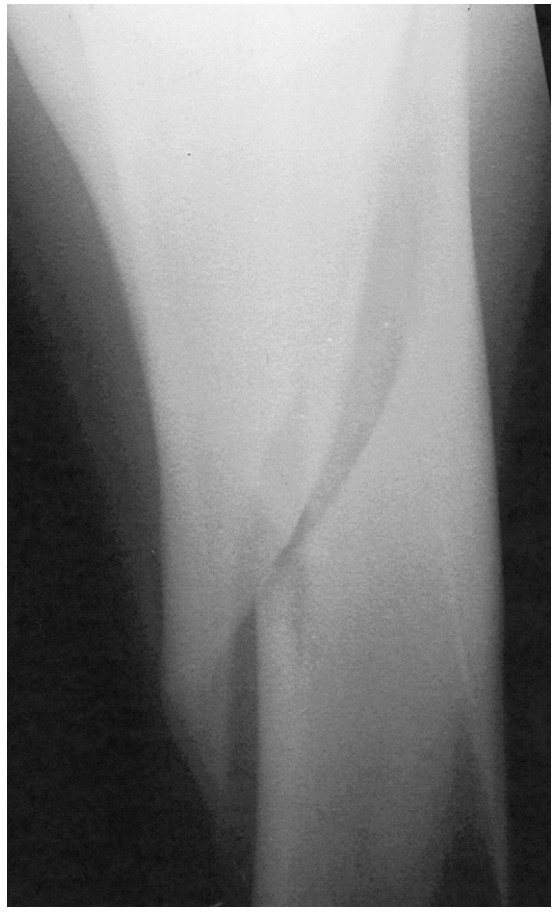

Fig. 3-4 Craniocaudal radiograph of the left antebrachium of a 5-year-old Thoroughbred mare showing a displaced, long oblique fracture of the radius. This filly had sustained a small puncture wound to the lateral aspect of the antebrachium 5 days earlier but was sound. The mare was turned out and developed acute, severe lameness and was later euthanized.

Catastrophic failure of long bones occurs even when initial radiographs show no or minimal cortical trauma. The radius appears to be at greatest risk (Fig. 3-4).

New problems may and often do arise despite a long history of recurrent lameness. Comprehensive reevaluation is the best and safest approach to avoid delays in proper diagnosis and treatment.

FURTHER INFORMATION

Full understanding of the horse's use, type and level of sporting activity, and value, all of which help the veterinarian assess prognosis, requires specific information. If the horse previously was under the veterinary care of another individual in the same or different practice, it is important to obtain accurate case records and view previous radiographs and other images.

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CHAPTER • 4

Conformation and Lameness

Mike W. Ross

The idea of a good horse with poor legs is a misnomer, the legs are the essence of the horse, and every other part of the equine machine is of only subservient and tributary importance.
A. Liautard[1]

That the way a horse is conformed determines the way it moves is well accepted. The relationship of conformation, especially of the distal extremities, and lameness also is well recognized. "Conformation determines the shape, wear, flight of the foot, and distribution of weight."[2] Veterinarians often are asked to comment on conformation during lameness and prepurchase examinations, especially with regard to the suitability of the horse to perform the intended task. In some instances, as in the case of presale yearling evaluations, the veterinarian's opinion is paramount, and purchase is contingent on judgment of the yearling's potential to perform as a racehorse, given its conformation, or in some instances its conformational faults (see Chapter 102). "It is by a study of conformation that we assign to a horse the particular place and purpose to which he is best adapted as a living machine and estimate his capacity for work, and the highest success in this connection will be best attained by the judicious blending of practice with science."[3] Evaluation of conformation and its influence on lameness is based largely on observation, experience, and pattern recognition. Recognizing desirable conformational traits in horses suited for a particular sporting activity and learning when to overlook a minor fault that has little clinical relevance is important.

RELEVANCE OF CONFORMATION EVALUATION

Conformation is one piece of the complex puzzle of a lame horse, although poor conformation does not necessarily condemn a horse to lameness: "faulty conformation is not an unsoundness…it is a warning sign."[2] All lameness diagnosticians should evaluate conformation briefly at the beginning of each examination. The association of lameness and faulty conformation will be obvious. The clinician must evaluate the horse from afar, assessing the whole horse for balance, angles and lengths, and posture and symmetry. The clinician must remember that horses come in all shapes, sizes, and types, and therefore conformation varies accordingly, but certain conformational faults produce predictable lameness conditions and are undesirable. However, good conformation is not synonymous with success, and although horses of certain body types tend to have longer strides and are more athletic than others, intelligence, aggression, "will to win," and other intangible factors are important. I am convinced that a well-bred horse from a successful family can endure faulty conformation much better than a poor- or mediocre-bred horse.

HEREDITARY ASPECTS OF CONFORMATION

Certain conformational faults appear to be highly heritable traits. Evaluation of broodmares and foals often reveals that the early conformational defects seen in a foal are present in the dam. The dam seems to contribute more to faulty conformation than does the sire, although the stallion also is important. This difference may be explained in part by the fact that fillies with faulty conformation may develop problems or be retired early and subsequently bred, whereas most stallions usually are proven performers with exceptional conformation. Conformational faults such as toeing in and toeing out commonly are passed down from generation to generation. Back-at-the-knee (calf-knee), offset (bench) knee, tied-in below the knee, sickle-hock, and straight behind conditions are also highly heritable.

Certain lameness conditions are common in horses with faulty conformation, but similar lameness conditions develop inexplicably in some breeding lines year after year in offspring with apparently acceptable conformation. Lameness of the carpus or tarsus appears to be most important. For example, in Standardbreds (STBs), siblings commonly develop similar lameness conditions, such as proximal suspensory avulsion injury, carpal osteochondral fragments, distal hock joint pain, or curb.

OBJECTIVE EVALUATION OF CONFORMATION: IS IT POSSIBLE?

A recent attempt to quantify conformation used a linear assessment trait evaluation system that allows the observer to assess where, given a particular trait, a single animal falls within a population of animals.[4] A population of 101 Irish Thoroughbred (TB) flat racehorses and 19 top stallions was used and 27 common conformational traits were evaluated, including various heights, lengths, angles, and distal extremity conformation (Figs. 4-1 to 4-3). Of the 27 traits, 6 were significantly linked to age, and 5 were linked to sex (head and neck shape; neck size at poll and larynx and at withers, and manubrium of the sternum; and forelimb hoof pastern axis). Most traits exhibited large phenotypic variation within the population, but 21 of 27 non–age-linked traits were judged suitable for possible inclusion in a linear assessment protocol.[4] Researchers judged a high percentage of horses to be toed out, suggesting this trait may even be desirable.

More recent studies have used video-image analysis, but direct physical measurements may be more accurate than those obtained by analyzing videotapes or photographs.[5] Potential errors in image analysis occur because of movement of skin markers over selected bony protuberances, a phenomenon more common in the upper limb and in motion studies.[5,6] Skin marker location is critical for evaluation of joint angulation and movement during locomotion or conformation analysis. Instant center or axis of rotation (ICR) is defined as the point with zero velocity during movement of that joint; accurate measurements of joint angulation require positioning markers at the ICR.[7] Conventional positions of skin markers and ICR in most joints agree well, but use of traditional marker sites on the scapulohumeral and femorotibial

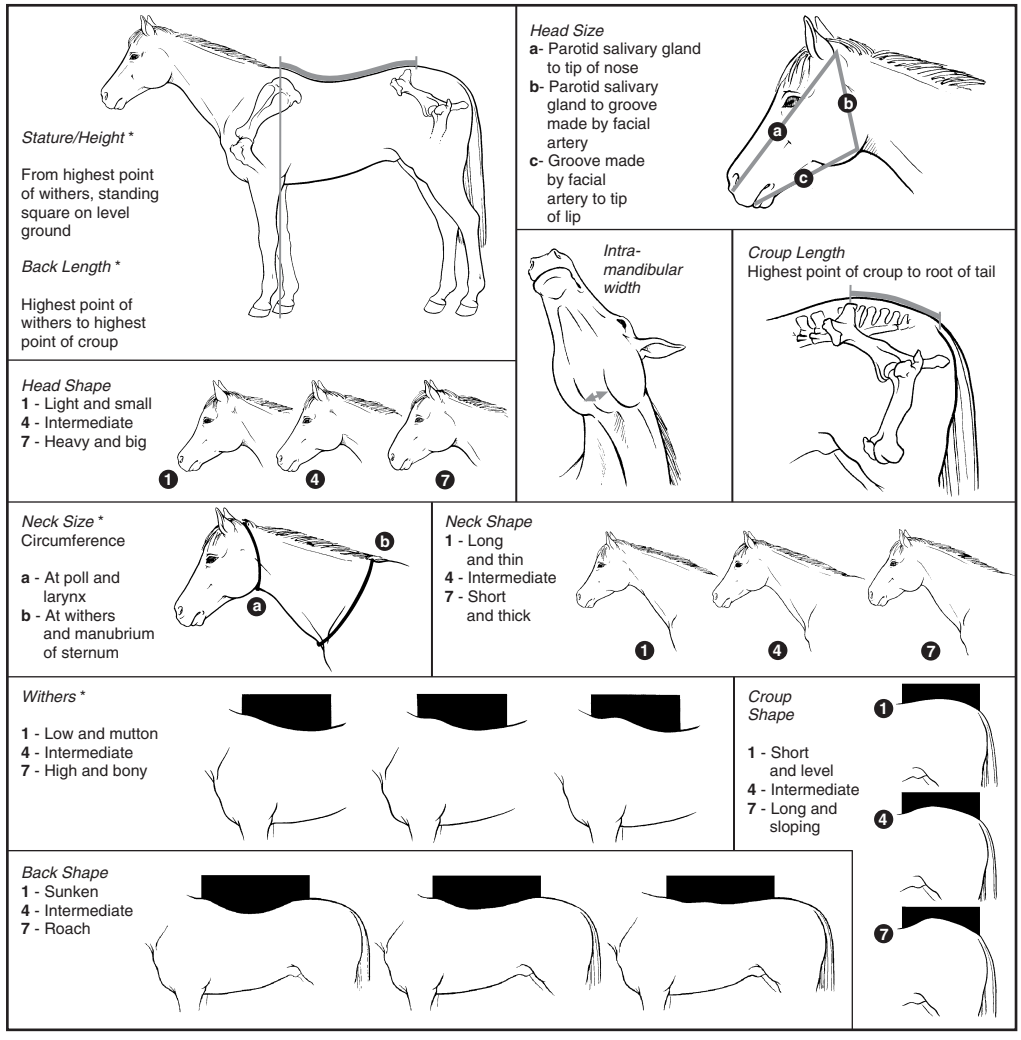

Fig. 4-1 Conformation traits used for linear assessment of conformation in a population of Irish Thoroughbreds. Asterisks indicate traits significantly linked to age. (Modified from Mawdsley A, Kelly AP, Smith FH, et al: Linear assessment of the Thoroughbred horse: an approach to conformation evaluation, *Equine Vet J* 28:461, 1996.)

joints results in overestimation and underestimation, respectively, of caudal joint angles.[8] Although video-image analysis may be fraught with potential or in some instances real error, objectivity is a major advantage. Other advantages include the ability to replay images, reduction of observer fatigue, elimination of observations and measurements in real time, and permanent recording of the observation. Ideally, objective measurements would withstand statistical evaluation, distinguish between desirable and undesirable traits, and account for differences among different types of horses.[5]

A combination of direct measurement and photography has been used to to evaluate conformation of Swedish Warmblood and elite sport horses.[9,10] Whereas most of the conformational defects were mild or moderate, 80% of Warmblood (WBL) horses were toed out behind, suggesting this may be a normal finding in this breed as in the STB trotter. More than 50% of horses had bench knees and 5% were toed out in front, contrary to findings in STB trotters.[9,11] Many of the elite horses were bucked kneed, whereas the riding school horses tended to have calf knees. It was speculated that this occurred because elite horses were evaluated after competition and muscle fatigue may have contributed to the tendency to be over at the knee. Sex had significant influence on conformation; females were smaller and had longer bodies and smaller forearms and metacarpi. There were interesting findings regarding hock angle. A sickle hock is defined as a hock angle of 53° or less; a large hock angle is referred to as straight behind. Sickle-hocked conformation was nearly absent in elite horses, and it was hypothesized that sickle-hocked conformation either predisposed a horse to lameness or impaired a horse's ability to achieve upper levels of competition.[9] A positive relationship between larger hock angles and soundness in STB trotters also exists.[12] In forelimbs of WBL show jumpers and the forelimbs and hindlimbs of STB trotters, smaller fetlock joint angles (less upright) were desirable.[9,12]

Radiography was used to assess the degree of hyperextension of the carpus to study the potential effect of back-at-the-knee (calf-knee) conformation on the subsequent development of carpal chip fractures.[13] Lateromedial radiographic views of 21 horses with carpal chip fractures and of 10 normal horses were obtained, with and without the contralateral limb raised. No relationship between measured carpal angle and carpal chip fracture formation existed, suggesting that this group of TB racehorses did not develop carpal chip fractures as a result of calf-knee conformation. The sample size was small, however, and a larger study may produce different results. Horses with severe calf-kneed conformation may develop other problems and not advance enough in training to develop carpal chip fractures. They may be judged poor surgical candidates, are not referred, or are slow.

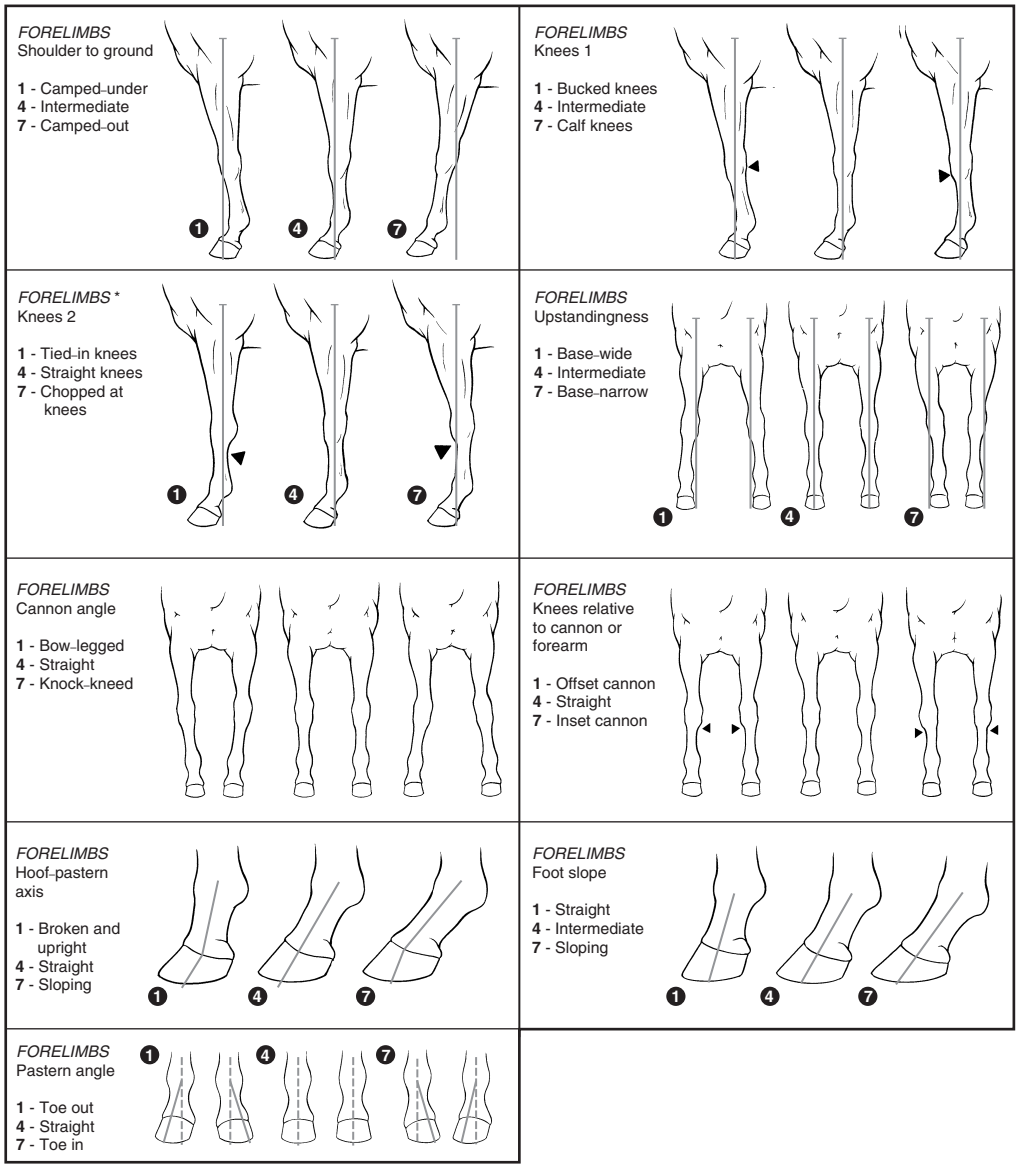

Fig. 4-2 Conformation traits used for linear assessment of conformation in a population of Irish Thoroughbreds. Asterisk indicates traits significantly linked to age. (From Mawdsley A, Kelly AP, Smith FH, et al: Linear assessment of the Thoroughbred horse: an approach to conformation evaluation, *Equine Vet J* 28:461, 1996.)

Two recent studies evaluated TBs and Quarter Horses (QHs) using skin markers, photography (three views: front, side, back), and computer-image analysis. The TB study evaluated the change in conformation with age; as expected, most body parts increased in length from birth to 2 years. Horses became more bench kneed from birth to 3 years, and the dorsal hoof angle decreased as horses grew (hoof wall became less upright). A strong relationship existed between long bone length and withers height, including the radius (taller horses have longer bones).[14] In the TBs, clinical observations (lameness) associated with conformational variables included effusion of the fetlock and carpal joints, carpal fractures, and other traumatic conditions of the fetlock joint. Risk of effusion of the fetlock joint increased with increased dorsal hoof angle, and the odds of carpal fracture decreased with scapular length. Carpus valgus decreased risk for carpal fracture, but fetlock lameness increased in horses with offset knees.[14]

In the second study, odds ratios were created for increase in bone length of 2.54 cm (1 inch) or joint angle of 1° and development of lameness. For every 1-inch increase in humeral length, odds for fracture of the proximal phalanx or carpal synovitis or capsulitis increased. Increased length from elbow to ground and increased toe length increased chances for carpal fracture, and in horses with offset knees greater than 10% the potential for carpal or fetlock synovitis or capsulitis increased. The potential for fracture of the proximal phalanx increased with an increase in shoulder angle.[14]

The relationship of many lower limb lameness conditions with limb length is interesting and somewhat unexpected because longer limb length generally is considered desirable. Although a relationship between longer toes and carpal fracture is not unexpected, the increased chance for fetlock lameness with more upright hooves is surprising. The relationship between offset knees and lower limb lameness was expected, but unexpected were fewer carpal fractures in TBs with this conformational fault. Because development of lameness, termed clinical outcomes in these studies, is complex, confounding variables such as track conditions, training regimen, breeding, individual horse ability, and experimental error could have contributed to outcome.

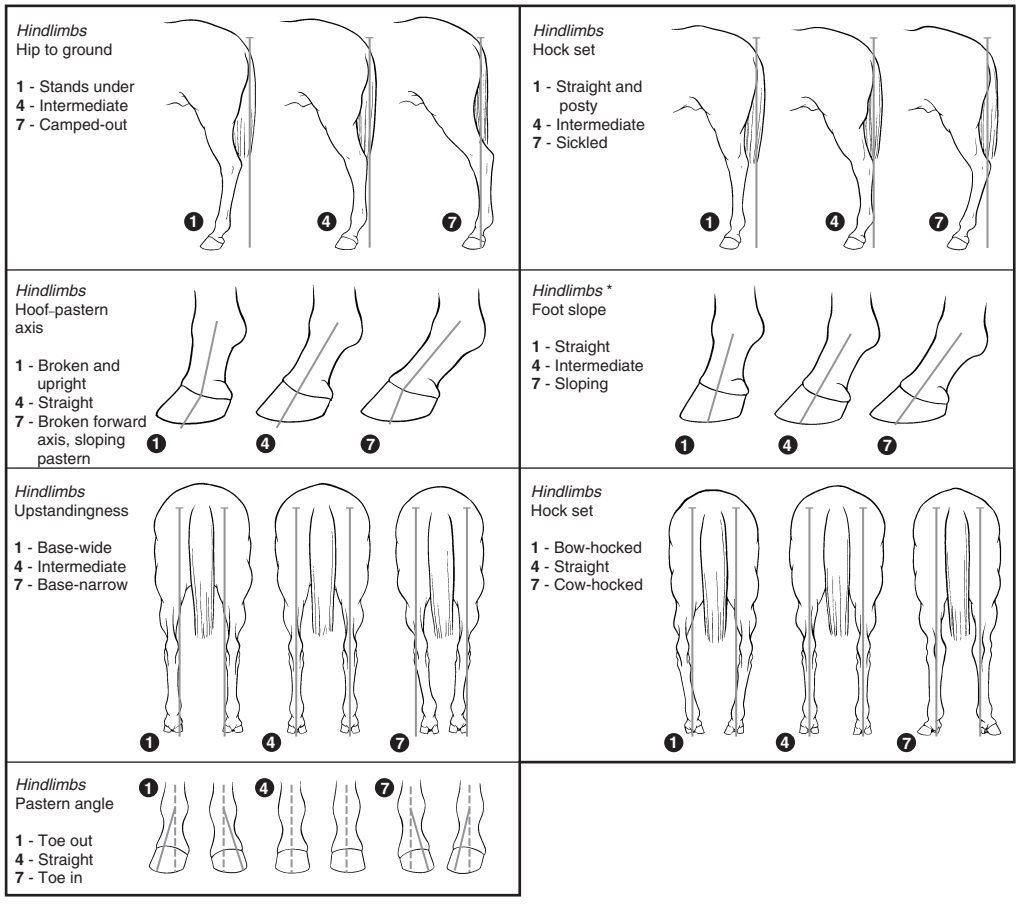

Fig. 4-3 Conformation traits used for linear assessment of conformation in a population of Irish Thoroughbreds. Asterisk indicates traits significantly linked to age. (From Mawdsley A, Kelly AP, Smith FH, et al: Linear assessment of the Thoroughbred horse: an approach to conformation evaluation, *Equine Vet J* 28:461, 1996.)

Little doubt exists that acquiring objective information is useful, not only to determine what is abnormal but also to define what is normal in a population. In both WBLs and STB trotters in Europe, toed-out conformation in the hindlimbs should likely be considered normal because a majority of both breeds have this conformational trait.[9,11] These populations differed, however, in forelimb conformation. Few STB trotters had bench-kneed conformation, a finding supported by my clinical observations that this conformational fault is highly undesirable in this breed (see Chapter 109).

EVALUATION OF CONFORMATION

Conformation determines the way a horse moves, and I am convinced that a relationship exists between faulty conformation and the development of lameness. Therefore assessment of conformation should be an integral part of lameness examination. Conformation evaluation has four basic components: (1) balance, (2) assessment of lengths, angles and heights, (3) muscling, and (4) conformation of the limbs. All are intertwined but should be evaluated separately, considering the whole horse not just the limbs, and then consolidated. The clinician should evaluate the horse on firm, level ground, preferably a smooth, nonslip surface that does not obscure the view of the feet. The horse should stand squarely with equal weight on all four limbs. Dynamic assessment of limb conformation while the horse is walking also is essential.

Balance

Balance is the way all parts of the horse fit together and is linked directly with assessment of lengths, angles, and heights. The horse should be proportional and thus well balanced. A horse may be visualized in thirds—the forehand, the midbody, and the hindquarters—by drawing three circles incorporating these areas (Fig. 4-4). The circles should overlap but not excessively. Horses in good balance are likely to be superior athletes. Horses with a short, thick (throatlatch and shoulder regions) neck are often heavy and straight in the shoulders (Fig. 4-5). A horse with a short back has naturally closer dorsal spinous processes and may be predisposed to impingement or over-riding, whereas a horse with a long back (Fig. 4-6) may have difficulty engaging the hindlimbs properly. The clinician must assess the relative heights of the withers and hindquarters. A horse that is taller behind than in front (rump height is greater than height at the withers) is predisposed to forelimb lameness. A horse that also is underdeveloped in the shoulders and upper forelimbs (weak up front) and heavy behind is more at risk. Limb lengths should be proportional to body size and height. In general, the body length (point of shoulder to point of rump) should be equal or slightly longer than withers height (see Fig. 4-4).

Head conformation is not relevant to lameness, but the size of the head relative to the body and the angulation between the head and neck influence the ease with which the horse can work "on the bit" (see Chapters 100 and 117). A horse's ability to see is important. A horse with ocular abnormalities may exhibit bizarre behavioral abnormalities or unusual head and neck

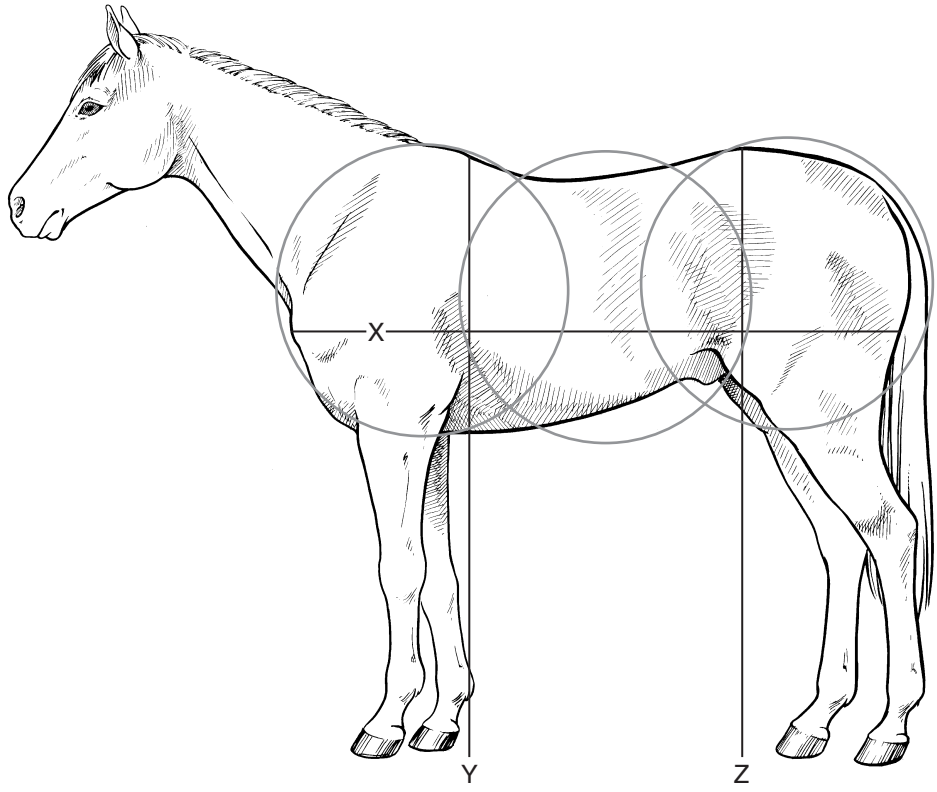

Fig. 4-4 Diagrammatic depiction of assessing balance during conformation evaluation. Three circles (from left to right: forehand, midbody, hind quarters) are visualized and should overlap by approximately one third. Excessive overlapping of circles (short coupled) or barely overlapping of circles (long, weak in back) are common conformational abnormalities. Body length (*X*) should be equal to or slightly longer than withers height (*Y*), and withers height and rump height (*Z*) should be the same.

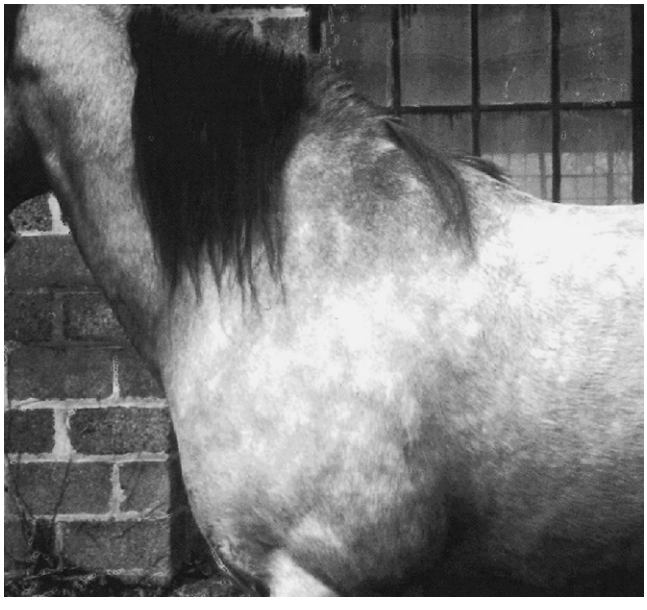

Fig. 4-5 Horse with short, heavy neck; heavy, short, and straight shoulder; and withers set forward. This horse is prone to forelimb lameness and likely to have a short stride.

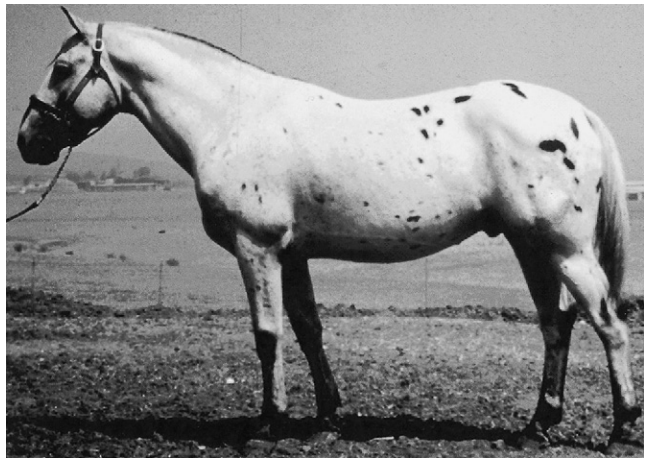

Fig. 4-6 Unbalanced Appaloosa gelding that is long and weak in the barrel (back). The rump height is slightly higher than withers height.

carriages, possibly misinterpreted as the result of pain. A rare cause of "being on a line" occurs in driving horses, such as a STB with unilateral blindness. The horse turns the head away from the blind side to see and thus is on the contralateral line (to straighten the head, the driver must pull on the contralateral line). This mimics a contralateral (to the blind eye) lameness.

Assessment of Lengths, Angles, and Heights

Body length is important in determining stride length (see Fig. 4-4). Short coupled conformation predisposes horses to short strides and problems with interference, especially in racehorses. If horses are too long, they can be weak in the back. The length of the neck is important in assessing balance and should be proportional to the overall body length. Some horses have long, weak necks. The neck may be "set on low" relative to the shoulder with a depression (ventral deviation) of the dorsal topline cranial to the withers (ewe necked), giving the appearance of prominent withers laid too far caudally. Horses should have adequate depth in the girth region (depth of girth).

Shoulder length (top of withers to the point of the shoulder) may be related directly to stride length, and horses with longer shoulders usually have longer strides (Fig. 4-7). Those with short shoulders usually have shorter strides (see Fig. 4-6). Shoulder length and shoulder angle often are related; long shoulders often are more sloping (smaller shoulder angle) and short shoulders often are straight. Good shoulder length appears to be important and desirable, but recent objective data from TB racehorses suggest that horses with long limbs may be at increased risk of lower limb lameness.[14]

In TBs particularly a long radius (forearm) and short, strong third metacarpal bone (McIII) are desirable for adequate strength and maximum stride length. Chest width should be commensurate with overall body size. A wide chest with a base-narrow (front feet close when evaluating the horse from the front) limb stance or a narrow chest with a base-wide stance are undesirable. In STB pacers a good chest width is desirable, but in trotters a narrow chest is preferred (see Chapter 109).

Rump length also is important in determining stride length, and a longer length of the rump is desirable. Many horses with long rumps have larger rump angles (flat croup) and those with short rumps have smaller rump angle (steep croup). Long, flat croup regions are desirable. The ideal horse should have a long gaskin (crus) and a short, strong metatarsal region (hocks close to the ground) to maximize stride length.

The angles of the shoulder and rump are important factors in determining stride length and balance. Undesirable shoulder and rump angles often accompany other conformational faults and may predispose to lameness. The ideal angle of the shoulder (relative to the ground) has classically been determined as 45° (see Fig. 4-7). Horses with steep shoulder angles (>50° to 55°) usually have short shoulders and short, upright pasterns, which predispose to lower limb lameness. The forelimb pastern angle should be equal to the shoulder angle. Steep shoulder angle shifts the center of gravity forward, predisposing to forelimb lameness. Horses with a flat rump or croup generally have longer rump lengths and longer strides (Fig. 4-8). Horses with short, steep rumps (goose rump), often have short, choppy gaits, and many have hindlimb lameness (Fig. 4-9). A steep rump angle shifts the center of gravity caudally, predisposing to hindlimb lameness.

Limbs

It is critical to evaluate limb conformation with the horse standing squarely on a firm, flat surface and with an experienced handler who can make the horse cooperate. If the clinician observes a fault that may result from how the horse is standing, he or she should reevaluate the horse after repositioning. Horses often stand camped out, both behind and in front, simply as the result of improper positioning.

The plumb line concept allows evaluation of each limb from the front or back and the side (Fig. 4-10). For example,

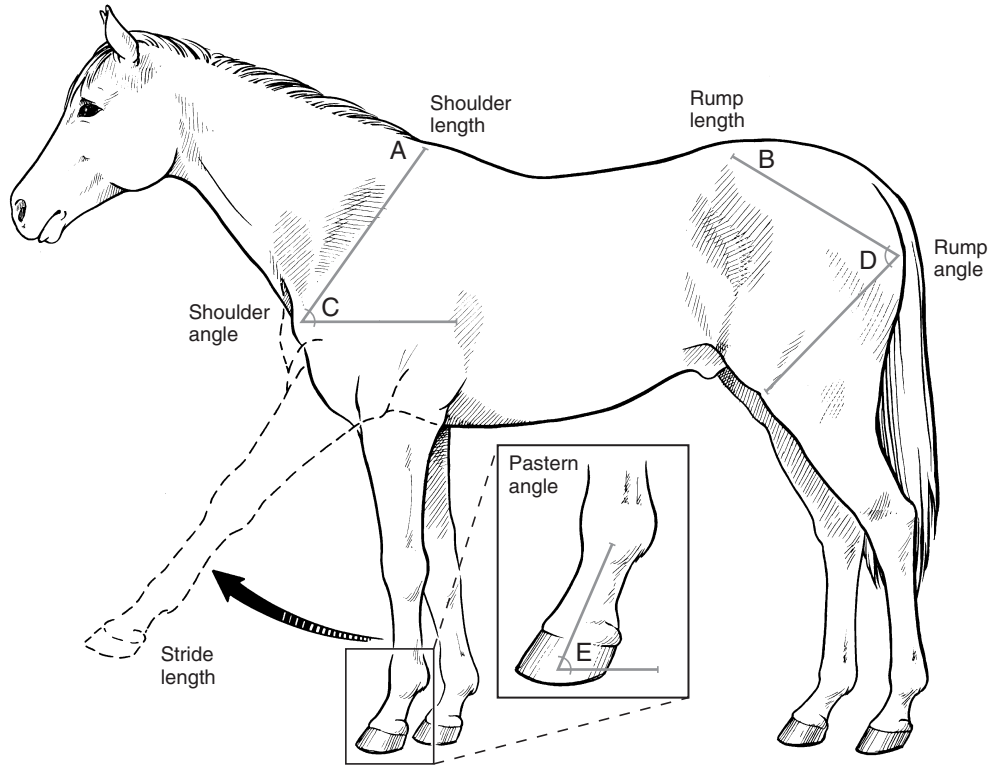

Fig. 4-7 Measurement of shoulder length (*A*), rump length (*B*), shoulder angle (C), and rump angle (*D*). The pastern angle (*E*) should be equal to the shoulder angle. Shoulder angle and length are important in determining stride length.

a vertical line from the point of the shoulder should bisect the limb. The clinician also should evaluate the horse while it is walking because some defects are dynamic. Horses that toe in or toe out, or those with fetlock or carpus varus deformities, may stand reasonably well, particularly with corrective trimming and shoeing, but the defect may be readily apparent while the horse is walking.

FORELIMB CONFORMATION

Front Perspective

Several forelimb conformational abnormalities are apparent when evaluating a horse from the front. *Base-wide* conformation may occur alone or in combination with toed-in or toed-out conditions (Fig. 4-11). Horses that are base wide stand with the forelimbs lateral to the plumb line and generally are narrow in the chest, resulting in overload of the medial aspect of the lower limb, predisposing to lameness. Horses that are *base wide, toed in* tend to wing out or paddle during protraction (Figs. 4-11, *B* and 4-12, *A*). Winging out predisposes trotters to interference with the ipsilateral hindlimb. *Base-wide, toed-out*

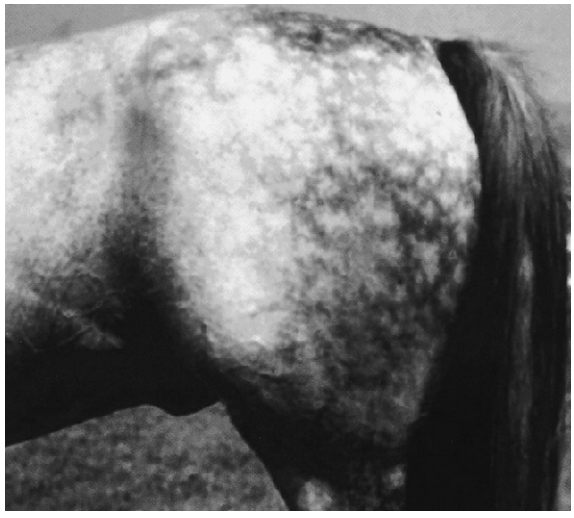

Fig. 4-8 Desirable hindlimb conformation. The flat rump angle and good rump length would likely increase stride length and allow good support and strength of the hindlimbs.

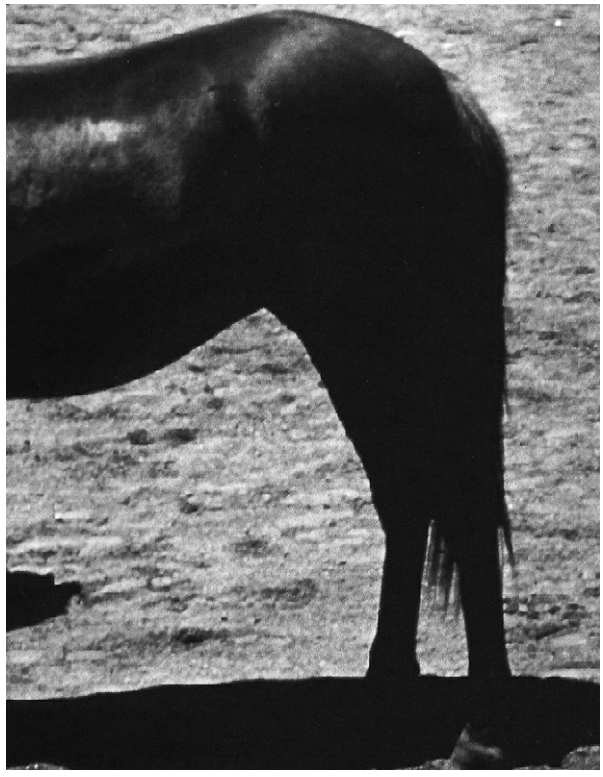

Fig. 4-9 Undesirable hindlimb conformation characterized by short, steep rump (goose rump), predisposing to hindlimb lameness.

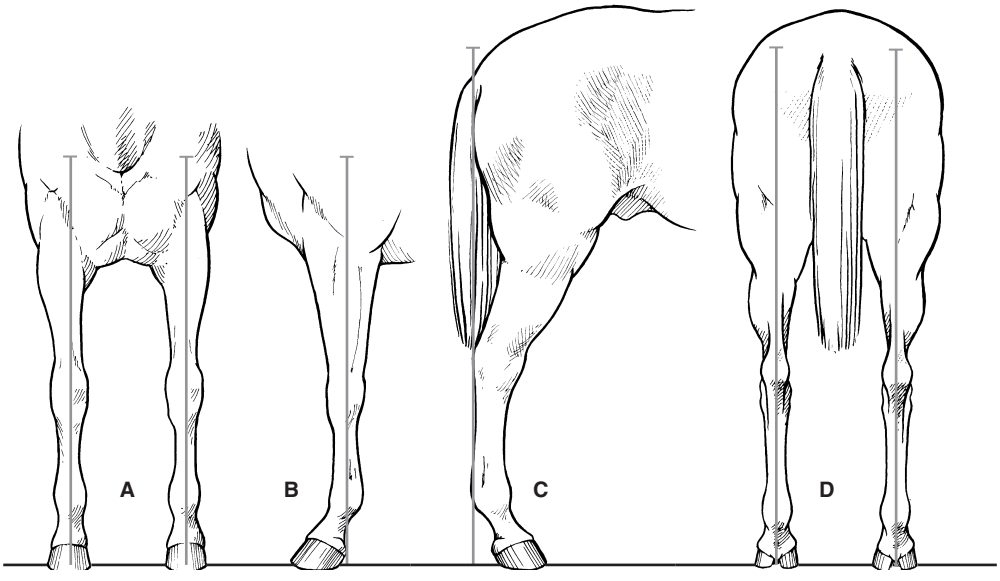

Fig. 4-10 Diagram demonstrating use of plumb lines to evaluate limb conformation from three perspectives. Vertical lines are visualized from the front (**A**) forelimb (line runs from point of the shoulder, bisecting the limb), side (**B**) forelimb (line bisects elbow joint, carpus, and fetlock joint and intersects the ground approximately 5 cm behind the solar surface of the heel), and side (**C**) hindlimb (line runs from point of rump to the ground, touching the point of the hock and plantar metatarsus and intersecting the ground approximately 7.5 to 10 cm behind the heel) and from the back (**D**) hindlimb (line drawn from point of rump, bisecting the hindlimb).

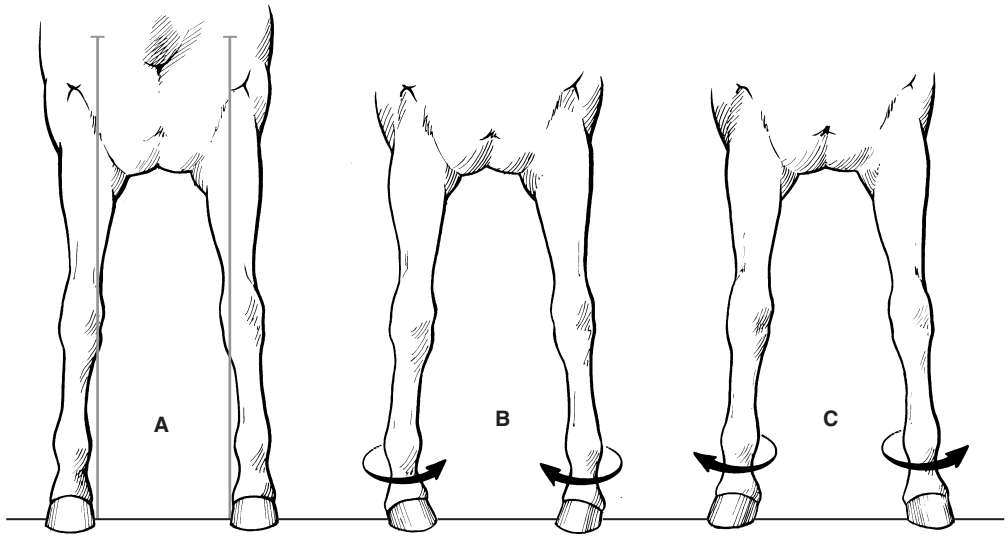

Fig. 4-11 Three variations of base-wide forelimb conformation, including (**A**) simple base wide, (**B**) base wide, toed in, and (**C**) base wide, toed out.

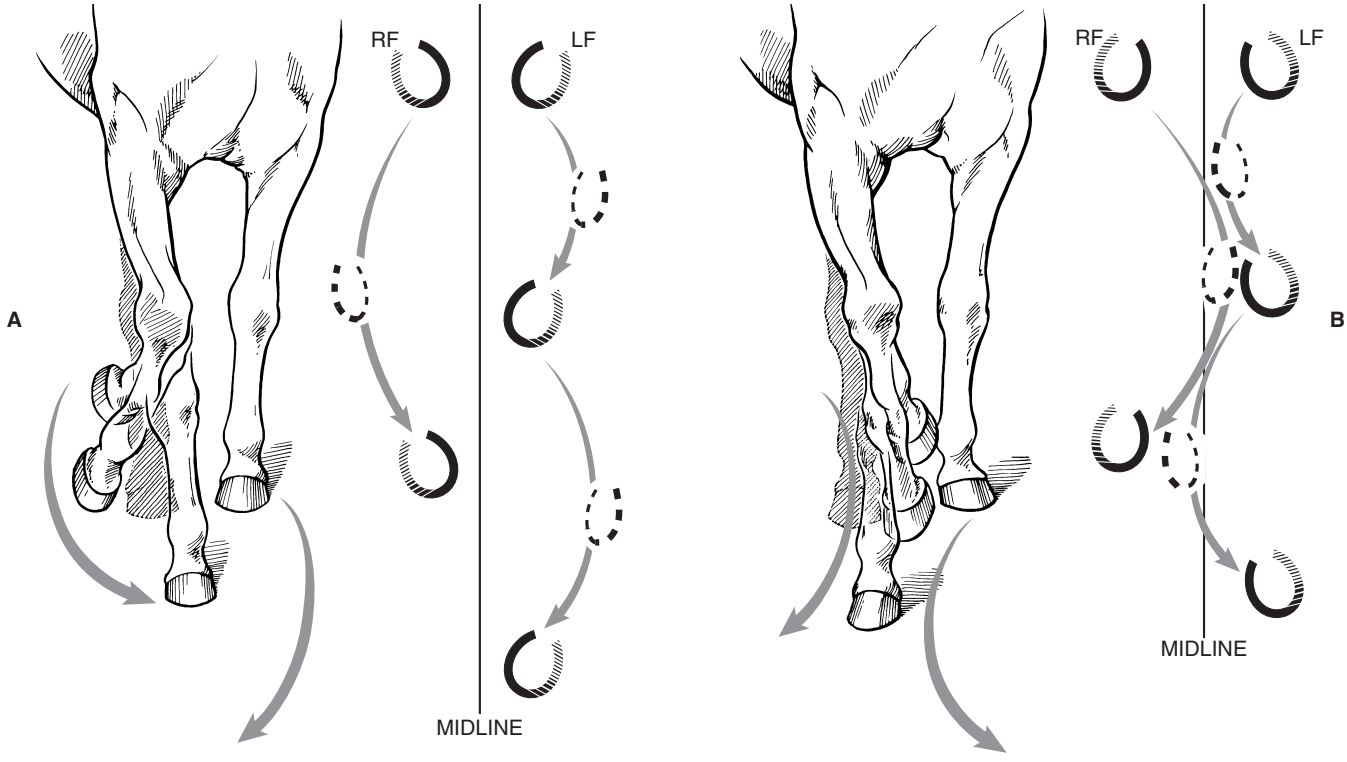

Fig. 4-12 **A,** Toed-in conformation often causes horses to wing out or paddle during advancement of the forelimb. **B,** Toed-out conformation causes horses to wing in, predisposing to interference with the contralateral forelimb.

conformation appears most often in horses with uncorrected carpus valgus deformities (Figs. 4-11, *C* and 4-12, *B*) and results in excessive loading on the medial aspect of the foot and misshapen feet. Interference with the contralateral forelimb may occur in severely affected horses.

Base-narrow conformation may occur alone or in combination with toed-in or toed-out conformation (Fig. 4-13). Horses that are base narrow stand with the forelimbs inside the plumb line and overload the outside of the lower limb and foot. Horses that are *base narrow, toed in* tend to wing out, and those that are *base narrow, toed out* tend to wing in during protraction (see Figs. 4-12 and 4-13).

With *in-at-the-knee*, knock-kneed, or carpus valgus conformation (Fig. 4-14) the carpi are medial to the plumb line, creating an angular deformity and concentrating the weight of the horse on the medial aspect of the carpus and proximal metacarpal region. This condition, if severe, may predispose the horse to carpal lameness and splints. In some horses, particularly foals, carpus valgus may be accompanied by external rotation of the entire limb or just the distal aspect (toed out). Severely affected horses wear the inside aspect of the hoof or shoe abnormally.

Out-at-the-knee (bow-legged, bandy-legged) conformation usually is a consequence of early carpus varus and often is career

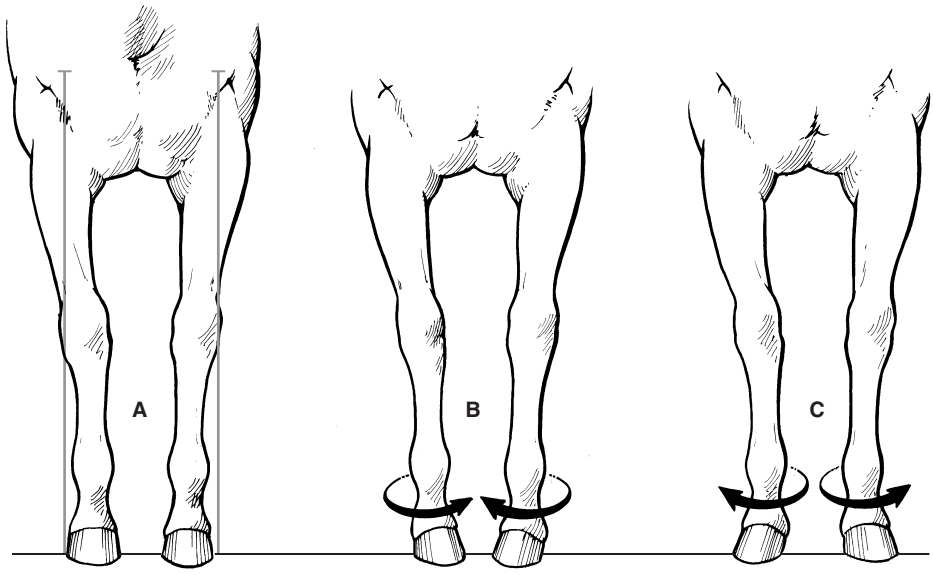

Fig. 4-13 Three variations of base-narrow forelimb conformation including (**A**) simple base narrow, (**B**) base narrow, toed in, and (**C**) base narrow, toed out.

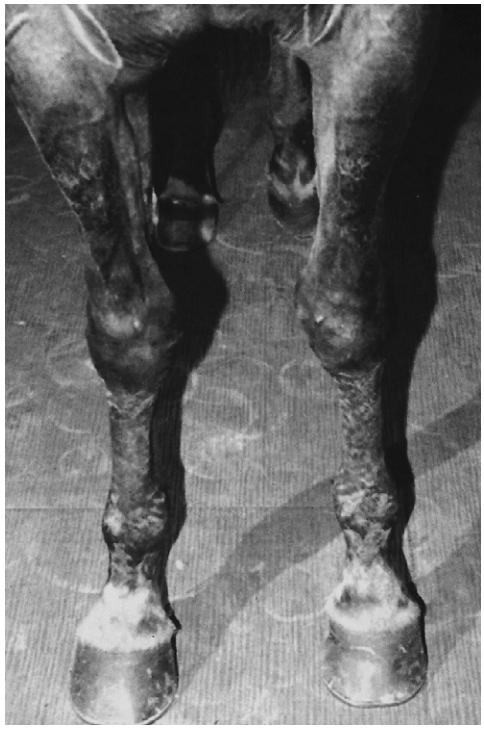

Fig. 4-14 A horse with in-at-the-knee conformation that is worse in the right forelimb.

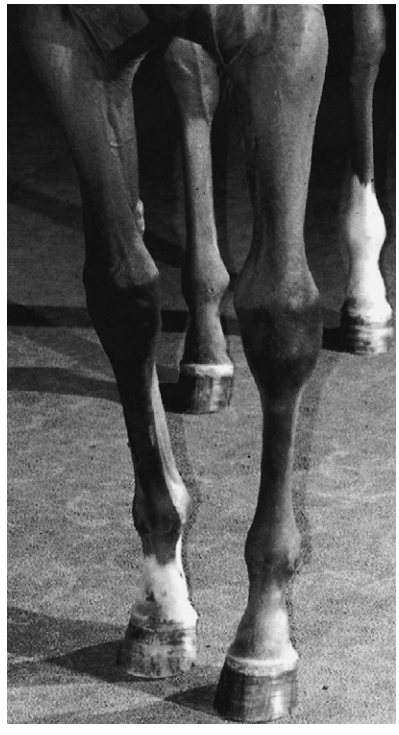

Fig. 4-15 Two-year-old Thoroughbred filly with moderate to severe carpus varus (out-at-the-knee) conformation in the left forelimb and mild deformity in the right forelimb. The filly also is toed in, a common finding in horses with this type of conformation. Another common finding is lateral deviation or bowing of the elbow, prompting clinical suspicion that a deformity also exists at this joint.

limiting, particularly if the fault is pronounced (Fig. 4-15). The carpus is bowed outward, lateral to the plumb line. Many horses are also toed in, accentuating abnormal forces on the lateral aspect of the entire distal forelimb, predisposing to osteoarthritis of the carpus or fetlock or lateral suspensory branch desmitis and sesamoiditis. In most foals with this deformity, lateral deviation of the elbow usually is present. Correction is difficult.

Offset or *bench-knee* conformation is classically defined as lateral positioning of the metacarpal region relative to the central axis of the radius (Fig. 4-16). However, radiographs demonstrate that the actual displacement usually is at the antebrachiocarpal joint. Displacement may be unilateral or bilateral with differing degrees of severity on each side. This conformation often is associated with carpal or metacarpal lameness (Fig. 4-17). Many 2-year-old STBs with offset knees are precocious during early training but often develop carpal lameness at 3 or 4 years of age. TBs with offset knees are believed to perform better on the soft turf tracks in Europe, as opposed to the firm turf or dirt tracks in the United States.

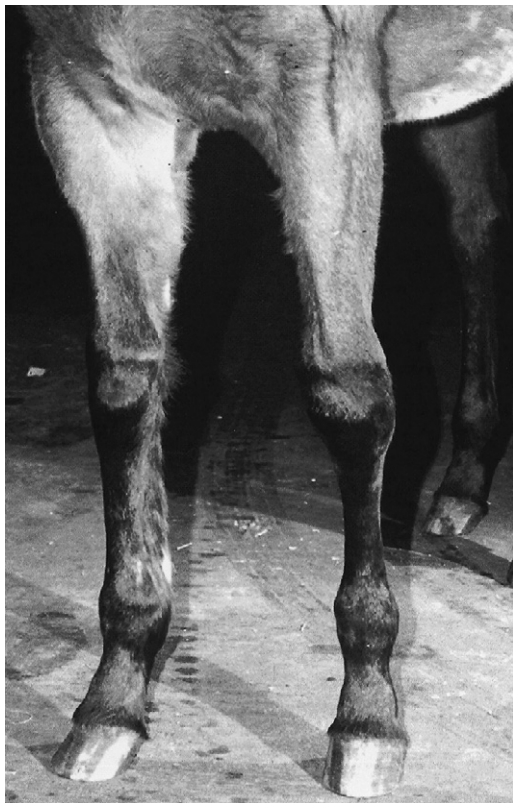

Fig. 4-16 Standard Thoroughbred with a prominently offset (bench) knee in the left forelimb. An apparent lateral deviation or shifting (offset) of the cannon bone and distal extremity occurs relative to the plumb line dropped through the left radius.

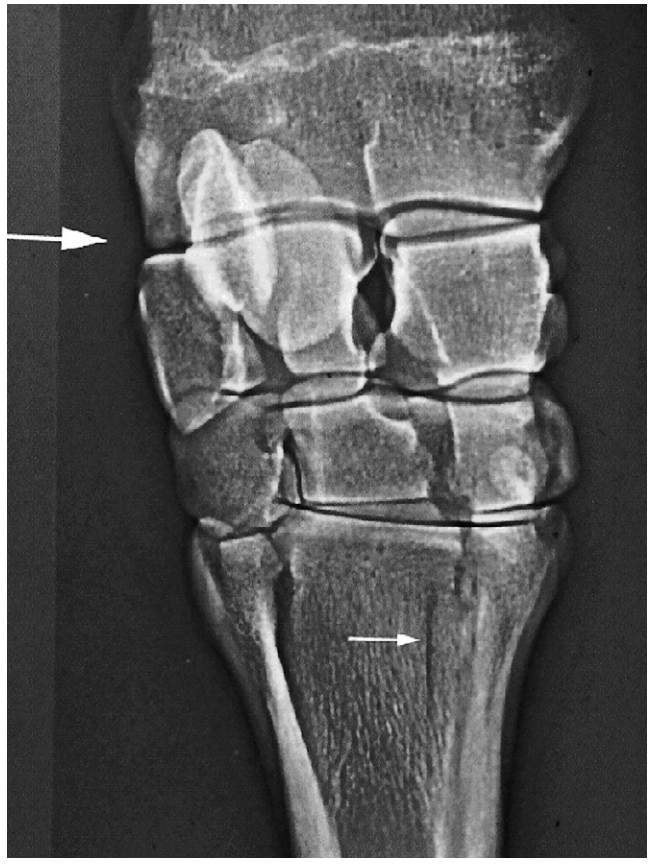

Fig. 4-17 Dorsopalmar xeroradiograph of 3-year-old STB colt with longitudinal fracture of the third metacarpal bone (*small arrow*). Medial is to the right. The lateral displacement ("step") at the antebrachiocarpal joint (*large arrow*) gives the clinical appearance of an offset (bench) knee.

Toed-in conformation, or internal rotation of the distal extremity, exists alone or in combination with abnormalities of stance (base-narrow or base-wide), other conformational faults such as carpus varus, and being wide in the chest (see Fig. 4-15). Horses that are toed in usually wing out (paddle) (see Fig. 4-12). Toed-in conformation is particularly undesirable in a trotter because of potential interference at speed. Toed-in conformation predisposes horses to lateral splints, lameness of the lateral aspect of the fetlock joint region (e.g., lateral branch suspensory desmitis), and osteoarthritis of the interphalangeal joints. Horses that are toed in wear the outside aspect of the foot.

Toed-out conformation, or external rotation of the distal extremity, is common and if mild may be considered normal or inconsequential (Fig. 4-18). Mild toed-out conformation appears in 50% of STBs and is common behind in STBs and WBLs.[9,11] It first develops in foals and, if pronounced, persists in the mature horse. In foals, toed-out conformation often accompanies carpus valgus deformities but may result from external rotation primarily from the fetlock down but, in more severe deformities, from further proximally (Fig. 4-19). Mild toed-out conformation usually resolves as a foal matures and with corrective trimming. Toed-out conformation results in abnormal wear on the inside aspect of the foot. Horses tend to wing in; if winging in is severe, particularly if accompanied by base-narrow conformation, it may interfere with the opposite forelimb (see Fig. 4-12). Exostoses (splints) on the second metacarpal bone (McII) or McIII may develop, requiring protective boots to be worn during exercise. In STBs, interference injury occurs as high as the distal radius.

Lateral Perspective

Horses camped out in front stand consistently with the entire forelimb ahead of the plumb line, but this conformation usually is a temporary problem with the horse's stance and can be corrected by repositioning the horse, or it reflects pain caused by laminitis, for example. C*amped under in front* is unusual and usually also results from temporary malpositioning of the horse (Fig. 4-20). If a horse prefers to stand camped under and is otherwise sound, however, this trait may be a sign of "extreme speed."

Back-at-the-knee or *calf-knee* (sheep-knee) conformation describes a concave dorsal aspect of the limb, with the carpus behind the plumb line (Figs. 4-21 to 4-23). On radiographs of a normal carpus the proximal and distal rows of carpal bones are aligned in a proximal to distal direction, and the dorsal faces of these bones are parallel to the radius and McIII (Fig. 4-22, *A*). With back-at-the-knee conformation the proximal row of carpal bones is set back (Fig. 4-22, *B*). Horses that stand back at the knee are considered predisposed to carpal injuries because of the natural tendency of the carpus to hyperextend (larger carpal angle) during fatigue. In my experience, TB racehorses are particularly at risk, despite limited contrary evidence (see page 16).[13]

In the STB, mild calf-knee conformation is common in pacers and acceptable, whereas in the trotter this defect is undesirable. In other breeds, mild calf-knee conformation may not directly lead to lameness. In young horses with lameness from unrelated sources such as osteochondrosis or fracture of the distal phalanx, back-at-the-knee conformation and clubfoot (small, upright foot) may accompany flexural deformity

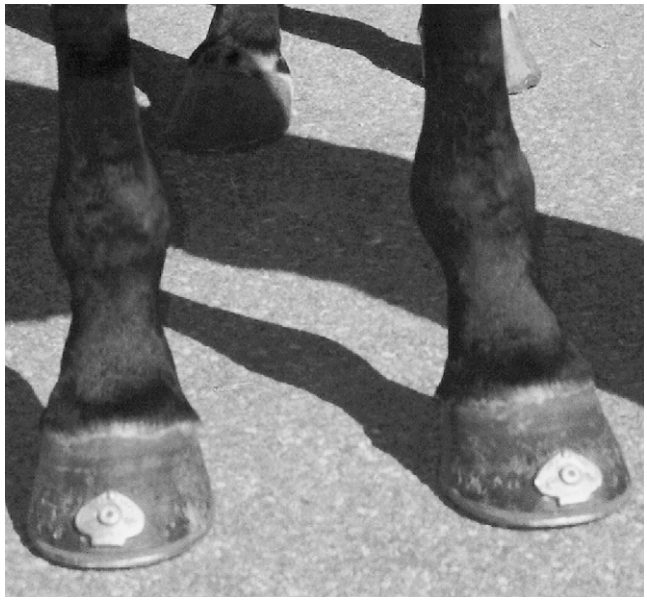

Fig. 4-18 Trotter showing inconsequential mild toed-out conformation. Toe weights often are used in trotters to balance gait and correct interference.

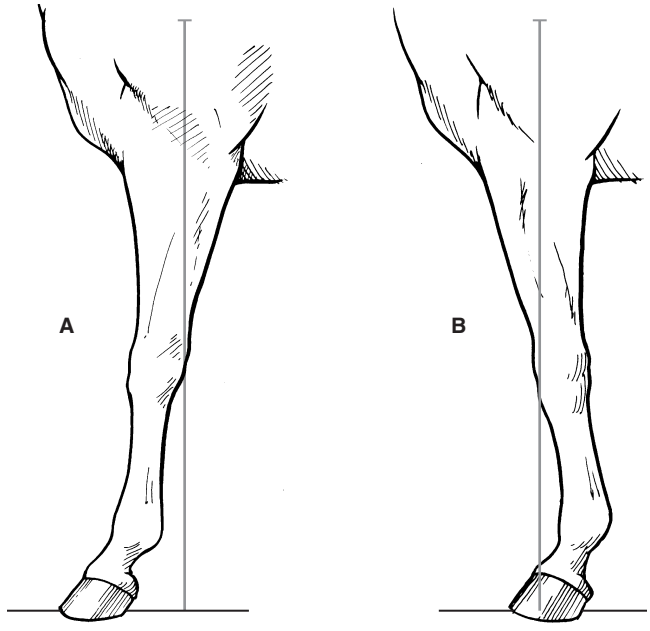

Fig. 4-20 Diagram showing (A) camped-out in front and (B) camped-under in front conformation, both in relation to plumb lines.

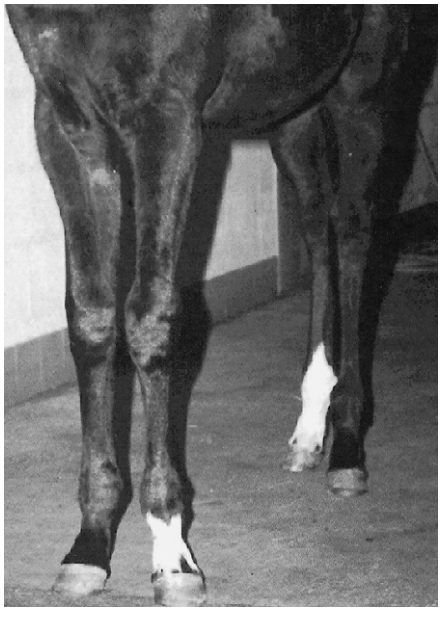

Fig. 4-19 Thoroughbred foal with pronounced external rotation or toed-out left forelimb limb conformation. The deformity involves the entire limb, beginning well above the carpus.

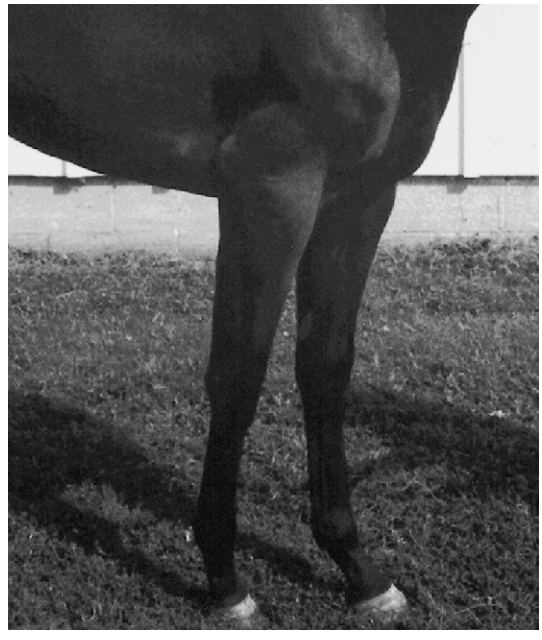

Fig. 4-21 Thoroughbred yearling with back-at-the-knee (calf-knee) conformation most noticeable in the left forelimb. This conformational fault is undesirable, particularly in racing breeds.

of the limb (see Fig. 4-23). This deformity is a combination of contraction (clubfoot) and laxity (calf knee) caused by chronic lameness and partial weight bearing and warrants a guarded prognosis.

Over-at-the-knee, bucked-knee (knee-sprung), or hanging-knee conformation describes a convex dorsal surface of the carpus, with the carpus in front of the plumb line (Fig. 4-24). In young, untrained horses, bucked-knee conformation may be a predictor of lameness, but in mature horses it appears to be an acquired characteristic and occurs primarily in horses that jump. Older cross-country horses, steeplechasers, jumpers, or field hunters are prone to buck knee and often stand over at the knee with no obvious lameness. These horses may exhibit a tendency to buck forward to such an extent that they appear on the verge of collapse or prone to stumbling yet show good stability. *Tied in below the knee* (Fig. 4-25) describes a distinct notch just distal to the accessory carpal bone on the palmar aspect of the limb. Normally, McIII and the flexor tendons are in parallel alignment from the accessory carpal bone to the proximal sesamoid bones. With tied-in conformation the flexor tendons appear to enter the carpus in a dorsoproximal direction. If the horse also is bucked kneed, the tied-in appearance

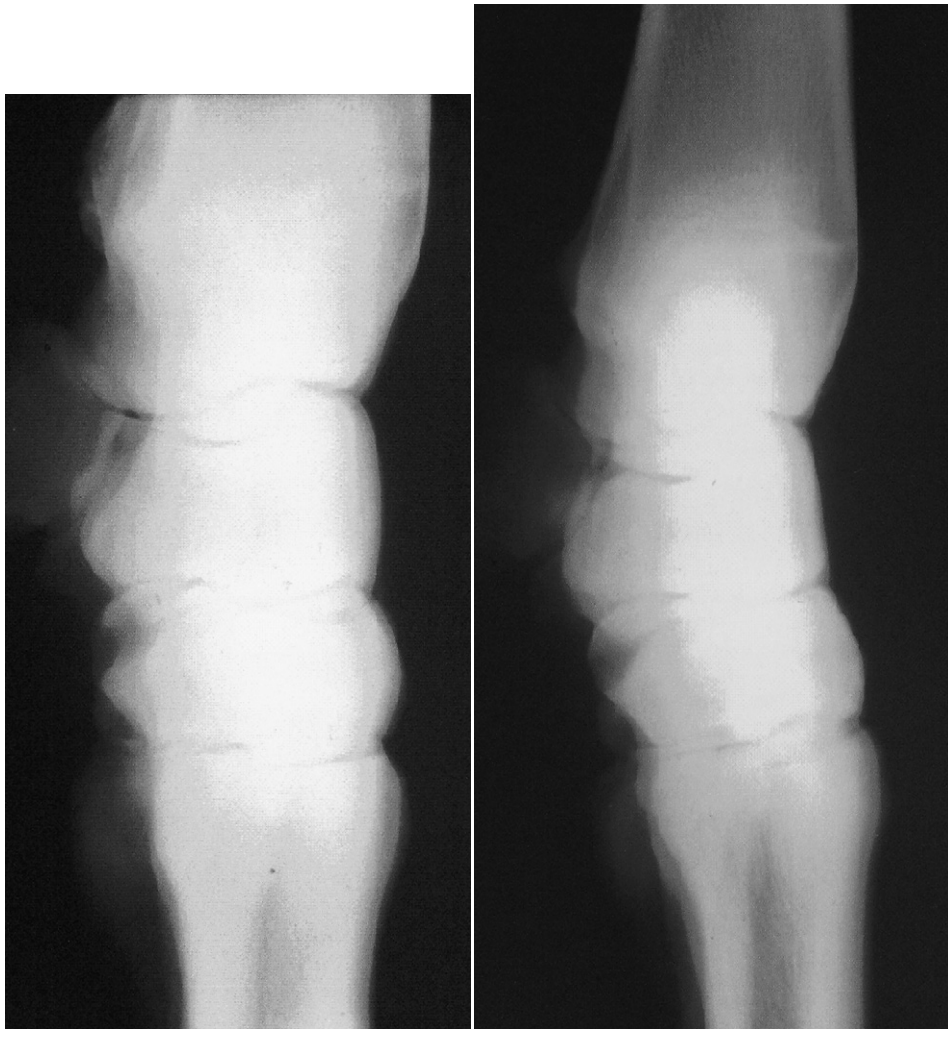

Fig. 4-22 Lateromedial radiographs of two different horses with carpal lameness. **A,** In a horse with nondisplaced slab fracture of the third carpal bone, normal carpal conformation is present: the proximal and distal rows of carpal bones are aligned and parallel to the radius and third metacarpal bone. **B,** In a horse with distal, lateral radius chip fracture, there is palmar deviation of the proximal row of carpal bones. This radiographic appearance is typical of back-at-the-knee conformation.

is accentuated. Young horses are prone to superficial flexor tendonitis. In STBs this defect is worse for a pacer than a trotter.

The junction of the carpus and McIII should be flat. C*ut out under knee* describes a notch under the dorsal surface of the carpus (see Fig. 4-25). In horses with this defect, McIII appears thin (dorsopalmar direction) and weak. Horses with this conformational abnormality also are often back at the knee, predisposing them to carpal and metacarpal problems. Some young racehorses, typically late yearlings or early 2-year-olds in training, appear to have distention of the middle carpal joint capsule or an unusually prominent distal radial epiphysis. These findings give the impression of an unusually large gap between these structures, described as open at the knee. I have not seen a correlation between this clinical observation and obvious radiographic changes, although in young horses with this conformation the distal radial physis remains visible. Whether this conformation is relevant to lameness in young racehorses is debatable.

O*ver-at-the-fetlock* usually is seen in young horses with flexor deformity of this joint (see Chapter 61). This conformational fault may persist in a mature horse, causing upright pasterns or knuckling of the fetlock joint. In some horses this condition causes a progressive, permanent deformity and severe lameness, whereas in others a dynamic, intermittent knuckling occurs and some of these horses remain surprisingly sound. Knuckling also may be a sequel to desmitis of the accessory ligament of the deep digital flexor tendon.

HINDLIMB CONFORMATION

Lateral Perspective

Hindlimb conformational faults generally are less numerous and problematic than those in the forelimb because of differences in weight distribution and center of gravity. Plumb lines also are useful in evaluating conformation of the hindlimb with the horse standing squarely, loading all limbs (see Fig. 4-10). Camped-out conformation is unusual and generally results from faulty positioning of the horse during the examination. Horses that are truly camped out usually have short strides and poor athletic ability. Camped under behind often is associated with sickle-hocked conformation but also appears in horses that are straight behind (Fig. 4-26). Horses with this type of conformation often have short, choppy strides (Fig. 4-27).

A particularly severe conformational fault that leads directly to lameness is *straight behind*, otherwise called *straight hocks* or *post* (posty) leg. Horses that are straight behind have larger stifle and hock angles but smaller fetlock joint angles compared

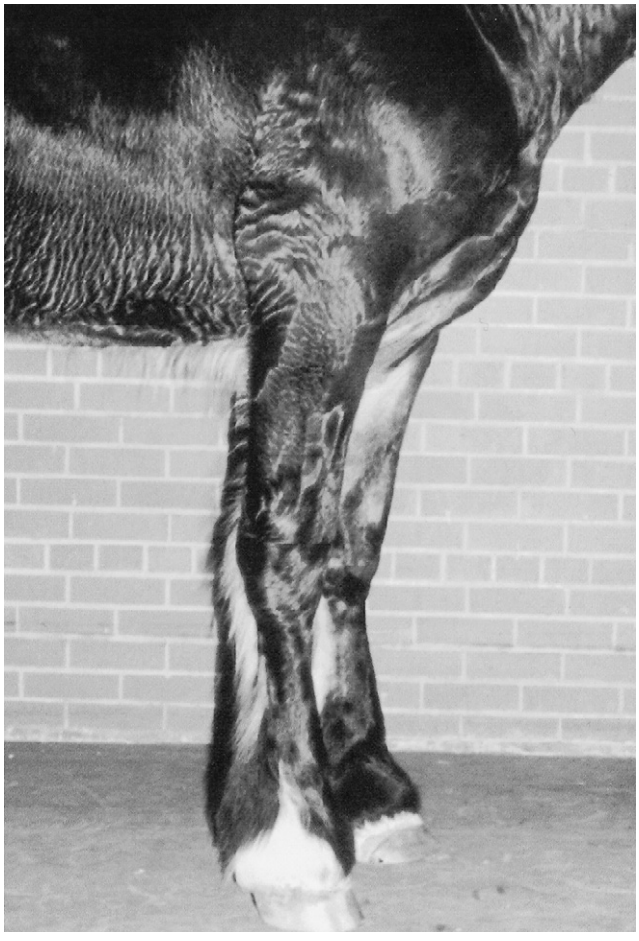

Fig. 4-23 Clydesdale yearling with calf-knee conformation and clubfoot secondary to osteochondrosis of the shoulder joint. This conformation is primarily the result of chronic lameness, decreased weight bearing, and the development of a flexural deformity.

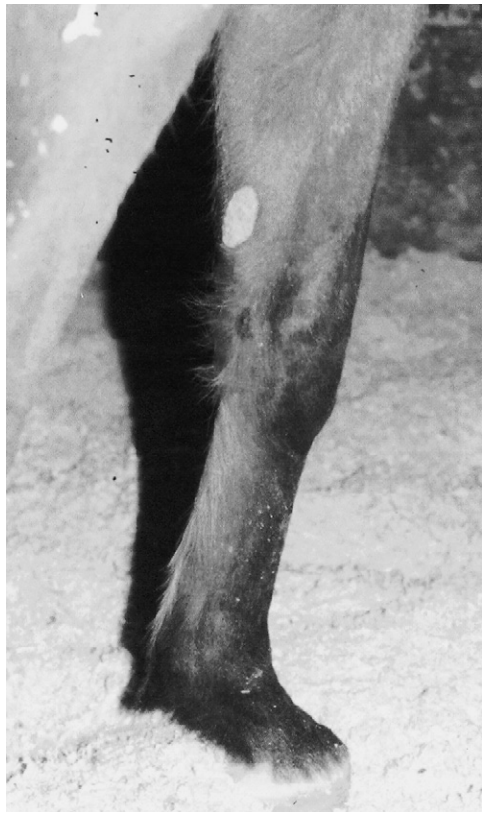

Fig. 4-24 Older horse without obvious lameness with over-at-the-knee (bucked-knee) conformation.

with ideal hindlimb conformation (see Fig. 4-26; Fig. 4-28). Straight behind, sickle-hocked, and in-at-the-hock conformation are the three most important hindlimb conformational faults and all may lead directly to lameness. Horses that are straight behind often develop upward fixation of the patella, a condition seen most often in WBLs. Suspensory desmitis and fetlock osteoarthritis also occur frequently. Horses with normal initial hindlimb conformation may become straight behind if they develop severe suspensory desmitis and lose support of the fetlock joint.

Sickle-hocked conformation is one of the most common conformational faults, and it leads directly to lameness of the tarsus and plantar soft tissues. Horses that are sickle-hocked stand with the lower hindlimb well ahead of the plumb line, with an exaggerated concave dorsal surface of the hindlimb (resembling a sickle), creating a smaller than normal hock angle (see Fig. 4-26; Fig. 4-29). This type of conformation is often called *curby* conformation because horses frequently develop curbs (see Chapter 79). Sickle-hocked conformation concentrates load in the distal, plantar aspect of the hock, predisposing to curb and to distal hock joint pain. In foals with incomplete or delayed ossification of the tarsal bones, marked sickle-hocked conformation may occur (see Chapter 45). Sickle-hocked conformation is undesirable, particularly in racing breeds, but if mild is not detrimental. In STB pacers, some prefer a mild degree of sickle-hocked conformation because horses can extend the hindlimbs farther forward without risk of interference. In STB trotters the condition

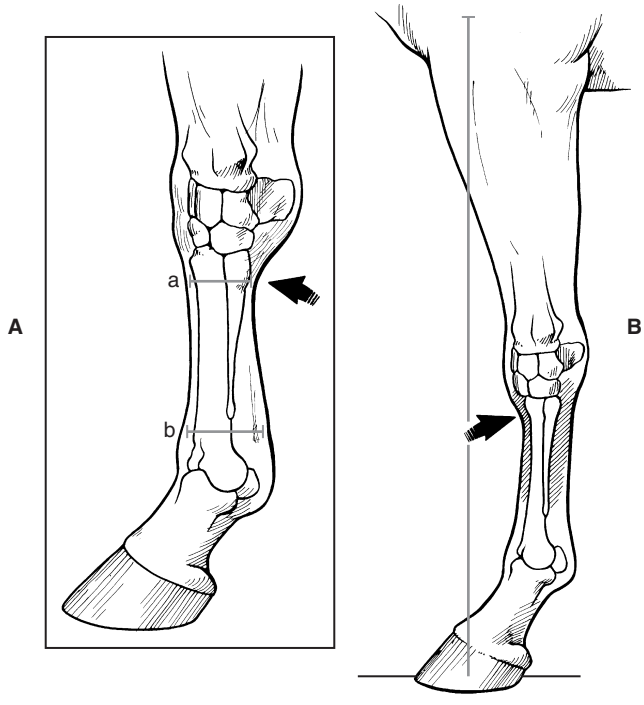

Fig. 4-25 **A,** Diagrammatic representation of tied in below the knee. The dorsal-palmar length of *a* is less than *b*, giving the appearance that the flexor tendons run obliquely, proximally to enter the distal carpal region more dorsal than expected. **B,** A horse that is cut out under the knee has a concave appearance of the dorsal aspect of the distal carpus and proximal metacarpal region.

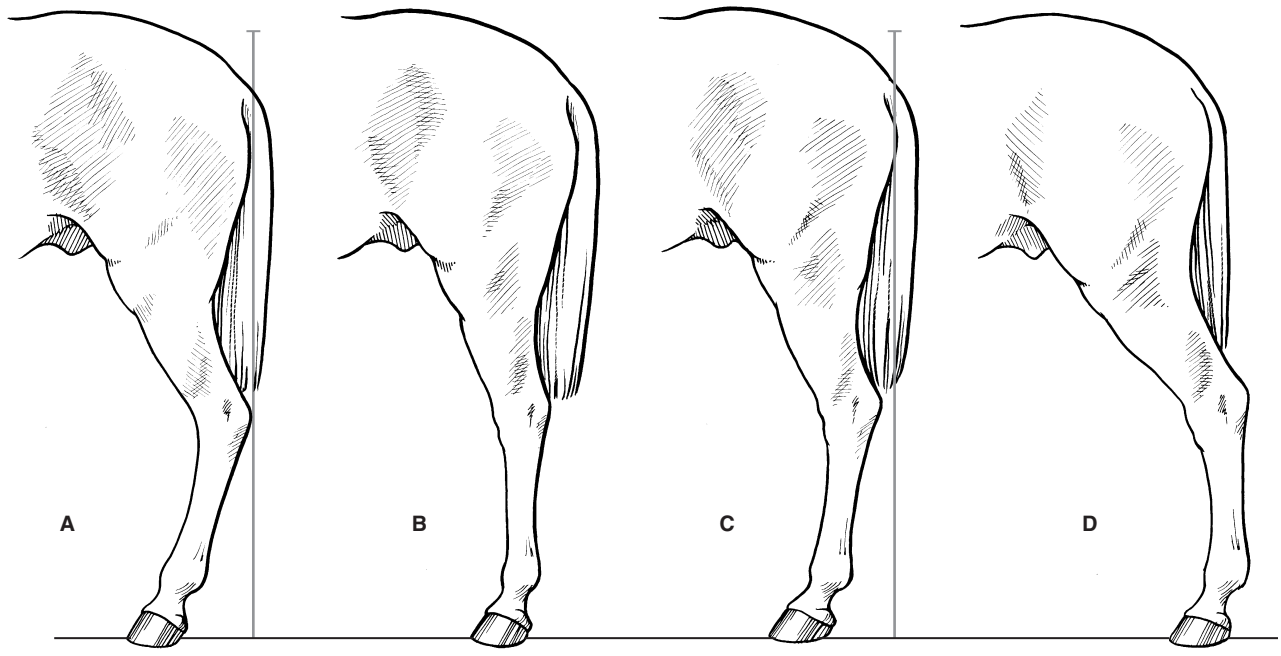

Fig. 4-26 Diagrammatic representation of conformational faults of the hindlimb from the lateral perspective. Compared with ideal conformation, horses with sickle-hocked conformation (**A**) have a concave dorsal surface of the limb with the distal extremity dorsal to the plumb line, but those that are straight behind (**B**) have large stifle and hock joint angles but smaller fetlock joint angles. Horses that are camped under (**C**) often are sickle-hocked as well. Camped-out conformation (**D**) is unusual and most often results from faulty positioning of the horse.

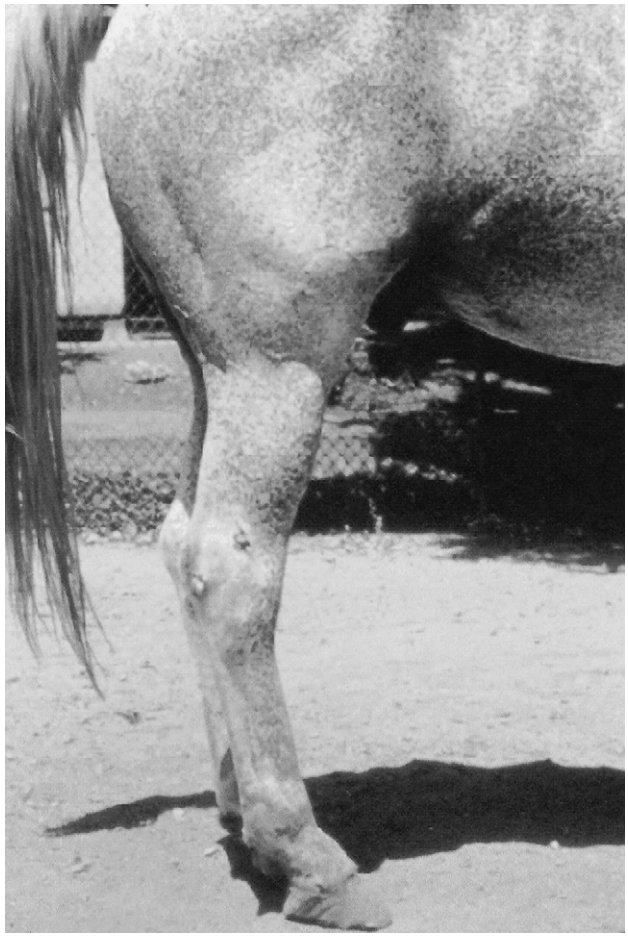

Fig. 4-27 Horse with camped-under and mild sickle-hocked conformation, a combination leading to a short, choppy gait.

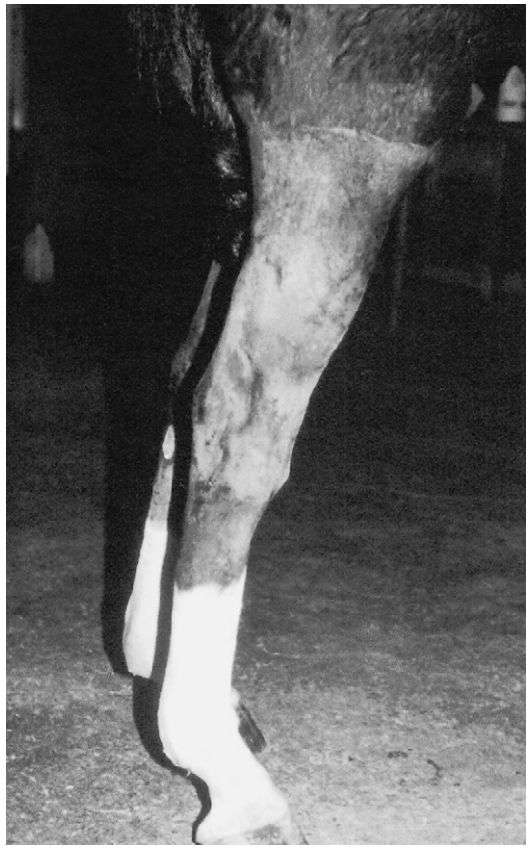

Fig. 4-28 Standardbred filly with straight hindlimb conformation and suspensory desmitis.

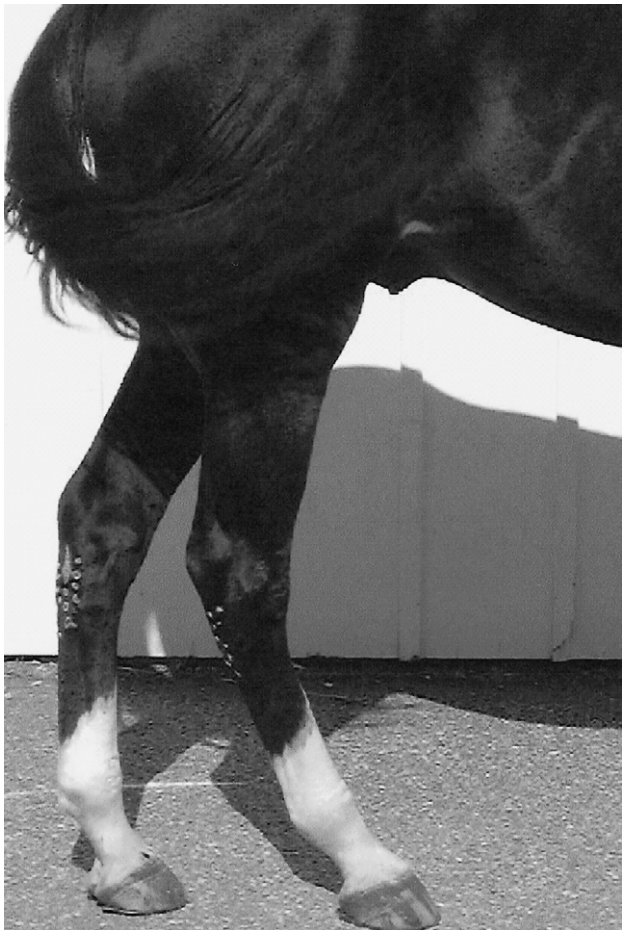

Fig. 4-29 This 4-year-old Standardbred has sickle-hocked conformation and has developed curb, which has been treated by freeze-firing, resulting in white marks on the plantar aspect of the hock.

often predisposes to distal hock joint pain and curb, but these horses tend to be fast, although unsound. In Western reining horses, sickle-hocked horses may be able to slide better.

Rear Perspective

A majority of STB and WBL horses toe out behind, which should be considered normal. Horses with mild external rotation of the distal extremity are called toed out and usually also have external rotation of hocks, causing the points of the hocks to be closer than normal. This fault is called *cow-hocked* conformation and is a *rotational* change of the hindlimb (Fig. 4-30). Cow-hocked conformation occurs in combination with base-wide or base-narrow deformities or independently. Cow-hocked and base-narrow conformation is most common. Base-wide and base-narrow conformation may occur without cow-hocked conformation. These conformational faults seldom lead to lameness but have a substantial effect on gait in some horses. Horses that are base narrow travel close behind, particularly at a walk. Some travel close at a trot, pace, or gallop, whereas others seem to widen out when going faster, thus avoiding interference. Those that travel close at speed often interfere, causing injury to the medial aspect of the contralateral hindlimb.

Bow-legged hindlimb conformation, in which the point of both hocks is truly outside the plumb line, is uncommon (see Fig. 4-30). Occasionally horses that are base-narrow appear to be bow-legged. Unilateral bow-legged conformation occurs in foals born with windswept deformity or in those with tarsus varus deformity. Bilateral tarsus varus deformity is unusual.

In-at-the-hock or tarsus valgus is an *angular* deformity (Fig. 4-31). The deformity can be corrected in foals. If it persists in a mature horse, particularly a racehorse with other conformational abnormalities, such as sickle-hocks, abnormal forces or load occur in the tarsal region, predisposing the horse to distal hock joint pain, curb, and proximal metatarsal lameness.

Horses can be toed in or toed out behind, but in general the conformational abnormality starts above the fetlock joint, causing the lower limb abnormality to be linked with the upper limb. Thus a horse that is toed in generally is bow-legged, and one that is toed out is cow hocked. In some foals, however,

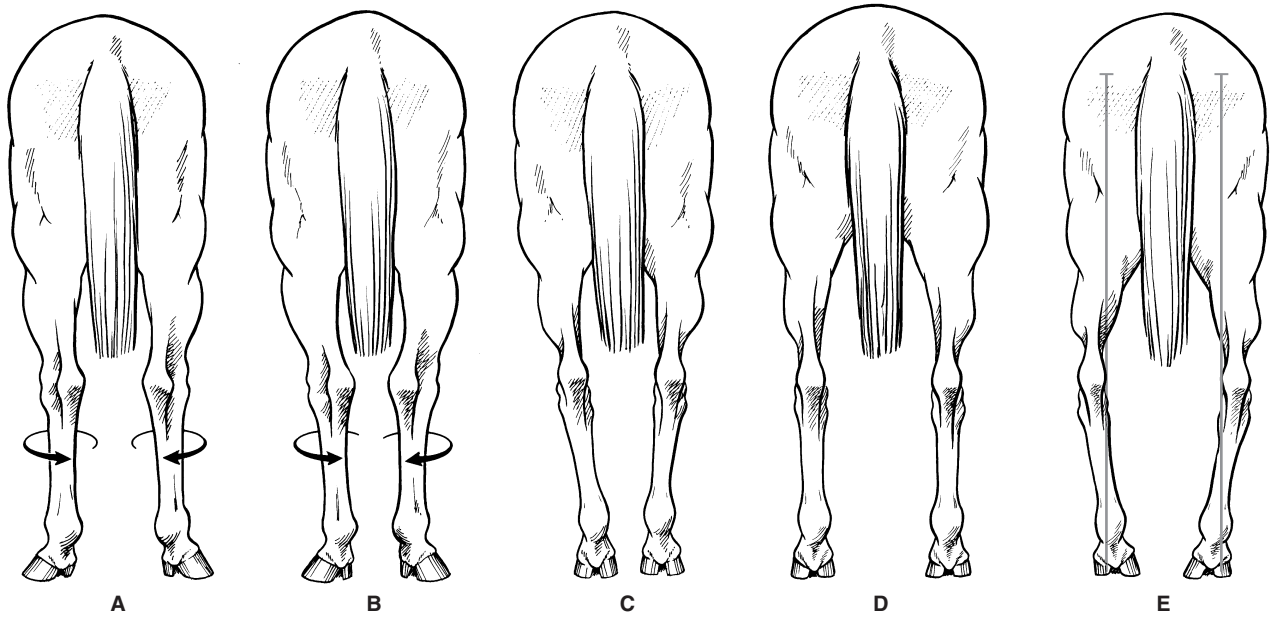

Fig. 4-30 Hindlimb conformational abnormalities viewed from the rear. **A,** Cow-hocked conformation is a common fault characterized by external rotation of the limb, usually without angular deformity, causing the hocks to be too close together. Mild external rotation of the hindlimbs is common and does not appear to cause lameness. **B,** Cow-hock, base-narrow conformation. **C,** Base-narrow conformation. **D,** Base-wide conformation is uncommon. **E,** Bow-legged conformation is uncommon and undesirable.

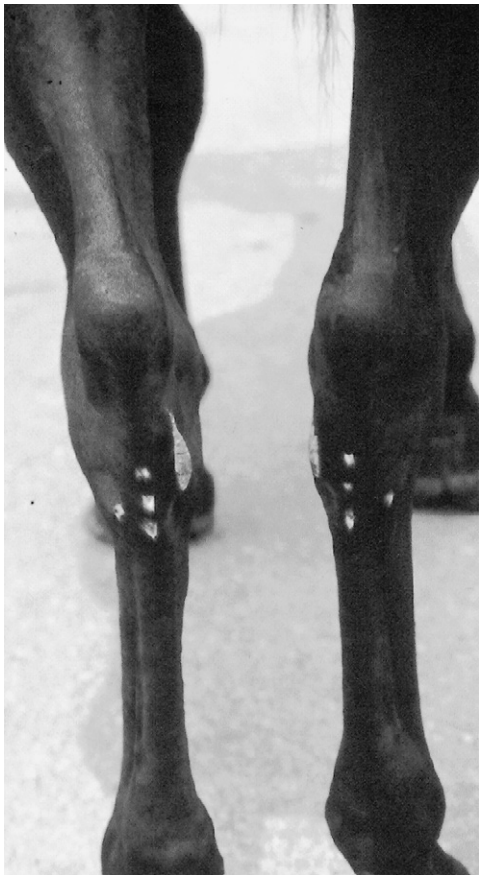

Fig. 4-31 Mature Standardbred racehorse with in-at-the-hock (tarsus valgus) conformation. This conformational abnormality is characterized by an angular deformity as opposed to the rotational deformity seen in cow-hocked conformation. The characteristic white marks were produced by cryotherapy for treatment of bilateral curb.

fetlock varus occurs independent of upper limb conformational abnormalities. This abnormality usually appears in windswept foals in which upper limb deformities have resolved, leaving a fetlock varus. This is an angular deformity, but with abnormal hoof wear, toed-in conformation can develop. Fetlock varus and the resulting toed-in conformational defect may cause osteoarthritis of the fetlock and interphalangeal joints and can be career limiting.

CONFORMATION OF THE DIGIT

More detailed aspects of conformation of the foot and limb flight characteristics are discussed in Chapters 5 and 7. Many of the changes in hoof growth or conformational changes in the hoof are the result of wear, shoeing, and exercise demands of training and performance and often are not present in a young horse.

The pastern angle is usually similar to the angle of the shoulder (see Fig. 4-7). The pastern foot axis should be straight. The pastern should be neither excessively sloped (low angle) nor upright (high angle). The angle of the pastern is important in determining the amount of load on the lower limb structures. In general, the more upright the pastern (steeper pastern angle), the shorter the stride and vice versa. Horses with upright pasterns appear to be prone to foot lameness and perhaps superficial digital flexor tendonitis. Those with long, sloping pasterns may be at risk to develop osteoarthritis of the fetlock joint and proximal phalangeal fractures. Horses with short, upright pasterns but relatively normal hoof angles have a broken foot-pastern axis—the foot axis is lower than the pastern axis—and are at risk of developing foot lameness (Fig. 4-32). If the pastern axis is lower than the foot axis, called *coon footed*, it causes undue strain on the soft tissue structures supporting the fetlock joint. This type of conformation may result from severe suspensory desmitis and loss of support of the fetlock joint.

Pastern length is important and usually is related to pastern angle. Horses with long pasterns commonly have more slope or lower pastern angles. The plumb line should drop approximately 5 cm behind the heel in a well-conformed horse. In horses with long, sloping, and weak pasterns, the line drops more than 5 cm behind the heel. Those with short pasterns usually have more upright pasterns, and the plumb line drops

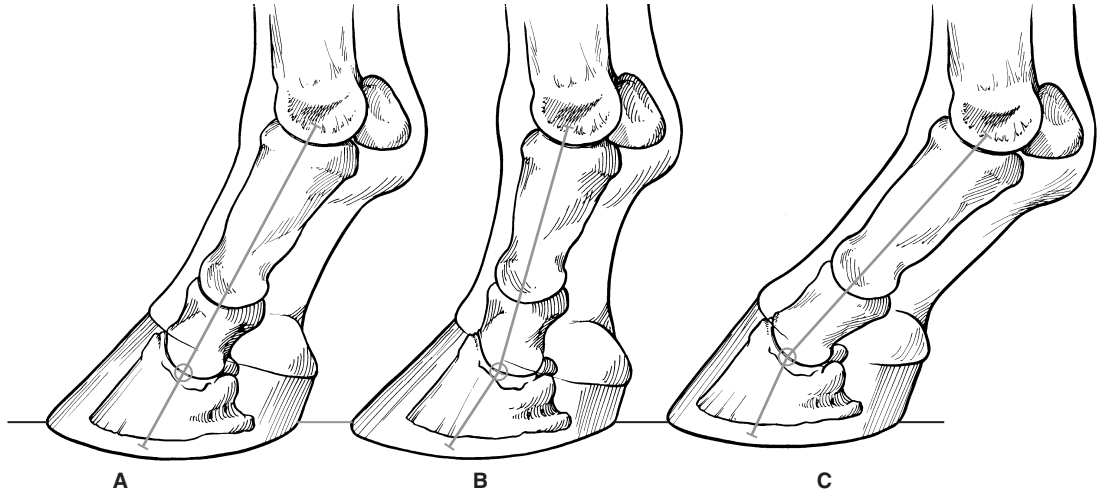

Fig. 4-32 Diagrammatic representation of ideal pastern and foot conformation and the concept of a broken pastern-foot axis. Ideally the foot and pastern angles (**A**) should be identical to allow full and even weight bearing on all aspects of the foot. A broken foot axis (**B**) occurs when pastern angle is more upright than that of the foot or vice versa (**C**). In both latter situations, uneven load distribution on the foot or soft tissue structures may cause lameness.

through the foot. A variety of pastern lengths and angles occur, but the pastern length should be in proportion to the overall length of the limb.

Viewed from the front, the plumb line may divide the pastern and foot asymmetrically with more pastern and foot laterally, which often is associated with some degree of distortion of the hoof capsule, with a steeper medial wall and some flaring laterally. This results in asymmetrical loading of the distal limb joints and may predispose to lameness.

Buttress foot is an acquired firm bulge or swelling at and proximal to the dorsal aspect of the coronary band and usually reflects osteoarthritis of the distal interphalangeal joint.

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CHAPTER • 5

Observation: Symmetry and Posture

Mike W. Ross

Assessments of symmetry and posture are important aspects of a lameness examination. Comparison between the normal and abnormal sides facilitates identification of abnormalities, unless the condition is bilateral so that no recognizable differences exist between the left and right limbs. The horse should be standing squarely on a flat surface in a quiet, insect-free environment. Horses with severe lameness often are reluctant to stand correctly, but information gained about symmetry and posture of severely lame horses is valuable. The veterinarian should look carefully at size, shape, contour, heights, and widths, and compare with the opposite side.

FORELIMB SYMMETRY

Muscle Atrophy

The symmetry of skeletal muscle in the forearm, pectoral, and cervical areas should be assessed. Muscle atrophy that occurs in horses with chronic lameness conditions is called *disuse atrophy* and in those with neurological disease is called *neurogenic atrophy*. Horses with muscle atrophy and lower motor neuron disease (see Chapter 11) may be lame, sometimes the result of muscle pain or nerve root pain, complicating differentiation between these causes of muscle atrophy. In most but not all horses with neurogenic atrophy, other clinical signs suggestive of neurological disease may be present. Horses with disuse atrophy resulting from chronic lameness usually have generalized atrophy of the ipsilateral forelimb. Muscle loss usually is not pronounced but involves the forearm (extensors are most commonly affected), triceps, and shoulder muscles. Shoulder muscle atrophy involving the infraspinatus and supraspinatus muscles generally is not pronounced, and lateral subluxation of the shoulder joint during weight bearing is not present (see Chapter 41).

Development of disuse atrophy resulting from chronic lameness generally takes weeks to months unless severe lameness exists. In horses with severe or non–weight-bearing lameness, atrophy may develop within 10 to 14 days. In horses with severe forelimb lameness, carpal contraction (flexor deformity of the carpus) may occur simultaneously with muscle atrophy. The most common cause of carpal contraction because of

through the foot. A variety of pastern lengths and angles occur, but the pastern length should be in proportion to the overall length of the limb.

Viewed from the front, the plumb line may divide the pastern and foot asymmetrically with more pastern and foot laterally, which often is associated with some degree of distortion of the hoof capsule, with a steeper medial wall and some flaring laterally. This results in asymmetrical loading of the distal limb joints and may predispose to lameness.

Buttress foot is an acquired firm bulge or swelling at and proximal to the dorsal aspect of the coronary band and usually reflects osteoarthritis of the distal interphalangeal joint.

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CHAPTER • 5

Observation: Symmetry and Posture

Mike W. Ross

Assessments of symmetry and posture are important aspects of a lameness examination. Comparison between the normal and abnormal sides facilitates identification of abnormalities, unless the condition is bilateral so that no recognizable differences exist between the left and right limbs. The horse should be standing squarely on a flat surface in a quiet, insect-free environment. Horses with severe lameness often are reluctant to stand correctly, but information gained about symmetry and posture of severely lame horses is valuable. The veterinarian should look carefully at size, shape, contour, heights, and widths, and compare with the opposite side.

FORELIMB SYMMETRY

Muscle Atrophy

The symmetry of skeletal muscle in the forearm, pectoral, and cervical areas should be assessed. Muscle atrophy that occurs in horses with chronic lameness conditions is called *disuse atrophy* and in those with neurological disease is called *neurogenic atrophy*. Horses with muscle atrophy and lower motor neuron disease (see Chapter 11) may be lame, sometimes the result of muscle pain or nerve root pain, complicating differentiation between these causes of muscle atrophy. In most but not all horses with neurogenic atrophy, other clinical signs suggestive of neurological disease may be present. Horses with disuse atrophy resulting from chronic lameness usually have generalized atrophy of the ipsilateral forelimb. Muscle loss usually is not pronounced but involves the forearm (extensors are most commonly affected), triceps, and shoulder muscles. Shoulder muscle atrophy involving the infraspinatus and supraspinatus muscles generally is not pronounced, and lateral subluxation of the shoulder joint during weight bearing is not present (see Chapter 41).

Development of disuse atrophy resulting from chronic lameness generally takes weeks to months unless severe lameness exists. In horses with severe or non–weight-bearing lameness, atrophy may develop within 10 to 14 days. In horses with severe forelimb lameness, carpal contraction (flexor deformity of the carpus) may occur simultaneously with muscle atrophy. The most common cause of carpal contraction because of

ipsilateral forelimb lameness is olecranon process fracture or other elbow lameness, but carpal contraction may occur in horses with severe shoulder or even lower limb lameness. Atrophy of the triceps muscles usually is recognized before other muscle atrophy in severely lame horses.

Horses with neurogenic atrophy may have profound atrophy of one or more muscles in the forearm, pectoral, or cervical regions. Atrophy often is much more pronounced than expected based on the degree of lameness, prompting suspicion of neurological disease. Pronounced, unilateral pectoral or triceps atrophy with mild atrophy of the forearm muscles suggests neurological disease. Severe atrophy localized to the infraspinatus or supraspinatus muscles without subluxation of the shoulder joint usually results from injury of the suprascapular nerve caused by external trauma. Atrophy and subluxation of the shoulder joint is associated with injury of the brachial plexus or nerve roots. Other muscles may also show atrophy.

Localized muscle atrophy or fibrosis occurs in horses with previous injury and subsequent scar tissue formation within muscle bellies. This condition is more common in the hindlimb, but occasionally occurs in the forelimb.

Swelling

Swelling, a common sign of inflammation, often causes asymmetry. Swelling within a joint capsule caused by excess joint fluid, *effusion*, is a general reaction of the joint to several traumatic or degenerative processes. Edema, cellulitis (lymphangitis), bleeding, fibrosis, and bony enlargement can cause soft tissue swelling. *Edema* usually signals acute inflammation and pits (a distinct impression is visible) when compressed by digital palpation (pitting edema). Horses develop edema around and often distal to the site of inflammation. In some horses, especially racehorses left unbandaged when accustomed to being bandaged, benign mild to moderate edema of the distal extremities develops. This process is called "stocking-up" and should not be misinterpreted as a pathological process. In these horses the edematous area is not painful and usually does not pit, and the horse is not lame.

Cellulitis describes infection within the tissue planes of the distal extremities (see Chapter 14) and is sometimes called *lymphangitis*. Lymphangitis, by definition, is inflammation of the lymphatic circulation of the limb, but the conditions are similar and the terms are used interchangeably. Swelling is firm, warm, and painful, and lameness is often pronounced. "Stove-pipe" swelling describes this condition ("the horse is all stoved-up"). Horses generally show systemic signs such as fever and elevated white blood cell count. This condition usually results from small puncture wounds that may be difficult to discover or occurs after articular, periarticular, or subcutaneous injections. Infection develops in subcutaneous tissues or deeper in the dense fascial planes and can be difficult to eradicate.

Blunt trauma or fractures may cause bleeding within tissue planes. Severe lameness and swelling accompany fractures of the scapula and humerus, because large vessels are nearby. Bleeding may be severe and cause a decrease in plasma protein and packed blood cell volume values. In horses with fractures located more distal in the limb, swelling is less pronounced but still prominent. The most likely location of injury is the swollen area, but swelling may occur distal to the site of injury because of venous and lymphatic congestion.

Fibrosis or scar tissue formation as the result of previous cellulitis or trauma causes asymmetry of the distal extremities but may not be the source of the current lameness. The veterinarian should avoid overinterpreting areas of scar tissue formation unless evidence of recrudescent inflammation exists. Horses may have scars caused by previous application of counterirritants or from healed wounds, leaving large, painless, and thus benign blemishes.

Bony swelling is a common cause of asymmetry. Proliferative change results in periosteal or periarticular new bone formation and accompanies a myriad of problems in the distal extremities. Bony changes may be active, causing the current lameness problem, or old and inactive, causing few or no clinical signs. For example, old inactive bony swelling of the shin or osselets (bony and fibrous swelling of the fetlock joint) may be prominent in ex-racehorses but have little to no relevance to current lameness.

Foot Size

Ideally both front feet should be identical in size and shape, or nearly so, and any asymmetry should be noted. Horses with chronic lameness may have disparity in foot size, usually with the smaller foot being ipsilateral to lameness. The small foot often is contracted and more upright (Fig. 5-1). Chronic reduction in weight bearing results in foot size disparity in some, but not all, horses. Mild disparity in foot size is a normal finding in some horses. Mild clubfoot conformation, acquired from previous flexor deformity, may be present incidentally in adult horses. Previous lameness may have caused contraction of the foot but has since resolved, resulting in disparity in foot

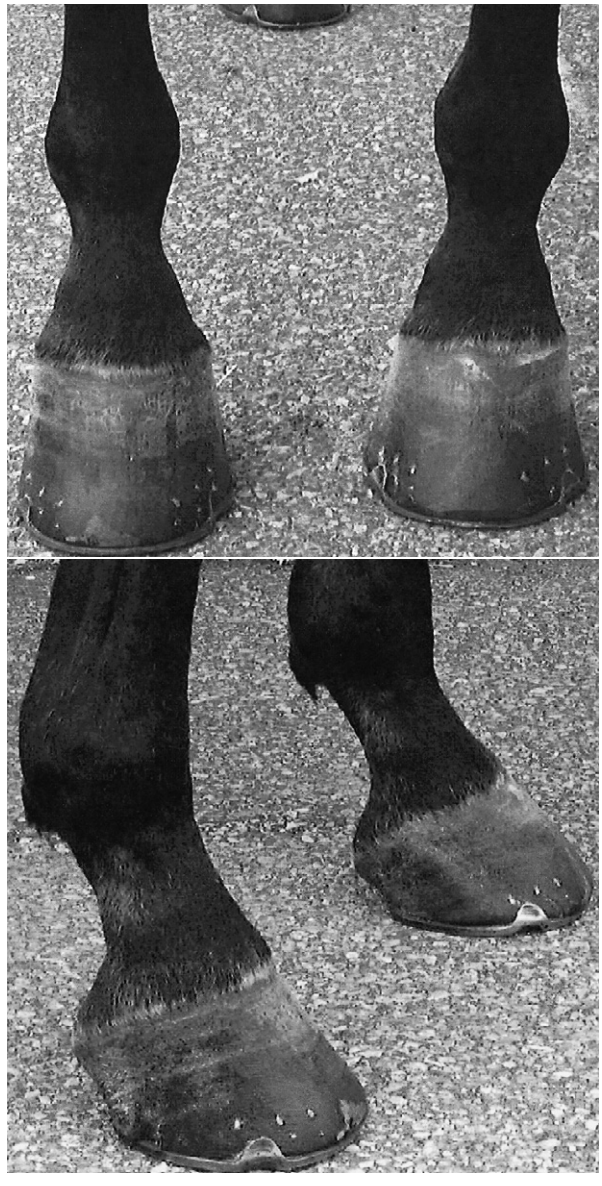

Fig. 5-1 A horse with disparity in front foot size caused by chronic lameness. The right forelimb foot is smaller compared with the normal left forelimb foot when viewed from the front **(A)** and more upright when viewed from the side **(B).**

size and shape but no residual lameness. Clubfoot conformation appears to be better tolerated in Thoroughbred (TB) than in Standardbred (STB) racehorses.

Fetlock Height

Fetlock position should be assessed in the standing horse and during movement. In a standing horse, fetlock height should be symmetrical, assuming the horse is loading the limbs equally. Horses with severe lameness commonly "point" or hold the limb in front of the opposite forelimb, thus taking weight off the limb. This standing posture obviously causes disparity in fetlock height but should be carefully interpreted. Loss of support of a fetlock in the standing horse causes the affected fetlock to drop and occurs most commonly with acute, traumatic disruption of the suspensory apparatus in racehorses but also appears with chronic, active desmitis (Fig. 5-2). Severe superficial digital flexor (SDF) tendonitis or lacerations resulting in fiber damage of the deep or superficial digital flexor tendons can cause similar clinical signs.

In horses with mild flexor deformity of the metacarpophalangeal joint, dynamic knuckling (buckling forward, flexion) of the fetlock joint may occur in the standing position (Fig. 5-3). Joint position usually returns to normal during movement. In horses with severe flexor deformity, normal fetlock position is never achieved. Knuckling of the fetlock also may result from desmitis of the accessory ligament of the deep digital flexor tendon.

Scapular Height

Disparity in scapular height is a rare clinical sign in a lame horse. The veterinarian must stand behind and above the horse to observe scapular height. The horse's mane may obscure observation from a distance, requiring closer examination by palpation. Traumatic or neurological conditions affect scapular height, causing either injury or dysfunction of the serratus ventralis muscle, respectively. With both conditions the dorsal aspect of the scapula is *higher* on the affected side. The veterinarian may place pieces of white tape or other suitable markers on both sides of the horse and stand back to compare height or may use two assistants to point to the locations.

HINDLIMB SYMMETRY

Muscle Atrophy

Asymmetry of bone and muscle mass in the hindlimbs and pelvis is a common clinical sign but must be differentiated carefully. The horse should stand squarely on a flat, even surface. The clinician must determine whether asymmetry exists, and if so, if the problem involves muscle, bone, or a combination of the tissues. Muscle atrophy is most common and, if unilateral muscle atrophy exists, easily can be confused with bony asymmetry caused by pelvic fractures or sacroiliac asymmetry.

Disuse and neurogenic muscle atrophy occur in the hindlimb. Horses with chronic hindlimb lameness develop ipsilateral gluteal muscle atrophy, but asymmetry may be subtle. Mild muscle atrophy usually first appears just lateral to the tuber sacrale. The veterinarian should differentiate muscle atrophy from disparity in height of the tubera sacrale (Fig. 5-4). Recognition of muscle atrophy helps determine the lame leg and provides some information about the duration of the problem. Severe muscle atrophy develops in horses with long-standing, severe lameness or in those with neurological disease (Fig. 5-5).

In horses with neurogenic atrophy of the gluteal muscles the degree of muscle loss is inappropriately severe compared

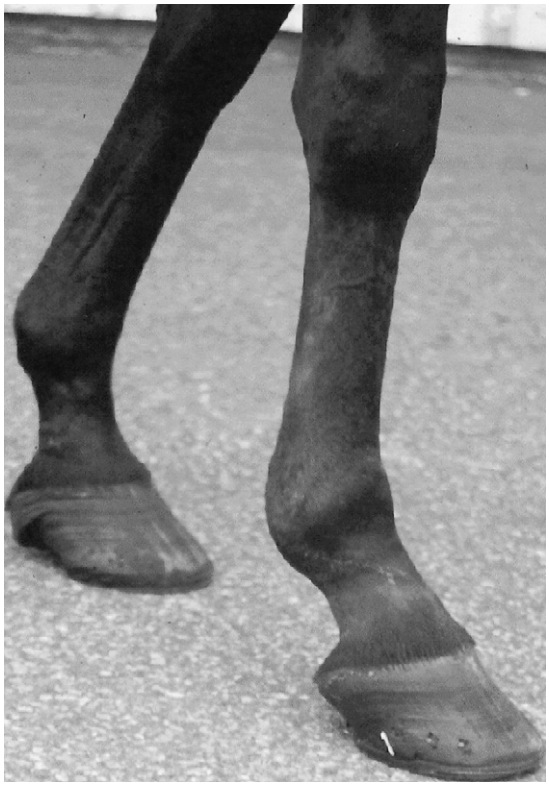

Fig. 5-2 A Standardbred racehorse with severe suspensory desmitis and a "dropped fetlock." The level of the right forelimb fetlock joint is lower than the left forelimb, caused by chronic, severe desmitis. Similar clinical signs and severe lameness appear in horses with acute traumatic disruption of the suspensory apparatus.

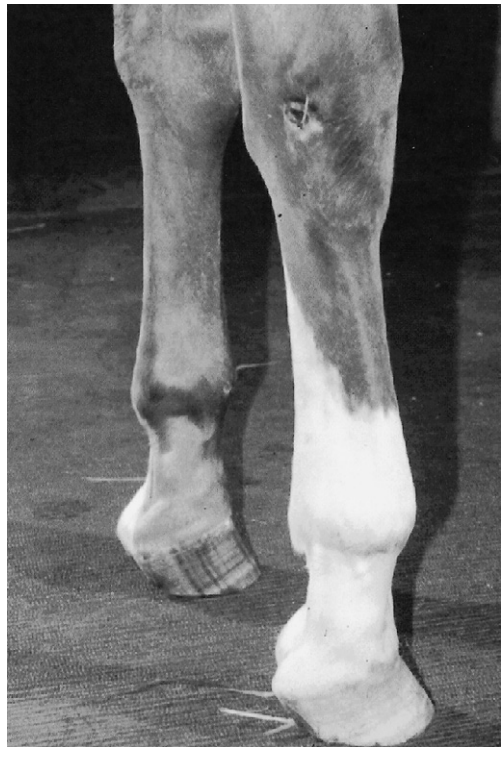

Fig. 5-3 Knuckling forward of the right front fetlock joint occurs in a standing position in this horse with mild flexor deformity of the metacarpophalangeal joint. This dynamic instability abates somewhat when the horse moves, but the left front fetlock also is straight, indicating the presence of bilateral flexor deformity.

Fig. 5-4 Three-year-old Thoroughbred filly with subtle disparity in tuber sacrale height. The left tuber sacrale is slightly lower (*arrow*) than the right, caused by a fracture at the base of the tuber sacrale. This clinical finding can easily be missed or confused with mild muscle atrophy.

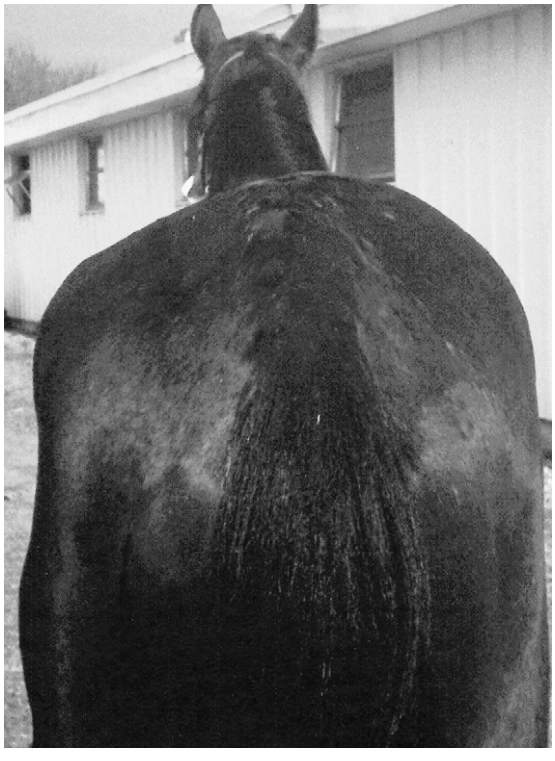

Fig. 5-5 A 4-year-old Standardbred with severe left gluteal atrophy caused by neurological disease. The presumptive clinical diagnosis was equine protozoal myelitis.

with observed lameness. Neurological signs such as weakness and proprioceptive deficits usually appear in horses with neurogenic atrophy, but early in the course of diseases such as equine protozoal myelitis (EPM) the only observable signs may be muscle atrophy and mild lameness.

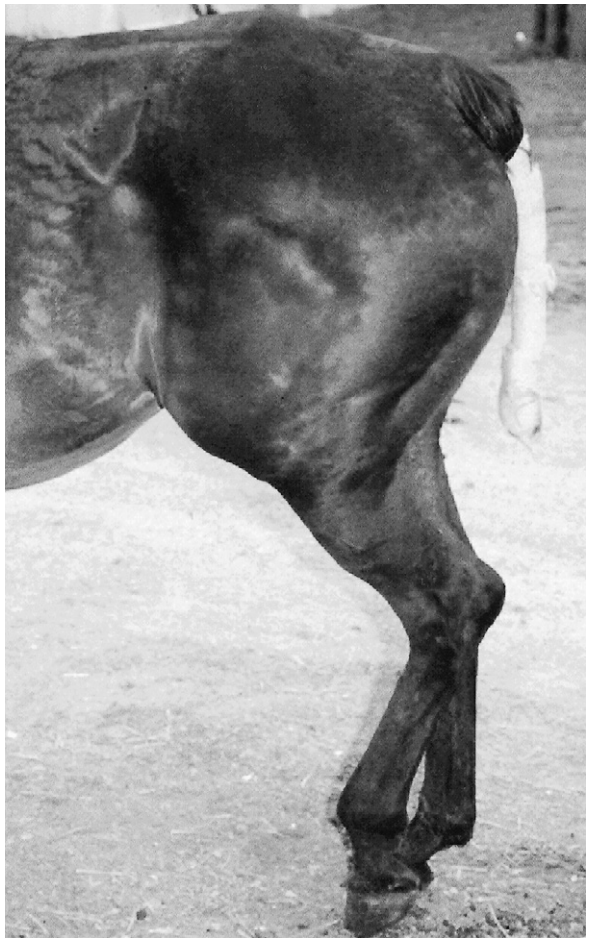

Fig. 5-6 A Thoroughbred broodmare with severe lameness and swelling of the left thigh caused by a comminuted femoral fracture.

Selective atrophy of individual muscles or muscle groups occurs in horses with neurological disease or injuries causing focal muscle loss and scarring. Horses with trauma involving fracture of the tubera ischii may develop focal muscle loss of the semitendinosus or semimembranosus muscles. A depression resulting from localized muscle atrophy replaces initial swelling of the point of the rump. Horses with *fibrotic myopathy,* which in most horses is believed to result from injury and scarring of the semitendinosus muscle, usually have palpable scars or defects of the caudal thigh muscles. Degenerative neuropathy of the nerves supplying the distal aspect of the semitendinosus muscle also may cause fibrotic myopathy[1] (see Chapter 49).

Swelling

Swelling is especially important in horses with acute, severe lameness when the clinician must differentiate between catastrophic injury, such as pelvic or long bone fracture, and more common conditions, such as cellulitis. Horses with pelvic fractures may develop mild swelling in the thigh, but swelling is not prominent in most horses. In horses with fracture of the tuber coxae or ilial wing or shaft, mild swelling may develop distally but usually is not prominent. Inappropriate lameness and lack of swelling should prompt the clinician to perform a rectal examination, checking for internal asymmetry or crepitus. Horses with femoral fractures develop acute, severe swelling of the thigh, accompanied by severe lameness, instability, and often crepitus (Fig. 5-6). Horses may develop severe swelling of the stifle and thigh resulting from trauma and secondary bleeding. Large stifle hematomas resemble the

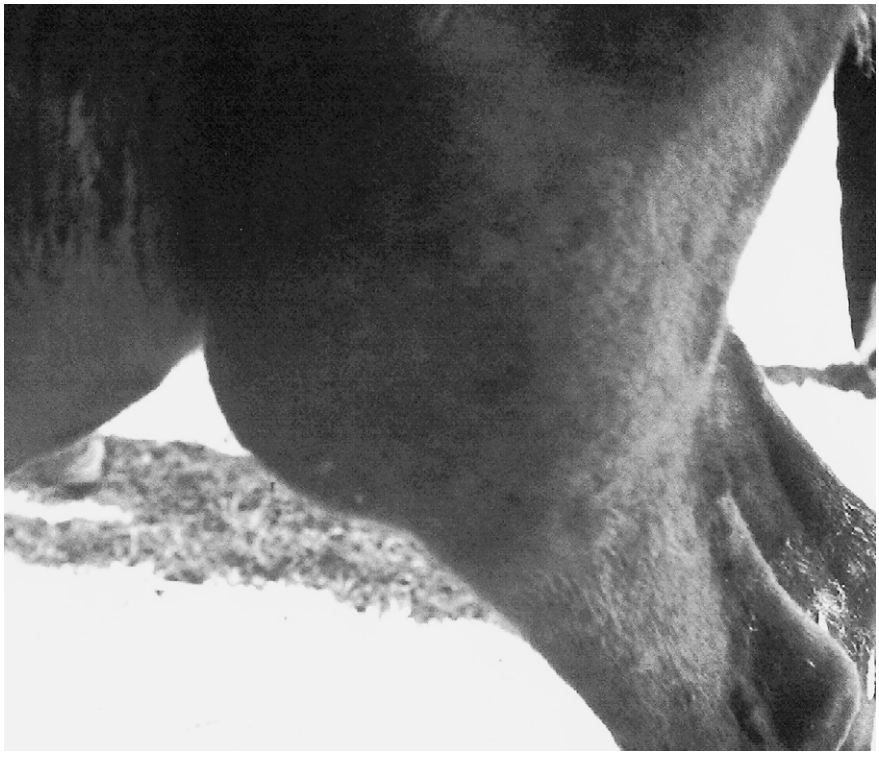

Fig. 5-7 Moderate, fluctuant soft tissue swelling over the stifle caused by subcutaneous bleeding (hematoma). In this situation, swelling is much more pronounced than expected for the observed degree of lameness.

swelling in horses with femoral fractures, and in some horses hematomas can be confused with severe femoropatellar effusion (Fig. 5-7). Excessive bleeding from subcutaneous vessels also may involve the ventral, lateral abdominal region. In horses with stifle hematoma, lameness is not as prominent as expected, and swelling fluctuates, which is useful in differentiating this cause of lameness from femoral fractures. Generalized, diffuse soft tissue swelling appears in horses with cellulitis or lymphangitis in the hindlimb (Fig. 5-8).

Bony Asymmetry

Comparison of the height of the tubera coxae is important to determine the nature and extent of pelvic bony injury (Fig. 5-9). Two assistants, one on each side of the horse, may point to the dorsal aspect of the tubera coxae, or the veterinarian may use temporary markers to compare the height. Determining the height of the tubera sacrale may be difficult and requires careful palpation to differentiate bone, ligament, and muscle asymmetry. Accurate determination may be possible only by ultrasonography. Estimating the midline-to-lateral pelvic width also aids in diagnosing acute or chronic pelvic fractures.

Tubera Coxae

Asymmetry in height of the tubera coxae accompanies many different pelvic fractures. The most common fracture involves the tuber coxae itself, often called *knocked-down hip.* Marked ventral and medial displacement of the fracture fragment occurs because of muscle attachment to the bony prominence. The veterinarian also must palpate the actual shape of the tuber coxae, because ventral displacement occurs with other pelvic injuries. Displacement and rotation occurs in horses with fracture of the wing of the ilium caudal to the tuber coxae without an obvious change in size or shape of the tuber coxae. However, with a partial fracture of the ventral aspect of the tuber coxae, there is a change in its shape, without displacement of the dorsal aspect of the bone.

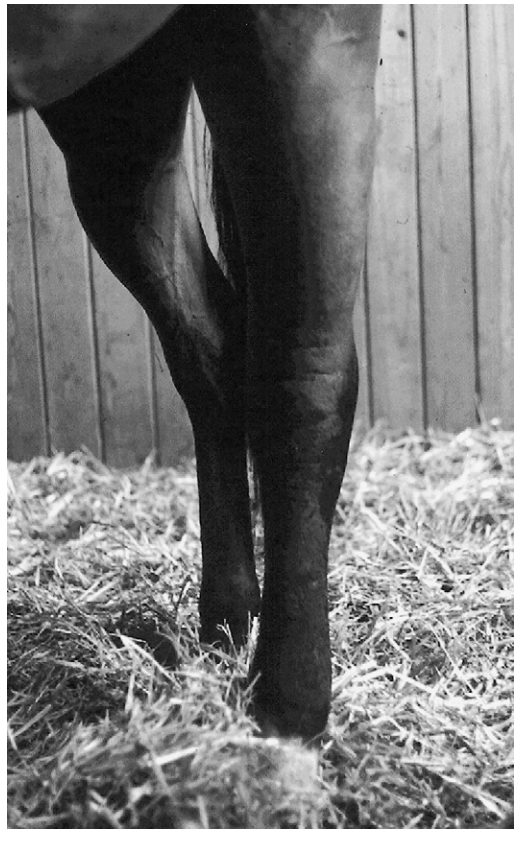

Fig. 5-8 Soft tissue swelling caused by cellulitis. Firm, painful swelling appears in the entire limb.

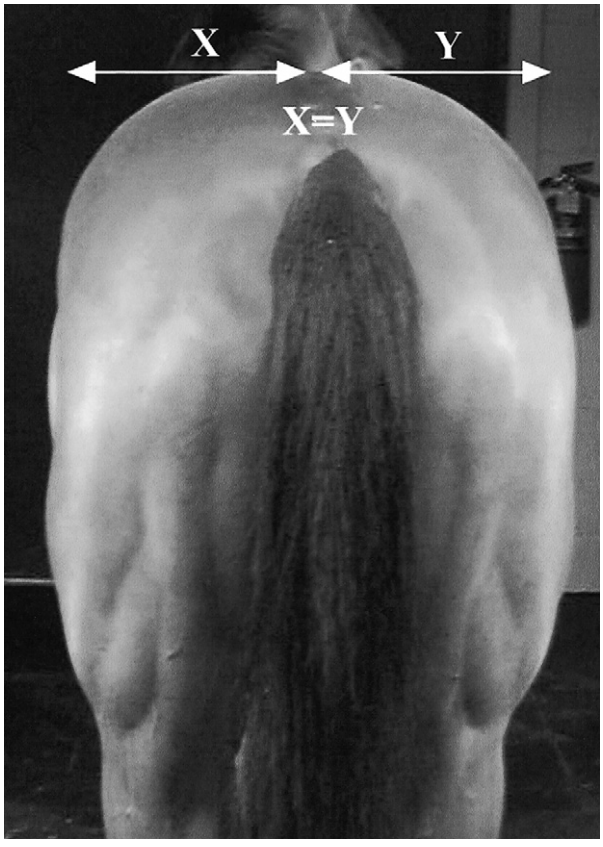

Fig. 5-9 A well-positioned horse to determine tubera coxae height and the midline-to-lateral pelvic width (*X*, *Y*). In a normal horse, *X* = *Y*.

Tubera Sacrale

The term *hunter's bump* describes the prominence of the tubera sacrale. This finding may reflect the horse's conformation, poorly developed surrounding musculature, or a change in position of one or both tubera sacrale. Increase or decrease in size of the overlying dorsal sacroiliac ligament also results in apparent asymmetry. Many clinically normal horses have slight apparent asymmetry of the tubera sacrale. Asymmetry in height of the tubera sacrale occurs in horses with acute or chronic sacroiliac joint disruption (Fig. 5-10). In horses with acute fractures of the base of the tubera sacrale, the affected side is lower[2] (see Chapter 51). Ultrasonography and nuclear scintigraphy may help identify the cause of asymmetry.

Midline-to-Lateral Pelvic Width

A change in the relative width of each hemipelvis is a subtle but important clinical sign of pelvic injury. In most horses with pelvic fractures, the injured side is narrower than the normal side. Over-riding and displacement of fracture fragments results in compression on the injured side.

Swelling over the Greater Trochanter

Mild swelling over the lateral aspect of the coxofemoral joint may be a subtle clinical sign of acetabular or proximal femoral fractures. When standing behind the horse, the veterinarian should carefully observe for enlargement over the affected hip joint. This clinical sign usually is not noticeable initially, but soft tissue enlargement is visible within 2 to 3 weeks after intra-articular fracture. The groove between the greater trochanter and the biceps femoris muscle should be compared carefully; usually a slight bulge or subtle enlargement on the affected side is visible.

Crepitus

Bone-on-bone grating is a valuable clinical sign, particularly in horses with pelvic injury. Crepitus can be heard (with or without a stethoscope) or felt (external or rectal palpation) and most often is caused by movement of bone fragments in horses with displaced fractures (see Chapter 6). In horses with pelvic fractures, crepitus usually is not observed for several days to weeks after injury because muscle tone and fracture hematoma apparently stabilize fracture fragments and delay onset. Crepitus also can be felt or heard in horses with end-stage osteoarthritis.

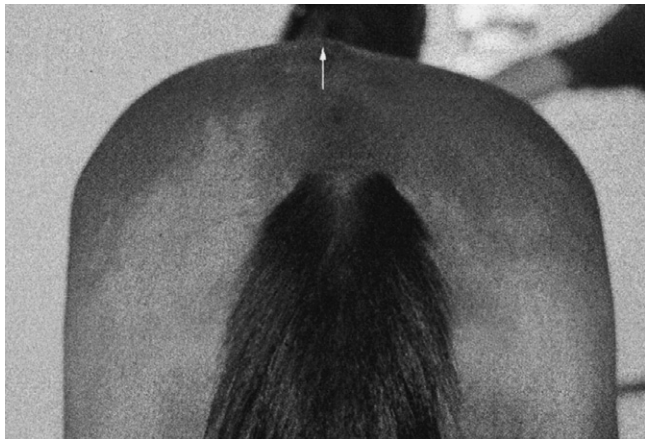

Fig. 5-10 Asymmetry of the tubera sacrale. The left tuber sacrale (*arrow*) is higher than the right. This horse has chronic left sacroiliac subluxation.

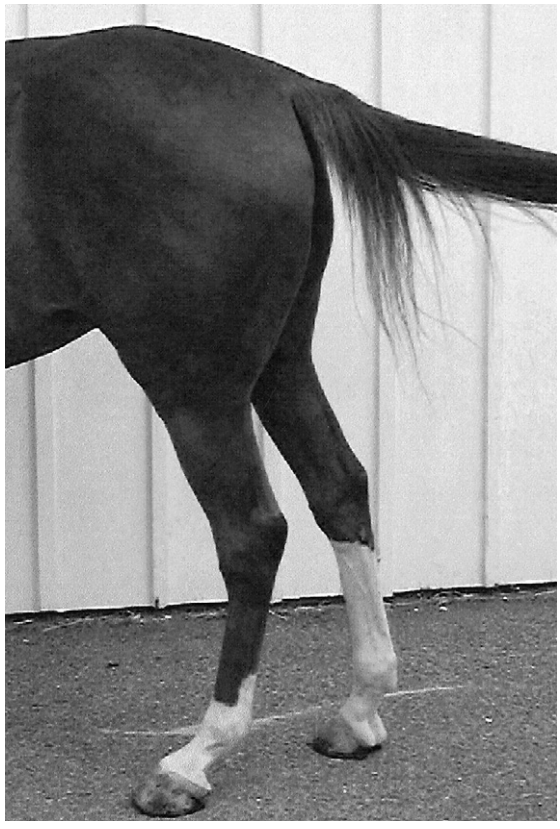

Fig. 5-11 A horse with partial disruption of the left gastrocnemius. An injury to the origin of the lateral head of the gastrocnemius muscle in this horse caused an unusual gait deficit, lameness, and mild distal displacement (drop) of the hock and fetlock.

Calcaneus

The points of the hock should be of equal height when observed from the side or from behind. There is dramatic lowering of the point of the hock with complete disruption of the

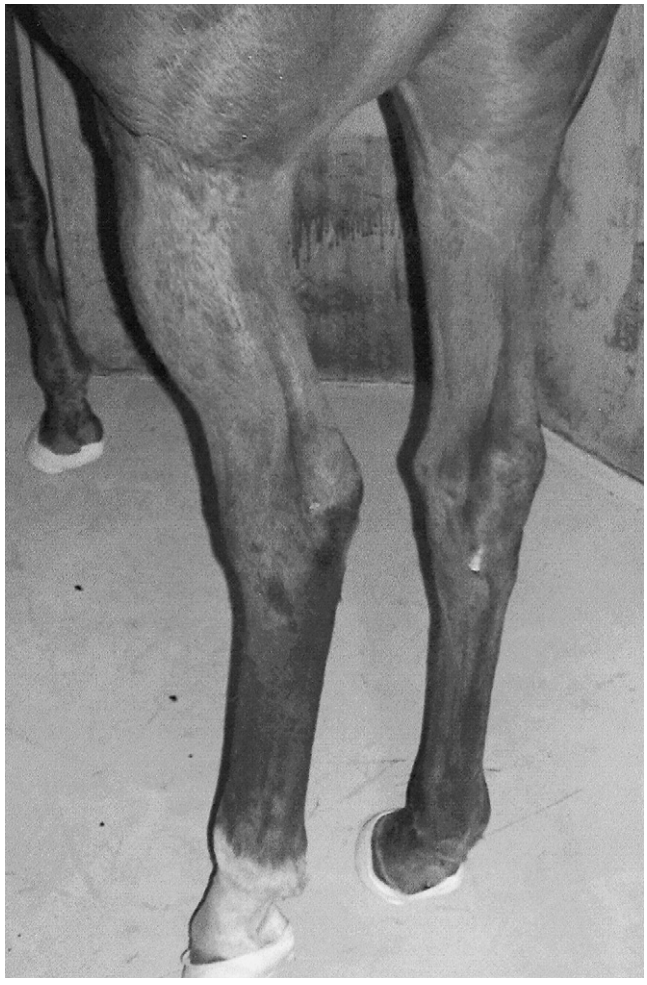

Fig. 5-12 A horse with a comminuted fracture of the left femur. The point of the left hock is higher than the right as a result of over-riding of fracture fragments and muscle contraction, effectively shortening the limb.

common calcaneal tendon or gastrocnemius tendon alone. Partial injury of the gastrocnemius muscle origin, the musculotendonous junction, or the tendon itself causes varying degrees of asymmetry in height of the point of the hock, both in a standing horse and during movement.[3] Other gait abnormalities such as unusual rotation or instability of the limb usually are present (Fig. 5-11).

The point of the hock is elevated in horses with severe pelvic fractures involving the acetabulum, luxation of the coxofemoral joint, and some femoral fractures (Fig. 5-12). Evaluating elevation is difficult because horses with severe lameness usually cannot bear weight, causing a dramatic alteration in limb position. However, in horses with true elevation in the point of the hock, the hock is extended, whereas with most non–weight-bearing conditions, the hock is flexed.

Fetlock Height

Assessment of fetlock position is as important in the hindlimb as in the forelimb. Horses with excessively straight hindlimb conformation (straight hocks) may have more obvious excursion of the fetlock (fetlock drop) while moving or shifting position during standing. Pathological fetlock drop generally accompanies suspensory desmitis (Fig. 5-13) but also occurs with partial disruption of the gastrocnemius and other ligamentous and tendonous injuries.

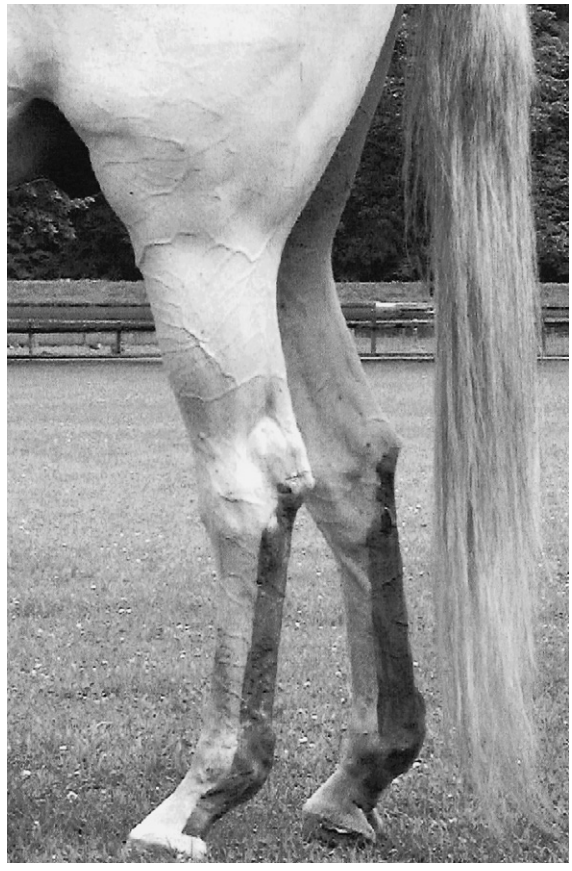

Fig. 5-13 Warmblood gelding with chronic, severe, bilateral hindlimb suspensory desmitis causing noticeable fetlock drop in the left hindlimb.

POSTURE

Body posture provides important clues to the source of lameness, but some abnormalities may be missed unless the horse is observed over long periods. Normal horses tend to rest one hindlimb and may alternate between limbs. Resting a forelimb is uncommon but does occur. Distractions in the environment may make a horse stand normally despite pain. However, abnormal posture because of mechanical or neurological dysfunction usually is evident.

The horse has a well-developed stay apparatus in both the forelimbs and hindlimbs.[4] It is assumed that the main purpose of the stay apparatus is to allow the horse to remain standing for long periods. The stay apparatus in the hindlimbs is better developed than in the forelimbs and includes ligamentous and tendonous structures dictating predictable movement of joints in the limb. If intact, the hindlimb stay apparatus demands reciprocal movement of the hock and stifle and often is called the *reciprocal apparatus*. A change in posture usually means a part of the reciprocal apparatus is broken.

FORELIMB POSTURE

Pointing

Horses that are severely lame often point or hold the affected forelimb ahead of the unaffected forelimb. These horses usually are severely lame at a walk. Horses with severe, bilateral forelimb lameness caused by laminitis may stand camped out in front, attempting to point with both forelimbs simultaneously. However, pointing is not synonymous with the presence

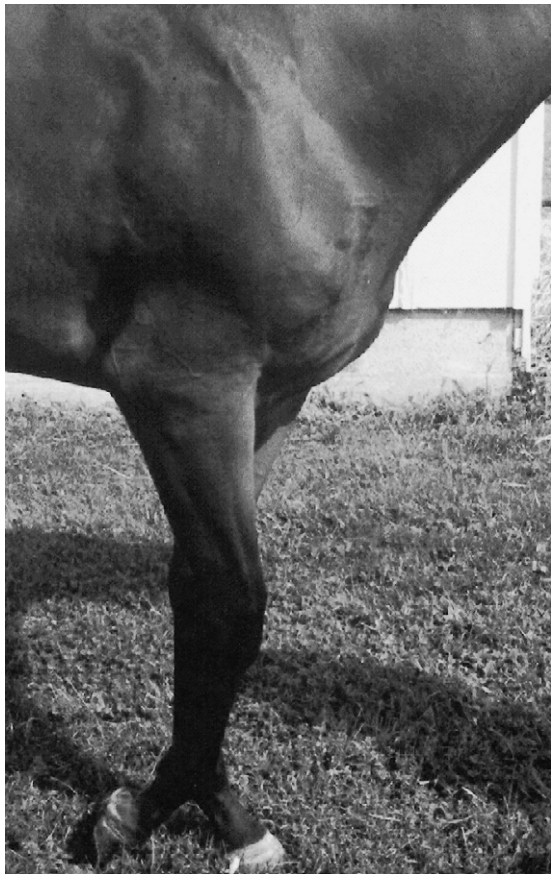

Fig. 5-14 Classic forelimb posture most often called radial nerve paresis or paralysis. The horse cannot extend or fix the elbow, causing the appearance of dropped elbow. The inability to fix the elbow (loss of triceps apparatus) commonly occurs in horses with olecranon fractures.

of pain or lameness or its degree. Some horses prefer to point one forelimb or another and walk and trot normally. In general, pointing is unusual and often signals resting pain or subtle pain relieved by adopting this posture. Some horses stack bedding under the heels to stand in a toe-down position, indicating a degree of unilateral or bilateral foot pain. These horses often are not as lame as expected based on the degree of postural change seen at rest.

Treading

Constant shifting of weight from one forelimb to the other may indicate bilateral forelimb lameness. Laminitis, severe soft tissue injuries such as tendonitis or suspensory desmitis, or severe osteoarthritis may cause treading. Horses with chronic, severe unilateral forelimb lameness often stand with little weight on the affected limb, overload the unaffected limb, and seldom tread. The development of treading in such circumstances is an ominous sign because the horse now is trying to shift weight from the previously unaffected limb, probably because of laminitis.

Buckling Forward at the Knee

Horses with bucked-knee or over-at-the-knee conformation may buckle forward at the knee while standing. In clinically normal older field hunters or other heavily used riding horses with bilateral over-at-the-knee conformation, this may be particularly obvious. The carpus is locked in extension primarily by the action of the extensor muscles. Neurological disease (e.g., EPM) affecting forelimb extensor muscles is a rare cause

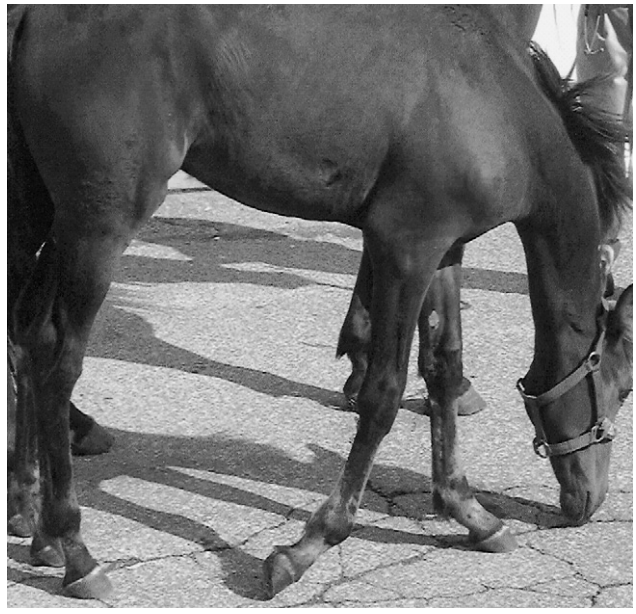

Fig. 5-15 Forelimb posture in a foal with infectious osteomyelitis (scapula) and arthritis (shoulder joint). With severe lameness of the shoulder or bicipital bursa, horses are reluctant to stand or move normally and often hold the limb caudally. This posture may be difficult to differentiate from that seen with loss of the triceps apparatus (see Fig. 5-14).

of buckling forward at the knee, both at rest and during movement. Specific injury of the distal aspect of the radial nerve causes similar clinical signs. In foals, rupture of the common digital extensor tendon or other extensor tendons may cause this posture (see Chapter 78).

Dropped Elbow

A dropped elbow results from failure of the triceps apparatus to maintain elbow extension (Fig. 5-14) and usually results from fracture of the olecranon process. It also may result from injury to the radial nerve or brachial plexus. Similar clinical signs appear in horses with lesions of the nerve roots (neuritis, radiculopathy) or nerve cell bodies in the cervical intumescence, usually the result of lower motor neuron disease. A most unusual cause of this posture appears in horses with "root signature" (see "Neck Pain").

Severe Lameness of the Shoulder Region

Horses with severe shoulder pain may stand with the affected limb more caudal than usual (Fig. 5-15) and often drag the limb even with the slightest movement. This posture is similar to dropped-elbow posture, but in horses with a dropped elbow the limb is held at, or even slightly cranial to, the expected position.

Neck Pain

Horses with neck pain often hold the head and neck lower than expected at a level equal to or slightly lower than the withers (Fig. 5-16). In horses with severe pain muscle tremors or spasms are visible, especially when approaching the horse or causing the horse to move, and the horse stands in a guarded position. The horse may be reluctant to turn or move and may be unable or unwilling to eat food from the ground or an elevated position. An unusual but characteristic sign of neck pain is posturing of a single forelimb, usually on the side of the lesion. The limb is held extended or pointed in front of the other forelimb; rarely the limb is held in slight flexion (see Fig. 55-10, *A*). This sign appears in dogs with cervical pain, most commonly from intervertebral disk disease, and is

Fig. 5-16 Yearling Standardbred with neck pain on the left side showing typical stance and head and neck posture.

termed *root signature*.[5,6] Pain associated with the nerve roots supplying the brachial plexus may be the cause. Some horses with cervical pain also have unilateral forelimb lameness.[7]

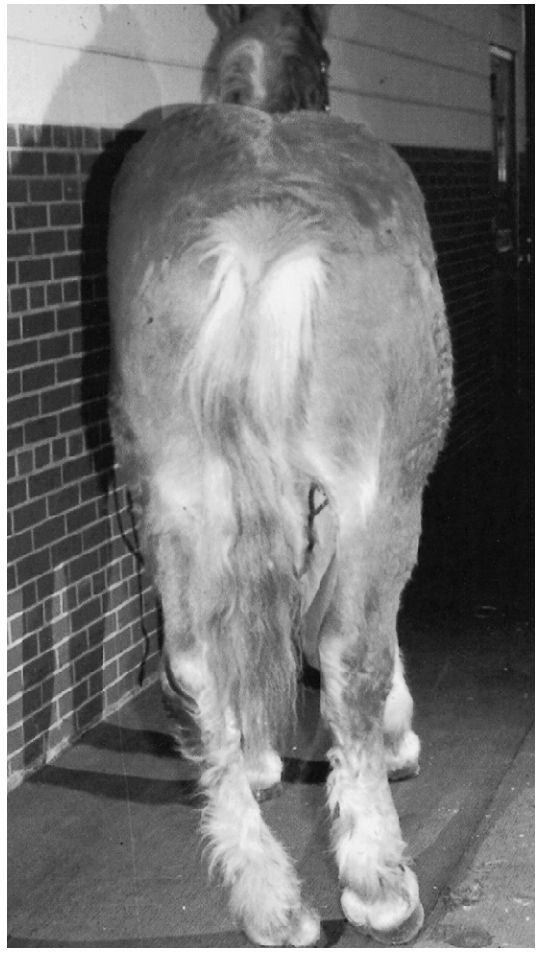

Fig. 5-17 A 2-year-old Belgian gelding with a fracture of the right femoral head and neck exhibits external rotation of the right hindlimb. Varus deformity of the left hindlimb also is visible.

HINDLIMB POSTURE

Resting a Hindlimb

Normally a horse rests one hindlimb or another, but immediately resting a hindlimb after work, or a combination of resting the hindlimb and trembling in the flank or stifle region, may indicate lameness.

Abnormal Tail Position

Horses may carry the tail in an abnormal position during movement, often alerting an observer to possible hindlimb pain. The tail usually is carried away from the lame limb, but this finding is inconsistent. Horses seldom have an abnormal tail posture at rest unless the tail has been traumatized or set, or there is severe hindlimb lameness.

External Rotation of the Hindlimb

Cow-hocked conformation is common, but unilateral external rotation may reflect pelvic injury (Fig. 5-17). The veterinarian should verify this change in posture by moving and reevaluating the horse. Horses with pronounced unilateral external rotation usually have fractures of the acetabulum or proximal femur but may have non-articular ilial shaft or wing fractures.

Hindlimb Varus Posture

Horses with chronic, severe unilateral hindlimb lameness may develop varus conformation of the contralateral limb (see Fig. 5-17). This posture most often appears in foals and may develop 7 to 10 days after onset of lameness.

Treading

Constant shifting of weight between the hindlimbs, or treading, is an unusual clinical sign and usually indicates pronounced bilateral lameness. Horses with bilateral hindlimb laminitis or severe osteoarthritis of any joint may tread. Horses with chronic, severe unilateral hindlimb lameness can endure 4 to 6 weeks or more of weight bearing on the contralateral limb, but treading may be the earliest sign of traumatic laminitis in the supporting limb.

Camped Under

Camped under appears only in horses with bilateral hindlimb laminitis and is rare. Horses often tread and exhibit an unusual hindlimb gait (shortened caudal phase of the stride) when moved.

Soft Tissue Injuries Altering Hindlimb Posture

Upward fixation of the patella causes rigid extension of all hindlimb joints in the standing horse (Fig. 5-18). Patellar dysfunction resulting in fixed extension of the stifle also causes extension of the tarsus and lower limb joints because of the hindlimb reciprocal apparatus. The horse may maintain this posture during movement, or the posture may be intermittent and resolve when the horse is moved. Occasionally a horse with severe hindlimb lameness assumes a similar posture, apparently hanging the limb, but the limb is not locked

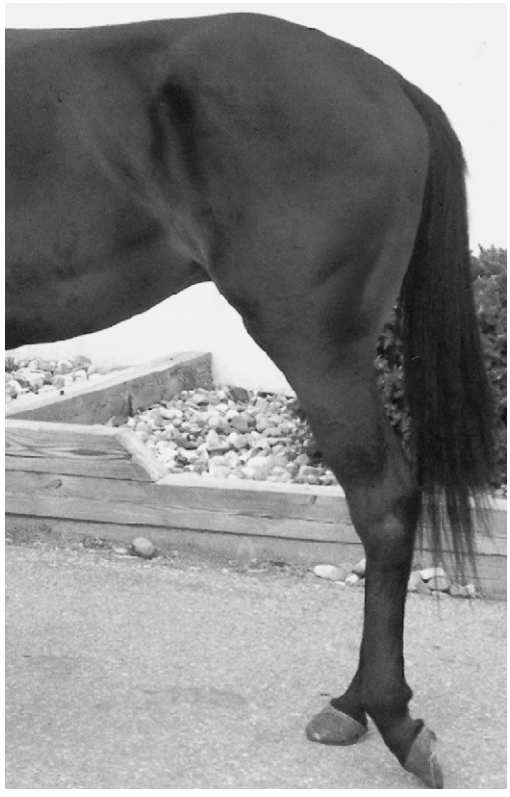

Fig. 5-18 A horse with the classic posture seen with upward fixation of the patella. All joints are held in rigid extension, and the horse is forced to rest or bear weight on the dorsal aspect of the hoof wall.

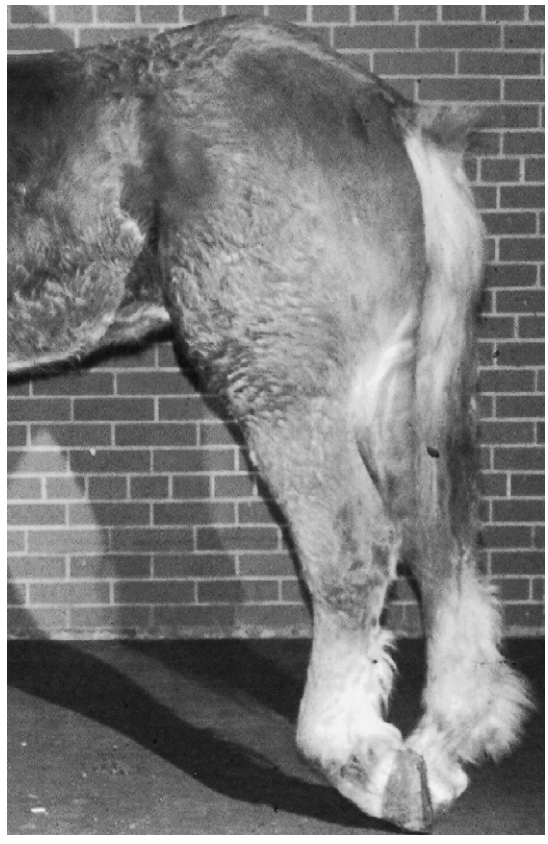

Fig. 5-19 Belgian gelding shown in Fig. 5-17. This horse occasionally rested the left hindlimb in this extended position, similar to that of upward fixation of the patella. Most normal horses, or those with severe lameness, prefer to rest the limb with the sole facing the ground.

in extension (Fig. 5-19). Normal horses simply resting a hindlimb, or those with severe lameness, usually keep the sole facing the ground.

Foals with unilateral or bilateral lateral patellar luxation have an unusual crouched hindlimb posture similar to femoral nerve paresis. The foal may have difficulty in rising if the condition is bilateral and severe or long-standing.

Disruption of fibularis (peroneus) tertius allows the hock to extend abnormally during weight bearing. Foals occasionally stand excessively straight in the hock (extended) in the affected hindlimb. In foals the injury usually causes tearing at the origin of fibularis tertius from the distal femur, but in adults injury may occur in the crus or at the distal aspect of the ligament as it courses over the hock. Swelling and excessive hock extension may occur with the latter injury (Fig. 5-20). The diagnosis is confirmed by manipulation: the hock can be extended while the stifle is flexed.

Rupture of the gastrocnemius tendon, or severe injury at any level of the muscle-tendon unit, causes mild or severe hindlimb postural change. During weight bearing, the hock flexes excessively as the stifle is held in extension, so the point of the hock drops. This injury is called *disruption of the caudal component of the reciprocal apparatus.*[8,9]

Peripheral Nerve Deficits

Sciatic nerve damage is rare. It occurs in foals as a result of injections into the thigh or rump or may occur transiently after injection of local anesthetic solution caudal to the coxofemoral joint. Horses with sciatic nerve damage support weight but appear to be crouched behind, because innervation to the gastrocnemius, flexor, and extensor muscles causes the hock to drop and fetlock to knuckle forward. Careful observation of stifle action and the ability to support weight are useful when attempting to differentiate this deficit from femoral nerve paresis.

Horses with femoral nerve paresis also assume a crouched hindlimb posture but are unable to bear weight, and the stifle drops substantially (Fig. 5-21). Because the reciprocal apparatus is intact, the inability to fix the stifle leads to hock flexion and knuckling (flexion) of the fetlock joint. If the condition is bilateral, the horse is unable to rise for more than a few seconds. Femoral nerve paresis may occur unilaterally or bilaterally after general anesthesia or may result from lower motor neuron disease or injury.

Solitary tibial nerve injury is rare. Fibular (peroneal) nerve injury usually is recognized after general anesthesia and causes characteristic knuckling of the fetlock joint. Tibial nerve injury is differentiated from sciatic injury by lack of involvement of the tibial nerve, and thus normal positioning of the hock, and from femoral nerve injury, because horses are able to support weight and fix the stifle.

Other Unusual Leg Positions

Horses with severe lameness occasionally rest a hindlimb back, forward, or abducted. Often these positions also are maintained during movement. Horses with caudal thigh or pelvic pain prefer to keep the affected hindlimb back, behind the unaffected limb. Horses with shivers may stand with the limb slightly abducted and more caudal than expected, with elevation of the tail head (see Chapter 49).

Horses with pelvic fractures, in particular those involving the acetabulum, may stand with the limb slightly forward

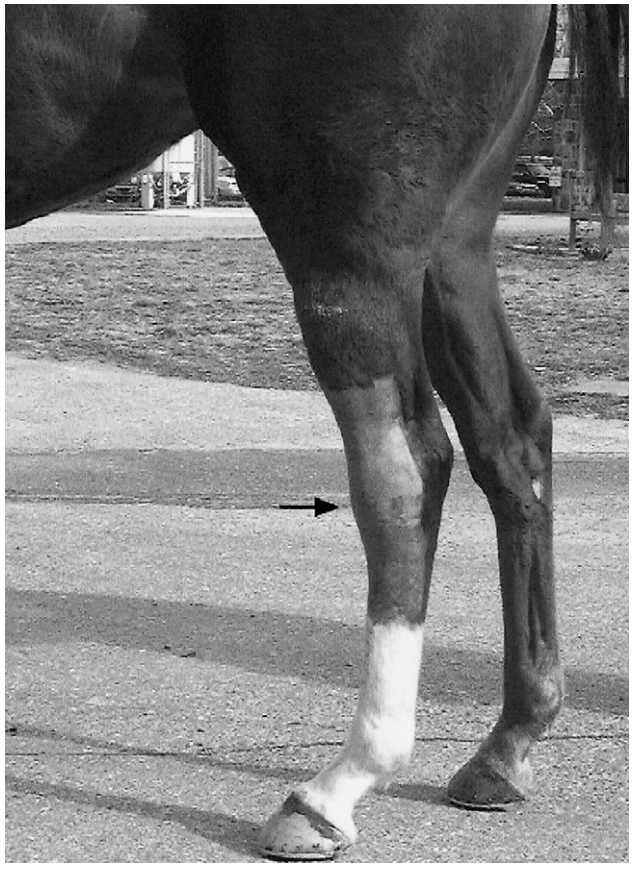

Fig. 5-20 This Thoroughbred racehorse with fibularis tertius injury has swelling over the dorsal aspect of the hock (*arrow*) and straight-in-the-hock conformation.

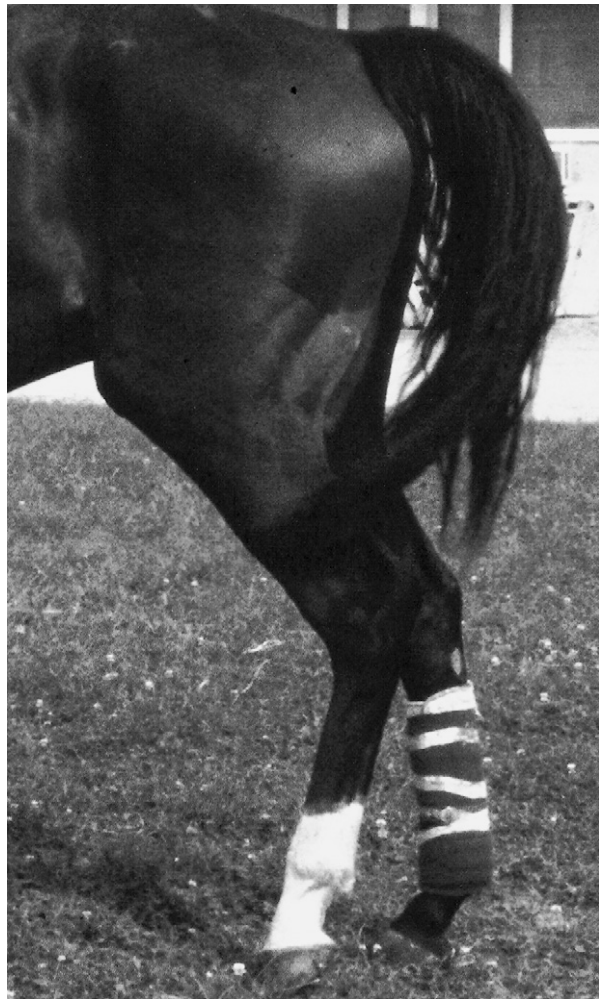

Fig. 5-21 Thoroughbred with transient, post-anesthesia, unilateral (left hindlimb) femoral nerve paresis. The crouched posture of the left hindlimb includes flexion (knuckling) of the fetlock joint.

and often are reluctant to place the limb behind the unaffected limb, thus reducing the caudal phase of the stride at the walk. Horses with pain in the medial thigh and groin area stand with the limb abducted and may travel in this manner. Adductor muscle damage, medial thigh abscessation, and scirrhous cord or other inguinal problems also cause this posture.

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CHAPTER • 6

Palpation

Mike W. Ross

Palpation is an important part of a lameness examination. In some sport horses, it becomes more important because, for example, suspensory desmitis often is not associated with overt lameness but may compromise performance. The veterinarian must develop a system to evaluate comprehensively all parts of the musculoskeletal system. I palpate in order each forelimb, the neck, back, pelvic regions, and then the hindlimbs. Each limb should be assessed when bearing weight and then again with the limb elevated from the ground. Deep palpation is used to describe direct, digital palpation, with the limb in an elevated position.

If time permits, palpation should be completed *before* the horse is moved, because if the lame limb is identified first, the other limbs may be overlooked and compensatory problems may be missed. For example, in a Thoroughbred (TB) racehorse, superficial digital flexor (SDF) tendonitis is a common compensatory problem caused by contralateral limb lameness resulting in overload. If a lame horse with left forelimb (LF) lameness is first examined while the horse is moving, and subsequent palpation of the limb reveals signs of possible fetlock osteoarthritis, mild swelling of the right forelimb (RF) SDF tendon may be missed. Comprehensive palpation may allow the clinician to make predictions about lameness, to "read" the horse. Palpation before exercise also facilitates identification of localized heat or swelling, because limb temperature increases with exercise and swelling often decreases.

THE ART OF PALPATION

The veterinarian should palpate and manipulate every possible anatomical structure, using the fingers and hands to push, prod, and feel. Interpretation of an abnormal response requires appreciation of the normal response. There are nerves beneath or adjacent to many structures, and direct pressure may elicit an apparently positive response. Such false-positive responses often occur during palpation of the origin of the suspensory ligament (SL) or the proximal sesamoid bones (PSBs). Care should be taken to apply pressure only in the desired location. During palpation of the PSBs, distal aspect of the SL, and flexor tendons, it is easy to apply pressure over the dorsal aspect of the third metacarpal bone (McIII), and a painful response may actually reflect sore shins.

The clinician should look for signs of inflammation: heat, pain, redness, swelling, and loss of function. One side of the horse should be compared with the other, but it should be remembered that both sides may be abnormal. Heat is one of the earliest clinical signs to develop with articular or nonarticular problems and may be the only sign. Subchondral remodeling and sclerosis of the third carpal bone often cause lameness in young racehorses, but effusion of the middle carpal joint and a positive response to flexion are found inconsistently. Usually prominent heat is detectable on the dorsal aspect of the carpus. It is important to recognize normality. A normal horse may have disparity in foot temperature. Horses often have two or three cold feet, but the other feet feel warm. A few hours later, feet that previously were cool may feel warm. Foot temperature often reflects variations in ambient temperature, and care must be taken not to over-interpret this normal finding. In general, palpation is done with the palm side of the hand, although the back of the hand may be more sensitive to detection of warmth.

The veterinarian should assess the quality or strength of the digital pulse. In a normal horse, reliable detection of a digital pulse may be difficult, especially in cold weather or in horses with a thick hair coat. Increased or elevated digital pulse refers to the detection of increased strength or the bounding nature of the digital pulse. Inflammatory conditions in the foot or pastern region, such as abscesses, laminitis, hoof avulsions, or cracks, are the most common causes of increased digital pulse. Complete absence of hindlimb digital pulse may occur with aortoiliac thromboembolism or other vascular problems, but care should be taken when interpreting weak or near absent hindlimb digital pulses, because hindlimb digital pulses can be difficult to feel in normal horses.

Redness is difficult to perceive in the horse because of skin pigmentation, but in the foot, solar bruising or redness at the coronary band can be observed, especially in horses with nonpigmented feet. *Swelling* is often detected by observation, but subtle enlargement of structures such as the SL, or presence of effusion may be determined only by careful palpation.

Loss of function of tissues and regions can be assessed during palpation. Manipulation, flexion, and extension of the joints or soft tissues provide a better idea of function or loss of function. Static flexion and extension determines the range of motion of a joint and the horse's response to the procedure. Chronic osteoarthritis of the fetlock or carpal joints often results in reduced range of flexion. However, many horses in work but without lameness resent hard flexion of the lower limbs. Good correlation between a reduction in fetlock flexion range, lameness and severity of osteoarthritis was found in TB racehorses.[1] A reduction in fetlock flexibility in young Warmbloods may be a predictor of future lameness.[2] The response to rotation of joints also should be assessed.

Crepitus, the grating or crackling sound made by bone rubbing on bone, is an unusual and ominous clinical sign usually determined by palpation, although in horses with prominent osteoarthritis or fractures, a grating sound may be heard. A stethoscope may be useful for detection of subtle crepitus.

Other factors may confound the results of palpation. Clipped areas usually are warmer than an adjacent area with normal hair length. Blistering or freeze firing can cause localized pain for weeks after application, even if lameness has resolved. Any type of skin lesion, such as those found in horses with scratches or boot rubs, can cause extreme soreness to palpation but no signs of lameness. Some individual horses are more sensitive to palpation than others, and interpretation of apparent pain can be frustrating.

PALPATION OF THE FORELIMB

Foot

The importance of the foot cannot be overemphasized, and it is for this reason that palpation of the forelimb begins here. The feet are included in evaluation of conformation, symme-

Fig. 6-1 Instruments needed to examine the hoof, remove a shoe without tearing the hoof wall, and prepare the hoof for radiographic examination. Shown are apron, rasp, shoe pullers, nail pullers, clinch tool, hoof knife, hammer and hoof pick, and wire brush.

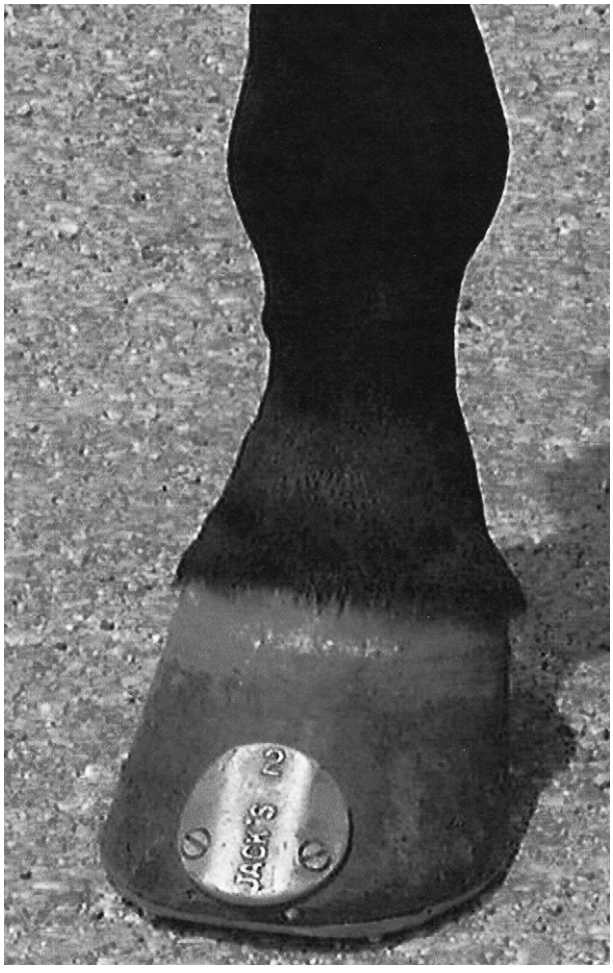

Fig. 6-2 The coronary band is uneven compared with the ground in this trotter's unbalanced hoof. The medial wall (*right*) appears to be shorter than the lateral wall.

try, and posture. Detailed static examination (examination at rest) of the foot must always be supplemented with, and correlated to, dynamic observations of foot flight and foot striking patterns. Some horses continually attempt to pick up the limb as the clinician tries to evaluate it with the horse in the

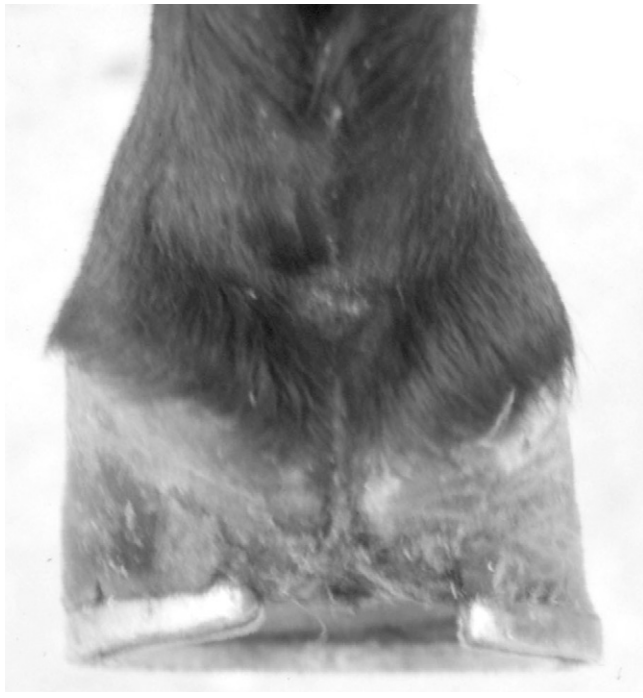

Fig. 6-3 Elevated foot viewed from the palmar aspects shows that the hairline at the medial bulb of heel (on the right) is displaced proximally compared with the lateral heel bulb. The medial wall is longer. Note also the prominent cleft between the heel bulbs. These features are typical of sheared heels.

standing position; it may be necessary to stroke the contralateral limb to divert attention. A hoof pick, wire brush, hoof knife, shoe-removing equipment, and hoof testers are required (Fig. 6-1). The sole and frog and wall of the foot should be cleaned thoroughly. Removal of the shoe at this stage in the examination usually is indicated only if a sub-solar abscess is suspected. The veterinarian should take care to preserve the hoof wall, and if cracked, protect it with tape.

Foot and hoof balance are assessed by evaluating toe and heel length, hoof capsule conformation, condition and integrity, type of shoe and shoe position relative to the hoof capsule, hoof and pastern angle (axis), medial to lateral hoof balance, coronary band conformation, and distal interphalangeal (coffin) joint capsule distention and response to hoof testers. The coronary band should normally be parallel to the ground surface. Deviation from parallel often indicates mediolateral foot imbalance (Fig. 6-2). Medial and lateral wall lengths should be assessed while the horse is standing and again with the limb off the ground, viewing the foot from palmar to dorsal along the solar aspect. The limb is lifted and held in neutral position so the solar surface is perpendicular to the ground. Sheared and underrun heels are commonly associated with lameness (Fig. 6-3). Deformation of the hoof capsule is not necessarily a cause of lameness. Many horses with proximal displacement of the medial heel bulb have level foot strikes and otherwise balanced feet. Toe and heel length should be assessed, and the hoof-pastern axis should be determined. The angle of the hoof and pastern should be equal to allow equal loading of all portions of the foot. Forelimb hoof-pastern angles normally range from 48° to 55°, but the absolute angle should not be overemphasized. A straight pastern-foot axis is more important. A long-toe, underrun heel foot conformation causes a broken foot axis and predisposes to palmar heel pain (Fig. 6-4).

The conformation, condition, and integrity of the hoof capsule should be assessed. It is easy to miss hoof wall defects on the medial aspect. Small quarter or heel cracks and defects at the

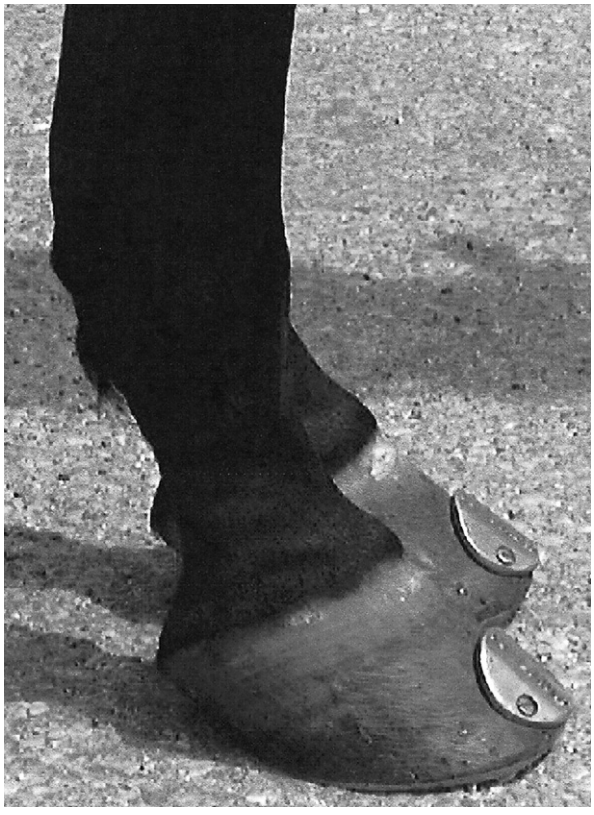

Fig. 6-4 This trotter has long toe, under-run heel hoof conformation, and broken hoof-pastern axis.

coronary band should not be overlooked. The clinician should evaluate the solar surface, bars, and frog. Thrush, although a reflection of poor management, rarely causes lameness.

The shoe type, shoe wear patterns, and the shoe size relative to the foot need to be assessed. The clinician should note the presence of pads or additions to the shoe, such as toe grabs, borium, and heel caulks. There is an association between toe grabs and suspensory apparatus failure in TB racehorses.[3] Low heel angle also has been associated with injury.[4] Shoe wear is important, because it reflects how the horse has been moving over the last several weeks. The clinician should note the breakover point and whether one branch of the shoe is worn more than the other. Shoe size should be assessed relative to foot size and the fit of the shoe. A shoe that is too small or set too close to the frog may predispose to lameness.

Careful palpation of the coronary band in the standing and non–weight-bearing position is critical in detecting foot soreness (Fig. 6-5). In horses with sore feet, heat and pain often are detected on the sore side of the foot, and a prominent digital pulse usually is present. Effusion of the distal interphalangeal joint capsule accompanies many abnormalities of the foot, from early synovitis to chronic osteoarthritis of the distal interphalangeal joint, and those with non-specific foot soreness. The clinician places one finger lateral to, and another medial to, the common digital extensor tendon and gently pushes in on the joint capsule, first laterally and then medially. *Ballottement* is a useful technique to detect effusion in many synovial structures: with effusion, pushing in on the capsule on one side of the tendon causes elevation of the capsule on the other side.

The clinician should palpate the cartilages of the foot, either with the horse standing or with the limb elevated. *Sidebone,* mineralization of the cartilages of the foot, rarely causes lameness. The cartilages of the foot normally are pliable and readily compressed axially. Fracture at the attachment of the cartilage of the foot to the distal phalanx is an occasional cause of lameness, and compression of the heel with hoof testers may elicit pain in some horses.

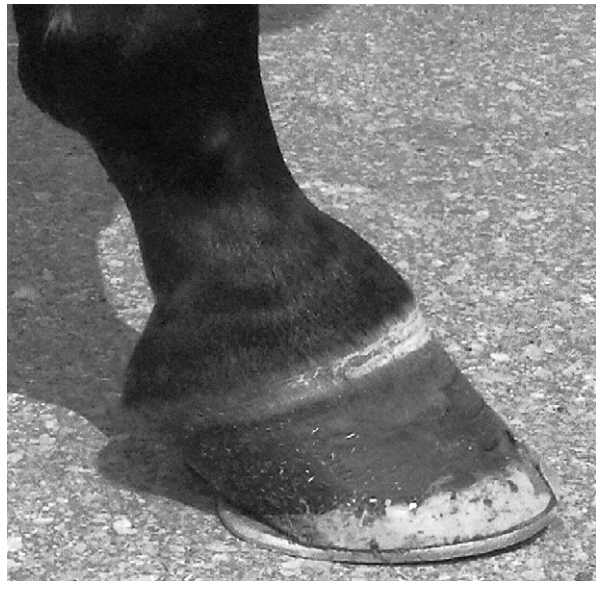

Fig. 6-5 Palpation of the coronary band should include assessing the dorsal joint pouch of the distal interphalangeal joint. In this horse, distal interphalangeal effusion and fibrosis appear as a bulge just proximal to the coronary band, dorsally.

Hoof Tester Examination

"…I feel naked going into a stall without my hoof testers!"[5] Hoof testers are essential for evaluation of the foot and are a basic requirement for all lameness examinations. Many types of hoof testers are available (Fig. 6-6), but I favor one that is adjustable and can be applied with one hand. A proper evaluation of the foot with hoof testers cannot be done with a pad in place, although useful information can be acquired. The instrument can be applied with or without a shoe in place. The amount of force to apply varies from horse to horse and by region of the hoof, and both false-positive and false-negative responses occur. More force is required when the instrument is used across the heels than when used from sole to quarter. The foot should be held between the clinician's legs in a relaxed manner. The clinician must be able to feel the horse react to subtle pressure, and if the leg is held too tightly or the horse is not calm during the examination, it is difficult to feel a response. The veterinarian should be careful not to place the outside jaw of the instrument too close to the coronary band, because this may cause a false-positive result. Sole sensitivity is assessed by applying the instrument to three to five sites from heel to toe, on both the medial and lateral aspects of the foot, starting from the angle of the sole (seat of the corn) and proceeding dorsally (Fig. 6-7). The responses should be compared. If the sole is readily compressible, pain from bruising, a sub-solar abscess, laminitis, fracture of the distal phalanx, and other injuries may be elicited, but in horses with hard horn the response may be negative. To evaluate sensitivity of the frog and underlying deeper structures, the hoof testers should be applied from the lateral aspect of the frog to the medial wall, and from the medial aspect of the frog to the lateral wall, each in the palmar, mid-portion, and dorsal aspects of the frog (Fig. 6-8). Pain over the middle third of the frog has been attributed to navicular disease or navicular syndrome, but the specificity of this association is questionable and there are many false-negative responses. Horses with generalized foot soreness or any other cause of palmar

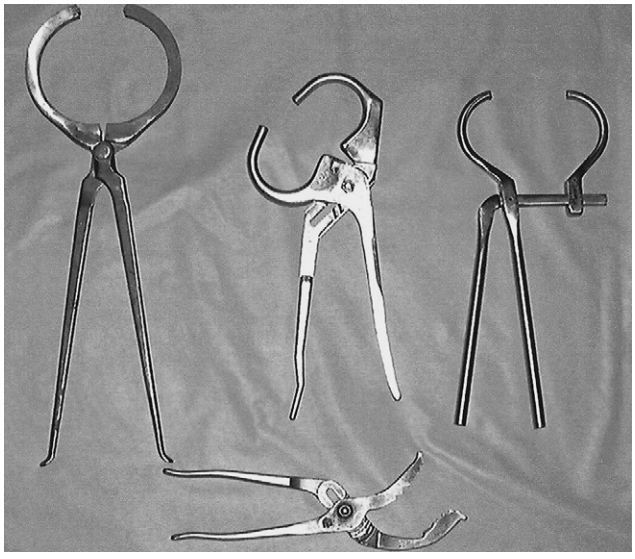

Fig. 6-6 A variety of hoof testers are available for lameness examinations. I prefer hoof testers that are easily adjusted and used in one hand (*two pairs on the right*). Large hoof testers (*left*) can be applied only with two hands, and small hoof testers (*bottom*) are inappropriate for medium to large hooves.

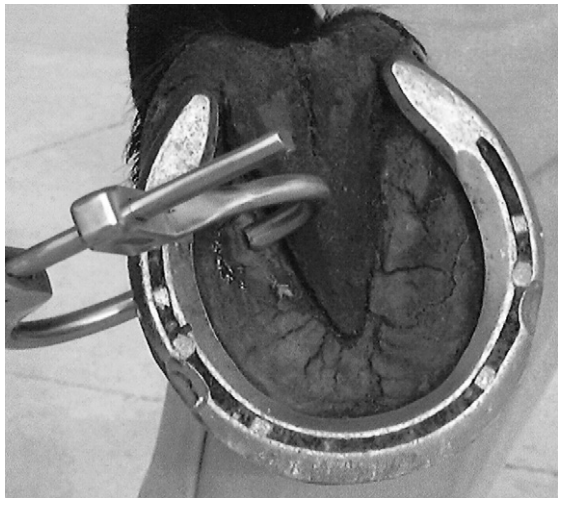

Fig. 6-7 Hoof testers should be applied from the sole to the wall, from the heel to toe, and to both sides of the hoof.

heel pain may respond positively or not at all. Only 19 of 42 horses with navicular region pain responded positively to hoof tester examination in the middle third of the frog, with 50% specificity, 50% positive predictive value, and 48% accuracy.[6] Horses with palmar heel pain caused by other conditions were as likely to respond to the test, a finding that obviously prompts questioning of the value of hoof tester examination.[6] It is difficult if not impossible to create adequate pressure to cause pain in large breed horses or if the horn is hard. Application of a poultice or soaking the foot may be necessary to soften a hard foot, and re-examination after several days may be rewarding. Hoof tester application to the small feet of foals or ponies may elicit a false-positive response, and hoof tester size or amount of compression may require adjustment.

Application of hoof testers across the heels may cause pain in horses with palmar heel pain but is not specific (Fig. 6-9). Application of the hoof tester to the area of the sole adjacent to each nail, nail hole, or defect in the sole or white line, is useful

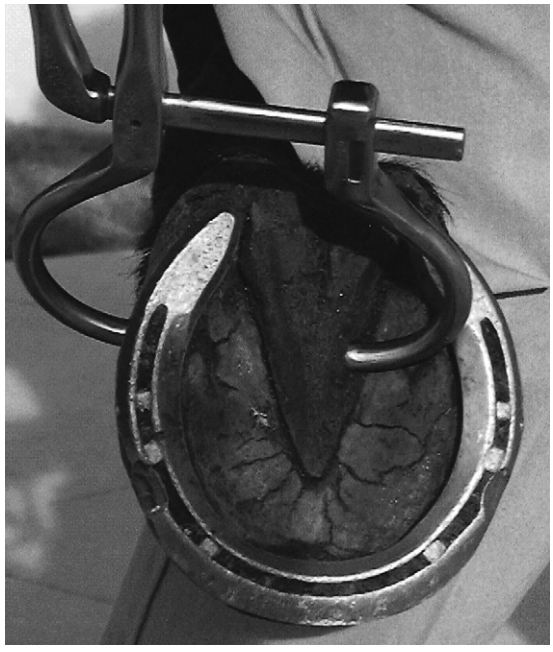

Fig. 6-8 Hoof testers applied from the middle of the frog to the contralateral hoof wall put pressure on the navicular region. Horses with many abnormal conditions of the hoof may manifest a positive response.

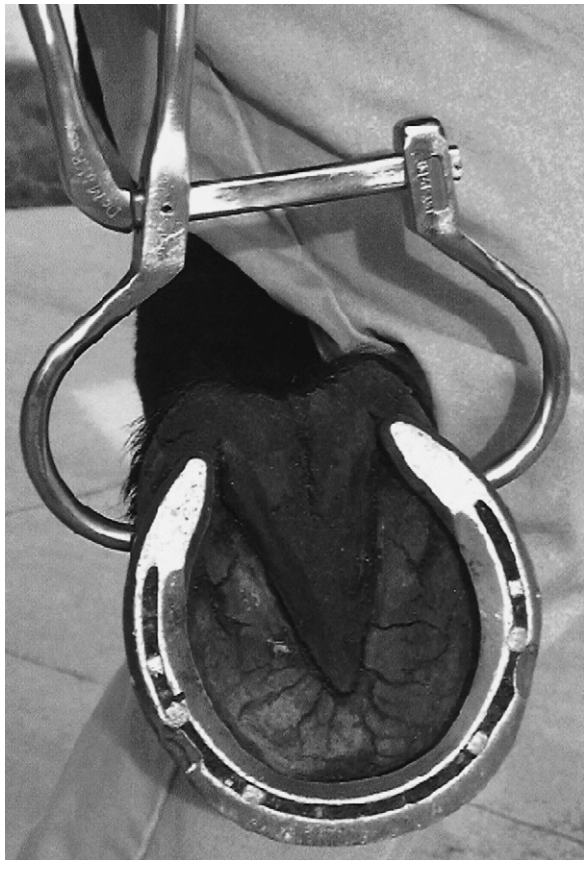

Fig. 6-9 Adjustable hoof testers are easily placed across the heels. I prefer to apply hoof testers in this manner to assess horses for palmar heel pain during static examination and as a provocative test for lameness.

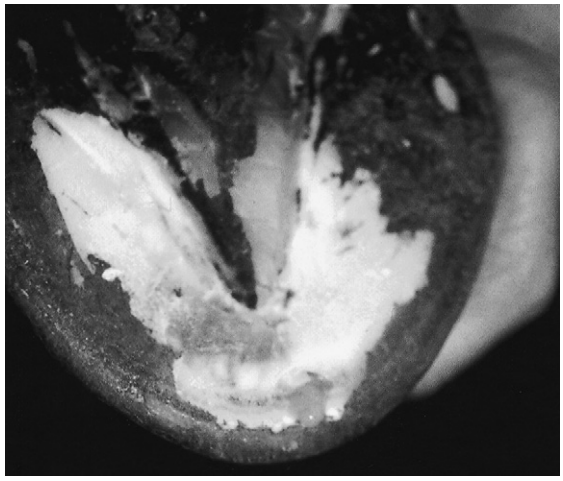

Fig. 6-10 Acute, severe lameness causing increase in digital pulse and profound hoof tester sensitivity in the toe region resulted from this hoof abscess. Exudate drains from the pared region at the toe. (Courtesy Greg Staller, Pottersville, New Jersey.)

to detect a sub-solar abscess or a close nail (Fig. 6-10). Areas of pain can be gently explored with a hoof knife, but unless clearly indicated, the veterinarian should refrain from digging too deep. The hoof tester can then be used as a hammer to percuss each nail in the shoe and the frog and toe regions.

After completing the hoof tester examination, the clinician should re-assess the digital pulses. In horses with foot pain the digital pulse may now be bounding. Horses that have recently been shod or trimmed, or have raced or performed recently, especially on hard surfaces, may have mild elevations in digital pulse and may show hoof tester sensitivity normally. Pain causing lameness may not be in the foot.

Pastern

The proximal interphalangeal (pastern) joint capsule is assessed by ballottement, although severe effusion must be present to perceive fluid distention. Bony swelling associated with this joint, proximal or high ringbone, is a classic cause of lameness yet an unusual clinical finding. Osteoarthritis of the proximal interphalangeal joint is a common diagnosis, but one made by a combination of clinical findings, diagnostic analgesia, radiography, and sometimes scintigraphy. The distal extent of the digital flexor tendon sheath (DFTS), deep digital flexor tendon (DDFT), and distal sesamoidean ligaments are palpated. Deep pain associated with the origin and insertion of these structures is assessed by palpation with the limb in flexion (Fig. 6-11). The oblique sesamoidean ligaments are difficult to differentiate from the branches of the SDFT, but injury of the SDFT is more common. Distal sesamoidean desmitis or chronic suspensory desmitis may result in subluxation of the proximal interphalangeal joint (Fig. 6-12). The proximal interphalangeal joint is manipulated in a medial to lateral direction to assess pain and collateral ligament integrity and is flexed independently of the fetlock joint. The proximal, dorsal aspect of the proximal phalanx is palpated (Fig. 6-13). Horses with short, mid-sagittal fractures of the proximal phalanx may show pain. Occasionally enthesophyte formation at the common digital extensor tendon attachment, seen most commonly in older, ex-racehorses with chronic osteoarthritis of the fetlock joint, results in prominent bony and soft tissue swelling and pain on palpation.

Fetlock

The clinician palpates the joint capsule of the metacarpophalangeal (fetlock) joint with the limb bearing weight, bearing in mind that pain associated with the joint can be present

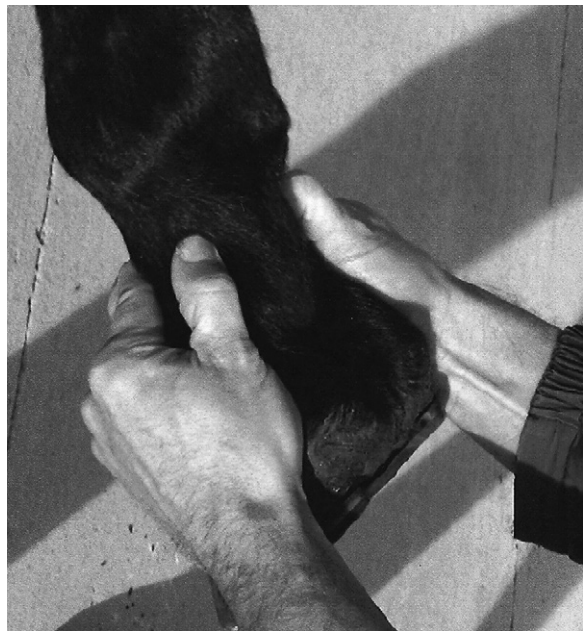

Fig. 6-11 Palpation of oblique distal sesamoidean ligaments.

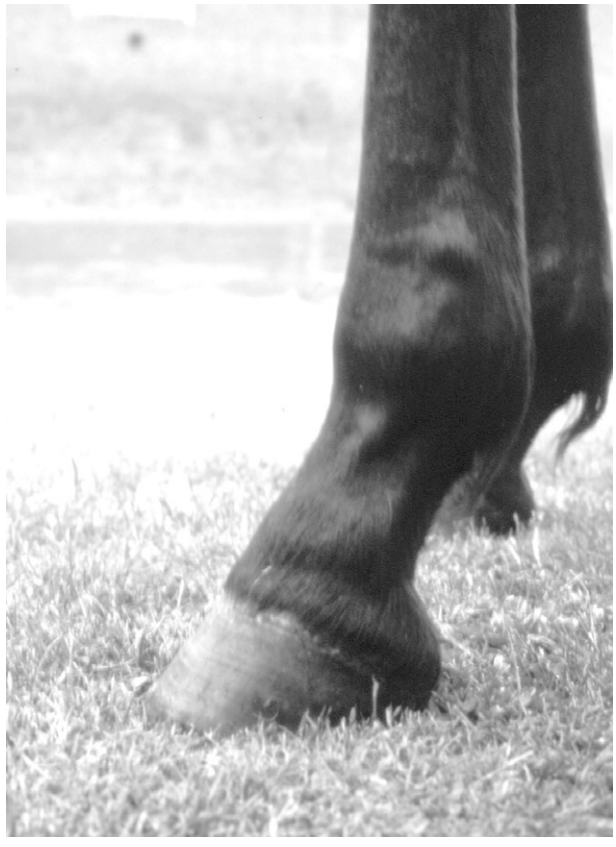

Fig. 6-12 Subluxation and osteoarthritis of the left front proximal interphalangeal joint resulted from primary suspensory desmitis.

without localizing clinical signs. The dorsal aspect is palpated using ballottement on either side of the common digital extensor tendon. The clinician should determine whether localized heat is present. *Osselets* is a North American term used to describe early osteoarthritis of the metacarpophalangeal joint in young racehorses, with firm bony and soft tissue swelling on

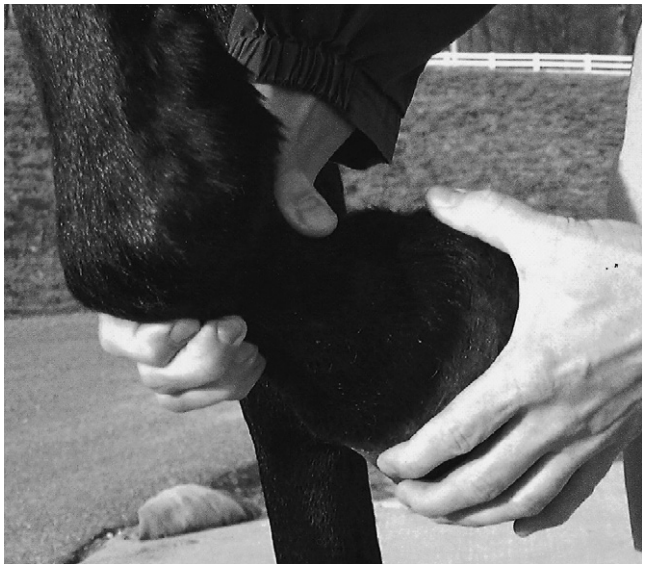

Fig. 6-13 Proliferative changes at the common digital extensor attachment or pain from mid-sagittal fracture of the proximal phalanx should be palpated along the dorsal, proximal aspect of the proximal phalanx.

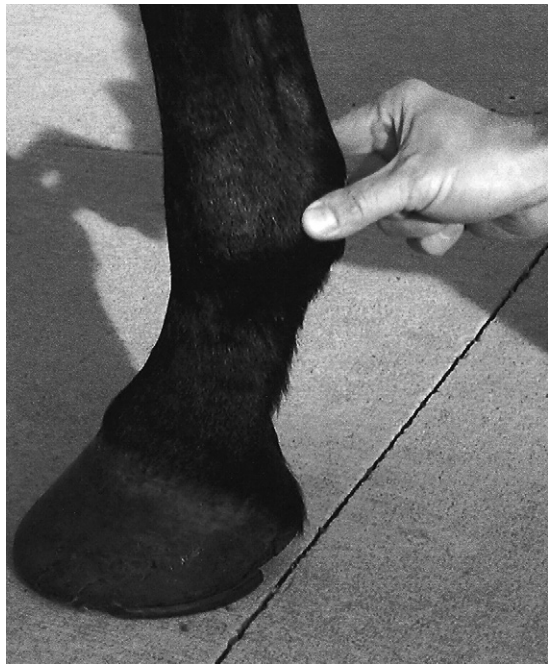

Fig. 6-14 Digital pulse quality can be assessed easily at the level of the proximal sesamoid bones.

the dorsal, medial aspect of the proximal phalanx, and the distal aspect of McIII, caused by traumatic capsulitis and early enthesophyte formation. Occasionally in horses with prominent effusion of the metacarpophalangeal joint, a soft tissue swelling can be palpated in the proximal, dorsal aspect of the joint from excessive proliferation of the dorsal synovial pads, called *proliferative* or *villonodular synovitis*. The palmar pouch of the metacarpophalangeal joint is palpated dorsal to the SL branches, both medially and laterally. Mild effusion may be present without associated lameness, especially in older performance horses. The PSBs are palpated and assessed for mild swelling and heat, clinical signs of sesamoiditis, or SL avulsion injury. The digital pulse is re-assessed by placing fingers both medially and laterally, abaxial to both PSBs (Fig. 6-14).

The DFTS extends from the distal metacarpal region to the distal palmar pastern. Usually no palpable fluid is found. Effusion of the DFTS (tenosynovitis) causes swelling in the palmar fetlock region that must be differentiated from effusion of the metacarpophalangeal joint. Tenosynovitis causes swelling *palmar* to the branches of the SL medially and laterally. Fluid can be compressed from medial to lateral. With severe effusion, distention is found in the palmar pastern, but there may be distention proximal to the palmar annular ligament without obvious distention distally. *Wind puffs* or *wind galls* describe incidental fluid distention of the DFTS, commonly seen in older performance horses unassociated with lameness. Tenosynovitis can cause lameness, but additional diagnostic techniques are required to confirm the diagnosis.

The limb is elevated to assess range of joint motion and the horse's response to flexion. Normally the fetlock can be flexed to 90° (the angle between the proximal phalanx and McIII) or slightly more. A reduction in fetlock flexion range is indicative of chronic fibrosis but is not necessarily a cause for concern. A pronounced response to static flexion is noteworthy, but many horses resent static flexion but do not show a positive response to dynamic flexion (lower limb or fetlock flexion tests; see Chapter 8). Horses with clinically relevant tenosynovitis usually strongly resent fetlock flexion. With the limb in flexion, the clinician palpates the PSBs and the branches of the SL, avoiding compression of the palmar digital nerves.

Metacarpal Region

The clinician should assess the dorsal aspect of McIII for heat and swelling. This is a common area for traumatic injury (barked shins) or stress-related bone injury (bucked shin syndrome). Many ex-racehorses have incidental, prominent, chronic, and non-painful swelling of McIII caused by extensive modeling and remodeling of the dorsal cortex while in race training. Racehorses currently in training may have heat and pain on deep palpation (performed with the limb elevated), but prominent swelling may be lacking. Any combination of palpation findings is possible in horses with stress-related bone injury of McIII. It is difficult to apply deep pressure to the dorsal aspect of McIII without concomitant pressure to the palmar soft tissue structures or PSBs, so the responses should be assessed carefully.

The entire length (abaxial surface) of the second and fourth metacarpal bones (McII/IV) should be palpated with the horse in the standing position to detect exostoses, callus, or fractures. Swelling of the SL branches or body may make this difficult. Palpation of McII/IV should be repeated with the limb elevated, because the axial aspect of these bones is impossible to assess in the weight-bearing position. Splint exostoses are common, particularly in young horses. Therefore the presence of even large bony swellings is not unusual. Exostoses detected axially, possibly impinging on the SL (or so-called blind splints) should be carefully noted. Pain from even small exostoses of McII/IV usually is more accurately assessed immediately after training or racing, because pain and lameness resulting from these swellings can be subtle and transient. The clinician should carefully palpate the SL branches. Differentiation of branch or SL body injuries is important: the latter injuries usually are more serious and have a worse prognosis.

The medial and lateral palmar digital vein, artery, and nerve, in dorsal to palmar orientation, respectively, are located between the SL and DDFT. The accessory ligament (distal or inferior check ligament) of the DDFT (ALDDFT) normally is difficult to palpate and even when enlarged cannot easily be differentiated from the DDFT, but injuries of the ALDDFT are more common. All soft tissue structures should be palpated

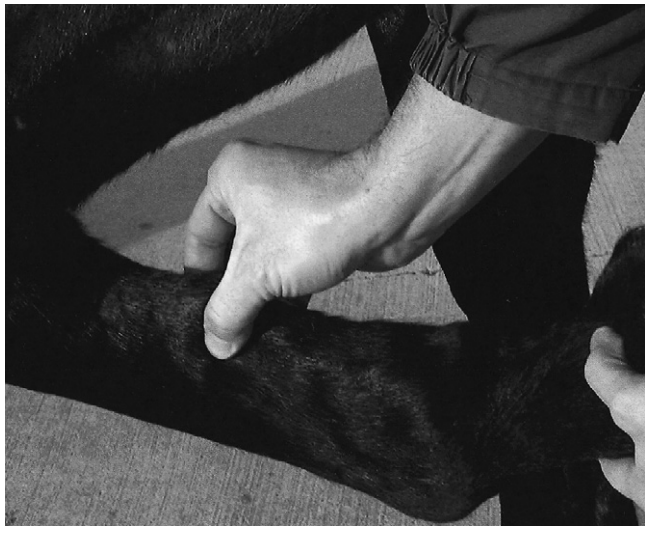

Fig. 6-15 The soft tissue structures in the palmar metacarpal region should be carefully palpated with the horse in standing and flexed (shown) positions for heat, pain on compression, and swelling. Most ridden horses have mild pain, but in racehorses a painful response is an early sign of tendonitis or desmitis.

carefully, using digital compression, with the limb elevated (Fig. 6-15). Acute or chronic swelling should be assessed, as should the horse's response to deep palpation. Obvious swelling and pain indicate the presence of tendonitis or desmitis. In some horses with acute severe tendonitis or desmitis the structure feels "mushy" or soft in the area of fiber damage. This finding, especially in horses with fetlock drop, indicates near rupture of the structure. There are many false-positive and even false-negative responses to palpation of the flexor tendons and SL. In most ridden performance horses a mild painful response (false-positive) to deep palpation of the SL is normal. However, in racehorses, false-positive responses are less common, and a painful response to deep palpation may indicate the presence of early desmitis or tendonitis. In many horses with foot lameness, secondary, mild suspensory desmitis is common. There is a painful response to palpation of the body and origin of the SL. It may be difficult to decide whether this is a true or false-positive response, a determination that often is made in hindsight after the lameness examination is finished. False-positive and false-negative responses to palpation of the proximal palmar metacarpal region also occur. This is a common site of lameness and should be examined carefully. Palpation must be done with the limb in flexion, and the presence of swelling and pain must be carefully interpreted (Fig. 6-16). Horses with acute injuries, such as proximal suspensory desmitis (PSD), avulsion or longitudinal fracture of McIII, and stress reaction of McIII at the origin of the SL, may have swelling and pain. Deep palpation may create pressure on the palmar metacarpal nerves resulting in a false-positive pain response, and many horses with PSD have no localizing signs.

The proximal dorsal aspect of McIII should be palpated in a flexed position (Fig. 6-17). Occasionally, dorsomedial articular fracture of McIII results in a subtle painful swelling. Swelling (effusion) of the carpal sheath may be detected in the proximal medial metacarpal region, but large veins (medial palmar, accessory cephalic, and cephalic veins), may interfere with accurate palpation. With mild tenosynovitis, effusion may be difficult to discern.

Carpus

Detection of warmth on the dorsal aspect of the carpus is a reliable indicator of underlying inflammation. Obviously, one side should be compared with the other, but bilateral conditions exist commonly. Previous application of counterirritants interferes with the reliable detection of warmth. Carpal joint lameness without obvious signs of synovitis is common, but if present, effusion is easily palpated using ballottement. With the horse in the standing position, a finger is placed dorsolaterally between the extensor carpi radialis and common digital

Fig. 6-16 Palpation of the proximopalmar metacarpal region is essential in diagnosing proximal suspensory desmitis and other conditions of the suspensory origin and differentiating lameness in the region from carpal lameness. (Courtesy Ross Rich, Cave Creek, Arizona.)

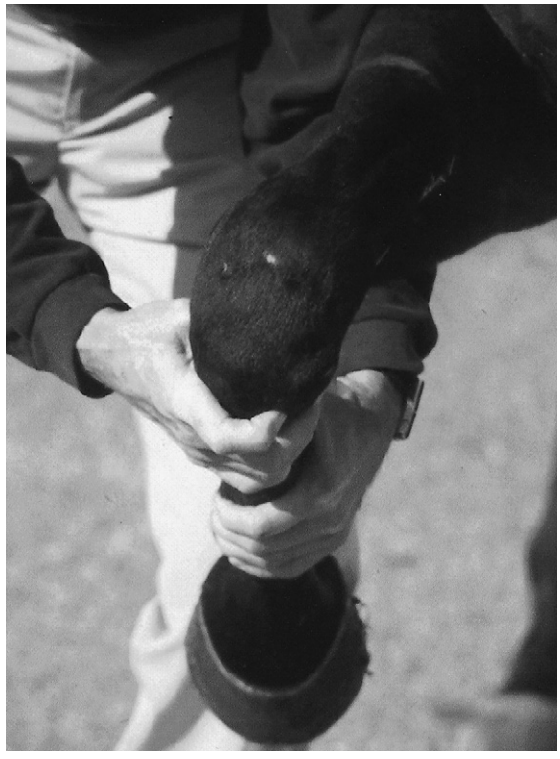

Fig. 6-17 Careful palpation of the proximal, dorsal metacarpal region identifies pain associated with dorsomedial articular fracture or other fractures of the proximal aspect of the third metacarpal bone. (Courtesy Ross Rich, Cave Creek, Arizona.)

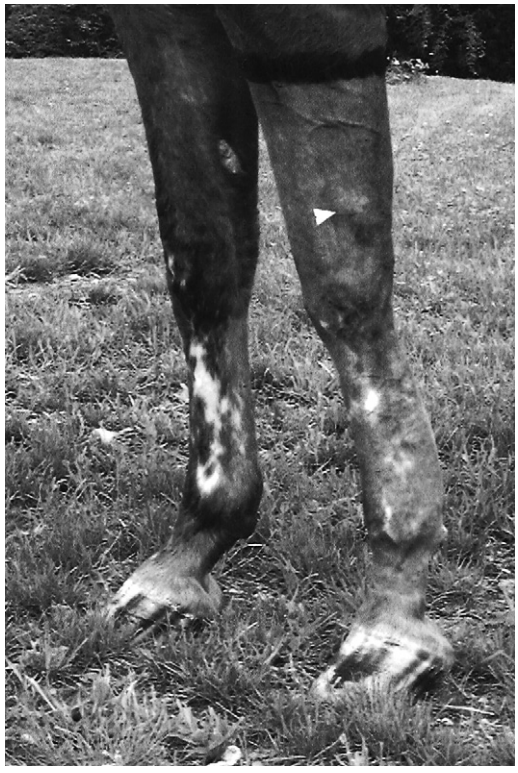

Fig. 6-18 Carpal tenosynovitis must be differentiated from effusion of the antebrachiocarpal joint. This horse with severe superficial digital flexor tendonitis has moderate distention of the carpal sheath (*arrow*) in the caudal, distal aspect of the antebrachium and medially in the proximal metacarpal region (not shown).

extensor tendons, and another finger is placed just medial to the extensor carpi radialis tendon. These openings are used for palpation and arthrocentesis of both the middle carpal and antebrachiocarpal joints. The middle carpal and carpometacarpal joints always communicate, but a small synovial compartment and dense overlying soft tissue structures limit palpation of the carpometacarpal joint. Both the middle carpal and antebrachiocarpal joints have a palmarolateral pouch that may be distended if effusion is severe. If swelling is detected just caudal to the radius, it is necessary to differentiate distention of the palmarolateral pouch of the antebrachiocarpal joint from the carpal sheath (Fig. 6-18). In horses with antebrachiocarpal joint effusion the dorsal outpouchings also should be prominent, whereas in those with carpal sheath effusion, fluid distention is restricted to the palmar aspect and also detected medially, both proximal and distal to the accessory carpal bone.

Tenosynovitis of the extensor carpi radialis, common digital extensor, or lateral digital extensor sheaths results in vertically oriented swellings that traverse the carpal joints, may extend proximal or distal to the carpus, and usually are multi-lobed, being divided by bands of extensor retinaculum located dorsally and laterally.

Normally the carpus can easily be flexed completely, so that the palmar metacarpal region and bulbs of the heel touch the caudal antebrachium. Reduced flexion may be caused by pain with or without chronic fibrosis associated with osteoarthritis. Pain during carpal flexion is a reliable indicator of carpal region lameness but does not indicate the cause. The carpal sheath is compressed and the extensor tendons are stretched during this maneuver, and conditions involving these structures and the accessory carpal bone can cause pain during

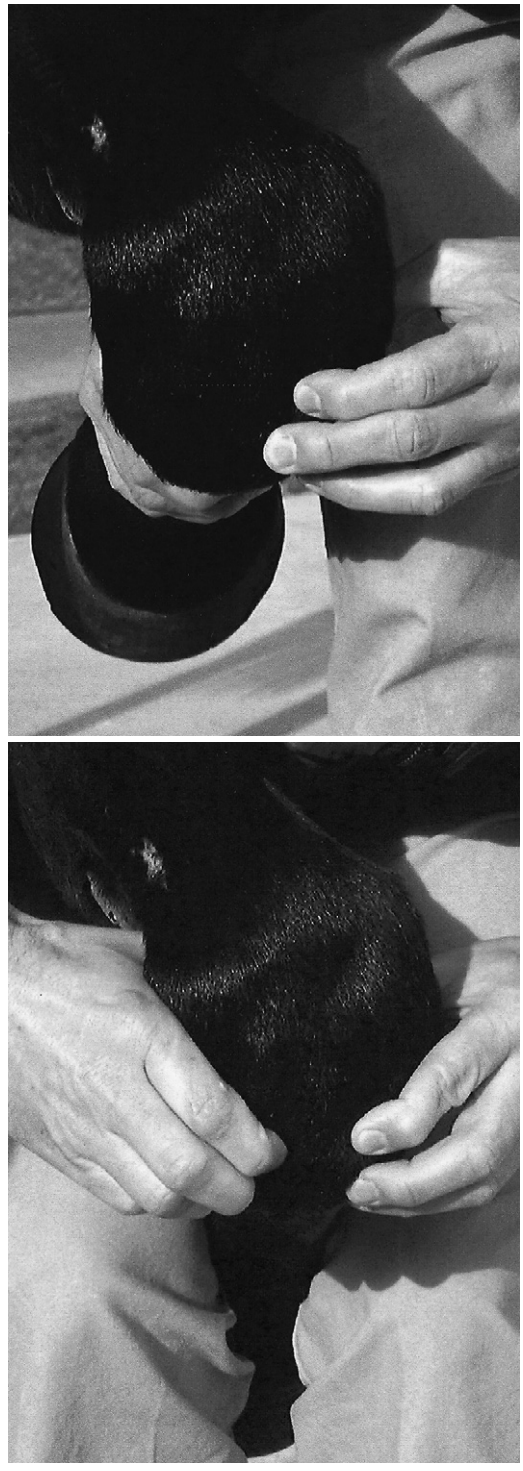

Fig. 6-19 Careful palpation of the dorsal aspect of each carpal bone can be done with one hand (**A**) or by placing the distal limb between the clinician's legs and using both hands (**B**). A painful response indicates an osteochondral fragment or an osteophyte. Occasionally a loose osteochondral fragment can be palpated.

flexion. The elbow joint is flexed simultaneously; therefore a positive response to carpal flexion can rarely result from elbow pain. The examiner should palpate the dorsal surfaces of the carpal bones with the limb in partial flexion (Fig. 6-19). Many pathological conditions associated with the carpus are manifested dorsally, and pain associated with osteochondral fragmentation, slab fractures or other severe injuries, or

osteoarthritis can be assessed with the limb in this position. Focal pain can be identified, and occasionally loose fragments associated with the third carpal bone or distal lateral radius can be identified.

Antebrachium (Forearm)

Digital palpation of the forearm usually is performed with the limb bearing weight. The examiner should look primarily for muscle atrophy, wounds, or mild swelling associated with the radius. Small wounds in the antebrachium may look innocuous, but inappropriately severe lameness and pain on palpation may reflect a spiral radial fracture. The examiner should pay particular attention to the medial aspect of the limb; this area is easily overlooked when palpating from the lateral side. Distally, fluid distention of the carpal sheath or acute swelling associated with injury of the accessory (proximal or superior check ligament) ligament of the SDFT, or the flexor muscles and tendons can occur. The amount of muscle in the extensors and flexors should be compared with the contralateral limb, because subtle atrophy may be missed during observation.

Elbow

Frank swelling and prominent lameness accompanies many injuries of the elbow region, but other problems of the elbow joint are discovered only after diagnostic analgesia has localized pain to this area or by use of advanced imaging modalities. It is nearly impossible to use diagnostic analgesic techniques to abolish pain in the distal humerus and proximal radius and ulna; therefore advanced imaging techniques are often required to identify problems in these structures. The clinician should palpate the olecranon process and the lateral and medial collateral ligaments with the limb bearing weight. Effusion is difficult to detect, but excess fluid occasionally can be found using ballottement, by placing fingers both cranial and caudal to the lateral collateral ligament. The elbow is flexed by pulling the distal limb in a cranial and proximal direction, and then extended, by pulling the lower limb in a caudal direction. The shoulder joint is undergoing the opposite reaction during this manipulation, and pain associated with that joint or the bicipital bursa can cause a positive response during elbow manipulation.

Brachium (Arm) and Shoulder

The shoulder and intertubercular (bicipital) bursa are regularly blamed as the cause of lameness yet are seldom involved. Normal horses may resent palpation of this area. Pain in the muscles surrounding the shoulder joint may develop *secondary* to primary lower limb lameness. In Standardbred (STB) racehorses with carpal lameness, secondary pain often is detected when palpating the bicipital bursa.

Palpation of the arm is limited because overlying muscles obscure much of the humerus. Horses with displaced humeral fractures usually are unwilling to bear weight and have severe soft tissue swelling. Those with humeral stress fractures usually have no localizing signs except a positive response to upper limb manipulation. A normal intertubercular (bicipital) bursa is not palpable. Horses with bicipital bursitis usually resent direct compression of the greater tubercle of the humerus and fluid distention may be palpable, but ballottement is usually limited. Effusion of the scapulohumeral (shoulder) joint is palpable only if severe and even then is easily overlooked.

Upper limb manipulation, including static flexion and extension to assess the range of motion of the shoulder and elbow joints and the presence of a painful response, should always be performed. This can be done during palpation of the elbow or later when the clinician finishes the shoulder region. Most horses with shoulder joint lameness or bicipital bursitis show a painful response when the limb is pulled back (shoulder flexion, elbow extension), whereas those with elbow lameness may show a painful response when the limb is pulled forward (shoulder extension, elbow flexion).

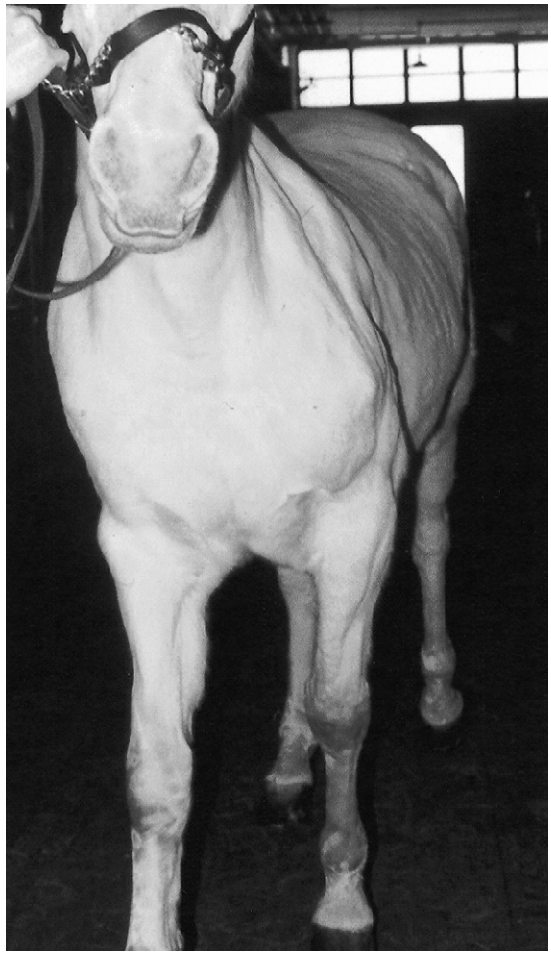

Fig. 6-20 This horse shows atrophy of the supraspinatus and infraspinatus muscles with concomitant lateral subluxation of the left shoulder joint.

The examiner should palpate the scapular area and move the mane if necessary. Atrophy of the infraspinatus and supraspinatus muscles may indicate suprascapular nerve or brachial plexus injury (Fig. 6-20). Muscle atrophy of these and other forelimb muscles can be caused by other neurogenic causes or by disuse. Upper limb palpation often is used to confirm those findings recognized during observation of the horse. Scapular height is compared manually. Although rare, damage to the innervation of the serratus ventralis muscle allows abnormal elevation of the injured side when the horse is standing or during movement. Pectoral muscle atrophy can easily be missed during observation, and the pectoral region should be palpated to assess pectoral muscle mass and identify swellings or wounds that may cause lameness.

PALPATION OF THE CERVICAL AND THORACOLUMBAR SPINE

Cervical Spine (Neck)

Palpation of the neck is limited. I usually palpate the brachiocephalicus muscle after shoulder palpation and manipulation, a procedure thought to have predictive, but non-specific value in horses with forelimb lameness.[8] The muscle is squeezed just cranial to the shoulder joint; most horses flinch, but some

horses with ipsilateral forelimb lameness show a marked painful response. The examiner should palpate both sides of the neck, noting any swelling or muscle atrophy. Cervical abscessation can cause signs of neck pain and forelimb lameness. Muscle atrophy may indicate long-standing cervical pain or ipsilateral forelimb lameness. Muscle development of the neck may be asymmetrical especially if viewed from above (the perspective of a rider).[2] Palpation of the poll region is important because undue soreness cranial to the wings of the atlas may be associated with poor performance.[2] The head should be moved from side to side to evaluate the horse's willingness to move the neck. One hand is place on the mid-cervical region to use as a fulcrum, and the other hand is used to bend the head and neck toward the examiner. Food also can be used to entice the horse to move the head and neck from side to side. Normally a horse can reach around to the girth region on either side to ingest food, and reluctance to do so may indicate neck pain. This procedure may more closely mimic the horse's natural head and neck movement than using a hand in the mid-cervical area as a fulcrum. A more comprehensive examination, including neurological or chiropractic evaluations, may be necessary after completion of the lameness examination. Up and down movement of the head also should be assessed. Although usually not a part of a routine lameness examination, evaluation of the temporomandibular joints and the mouth may be necessary in horses with poor performance.[2]

Thoracolumbar Spine (Back)

Additional detailed palpation of the back and pelvis may be necessary once the lameness examination is completed. Chiropractic manipulation and assessment of acupuncture points may be useful, but it usually is reserved for specific horses or when history and clinical signs warrant such an examination and if the clinician is qualified to complete it. The cranial thoracic spine has already been briefly evaluated during examination of the shoulder for scapular symmetry. The withers should be examined closely for conformational abnormalities, such as those seen with fracture of the dorsal spinous processes or fistulous withers. The presence of sores may indicate an ill-fitting saddle and can cause performance-related problems. Using a hand on each side of the spine the examiner should apply digital pressure to assess vertebral height, presence of pain, muscle atrophy, and to confirm symmetry (Fig. 6-21). Many horses resent deep and aggressive palpation of the epaxial muscles, and the response of normal horses should be learned before a pathological response is presumed. Most horses readily become mildly lordotic ("scootch") during deep digital palpation or by using a blunt object such as a pen. Some clinicians prefer to use the ends of the fingers to "run" (apply digital pressure while moving the fingers caudally) the muscles from cranial to caudal, parallel to the spinal column. When this is continued along the gluteal muscles and rump. most normal horses become somewhat kyphotic and move forward slightly. Aggressive use of blunt or sharp objects to assess pain should be avoided. Some horses are stoic during palpation, and it may be impossible to stimulate them to extend and flex the thoracolumbar region without the use of a blunt instrument.[2] In these horses, firmly stroking the ventral abdomen may stimulate movement.[2] With one hand on the horse's back during movement of the thoracolumbar spine, the clinician may be able to feel muscle "cracking" during the release of tension in the epaxial muscles.[2] The observation of muscle fasciculations during or after palpation usually indicates a degree of muscle pain. Failure to exhibit the normal lordotic or kyphotic responses, assuming a guarded posture, or vocalizing during the examination are further signs of back pain.

In many horses, back pain, and more specifically, muscle pain, is secondary to hindlimb lameness, resulting from altered gait and posture. Any site of pain in the hindlimb may alter the gait to cause secondary upper limb or back muscle pain. The use of diagnostic analgesia to confirm the primary source of pain (in the hindlimb or locally in the back) may be required to make the true diagnosis. Back pain often is complex and may be caused by many factors including ill-fitting saddles, poor riding, and other primary problems, such as overriding of dorsal spinous processes or other bony causes. The clinician should palpate carefully to detect localized swelling in the area of the saddle. Even small areas of hair loss without swelling may indicate a loose or ill-fitting saddle, abnormal movement of the saddle associated with hindlimb lameness, or a rider sitting crookedly.[2]

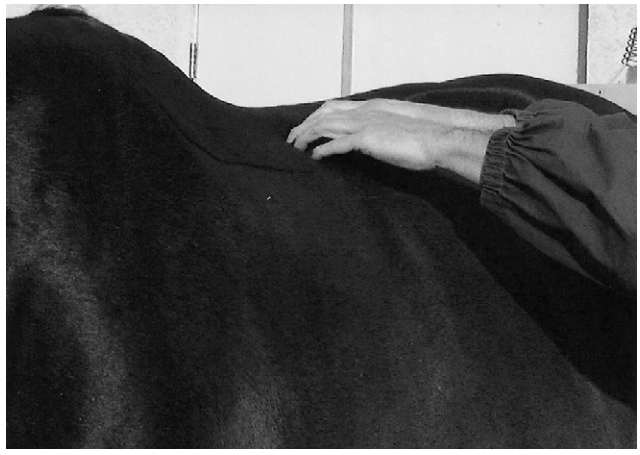

Fig. 6-21 Palpation of the thoracolumbar region should be performed in a quiet, careful manner. Many horses object to sudden or sharp stimuli applied to this region. Direct, even pressure is applied to the epaxial muscles (*shown*) and the summits of the dorsal spinous processes.

It is doubtful that muscular pain alone can cause unilateral hindlimb lameness. Back pain was induced in STB horses by injection of lactic acid into the left longissimus dorsi muscle and subsequently exercised and observed with high-speed cinematography.[8] Frank lameness was not observed, but there was slight modification of left hindlimb stride and reduced performance. This supports the clinical observation that back pain usually is the result, not the cause, of obvious hindlimb lameness, although it may result in slight alterations in gait. Severe vertebral abnormalities or an abscess in the epaxial muscles may result in lameness or neurological dysfunction.

PALPATION OF THE LATERAL AND VENTRAL THORAX AND ABDOMEN

History or observation of lameness, performance, or behavioral abnormalities seen only when a horse is ridden or wearing tack should prompt examination of the thoracic region. Irritation from an ill-fitting girth or other sores or wounds can contribute to poor performance, and injury of the sternum or ribs can cause pain associated with saddling or being ridden. Traumatically induced hernias of the ventral abdomen can cause gait deficits or guarding of the abdomen.

PALPATION OF THE EXTERNAL GENITALIA

Testicular or inguinal pain should be considered as a cause of gait modification. Swelling, infection from previous castration, scirrhous cord, and mastitis can cause a change in gait. The veterinarian should determine the sex of the horse and the presence of one or both testicles.

PALPATION OF THE PELVIS

Palpation of the pelvis is performed to confirm previous observations. The horse should stand as squarely as possible. The clinician should palpate all bony protuberances, including the tubera coxae, tubera sacrale, and tubera ischii. The examiner stands behind the horse and palpates these paired protuberances simultaneously if it is safe to do so (Fig. 6-22). Fracture of a tuber coxae or ilial shaft may result in asymmetry, but if the ventral aspect of the tuber coxae is fractured, the height of the dorsal aspect may be equal to the contralateral side, but the anatomy of the ventral aspect is distorted. Small muscle defects may be associated with fracture or enthesopathy of the tubera ischii, but even with a displaced fracture, palpation of this area may be unrewarding (Fig. 6-23). If a pelvic injury or fracture is suspected, the clinician should gently rock (move) the horse from side to side. Subtle crepitus may be detected, but with many pelvic fractures this is not apparent until days to weeks after injury and only during the initial portions of the examination before muscle guarding supervenes. The veterinarian should grasp the tail and elevate it. Many horses resist this, but in those with fractures of the base of the tail (most commonly from sitting in the starting gate or trailer), a true painful response is elicited. Subtle swelling also may be present. Lack of tail tone may indicate neurological disease.

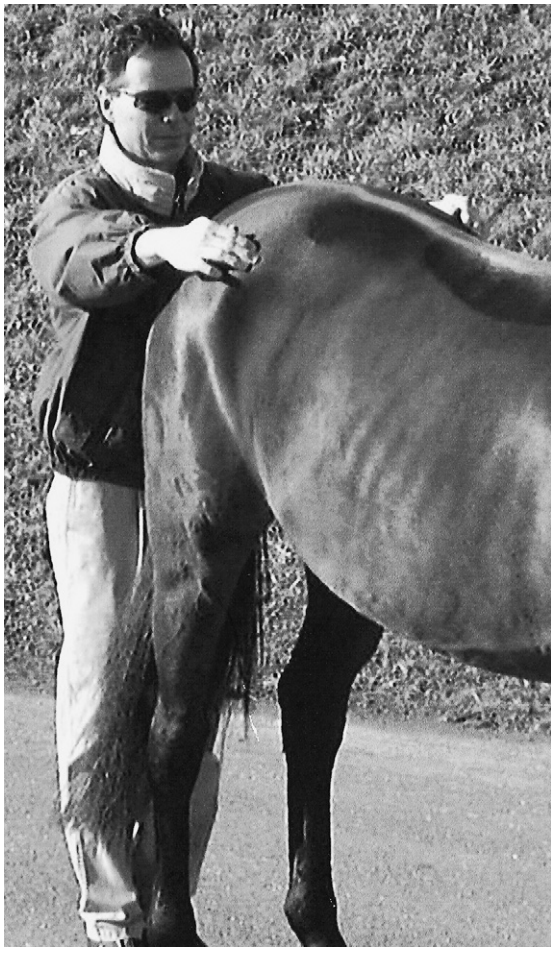

Fig. 6-22 Although the veterinarian must take care when standing behind any horse, this perspective is crucial in determining pelvic heights and widths. The height of each tuber coxae is compared in this photograph. Alternatively, an assistant on each side can be asked to point to a comparable location, or tape can be applied.

Muscle pain and muscle atrophy should be assessed. The clinician carefully examines the gluteal musculature, the origin of the caudal thigh muscles, and the tensor fascia lata (see Chapter 46 for further discussion of muscle assessment and palpation of the greater trochanter of the femur). Pain or soreness noted during palpation of the semimembranosus and semitendinosus muscles may be associated with injury of the ipsilateral tuber ischium.[2]

PALPATION OF THE PELVIS PER RECTUM

Rectal examination is not part of the routine lameness examination and should be reserved as a special examination procedure if pelvic fracture or aortoiliac thrombosis is suspected. With the wrist just inside the anus the veterinarian should palpate the medial and dorsal aspects of the acetabulum, comparing sides. In young horses, there is a membranous junction between pelvic bones in the center of the acetabulum; a defect and a small amount of motion normally can be felt. Just cranial to the acetabulum is the cranial aspect of the pubis (brim of the pelvis). With the arm at elbow depth the examiner should sweep the arm dorsally on each side to palpate the medial aspect of each ilium. The ventral aspect of the sacrum and sacroiliac region are compared. The clinician should compare the pulse quality between the right and left external iliac arteries and evaluate conformation and pulse quality of the terminal aorta and branches. Horses with aortoiliac thromboembolism have abnormal conformation and altered pulse quality. Crepitus may be felt more easily by gently rocking the horse from side to side, picking up one hindlimb, or walking the horse a short distance with the veterinarian's arm still within the rectum.

Asymmetry, swelling, actual fracture lines, fragments or callus, and crepitus are assessed. In horses with acute pelvic fractures, crepitus, fracture fragments or lines, and callus usually are not detectable, but hematoma and soft tissue swelling usually can be felt. In horses with ilial wing or shaft

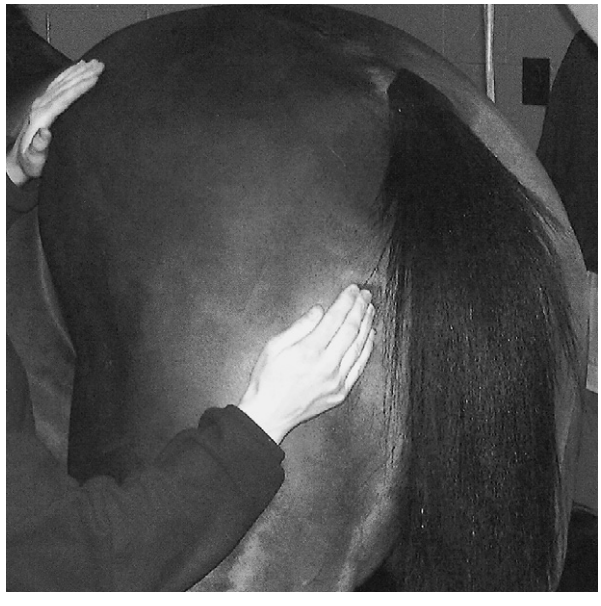

Fig. 6-23 The tubera ischii (*shown*) and third trochanters are palpated carefully. Enthesopathy or fracture causes lameness that is difficult to locate without careful palpation or scintigraphic examination. Occasionally, horses with small muscle defects located distal to the tubera ischii have chronic lameness from previous fracture. (Courtesy Carolyn Arnold, Kennett Square, Pennsylvania.)

fractures, large fracture hematomas often are present, but the absence of swelling does not preclude presence of ilial fractures. These horses are at risk to develop fatal hemorrhage. Edges of fracture fragments may be evident with comminuted or grossly displaced fractures. With chronic pelvic fractures, crepitus and callus may be more obvious.

PALPATION OF THE HINDLIMB

For safety reasons, I prefer to start proximally and work distally in a hindlimb, allowing the horse to become accustomed to palpation. Horses often object to palpation of the flank and stifle regions, and this should not be misinterpreted as a sign of pain. The clinician should grasp the tail and pull it gently toward himself or herself to keep the ipsilateral hindlimb bearing weight and reduce the chance of the horse kicking. It may be useful to pick up the ipsilateral forelimb. In the large majority of horses the entire limb can be safely examined while bearing weight, but pain in the lame limb or contralateral limb or the horse's behavior may make it difficult or impossible to pick up the limb. Reluctance to pick up the hindlimbs has been attributed to unilateral or bilateral sacroiliac pain.[2] Horses with shivers often are reluctant or anxious to pick up one or both hindlimbs. It may be necessary to spend a small amount of time coaxing the horse to elevate the hindlimb, at first just high enough to examine or pick out the hind foot and then progressing to full flexion. Although historically the hock and stifle joints have been regarded as the principal sources of pain causing hindlimb lameness, there are many other potential sites, and the metatarsal and fetlock regions in particular should be examined with care.

Thigh

The clinician should assess the thigh for swelling, muscle atrophy, or scarring. Horses with femoral fractures usually have obvious severe swelling, crepitus, and instability of the limb. The third trochanter of the femur is difficult to feel, and clinical abnormalities associated with enthesopathy of the insertion of the superficial gluteal muscle or a fracture usually are impossible to detect. Scarring associated with the semitendinosus, semimembranosus, and rarely, the biceps femoris can lead to mechanical gait deficits, known as *fibrotic myopathy*. The gastrocnemius muscle arises from the distal caudal femur, and acute tearing of this muscle may cause swelling in the caudal stifle area. This is difficult to perceive, but severe muscle injury results in a marked postural change, which should provoke more careful assessment of this region.

Stifle

Palpation of the stifle is limited to the cranial, lateral, and medial aspects; unfortunately, the caudal and proximal aspects of the joint are inaccessible. Many horses, especially fillies, object to palpation of the stifle, a normal response often misinterpreted as a painful reaction.

The veterinarian should palpate the stifle with the limb bearing weight. The foot should be flat on the ground. This may be impossible if the horse has severe pain prohibiting complete assessment of the stifle. The limb should be in a neutral and not in an abducted position, and should be perpendicular to the spine or slightly ahead of the other hindlimb. If the limb is retracted, it is more difficult to palpate the patellar ligaments and joint outpouchings. The middle patellar ligament is identified and followed proximally to the distal aspect of the patella. The clinician should feel the femoropatellar joint capsule between either the middle and medial or the middle and lateral patellar ligaments and should determine the presence of effusion. In horses with osteochondrosis dissecans (OCD), fluid distention can be pronounced. However, normal young horses (weanlings to early 2-year-olds) often have prominent bilateral fluid distention of the femoropatellar joint capsules.

The clinician should find the medial patellar ligament and follow it proximally and distally. At the proximal extent, the medial fibrocartilage of the patella can be felt medial to the medial trochlear ridge of the femur. It is this normal arrangement of the medial aspect of the patella and the medial trochlear ridge that allows the veterinarian to determine whether a horse has patellar luxation. The position of the patella is difficult to confirm if the horse is standing with the stifle flexed. True patellar luxation is rare. The examiner should determine if the medial patellar ligament is enlarged, which usually reflects previous desmotomy. Usually a distinct depression is present between the medial patellar ligament and the medial collateral ligament, but effusion of the medial femorotibial (MFT) joint may result in a substantial "bulge" (Fig. 6-24). This may be the only clinical sign indicative of MFT joint injury.

The lateral patellar ligament is palpated from its origin to insertion. Patellar desmitis is unusual, but does occur; it usually involves the middle patellar ligament and may cause mild swelling. Previous injection with counterirritants causes firm, fibrous areas over the patellar ligaments, a common finding in racehorses. Gently rocking the horse from side to side to assess motion of the patella may give some indication of the potential for intermittent upward fixation of the patella (IUFP). In horses prone to IUFP, jerky rather than the normal smooth motion of the patella sometimes is detected.[2] The

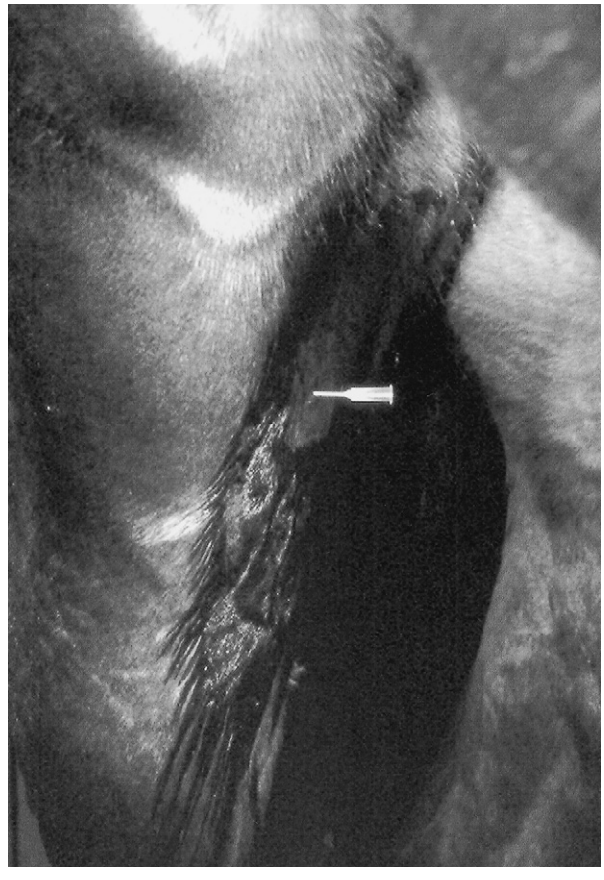

Fig. 6-24 The medial femorotibial joint (right stifle, cranial view) is the most common location for osteoarthritis of the stifle joint and is palpated medially (*needle in joint*) between the medial patellar ligament and the medial collateral ligament. Normally a depression is present at this location, but a bulge from effusion can be palpated.

lateral femorotibial joint capsule is accessed between the lateral patellar and lateral collateral ligaments, but even with severe effusion it is difficult to palpate. Overlying and adjacent soft tissue structures obscure palpation.

Deep palpation of the medial collateral ligament and the medial patellar ligament with the limb in flexion may elicit a painful response in horses with stifle lameness, but it probably is not specific for the source of pain in the stifle. The medial collateral stress test is perhaps the most reliable manipulative test of the stifle, although horses with lower limb lameness also may respond. With the leg in partial flexion, the shoulder or one hand is used as a fulcrum on the lateral aspect of the stifle, and the distal extremity is pulled laterally, thus placing valgus stress on the stifle (Fig. 6-25). Care should be taken because horses may resent this manipulative test. If possible, the valgus motion should be applied by using the shoulder as a fulcrum and both hands on the crus, thus eliminating possible false-positive results from the lower limb. Patellar manipulation may cause a painful response, particularly in horses with femorotibial joint disease. During this procedure, caudal movement of the femur also may exacerbate cruciate injuries. With the limb on the ground in a weight-bearing position, the clinician's hand is placed on the distal aspect of the patella, and the patella is forced upward (Fig. 6-26). Theoretically during this test, multiple movements of the stifle are induced. The patellar ligaments are stretched, the patella is forced proximally, and on release the patella rapidly moves distally against the trochlear ridges, and the femur is forced caudally. Tests to assess cruciate ligament damage have been described but are dangerous to perform, and I have not found them particularly useful.[9] Complete tearing of the cruciate or collateral ligaments is rare, and partial tearing does not cause clinically detectable instability. In horses with severe lameness and gross stifle instability, it is obvious that the stifle is the source of lameness and pain usually prohibits manipulation.

Fig. 6-25 The valgus stress test of the stifle is difficult to perform and is accomplished by using a hand (*shown*) or shoulder as a fulcrum. This test can be done during static examination or a provocative test followed by trotting (see Chapter 8).

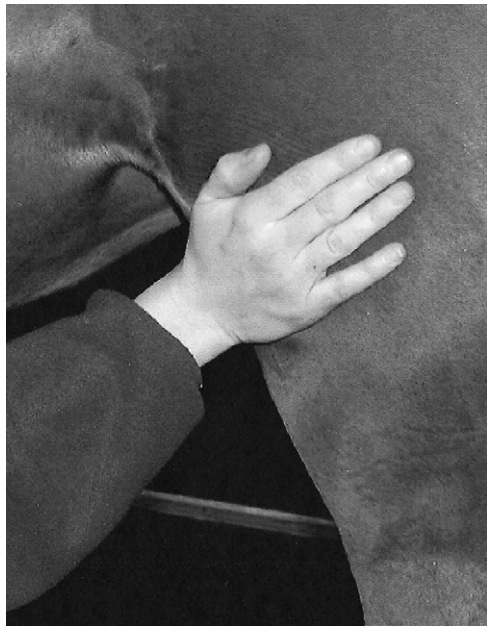

Fig. 6-26 Used as a static or provocative test, patellar manipulation is performed by placing the palm of the hand over the cranial aspect of the patella and manually forcing the patella proximally several times in succession. This maneuver can exacerbate pain from conditions of the patella and femoropatellar joint and forces the distal femur in a caudal direction. Pain from soft tissue injuries such as patellar, cruciate or collateral ligament tears, and osteoarthritis of the femorotibial joints can be exacerbated, but false-negative and false-positive results are common. (Courtesy Carolyn Arnold, Kennett Square, Pennsylvania.)

Crus

The examiner should palpate the crus using both hands with the limb bearing weight. Subtle swelling of the medial aspect of the tibia can be palpated, but palpation of the caudolateral aspect of the tibia, the area in which stress fractures are diagnosed most frequently, is limited. Often no palpable abnormality is associated with a stress fracture. Any small wound or any form of swelling should be thoroughly investigated for the possibility of underlying bone damage, such as an occult tibial fracture. The veterinarian should palpate the caudal soft tissues. Proximally, the musculotendonous junction of the gastrocnemius muscle is a rare site of pain. The common calcaneal tendon is assessed. Swelling may indicate damage to any one of the contributing tendons. Effusion of the tarsal sheath causes swelling just proximal to the tarsus, at the caudal aspect of the crus, and should be differentiated from bog spavin. Deep palpation of the medial and caudal aspects of the crus is performed with the limb elevated (Fig. 6-27). Horses with tibial stress fractures or those with spiral fracture or other tibial trauma may show a painful response, but false-positive responses are frequent. Tibial percussion, performed medially by using a clenched fist (knuckles) as a hammer, may elicit a painful response in horses with stress fractures, but many normal horses resent this test.

Tarsus

Five common swellings of the hock are important to differentiate, but hock swelling is not synonymous with hock pain. *Capped hock* is swelling located at the point of the hock (the proximal aspect of the calcaneus) and usually is an incidental finding, but in some horses the condition does cause lameness (Fig. 6-28). The most common form involves the development of firm, fibrous subcutaneous tissue in the false bursa that lies over the point of the hock. This is a common area for abrasions and excoriation, and fibrous tissue formation results in a blemish but usually no lameness. Horses may be sensitive to palpation if the area has been traumatized recently. Infection or trauma leading to osteitis of the calcaneus can cause a clinically important capped hock and severe lameness. In these horses the problem involves the calcaneal bursa, located between the common calcaneal tendon and the calca-

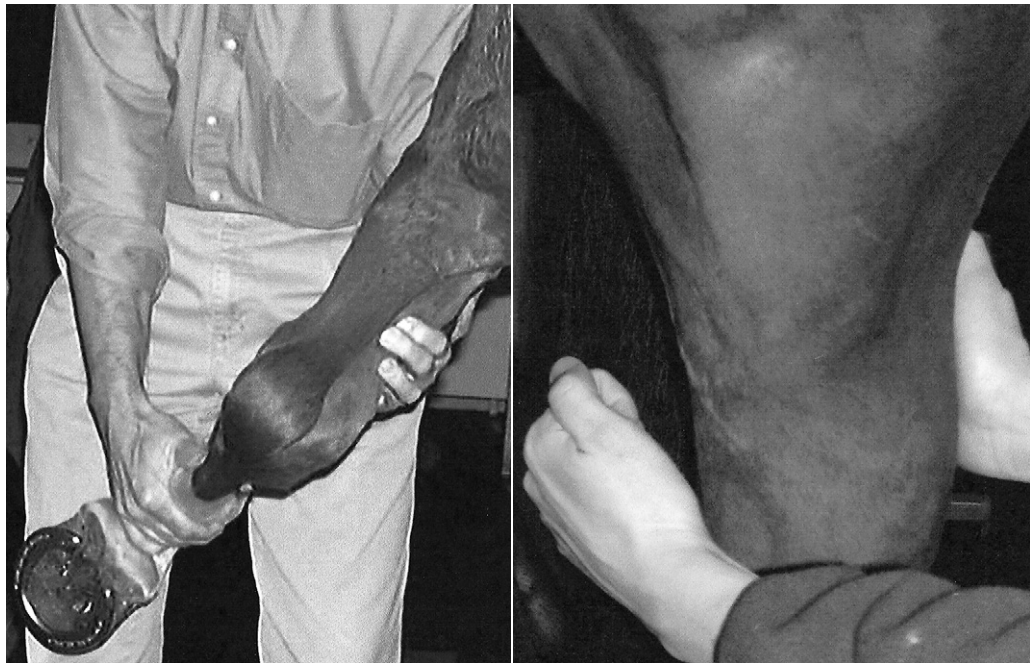

Fig. 6-27 **A,** Palpation of the medial aspect of the tibia in a flexed position or **B,** tibial percussion in the standing position sometimes elicits pain in horses with tibial stress fractures, but false-positive results are common.

neus. If surrounding soft tissue swelling is minimal, fluid distention of the calcaneal bursa may be felt by ballottement. The bursa can be felt both medially and laterally at the proximal aspect of the calcaneus. Lateral, or less commonly, medial dislocation (luxation) of the SDFT results in similar swelling, but in the acute situation, lameness is present. Careful palpation may reveal the SDFT coursing laterally (Fig. 6-29), unless excessive soft tissue swelling is present.

Effusion of the tarsal sheath, *thoroughpin*, must be differentiated from bog spavin (see following text). Tarsal tenosynovitis causes swelling both medially and laterally in the depression between the calcaneal tendon and caudal tibia (Fig. 6-30). With severe effusion of the tarsal sheath, fluid distention can be palpated distal to the hock on the medial aspect. Thoroughpin usually is an incidental finding, seen most commonly in Western performance horses, but acute lameness accompanied by tarsal tenosynovitis can indicate strain or injury of the sheath, often associated with adjacent bony injury. Unusually, swelling in the distal, caudal crus identical to that seen with classic thoroughpin is seen, but communication with and concomitant swelling of the tarsal sheath is absent.

The term *spavin* refers to "any disease of the hock joint of horses in which enlargements occur, often causing lameness…the enlargement may be due to collection of fluids or to bony growth."[10] *Bog spavin* is fluid distention of the tarsocrural joint capsule. The tarsocrural joint has four outpouchings: dorsolateral, dorsomedial, plantarolateral, and plantaromedial. All joint pouches may be distended, although the dorsomedial and plantarolateral pouches are large and most prominent (Fig. 6-31). Using ballottement, fluid can be pushed between pouches on the dorsal or plantar aspects, thus differentiating this condition from thoroughpin.

Bone spavin refers to fibrous and bony swelling that results from chronic osteoarthritis of the proximal intertarsal, centrodistal, and tarsometatarsal joints. This swelling usually is seen in older horses and can be palpated and observed on the medial side of the hock (Fig. 6-32). Although the bony enlargement is the result of proliferation, it does not necessarily mean the horse is lame as the result of the condition. Most horses with distal hock joint pain do not have palpable enlargement medially, and based on radiographic evaluation, the most common area of proliferation and bony change is dorsolateral. Blood spavin is an old term usually meaning enlargement of the saphenous vein,[3] but it also may have been used to describe a prominent saphenous vein in horses with bog spavin. Saphenous distention is rare and the term is not used today. *Occult* or *blind spavin* is an obsolete term used to describe horses with clinical signs of hock lameness but no observable

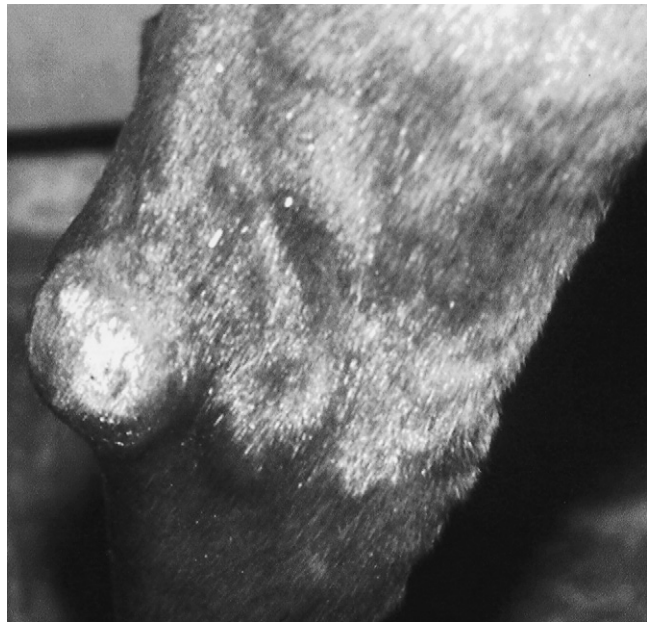

Fig. 6-28 Capped hock, a firm fibrous swelling of the proximal aspect of the calcaneus (point of the hock), is considered a blemish, but with effusion of the calcaneal bursa (not shown), lameness is substantial.

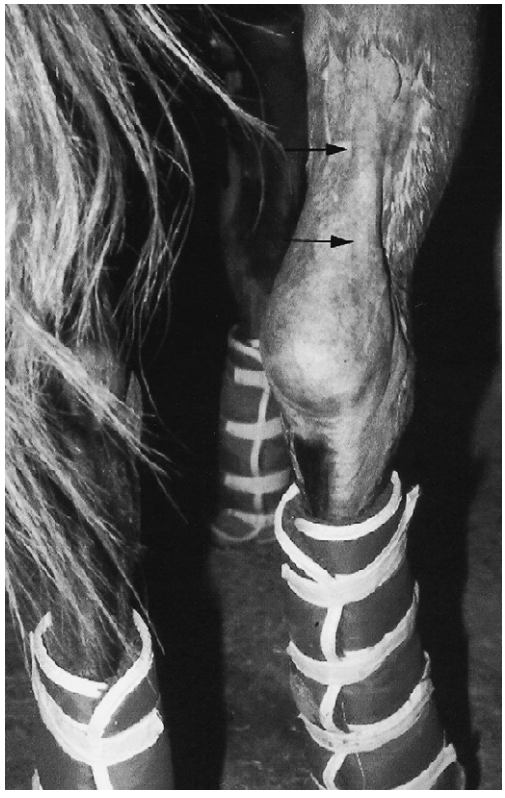

Fig. 6-29 Lateral dislocation (luxation) of the superficial digital flexor tendon. Instead of attaching to the tuber calcanei, the superficial digital flexor tendon (*arrows*) is now located lateral to the point of the hock. Initial swelling makes this diagnosis difficult.

Fig. 6-31 Moderate to severe tarsocrural effusion, bog spavin, in this draft filly was caused by osteochondritis dissecans of the distal intermediate ridge of the tibia. Distention of the large dorsomedial pouch and swelling of the dorsolateral, plantarolateral, and plantaromedial pouches was present.

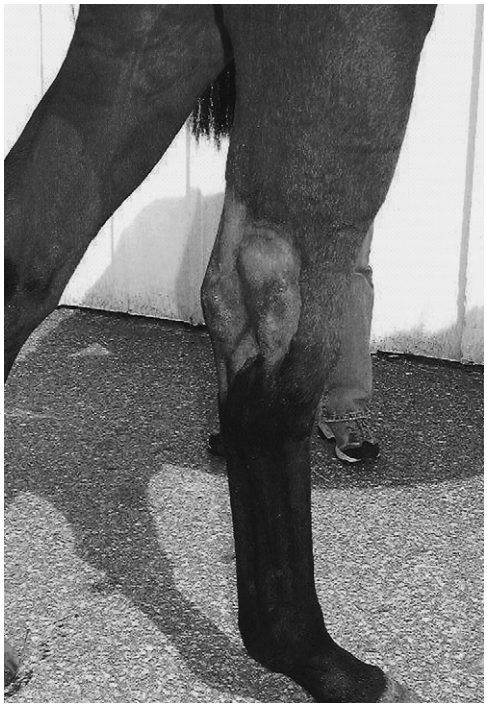

Fig. 6-30 Thoroughpin, swelling located in the distal, caudal crus, usually is caused by distention of the tarsal sheath and must be differentiated from effusion of the plantarolateral pouch of the tarsocrural joint (bog spavin).

bony swelling.[3] High spavin is also an obsolete term used to describe bone spavin located close to the tarsocrural joint.[3]

Curb describes swelling along the distal, plantar aspect of the hock and has often erroneously been blamed on long plantar desmitis. In most horses the swelling is actually enlargement of the SDF tendon or subcutaneous tissues. The swelling is often firm, but in some horses subcutaneous fluid can be present (see Fig. 79-1). In horses with acute severe injury the swelling may feel soft and mushy. In some normal horses the proximal aspect of MtIV is prominent and should not be confused with curb.

Swelling restricted to the medial or lateral aspect of the hock may reflect collateral ligament injury. Localized heat on the medial aspect of the hock or on the proximal aspect of the metatarsus may be important findings.

THE CHURCHILL HOCK TEST

• Dan L. Hawkins

The Churchill hock test was developed by Dr. E.A. Churchill in the 1950s as a rapid, non-invasive, specific method to screen and identify distal tarsal pain in athletic horses. Although the test has been used by Dr. Churchill and me primarily in STBs, TBs, and Three-Day Event horses, it is equally reliable when applied to other equine athletes.

Digital pressure is applied on the plantar aspect of the head of the second metatarsal bone (MtII) and fused first and second tarsal bones with the limb in a non–weight-bearing position. Abduction of the limb is a positive response. To

Fig. 6-32 Bone spavin (*arrows*), fibrous and bony swelling on the medial aspect of the distal hock joint (left hindlimb) caused by chronic osteoarthritis of the distal hock joints, sometimes appears in older sport horses but is rare in young racehorses. The presence of bone spavin should be noted and this area should be palpated carefully, but horses can have distal hock pain without bone spavin, and horses with bone spavin can have lameness elsewhere in the limb. Previous cunean tenectomy causes chronic fibrosis in this region.

examine the left tarsus, the clinician approaches the horse facing caudally. The left hindlimb is picked up and brought forward, supported by the clinician's right hand cupped under the fetlock or hoof. Holding the limb so that the hoof is approximately 25 to 30 cm above the ground is most comfortable for the horse. The heel of the left hand is positioned on the proximodorsal surface of the third metatarsal bone (MtIII) while the third phalanges of the index and middle or middle and ring fingers are placed around the medial side of the tarsus to engage the bony ridge formed by the head of MtII and the first and second tarsal bones (the area of insertion of the cunean tendon) (Fig. 6-33). The thumb is rested on the dorsal lateral aspect of the tarsus and proximal MtIII. Gentle, firm pressure is applied to the bony ridge by flexing the phalanges of *only the index and middle fingers* (Fig. 6-34). The hand does not squeeze the hock. Pressure is applied three times approximately 1 second apart, each time with increasing intensity to a maximum effort on the third time.

Proficiency requires patience and routine practice. Consistent diagnostic information can be obtained safely from

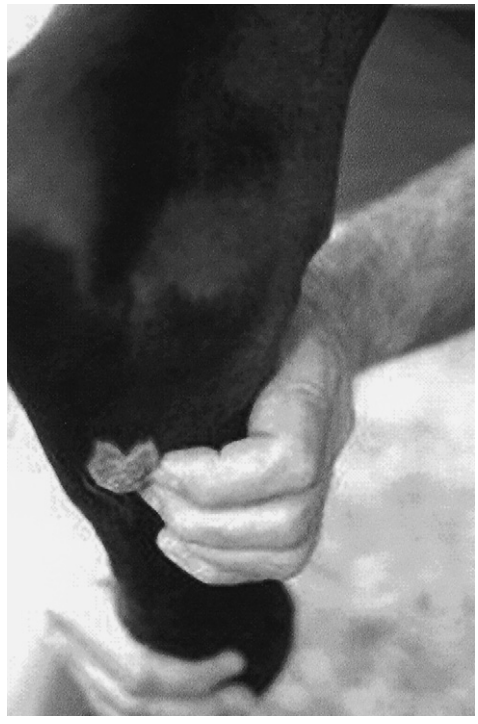

Fig. 6-33 Correct left hand placement in the left proximal metatarsal region to perform the Churchill hock test. (Courtesy Dan Hawkins, Dubai, United Arab Emirates.)

Fig. 6-34 The Churchill test is demonstrated on an anatomy specimen. The index and middle fingers are flexed and positioned on the bony ridge formed by the third metatarsal bone and the fused first and second tarsal bones, and the heel of the hand rests on the proximodorsal aspect of the third metatarsal bone. The thumb rests against the dorsolateral aspect of the tarsus. (Courtesy Dan Hawkins, Dubai, United Arab Emirates.)

more than 90% of fit racehorses. If the limb cannot be picked up, the test cannot be performed. Fussing and repeatedly flexing the hock and limb in an agitated manner while the procedure is performed should not be misinterpreted as a positive response.

The Churchill hock test is useful for horses that are not visibly lame, but the trainer or rider has a complaint that the horse is doing something uncharacteristic during work or competition associated with decreased performance. The horse may have a changed attitude toward work, lugs in, is rough in the turns, refuses to change leads, stops at jumps, jumps to one side, or is stiff going in one direction. Although these horses cannot be blocked out at a slow gait, the Churchill hock test may suggest hock pain.

SAPHENOUS FILLING TIME

• **Mike W. Ross**

The veterinarian should assess the saphenous vein filling time. Blood flow in the saphenous vein is prevented using digital compression in the proximal metatarsal region, and the blood accumulated in the vein over the tarsocrural joint is pushed proximally to completely collapse the vein. The finger compressing the vein distal to the hock is then removed, and the time it takes for the saphenous vein to fill is observed. Normally, it takes less than 1 second for the vein to fill, but in horses with reduced circulation, prolonged filling time is seen. Pulse quality of the dorsal metatarsal artery, located on the dorsolateral aspect of MtIII just dorsal to the fourth metatarsal bone (MtIV), can be useful, especially if the history suggests lameness is caused by vascular compromise. The arterial pulse quality is compared with the contralateral limb.

The Metatarsal Region

The veterinarian should palpate the flexor tendons and SL. Tendonitis is unusual in the hindlimb, but occasionally SDF tendonitis occurs in the proximal metatarsal region. This is most common in horses with curb, and tendonitis progresses distally to involve the metatarsal area. The hock angle is evaluated carefully. Occasionally horses with severe curb or those with SDF tendonitis of the metatarsal region have reduced hock angle (obvious unilateral sickle-hocked conformation), indicating loss of support in the SDFT. Once a general palpation for the presence of heat, swelling, exostoses associated with the MtIII, MtII, MtIV, and the proximal aspect of the DFTS is completed, the limb is lifted and deep palpation is performed. The clinician should carefully palpate the origin and body of the SL, keeping in mind that both false-positive and false-negative responses can occur (Fig. 6-35). Much of the palpation of the SL laterally is indirect, since MtIV hides the origin and proximal body. Because the presence of the splint bones and dense metatarsal fascia prevents substantial swelling, or at least the clinical recognition of swelling of the SL, even mild swelling in the proximal, medial metatarsal region should be carefully interpreted. With the limb in flexion, the axial borders of splint bones are palpated. The dorsal aspect of MtIII should also be assessed with the limb in flexion, because bony injury of MtIII does occur and includes dorsal cortical trauma from external injury or interference, dorsal cortical and spiral fractures, and proximal dorsolateral fractures.

Metatarsophalangeal Joint

Many of the common problems of the metatarsophalangeal joint, such as short, mid-sagittal fractures of the proximal phalanx, sesamoiditis, stress or non-adaptive remodeling of MtIII, and osteochondrosis, cause very few clinical signs and while palpation is quite important, diagnostic analgesia is often needed to localize pain to this area. Nonetheless, careful

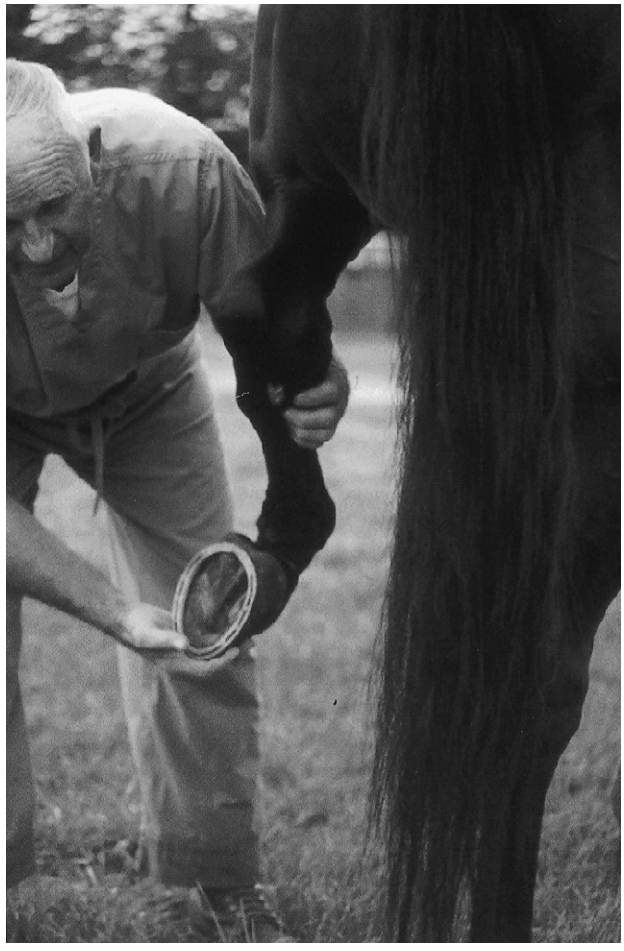

Fig. 6-35 Deep palpation of the proximal suspensory ligament can be performed only with the limb in flexion. The close association of the suspensory origin to the Churchill site explains the need to differentiate proximal plantar metatarsal pain from distal hock joint pain using diagnostic analgesia. (Courtesy Howard "Gene" Gill, Pine Bush, New York.)

palpation of the fetlock region is mandatory. Some horses have concurrent metatarsophalangeal joint and stifle pain and when suspicious findings exist in one site, the veterinarian should look carefully at the other for additional, secondary, or complimentary problems.

The metatarsophalangeal or hind fetlock joint, is evaluated with the limb bearing weight and in flexion. The clinician should assess the metatarsophalangeal joint capsule and the DFTS for the presence of effusion or fibrosis (Fig. 6-36). Incidental effusion of both the metatarsophalangeal joint and DFTS is common in the hindlimb of older performance horses therefore this finding should not be over-interpreted. In younger horses, particularly racehorses, the presence of effusion can be an important clinical sign associated with osteoarthritis or other problems and should be interpreted accordingly. The clinician should carefully palpate for the presence of heat and mild swelling over the surface of both PSBs, subtle but important signs of sesamoiditis. This problem is more prevalent in the hindlimb and causes few clinical signs. The digital pulse should be assessed.

With the fetlock joint in flexion, the veterinarian should palpate the proximal, dorsal aspect of the proximal phalanx for the presence of pain or exostoses (Fig. 6-37) and should apply pressure to the PSBs, avoiding aggressive compression

Fig. 6-36 The metatarsophalangeal joint region often is overlooked during lameness examination. This joint should be palpated carefully with the limb in the standing and flexed (*shown*) positions. (Courtesy Ross Rich, Cave Creek, Arizona.)

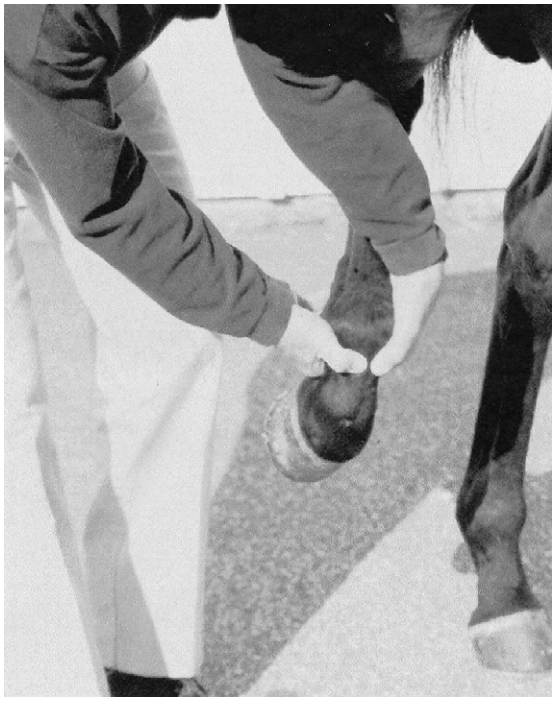

Fig. 6-37 Palpation of the proximal, dorsal aspect of the proximal phalanx can elicit pain in horses with incomplete mid-sagittal fracture of the proximal phalanx. In trotters, interference injury from the ipsilateral front foot causes pain and swelling in this region. (Courtesy Ross Rich, Cave Creek, Arizona.)

that may cause false-positive results. The range of motion of the metatarsophalangeal joint is noted.

Pastern

When the limb is elevated, the reciprocal apparatus causes constant flexion of the digit, which makes palpation of the plantar aspect of the pastern exceedingly difficult. Subtle swelling in the plantar pastern is easy to miss. Bony and soft-tissue structures should be palpated with the horse in the standing position and the veterinarian should note the same clinically important areas that were pointed out for the forelimb. High and low ringbone (osteoarthritis of the proximal interphalangeal and distal interphalangeal joints, respectively), osteochondrosis of the pastern joint, and soft tissue problems such as SDF branch tendonitis, distal sesamoidean desmitis, and plantar injury of the pastern joint occur, but with reduced frequency when compared with the forelimb.

Foot

A similar approach to the evaluation of the hind foot as that described for the front foot is used. I spend considerably less time evaluating the hind foot than the front foot, unless the history or horse type dictates otherwise, because this area is relatively infrequently the source of pain. In the Draft horse, hind foot pain is as common as in the forelimb, and therefore the hind feet merit considerable attention. Unless specifically indicated by the lameness history, or the horse is severely lame without an obvious cause in the upper limb, I do not routinely perform a hoof tester examination of the hind foot. Pressure with hoof testers over the frog and across the heels in the hind foot often causes a false-positive response in normal horses. The position needed to perform an unassisted hoof tester examination in the hindlimb can be dangerous. The presence of an assistant to elevate the limb may obviate some of the risk.

The examiner should assess the shape, balance, and contour of the foot, and observe the shoe (or lack of one) carefully. Hoof angle in the hindlimb ranges from 48° to 58°, and the hoof and pastern axis should be straight. A common finding is low or under-run heels. An interesting relationship between low heels and the presence of PSD has been noted.[2] In these horses a lateral radiograph of the foot shows the plantar aspect of the distal phalanx is lower than the dorsal aspect.[2] Shoe wear is extremely important in the hindlimb and can give clues to the source of lameness. For instance, horses with distal hock joint pain tend to stab the lower hindlimb during advancement, causing excessive wear of the lateral branch of the shoe (Fig. 6-38). Other lower hindlimb lameness, such as osteoarthritis of the metatarsophalangeal joint, can cause a similar gait, but usually abnormal shoe wear is less pronounced. Horses with stifle lameness often wear the medial branch of the shoe. The presence of heel and toe caulks or borium cause additional shear stress on many of the lower limb joints and can exacerbate lameness.

THE ROLE OF PHYSICAL EXAMINATION IN THE LAMENESS EXAMINATION

Body temperature may assist with a clinical diagnosis. The normal temperature range is 37.5° to 38.6° C (99.5 to 101.3° F), although in a foal the upper limit may normally be slightly higher. Body temperature in foals rises more abruptly than in adult horses in response to stress, infection, and inflammation. Thus transport of a foal may cause transient low-grade pyrexia, but fever in an adult horse after transport is abnormal. Localized infection in a foal usually causes pyrexia but rarely does in an adult. The examiner should not *exclude* infectious arthritis in an adult horse simply because fever is *not* present. However, adult horses usually are pyrexic during the early stages of cellulitis or lymphangitis.

Elevation in the pulse and respiratory rates often accompanies severe lameness because of pain. Systemic diseases such as endotoxemia may cause abnormal vital parameter findings in any horse and can lead to conditions such as laminitis.

It is important to remember that diseases of other body systems can cause clinical signs that mimic lameness or cause true gait deficits. For instance, abnormal or stiff gaits can be seen in horses with pleuritis and peritonitis, abdominal, sublumbar, inguinal, thoracic inlet, and pectoral abscesses or

Fig. 6-38 It is imperative to observe the hind shoes for wear during lameness examination. This right hind shoe (lateral is to the right) has wear along the dorsal and lateral aspects (lateral aspect of toe grab and fullering are worn) consistent with a lower hindlimb lameness, such as distal hock joint or proximal metatarsal region pain.

tumors. Proliferative new bone associated with hypertrophic osteopathy may be associated with a thoracic or abdominal mass. If an unusual situation arises, the veterinarian should step back and think of the exception rather than the rule, because the "red herring" may be just around the corner.

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CHAPTER • 7

Movement

Mike W. Ross

"The best time for examining a lame horse is while he is in action. An attendant should lead him on a trot, preferably on hard ground, in a straight line, allowing him freedom of his head, so that his movements may all be natural and unconstrained."

A. Liautard, 1888[1]

It would be difficult to improve on Liautard's insistence that the lame horse be examined during movement or his description for how it is best accomplished. Although all parts of the lameness examination are important, the key is the determination of the limb or limbs involved. Not all horses with musculoskeletal problems exhibit lameness that is perceptible under normal conditions, or even by use of high-speed or slow-motion cinematography, gait analysis, or other sophisticated imaging devices. Under most circumstances, however, lameness from pain or a mechanical defect in gait is discernible, and the essence of the lameness examination is to determine the source of the pain. This discussion includes relevant experimental findings to support clinical observations, but sometimes experimental finds are confusing rather than informative.

GAIT

Gait, defined as the "manner or style of walking"[2] or "the manner of walking or stepping,"[3] is used to describe the speed and characteristics of a horse in motion. The *natural gaits*, those exhibited when a horse is free in a field, are the walk, trot, and gallop.[4] The canter is a collected gallop. Other gaits including the pace, running walk, rack (a singlefoot or broken amble), fox trot, and amble are *artificial gaits*, although some

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pacers pace "free-legged" (without the use of hobbles) while on the track, either at a slow speed or racing speed, and occasionally a Standardbred (STB) paces free-legged in a field. In some instances a trotter switches from a trot to the pace, but this change usually is exhibited while the horse is performing at speed and may be associated with lameness or interference.

The term *beat* describes the number of foot strikes in a single stride cycle regardless of whether one or more feet strike the ground simultaneously. The following abbreviations are used for limbs: left forelimb (LF), right forelimb (RF), left hindlimb (LH), and right hindlimb (RH). The walk is a four-beat gait in which all four feet strike the ground independently without a period of suspension (in which no feet are on the ground). Depending on the part of the stride during which observations begin, the walk can appear to be lateral or diagonal. In general, in a lateral gait, both feet on one side strike the ground before the feet on the contralateral side. In a diagonal gait, one foot strike is followed by a strike of the foot located diagonal and contralateral to the initial foot (e.g., LF followed by the RH).

Lame horses should always be evaluated at the walk. Stride length should be evaluated and compared with observations at the trot. Stride length and sequence of footfalls are easier to see while horses are walking than while trotting. Horses with hindlimb lameness may be admitted for evaluation for failure to track up.[5] Horses normally track up, or over track. The hind foot is placed in or in front of the imprint of the ipsilateral front foot. Failure to track up usually is caused by hindlimb lameness or poor impulsion, and the hind foot imprint is seen behind that of the ipsilateral front foot.[5]

Backing is a diagonal, two-beat gait. Horses seldom back up naturally, but backing commonly is required of horses during performance events, while exiting from a trailer, or while driving. Backing is useful during lameness examination to evaluate certain gait deficits, such as shivers, stringhalt, and neurological disease.

The trot is a diagonal, two-beat gait and diagonal pairs of limbs move simultaneously. The trot is theoretically a symmetrical gait, meaning both "halves" (beats) of the stride are identical, and at low speed in a sound horse, symmetry is likely achieved. However, at speed perfect balance and fine management of weight (of the shoes) are necessary for a trotter to be perfectly symmetrical. There is a moment of suspension between impact of each diagonal pair of limbs. Hindlimb lameness is present in a higher percentage of horses that perform at speed at the trot compared with galloping horses because of differences in weight distribution in the trot and gallop. Compensating lameness develops in the diagonal paired limb. LF lameness predisposes to RH lameness.

The pace is a symmetrical, lateral, two-beat gait predominantly in STB racehorses and is characterized by movement of lateral pairs of limbs simultaneously (LH and LF; RH and RF), with a moment of suspension between lateral pairs. Pacers also have a high percentage of hindlimb lameness, but compensatory lameness usually develops in the lateral paired limb. RH lameness predisposes to RF lameness.

The gallop or run is a four-beat gait. In the gallop and the canter the horse leads with the left or right forelimb, the forelimb that strikes the ground last in the stride sequence. An unrestrained horse usually leads with the LF turning to the left, or the RF turning to the right. Fatigue also plays a role. A Thoroughbred (TB) racehorse racing counterclockwise leads with the LF on the turns, but immediately after entering the stretch, switches to the right lead. Failure to switch leads, or constantly switching leads in the gallop or canter, may reflect fatigue or lameness.

In a *left* lead gallop, the RH strikes the ground first, followed in sequence by the LH, RF, and LF followed by a period of suspension. When a horse is on the right lead, the RF strikes the ground last, propelling the horse into the suspension phase of the stride. It is often assumed that a horse with RF lameness is reluctant to take the right lead. However, bone stress measured in the radius and McIII is greater on the non-lead (trailing) forelimb, and thus a lame horse may change leads to protect the non-lead forelimb.[6] Ground reaction forces are greater in the trailing (non-lead) forelimb, a fact that supports the clinical observation that horses with forelimb lameness may select leads to protect the lame forelimb. A horse with a RF lameness may prefer the right lead, allowing the LF to assume the greater forces and bone stress.[5]

The canter (lope) is a three-beat gait. In left lead canter the RH strikes the ground first, then the LH and RF land simultaneously, followed by the LF and then a period of suspension. A horse reluctant to take a lead may be trying to compensate for hindlimb lameness. In the right lead the LH must absorb a considerable amount of concussion and then generate propulsive forces. Proneness of this limb to fatigue seems logical, but a consistent change in stride characteristics of fatigued horses to protect the LH was not seen.[7] Although the LH strikes the ground first, stance time, flexion of the upper limb joints, and ground reaction force are greater in the RH.[5] It could be assumed that a horse lame in the RH would be reluctant to take the right lead and may prefer the left lead.[5] Lead and stride characteristics of fatigued and lame horses are complex because of asymmetry of the gait, and forelimb and hindlimb problems could account for failure or reluctance to take a particular lead and inappropriate lead switching.

Young horses early in training or trained horses that are lame may exhibit a disunited canter. The horse may spontaneously change legs behind, but not in front. In changing from left to right lead canter, or vice versa, the forelimbs and hindlimbs should change simultaneously. Horses with back pain or hindlimb lameness may be reluctant to change leads, or may change in front, but not behind.

The Lameness Examination: Which Gait Is Best?

The trot is the most useful gait to determine the location of the lame limb or limbs. Forelimb lameness in particular is difficult to observe at the pace, especially in horses that are led in hand. Lame trotters may pace, supporting the supposition that the pace is an easier gait in a lame STB. I have seen horses with severe forelimb lameness at the trot that looked barely lame when pacing.

Relevance of Lameness at a Trot in Hand

Is lameness seen at a trot in hand the same lameness that compromises performance at speed? Is the lameness seen at a trot in hand in a jumping horse the same problem that causes the horse to refuse fences? The answer is usually, but not invariably, yes. For instance, I have seen many STBs show subtle unilateral hindlimb lameness at a trot in hand, but when the horse was later examined at the track and hooked to a cart, pronounced contralateral hindlimb lameness was noted. Differences include the track surface, pulling a cart, the additional weight of the driver, and a faster gait. Lameness often is evaluated on a smooth hard surface useful in exacerbating even subtle problems, but most horses perform on softer surfaces, when other problems may be apparent. More than one lameness problem may exist, one evident at a trot in hand and another while the horse is ridden or driven. Horses can show lameness from one problem when trotted in a straight line, but lameness from an entirely different problem while being trotted in a circle.

Horse Temperament and Lameness Examination

Safety of the handler, observers, and the horse must always be considered throughout a lameness examination, and with a difficult horse the examination may need to be modified,

especially on cold, windy days. In some female horses and geldings, judicious use of the tranquilizer acetyl promazine (0.02 to 0.04 mg/kg, IV) permits continuation of the examination. I avoid use of this tranquilizer in stallions, although the possibility of paraphimosis is remote. Low doses of sedatives such as xylazine can be used (0.15 to 0.30 mg/kg, IV) in stallions or other horses but can produce mild ataxia. Detomidine may be a better choice than xylazine, since the drug lasts longer, thus allowing diagnostic analgesic procedures to be performed.[5]

I try to avoid using tranquilization and sedation, although some clinicians use them frequently and report that lameness in most horses may be more pronounced and easier to observe. Mild muscle relaxation may reduce the tendency of the horse to guard the lame leg. In big moving, exuberant Warmblood horses, especially dressage horses (particularly stallions), sedation may be essential to accurately assess lameness.[5]

Leading the Horse during Lameness Examination

The horse must be led with a loose lead shank so that it can move the head and neck freely. It is impossible to see a head nod in a fractious or excited horse that is held tightly. Use of a chain lead shank over the nose facilitates control but is resented by some horses, and use of a bridle with a lunge line attached may be preferable.[5]

Horses should move at a consistent speed, not too fast and not too slow. A lazy horse may need encouragement with a whip. Constantly changing speed can make assessment of lameness difficult, but occasionally, assessing a horse during deceleration may reveal useful information about the existence of subtle lameness.[5] A horse may have to be trotted up and down many times. It is sometimes useful for the examiner to lead the horse to assess subtle forelimb lameness, since gait abnormalities may become more obvious.

Surface Characteristics and Lameness Examination

The horse should be examined on a smooth, flat surface. I prefer a hard surface, such as pavement or concrete, that creates maximal concussion and may exacerbate subtle lameness. However, the clinical relevance of mild lameness seen on hard surfaces, especially on turns, should not be over-interpreted. Many horses that are actively competing successfully show mild lameness on hard surfaces; it is important to understand that the horse does not perform on a surface of pavement, and foot strike patterns and gait could be much improved if the horse performs on firm, but forgiving surfaces. Crushed rock, cobblestone, deep sand, or undulating grassy areas and potentially dangerous slippery surfaces should be avoided.

It is important that the surface is non-slip because some horses appear to lack confidence while moving on hard surfaces and alter the gait. In these situations, horses may shorten the stride for protection rather than from lameness.[5] Horses with studs or caulks on the shoes may develop induced lameness unrelated to the baseline lameness when trotting on hard surfaces.[5]

Ideally the gait on hard and soft surfaces should be compared to help differentiate soft tissue from bony problems. Horses with foot pain usually perform worse on a hard surface. Lameness from soft tissue injuries, such as suspensory desmitis or tendonitis, tends to be worse on soft or deep ground.

DETERMINATION, GRADING, AND CHARACTERIZATION OF LAMENESS

Six basic steps are necessary to determine, grade, and characterize lameness. The clinician should determine the following:

1. Primary or baseline lameness or lamenesses
2. Possibility of involvement of more than one limb and presence of compensatory lameness
3. Classification of lameness as a supporting, swinging, or mixed
4. Grading of lameness or lamenesses
5. Alteration of the cranial or caudal phase of the stride
6. Presence of abnormal limb flight

The *primary or baseline lameness* is the gait abnormality before flexion or manipulative tests are used. The practitioner attempts to abolish baseline lameness using analgesic techniques. Lameness in more than one limb may complicate determination of the worst affected limb. It is important to trot a horse even if it is quite lame at a walk, unless an incomplete or stress fracture is suspected. A horse may take a short step with a limb at walk, or can appear very lame, but trot reasonably sound. Horses with scratches (palmar/plantar pastern dermatitis) or superficial wounds in the palmar or plantar pastern may appear quite lame at walk but trot well. A STB pacer may walk extremely short both in front and behind but pace or trot without lameness. However, only the degree of lameness usually differs between a walk and trot. A horse may appear sound at walk and trot in hand, but lameness may be apparent trotting in a circle, in hand or on the lunge, or while being ridden. *This lameness now becomes the baseline lameness*, and under these conditions, the results of nerve blocks should be evaluated. The clinician should try to recognize if the horse has bilateral forelimb or hindlimb lameness, which is manifest as shortness of stride or poor hindlimb impulsion, or if concurrent forelimb and hindlimb lameness is present. Moderate to severe hindlimb lameness can mimic ipsilateral forelimb lameness, although ipsilateral forelimb and hindlimb lameness also occurs. In these horses the veterinarian should perform diagnostic analgesia in the hindlimb first.

Compensatory Lameness

Compensatory (secondary or complimentary) lameness results from overloading of the other limbs as a result of a primary lameness. It must be differentiated from the stride-to-stride compensation by a horse to avoid interference injury because of a gait deficit, or lameness, or to shift weight (load) during examination. A compensatory problem develops as the result of predictable compensation a horse may make *over time* for a primary lameness in a single limb. However, a horse may compensate for lameness in one limb by shortening the stride in another, a stride-to-stride change in gait that is not the result of lameness. A horse with severe LF lameness is reluctant to extend the LF and must shorten the cranial phase of the LH limb, creating what appears to be a hike in the LH. If the veterinarian looks only at the hindlimbs, LH lameness may be diagnosed. A trotter performing at speed with a LF lameness is likely to develop compensatory lameness in the RF or RH, but not in the LH. However, the horse may appear to be hiking (lame) in the LH to avoid interfering with the LF. Elimination of obvious unilateral forelimb lameness usually resolves an ipsilateral pelvic hike. Experimental results appear to contradict this clinical impression. In 6 of 10 horses with stance phase forelimb lameness, compensatory movements of horses created a false lameness in the *contralateral* hindlimb (see following text).[8]

It is often difficult to know which lameness came first, but it is important to understand how horses compensate for lameness and which limbs are at risk to develop compensatory problems. Compensatory problems range from obvious lameness to only mild palpable abnormalities that may still compromise performance. Several predictable patterns of compensatory lameness are possible; the most common is bilateral forelimb or hindlimb lameness. Horses with a specific lameness in one forelimb are at risk to develop the same condition in the opposite forelimb. This tendency may not always be compensation for the primary lameness but may be simultaneous injury or degeneration of bone or soft tissue of both limbs. Abnormal loading of forelimbs or hindlimbs, faulty

bilateral conformation, and the same shoeing or foot conditions all likely contribute to bilateral, simultaneous lameness. In horses with bilateral lameness, eliminating lameness in one limb usually results in contralateral limb lameness. Bilateral lameness may affect both limbs equally, resulting in a short, choppy gait. The horse may be lame in one limb while being circled in one direction and lame in the contralateral limb in the opposite direction.

Racehorses that gallop are most likely to develop compensatory lameness on the contralateral limb or the ipsilateral forelimb or hindlimb. A TB racehorse with a left metatarsophalangeal joint lameness is most likely to develop a similar problem in the RH but may also develop LF lameness. In a trotter the contralateral limb is most at risk, followed by the diagonal forelimb or hindlimb. If a trotter has a right carpal lameness, the left carpus should be examined carefully; compensatory lameness also may occur in the diagonal LH limb. In a pacer the ipsilateral forelimb or hindlimb should be considered after the contralateral limb. In a pacer with LH lameness the RH and LF are at risk.

The most common compensatory lameness is the same problem in the contralateral limb. However, suspensory desmitis is a common compensatory problem in both the contralateral and other limbs. In a TB racehorse or a jumper with LF lameness, RF suspensory desmitis is common. Primary RH lameness may result in suspensory desmitis in the RF. It is logical that soft tissue structures are particularly vulnerable to the effects of over-load. Superficial digital flexor tendonitis may develop secondary to a primary problem in the contralateral limb. In trotters a common pattern is primary carpal lameness and compensatory osteoarthritis of the medial femorotibial joint in the diagonal hindlimb, or vice versa.

Compensatory lameness also can develop in the same limb. In horses with front foot lameness the suspensory ligament (SL) often is sore, and some horses have suspensory desmitis. In horses with lameness abolished by palmar digital analgesia, most with navicular syndrome, scintigraphic examination revealed increased radiopharmaceutical uptake (IRU) in the proximal palmar aspect of the third metacarpal (McIII) bone in 30% of horses, indicating possible abnormal loading of the proximal SL (Fig. 7-1).[9] Complete resolution of lameness may not be achieved until high palmar analgesia is performed.

Horses with primary metatarsophalangeal joint lameness often have associated ipsilateral stifle pain.[10] Determination of the primary site of lameness may be difficult without use of diagnostic analgesia and observing that blocking one site abolishes the majority of lameness. This phenomenon may be most common in trotters, but I have recognized it in all types of sport horses.

Supporting, Swinging, and Mixed Lameness

Lameness has classically been divided into three categories in an attempt to characterize the motion associated with the lame leg and to assign etiology to the lameness condition. These categories are described and discussed, but I firmly believe that adequate characterization of most lameness conditions is impossible and may be unnecessary.

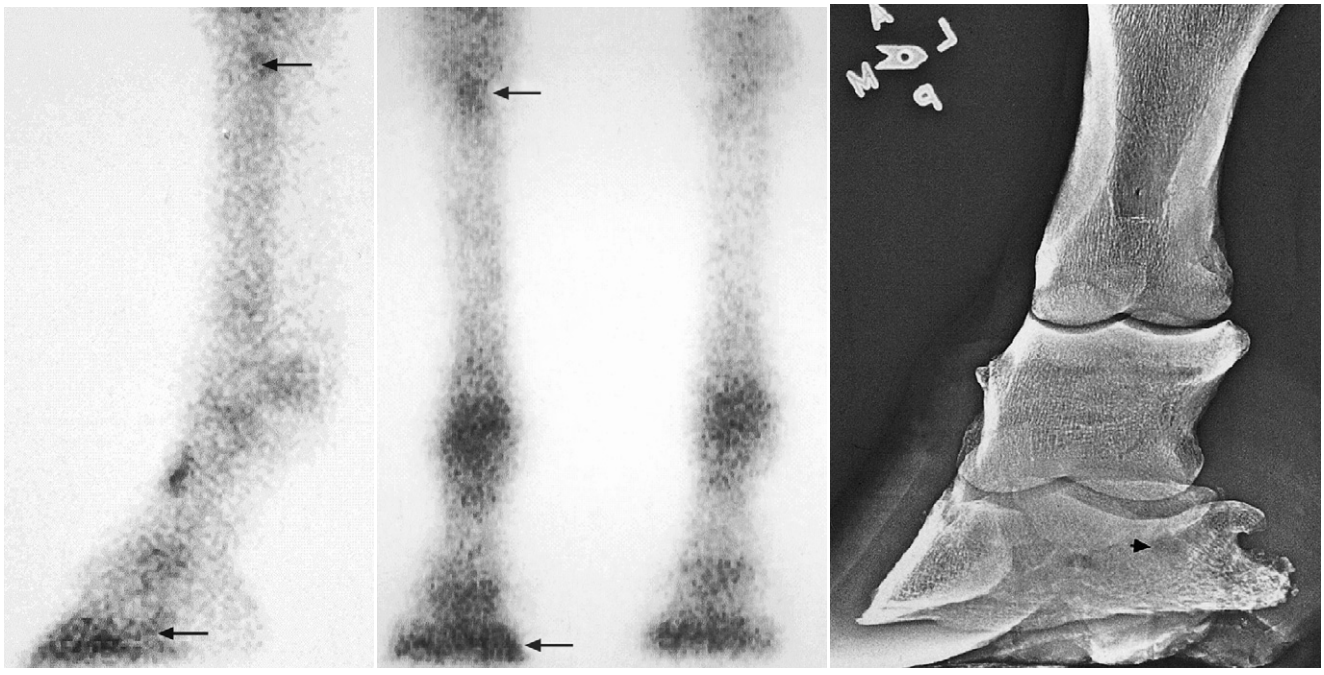

Fig. 7-1 **A,** Lateral delayed-phase scintigraphic view showing focal, mild increased radiopharmaceutical uptake (IRU) of the navicular bone *(bottom arrow)* and proximal aspect of the third metacarpal bone (McIII; *top arrow*). Normal modeling is seen in the dorsal aspect of the proximal phalanx. **B,** Dorsal delayed-phase scintigraphic image, and **C,** dorsomedial palmarolateral oblique xeroradiograph of a dressage horse with lameness abolished by palmar digital analgesia. IRU of the medial aspect of the distal phalanx *(bottom arrow)* corresponds to the area of subchondral radiolucency seen in the xeroradiographic projection (C, *arrowhead)*. Notice the focal area of mild IRU involving the proximal aspect of McIII *(top arrow, A* and *B)*. Abnormal loading of the suspensory ligament may occur as a compensatory problem in some horses with navicular syndrome or other sources of palmar heel pain.

Supporting limb lameness describes a lameness that is painful during the weight-bearing phase of the stride. Most lameness conditions are of this type. Supporting limb lameness also has been referred to as *stance phase lameness*, but this term is inappropriate because the swing phase of the stride is also altered.

Swinging limb lameness describes lameness that primarily affects the way the horse carries the lame limb. However, most horses with painful lameness conditions alter the swing phase of the stride in a typical and repeatable fashion, and it is difficult to make a clear separation between supporting and swinging limb lameness. Swinging limb lameness should be a term reserved for mechanical defects of gait, such as fibrotic myopathy, upward fixation of the patella, stringhalt, or other lameness conditions causing a mechanical restriction of gait. In these horses, lameness is manifested in the swing phase of the stride with no apparent pain. Unfortunately, the term swinging limb lameness often is used inappropriately to describe the gait deficit in horses with painful, supporting limb lameness. Lameness associated with osteochondrosis of the scapulohumeral joint is often described as a swinging limb lameness because of a marked shortened cranial phase of the stride. Dramatic improvement in the shortened cranial phase of the stride can be achieved by diagnostic analgesia, eliminating pain associated with lameness. Thus the gait deficit is the direct result of pain and no clear differentiation between supporting and swinging limb lameness can be made. Horses with painful forelimb lameness almost always shorten the cranial phase of the stride, although perhaps not to the extreme as in a horse with authentic scapulohumeral joint lameness. Because the terminology is confusing and often erroneous, I prefer to avoid use of these terms and simply describe lameness as accurately as possible. For instance, describing a horse as grade 2 of 5 LF lame, with a marked shortening of the cranial phase of the stride reminiscent of other horses I have seen with shoulder region lameness, gives the most accurate and useful information.

There is an erroneous tendency to equate a swinging limb lameness with one that is more evident when the lame limb is on the outside of a circle. Upper limb lameness is often presumed, yet not confirmed by diagnostic analgesia. It is logical that if a horse is reluctant to swing a limb forward, the lameness may be most prominent when the lame limb is on the outside of a circle. However, many horses with painful weight-bearing lameness show more pronounced lameness with the limb on the outside of the circle, a finding that neither suggests that lameness originates from the upper limb nor indicates the presence of swinging limb lameness (see following text). The outer limbs must stretch further and cover a larger circumference circle than the inside limbs. Slight temporal differences in the stance and swing phases of the inside and outside limbs are necessary to maintain gait symmetry.[5]

The results of cinematographic analysis of gait in lame horses seem to support reservation of the term swinging limb lameness for horses with authentic mechanical gait deficits, rather than those induced by painful lameness. In a horse with a supraglenoid tubercle fracture examined at a trot in hand, a marked decrease in the cranial phase of the stride (protraction) was observed, along with a marked head and neck nod. A markedly shortened stride could be equated with swinging leg lameness, but high-speed cinematography showed that the cranial and stance phases of the stride were shorter than in the sound limb.[11] A horse with unilateral semitendinosus fibrotic myopathy had a shortened stride length and a shortened cranial phase of the stride, but the stance phase did not differ from that of the unaffected contralateral limb.[12]

In my experience, most lameness conditions can be considered *mixed lameness*, with changes in gait during weight bearing or the stance phase and during the swing phase of the stride. With the exception of mechanical defects in gait, I have not been able to categorize the clinical characteristics of most lameness conditions into swinging or supporting limb types. However, it has been suggested that swinging limb lameness is caused by muscle injury, supporting limb lameness is caused by bone, tendon and ligament injury, and mixed lameness is caused by joint, tendon sheath and periosteal injury.[13] A shortened cranial phase of the stride is a common characteristic in forelimb and hindlimb lameness and should not be considered pathognomonic for the location or type of lameness.

DETERMINING THE LOCATION OF LAMENESS

The horse should be observed at both the walk and the trot from the front, behind, and side. I spend most of my time watching the horse move away and then back toward me. Medial-to-lateral limb flight and foot strike can be evaluated only from this perspective, although cranial and caudal aspects of the stride can be evaluated only from the side. Most important, evaluation of lameness from this perspective allows the veterinarian to use the horse as a frame of reference. I find it quite useful to evaluate forelimb lameness when the horse is traveling away from me and hindlimb lameness when the horse is traveling toward me. This perspective allows use of the horse's top line to see a subtle head and neck nod or pelvic hike. Only by observing the horse from the side can the cranial and caudal phases of the stride be determined. When first learning to assess lameness from the side, a linear frame of reference, such as a fence or wall in the background, may be helpful to notice head nod and pelvic hike against a non-moveable background. Application of pieces of tape or other markers to the horse's head or a fixed point on the pelvis can assist recognition of upward and downward movement of that body part.

Independent observation of the forelimbs and hindlimbs is needed to understand whether a horse has forelimb or hindlimb lameness or a combination. These observations then are amalgamated to form a final clinical impression.

Recognition of Forelimb Lameness

Forelimb lameness often is easier to recognize than hindlimb lameness. *Understanding the concept of the head nod is vital to the correct interpretation of equine lameness.* The head and neck elevate or rise when the lame forelimb is bearing weight or hits the ground and nod down or fall when the sound forelimb hits the ground. "…When the [forelimb] is the lame one, the movements of the foot and head occur somewhat in unison. When the lame foot is raised, the head is elevated, but only to fall when the sound leg is brought to a rest."[1] Some clinicians find it easier to appreciate the head nod, while others find it easier to recognize elevation of the head.

When evaluating slow-motion videotape of lame horses, it is immediately obvious that the elevation of the head and neck is much easier to see than the head nod down. In slow motion the horse appears to be elevating the head and neck just before the lame limb hits the ground, and then, during the later portion of the support or stance phase, the head and neck nod down. The head and neck nod occurs as the contralateral limb begins the support or stance phase. Both head elevation and falling are present, but head elevation is much easier to detect when it occurs in unison with the lame limb hitting the ground. It is likely that a combination of visual clues allows the clinician to decide the primary forelimb lameness. Quantification of lameness and description of the actions of the lame and compensatory limbs have been attempted using gait analysis systems. In horses with amphotericin-induced carpal lameness, head movements were the most consistent indicator of lameness, followed by sinusoidal

motion, or a rising and falling action, of the head and withers.[14] The motion of the lame limb was assessed, and a falling of the head and withers during the support phase of the lame limb was noted, contrary to clinical perception and evaluation of slow-motion videotape of lame horses. It was suggested that an uncoupling of the weight from the lame forelimb and a "free fall–like" phenomenon occurred during weight bearing.[14] The problem with this description is that it considers only the lame limb and is confusing. When evaluating a lame horse, the observer "sees" both forelimbs. During the later portion of the support phase of the lame limb, the sound limb is in the later portion of the swing phase and beginning the support or stance phase. Thus the head and withers drop described experimentally appears to occur concomitant with the sound limb hitting the ground. The observer perceives the early portion of the stance or support phase.

In general a good correlation between clinical evaluation of forelimb lameness and that described using motion analysis has been observed. There was complete agreement between clinical determination of location of forelimb lameness and that detected by motion analysis using a computerized three-dimensional motion measurement system. However, the degree of lameness differed in 6 of 29 horses.[15] The maximal vertical acceleration of the head was the best indicator of forelimb lameness.[16] Although horses with forelimb lameness shifted weight in a caudal direction to the diagonal hindlimb, the amount of withers motion was minimal. The authors reasoned that the tremendous mobility of the head and neck, allowing the horse to asymmetrically elevate the neck and thus load the non-lame forelimb, accounted for the lack of withers movement and the horse's adaptation to forelimb lameness.[16] A similar compensatory ability is not present in the hindlimb. Vertical displacement of the tuber coxae and forward motion or translation of the pelvis occur in horses with hindlimb lameness, since a mechanism such as head and neck movement does not exist.[16] In a computer-generated model of a trotting horse the dynamic effects of head and neck movement accounted for the majority of load shift to the contralateral forelimb and diagonal hindlimb in horses with unilateral forelimb lameness.[17] Load shift and compensation by the diagonal hindlimb in horses with unilateral forelimb lameness lends support to the clinical findings of compensatory lameness in the diagonal limbs in trotters.

Instrumented shoes have been used experimentally to study motion in horses by quantifying ground reaction forces (GRF) but have had limited clinical use.[18,19] Although this system is not currently widely available, in the future this or similar systems may be useful to objectively assess lameness and the response to diagnostic analgesic techniques in clinical patients.

Recognition of Hindlimb Lameness

Historically descriptions of hindlimb lameness have been confusing. An important principle in the recognition of hindlimb lameness is the *concept of the pelvic hike* or asymmetrical movement of the pelvis. This has also been termed *hip hike*, but I prefer the term *pelvic hike* because it accurately describes how the pelvis moves in a horse with unilateral hindlimb lameness. The entire pelvis, not just the lame side of the pelvis, appears to undergo elevation. Because the horse has two "hips" and only one pelvis, the term pelvic hike seems preferable. Pelvic hike is the vertical elevation of the pelvis when the lame limb is weight bearing. In other words, the pelvis "hikes" upward when the lame limb hits the ground and moves downward when the sound limb hits the ground. "… the haunch settles downward when the sound leg touches the ground…".[1] Some clinicians find it easier to see the downward movement of the pelvis, on the side of the lame limb, rather than the pelvic hike.[5] It may be simpler to determine which side has the most movement, rather than looking for either a hike or a drop.[5] The clinician must keep in mind that the pelvic hike is the clinical impression of *the change in height of the pelvis*, not the absolute or measured height. It is the shifting of weight or load that occurs as the horse tries to reduce weight bearing in the lame limb and transfer weight to the sound limb.

Another explanation for asymmetrical movement of the pelvis involves one of the protective or compensatory mechanisms used by the horse to assist in break over and minimize load on the lame limb. Many horses with hindlimb lameness drift away from the lame limb toward the sound limb. Drifting may decrease the magnitude of the observed pelvic hike, but more important, makes the lame side look lower than the sound side. This is why it is important to watch the entire pelvis as a unit rather than the individual sides of the "hips."

In most horses with hindlimb lameness, particularly those without a substantial tendency to drift away from the lame limb, the elevation of the pelvis (pelvic hike up) when the lame limb hits the ground surpasses that when the sound limb is weight bearing. This elevation can be seen readily in real-time and slow-motion videotape analysis, but it may not be as obvious during clinical examination. Observing horses with hindlimb lameness from the front as the horse trots toward you may be useful. This approach allows the pelvic hike to be seen clearly using the horse's top line as a frame of reference. Subtle pelvic elevation is best seen from this perspective. The use of markers on a fixed part of the pelvis can help to identify asymmetry. Stride length characteristics, height of foot flight, sound, and fetlock drop are also helpful (see following text).

Horses with bilateral hindlimb lameness may have a short, choppy gait that lacks impulsion, but they may have no pelvic hike. Other methods to exacerbate the baseline lameness should be performed, such as circling the horse at a trot in hand or while on a lunge line. Lameness may be accentuated when the lame or lamer limb is on the inside or outside of the circle (see following discussion).

Hindlimb Lameness Confused with Forelimb Lameness

It is important to understand how a horse with unilateral hindlimb lameness modifies its gait so that *hindlimb lameness can mimic forelimb lameness at the trot*. When the lame limb hits the ground, the horse shifts its weight cranially to transfer load away from the lame limb. This causes the head and neck to shift forward and nod down at the same time. The contralateral forelimb bears weight simultaneously with the lame hindlimb and the head nod coincides, thus mimicking lameness in the forelimb ipsilateral to the lame hindlimb. Head and neck movement in horses with hindlimb lameness is not always observed. Horses generally must have prominent (≥3 out of 5, see later grading discussion) hindlimb lameness before compensatory head and neck movement develops. At the pace, a lateral gait, LH lameness mimics RF lameness and RH lameness mimics LF lameness.

Horses can have a head and neck nod at the trot caused by singular forelimb lameness, singular ipsilateral hindlimb lameness, or concurrent forelimb and ipsilateral hindlimb lameness. A prominent head nod is seen in horses with simultaneous LF and LH lameness. The examiner first must determine whether both limbs are affected. Problems arise because a horse with only LF lameness may shorten the LH stride at the trot, leading the veterinarian to question whether LH lameness also exists. Horses with only LH lameness can have a rather pronounced head nod, and thus the veterinarian may question the existence of LF lameness. Although a horse with LF lameness may have a compensatory shortened stride of the LH, in the absence of lameness a marked pelvic hike should not be

present. A head nod consistent with a LF lameness may be inappropriately severe to be caused by mild LH lameness. If a horse has simultaneous LF and LH lameness, it is essential to perform diagnostic analgesia in the hindlimb first, because moderate to severe hindlimb lameness produces head and neck nod that is not abolished unless the hindlimb lameness is resolved. Resolution of the pelvic hike and reduction in the head nod should be expected with resolution of the hindlimb lameness.

Simultaneous lameness of a diagonal pair of limbs is less common than simultaneous ipsilateral lameness, except in trotters, because many horses perform at gaits that induce compensatory lameness either in the contralateral or ipsilateral limb. With simultaneous LH and RF lameness the head nod reflects the forelimb component, a mandatory clinical sign for perception of RF lameness. The horse may drift away from the LH with shortening of the cranial phase of the stride. The horse may have a short, choppy stride in the forelimbs and hindlimbs. The horse may have a rocking gait. It cannot shift weight or compensate from stride to stride in the usual manner and thus tends to rock back and forth from the hindlimbs to the forelimbs.

Reasonable agreement generally exists between clinical recognition of hindlimb lameness and that found experimentally. The use of markers placed on each tuber coxa of 13 horses with unilateral hindlimb lameness showed a consistent increase in vertical displacement of the pelvis during early weight bearing of the lame limb.[20] Although the rise and fall of the pelvis was readily apparent and occurred consistently with weight bearing of the lame and sound limbs, respectively, the absolute height of the pelvis on the lame side did not rise above that of the lame limb.[20] These findings are consistent with my clinical impressions. A head nod down when the diagonal forelimb was bearing weight further confirmed clinical observations that hindlimb lameness can mimic lameness of the ipsilateral forelimb.[21] In a kinematic study using a three dimensional optoelectronic locomotion system, hip acceleration quotient increased in horses with hindlimb lameness.[21] Vertical displacement corresponded to the pelvic hike up on the lame leg, with a simultaneous forward movement of the head and neck during the stance phase of the lame limb.[21]

GRF has been measured in normal horses and those with forelimb and hindlimb lameness.[22-25] GRF is reduced in the lame forelimb or hindlimb with compensation by the other limbs. In horses with unilateral forelimb lameness, decreased horizontal GRF in the lame limb is compensated by increased GRF in the contralateral forelimb and ipsilateral hindlimb.[26] Decreased vertical GRF in the lame limb is compensated by increased vertical GRF in the contralateral forelimb during the swing phase of the lame limb, and increased vertical GRF in both the ipsilateral and contralateral hindlimbs during the stance phase of the lame limb.[26] During unilateral hindlimb lameness the decreased GRF in the lame limb is compensated by increased GRF in the contralateral hindlimb and the contralateral and ipsilateral forelimbs.[26] These experimental data support the clinical impression that a lame horse adapts by shifting load to the contralateral limb or by shifting load in a caudal direction for forelimb lameness and in a cranial direction for hindlimb lameness.

A study using an optoelectronic motion measurement system and expert vision analysis high-speed video system for motion analysis confirmed that horses with hindlimb lameness show false lameness in the ipsilateral forelimb. However, contrary to my clinical observations, 6 of 10 horses with severe forelimb lameness showed "false" lameness of the diagonal (contralateral) hindlimb.[8] Review of videotape of lame horses reveals false lameness in the diagonal or ipsilateral hindlimb depending on several factors. Horses with pronounced forelimb lameness may look lame in the diagonal hindlimb. Horses with marked shortening of the cranial phase of the stride may appear lame in the ipsilateral hindlimb. Horses with forelimb lameness circled with the lame limb on the inside may look lame in the ipsilateral hindlimb. Thus analysis of lameness can be complex, and determination of the lame limbs may not become clear until diagnostic analgesia is performed.

Bilaterally Symmetrical Forelimb or Hindlimb Lameness

Bilateral lameness is a common cause of poor performance and may go unrecognized without additional movement, such as circling, lunging or riding. Horses with bilaterally symmetrical forelimb lameness may have a short, choppy gait when trotted in straight lines. Horses with hindlimb lameness may lack lift to the stride, have a subtle change of balance, or reduced hindlimb impulsion.[5] If bilaterally symmetrical lameness is suspected, the veterinarian should select one limb and begin diagnostic analgesia. Horses often show pronounced lameness in the contralateral limb when the source of pain is desensitized.

THE LAMENESS SCORE: QUANTIFICATION OF LAMENESS SEVERITY

I believe it is important to have a standardized lameness scoring system that allows the clinician to quantify lameness within and between horses. Ideally it should be consistent worldwide, but currently a scale from 0 to 5 generally is used in North America, and a scale from 0 to 10 is often used in Europe. Definitions vary within the grading systems. The system adopted by the American Association of Equine Practitioners (AAEP) provides a framework.[27]

- Grade 1 lameness is difficult to observe and not consistently apparent regardless of circumstances (such as weight carrying, circling, inclines, hard surfaces).
- Grade 2 lameness is difficult to observe at a walk or trotting a straight line but is consistently apparent under certain circumstances (such as weight carrying, circling, inclines, hard surfaces).
- Grade 3 lameness is consistently observable at a trot under all circumstances.
- Grade 4 lameness is obvious lameness with marked nodding, hitching, or shortened stride.
- Grade 5 lameness is characterized by minimal weight bearing in motion or at rest and the inability to move.

The AAEP system is potentially confusing because it grades lameness at both the walk and trot. It does not account for a horse that has a shortened stride at walk that trots sound. In my experience, many lame horses show consistently observable lameness at a trot and therefore would have to be scored at least 3, leaving only grades 3 and 4 for use for the majority of lame horses. Horses with bilateral lameness and a shortened stride but no obvious head nod or pelvic hike are difficult to score based on this system.[5] It does not permit grading under different circumstances, such as straight lines, circles on the soft in each direction, and circles on the hard.[5]

An alternative lameness scoring system is listed in Box 7-1. Lameness is only scored at a trot, and the grading system is used most often to describe lameness at a trot in hand. The system is useful for both forelimb and hindlimb lameness and is based on a range of 0 (sound) to 5 (non–weight-bearing). In this system a horse with unilateral hindlimb lameness of grade 3 or worse would have a head nod that mimics ipsilateral forelimb lameness. There is a practical difference between this scoring system and that put forth by the AAEP. A horse with lameness grade 1 in this modified scoring system would have a lameness grade between 2 or 3 in the AAEP system. The

Box • 7-1

Lameness Scoring

Lameness grades from 0 to 5 are based on observation of the horse at a trot in hand, in a straight line, on a firm or hard surface.

0 Sound
1 Mild lameness observed while the horse is trotted in a straight line. When the lame forelimb strikes, a subtle head nod is observed; when the lame hindlimb strikes, a subtle pelvic hike occurs. The head nod and pelvic hike may be inconsistent at times.
2 Obvious lameness is observed. The head nod and pelvic hike are seen consistently, and excursion is several centimeters.
3 Pronounced head nod and pelvic hike of several centimeters are noted. If the horse has unilateral singular hindlimb lameness, a head and neck nod is seen when the diagonal forelimb strikes the ground (mimicking ipsilateral forelimb lameness).
4 Severe lameness with extreme head nod and pelvic hike is present. The horse can still be trotted, however.
5 The horse does not bear weight on the limb. If trotted, the horse carries the limb. Horses that are non—weight bearing at the walk or while standing should not be trotted.

modified scoring system is more flexible and allows clear differentiation between most lameness conditions. However, it does not account for a bilaterally symmetrical gait abnormality and may be difficult to apply in a horse with lameness in more than one limb. Many horses evaluated for subtle lameness or poor performance have a score between 0 and 1 because consistent lameness is not observed. Use of half grades provides greater flexibility and supports adoption of a scoring system from 1 to 10, assuming 0 denotes soundness.

LAMENESS DETECTION

Fetlock Drop

Assessment of fetlock drop, or extension of the metacarpophalangeal and metatarsophalangeal joints, may be helpful in recognition of the lame limb. In general, *the fetlock joint of the sound limb drops farther when this limb is weight bearing than does the fetlock joint of the lame limb*, because the horse is attempting to spare the lame limb by increasing load in the sound limb. This may be easier to detect by video analysis than in a clinical situation and may be more recognizable at the walk than a trot. However, in some horses with moderate or severe unilateral suspensory desmitis or tendonitis, the fetlock drops markedly on the lame limb when the horse is walking, but at a trot fetlock drop usually is more pronounced in the sound limb. With bilateral suspensory desmitis or severe tendonitis the fetlock may drop further in the lamer limb.

Use of Sound

Sound can be useful in lameness evaluation. *A lame horse usually lands harder on the sound limb, resulting in a louder noise.* To appreciate this sound, the horse must be trotted on a firm or hard surface such as pavement or concrete. However, the sound a horse makes while landing depends greatly on symmetry of the front or hind feet, and the loss of one shoe, different shoe types, or disparity in foot size confounds interpretation. Listening for regularity of rhythm and sound of footfall are important, especially when evaluating the response of lame horses to diagnostic analgesia, particularly in horses with subtle lameness.[5]

Drifting

Horses with hindlimb lameness generally *drift away from the lame limb.* Drifting is one of the earliest adaptive responses of a horse with unilateral hindlimb lameness, allowing the horse to break over easier or reduce load bearing. Drifting may alleviate the need for extensive pelvic excursion (hike). It may make pelvic drop on the lame side more obvious. The horse may mask the lameness by reducing pelvic excursion. In some horses a pelvic hike is undetectable or subtle, but consistent drifting away from the lame side indicates the presence of hindlimb lameness. Many driven STBs with hindlimb lameness drift away from the lame limb, or are "on the shaft." Horses with LH lameness have a tendency to be on the right shaft and vice versa. Drifting away from the lame limb may be most evident when horses have pain from the tarsus distally, although some clinicians have different experiences.[5] Drifting may result in the horse moving on three tracks.

Horses with severe forelimb lameness also tend to drift away from the lame limb, but this tendency usually is less obvious than in horses with hindlimb lameness. Drifting is most common with carpal lameness when the horse tends to abduct the limb during the swing phase of the stride and appears to push-off with the limb, forcing the horse away from the lame side. Racehorses with either forelimb or hindlimb lameness tend to drift away from the lame limb while training or racing at speed. This finding is an important piece of the lameness anamnesis.

Drifting toward the lame hindlimb is an unusual but important clinical sign. In horses that drift toward the lame limb, I suspect weakness and lameness exist simultaneously, suggesting a neurological component to the gait abnormality. However, a jumping horse at takeoff may push off more strongly with the non-lame hindlimb and drift across the fence toward the lame limb.[5]

EVALUATION OF LIMB FLIGHT

Observation and characterization of limb flight can be useful in determining the lame limb or limbs and possibly the location of lameness within the limb. Abnormal limb flight also may predispose to lameness, especially in horses with faulty conformation. In my opinion, it is impossible to predict the site of lameness accurately based on limb flight and other characteristics, although some abnormalities lead to a high index of suspicion. I believe strongly that the location of pain should always be confirmed by diagnostic analgesic techniques whenever possible. Some abnormalities are consistently associated with specific lameness conditions, whereas others are general patterns of limb flight seen with many different conditions.

Cranial and Caudal Phases of the Stride

Changes in limb flight in the cranial and caudal phases of the stride can be seen only when the horse is evaluated from the side. In a normal horse the length of the stride of the paired forelimbs and hindlimbs, measured from hoof imprint to hoof imprint, is nearly identical from side to side. Extension and flexion of the limbs is also similar. From a clinical perspective the length of the stride of the affected limb cranial to the stance position of the contralateral limb is called the *cranial phase* of the stride, and the length of the stride caudal to the stance position of the contralateral limb is called the *caudal phase* of the stride. Obviously in a normal horse these individual parts of the

stride are symmetrical. In a lame horse the overall stride length does not appear to change. If stride length changed, the horse could not trot in a straight line. Drifting is associated with lameness and could be explained by a change in stride length, but shorter stride length would be expected in the lame limb, causing the horse to drift toward the lame side, in contrast to the usual observation, drifting away from the lame limb. In racehorses, some of the tendency to drift away or toward the inside of the track could be easily explained by mild differences in stride length. However, at a trot in hand we can assume that total stride length does not change.

In many lame horses the cranial phase of the stride of the affected limb is shortened. The caudal phase is lengthened to maintain a near equal overall stride length side to side. Shortening of the cranial phase of the stride appears to be a learned response of the horse to reduce the time spent during the stance phase and to help during break-over. Loss of propulsion, or an unwillingness to push off with the lame limb, could also explain reduction in the cranial phase of the stride. Because most lame horses have a shortened cranial phase of the stride, this finding is not particularly useful in localizing or classifying lameness and is not synonymous with swinging limb lameness. It is also important to recognize that pain causing lameness results in altered proprioceptive responses, to protect the painful area, and these responses may persist for some time after pain has resolved.[5] A classic example of attenuation of the cranial phase of the stride in the hindlimbs occurs mechanically in horses with fibrotic myopathy. This authentic swinging limb lameness causes a marked abrupt change in the later portion of the protraction phase of the affected hindlimb, shortening the cranial phase and causing a sudden downward and backward action of the limb.

The caudal phase of the stride is lengthened in most lame horses since, to maintain overall equal stride length, this portion of the stride must compensate. I generally have not found evaluation of the caudal phase of the stride at the trot in hand clinically useful, but it is sometimes a useful observation in horses at a walk (see following text). Some horses with severe palmar foot pain have a shortened caudal phase of the stride at both walk and trot.[5]

Contrast of the cranial and caudal phases of the stride in the lame limb at a walk and a trot is useful. In most horses with forelimb lameness the cranial phase of the stride is slightly shortened at the walk but markedly shortened at a trot. Obviously, in horses with subtle lameness, this clinical sign is absent at the walk and only mildly apparent at the trot. Horses with pain in the dorsal aspect of the foot, such as hoof abscessation or laminitis, may have a shortened caudal phase of the stride at a walk. This response is an attempt to protect the painful area and to shorten during break-over. These horses walk with a marked camped-out appearance in the forelimbs. At the trot, however, the cranial phase of the stride is likely to be shortened, a clinical contrast useful in localizing lameness to the dorsal aspect of the hoof.

Most horses with hindlimb lameness have a reduction in the cranial phase of the stride at the walk and the trot. Horses with pelvic fractures involving the acetabulum prefer to keep the lame limb ahead of the contralateral limb at the walk and have marked shortening of the caudal phase of the stride, but at the trot the horse has a shortened cranial phase of the stride. Horses with hoof abscessation, most commonly of the dorsal aspect of the hoof, walk similarly, only to trot with a pronounced shortening of the cranial phase of the stride. Unilateral or bilateral laminitis is rare in the hindlimbs and can cause similar clinical signs.

Shortening of the cranial phase of the stride does not always indicate that lameness is present in that limb. At speed a trotter with forelimb lameness shortens the cranial phase of the stride in the ipsilateral hindlimb to avoid interference with the lame limb. This observation sometimes is also made in horses being trotted in hand. This compensatory movement gives the impression that the horse may be lame in the ipsilateral hindlimb, with a subtle pelvic hike. Lameness of the foot and carpus in trotters most often causes this compensatory ipsilateral hindlimb pelvic hike. Once lameness is abolished in the ipsilateral forelimb, the pelvic hike and shortened cranial phase of the stride in the hindlimb abate. In trotters a shortened cranial phase of the stride and a pelvic hike may be related to faulty weight distribution and interference problems and not to lameness at all.

ABNORMALITIES OF LIMB FLIGHT

Abnormalities of limb flight can cause interference of one limb with another, particularly in trotters and pacers (Fig. 7-2). However, horses performing at speed at any gait and those with faulty conformation also are at risk to develop interference injuries. In some horses, interference is of no consequence, but in others, especially trotters, it causes gait deficits. Skin lacerations, bruising, and underlying bone and soft tissue damage (interference injury) may occur. Various boots and other protective devices have been developed to protect the limbs from potential trauma. It is important to assess the presence and location of interference injuries. In some horses, only mild evidence of hitting is found, but other horse may have many painful areas. Chronic interference can be the sole reason for lameness or poor performance.

Front Foot Interference

Front foot hitting the contralateral forelimb. Horses with toed-out conformation tend to wing-in during movement, predisposing to interference injuries. Horses with base-narrow conformation or those with a combination of base-narrow and toed-out conformation also are at risk. However, many horses with these conformational abnormalities do not interfere.

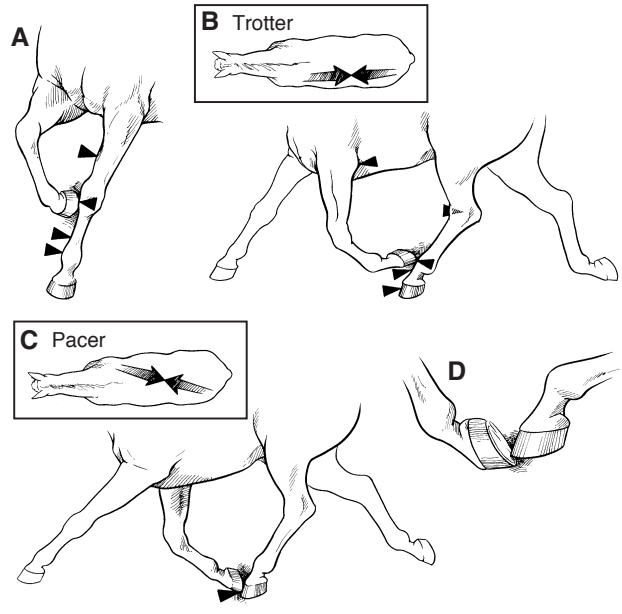

Fig. 7-2 Types of interference. **A,** Horses at any gait can be injured by interference of a forelimb with the opposite side. **B,** Interference is common in the trotter and usually involves the ipsilateral forelimb and hindlimb. Interference within a forelimb can be seen in horses that hit the elbow of the same limb. This usually occurs because of high action, excessive weight of the shoes, or a combination of these factors. **C,** Interference in the pacer can involve the forelimb and diagonal hindlimb, commonly called cross-firing. **D,** Forging occurs during trotting when the toe of the hind foot strikes the bottom of the ipsilateral front foot.

Some horses walk very closely but widen out at faster gaits. Interference injury from one hind foot hitting the medial side of the contralateral hindlimb occurs infrequently.

All types of horses, especially STB racehorses, are at risk of interference. Interference can involve any level of the limb from the foot to the proximal antebrachium. Mild interference of this type is called *brushing*. STBs often "hit their knees," which causes swelling, bruising, and lacerations of the skin on the distal medial aspect of the radius. In some horses, large, chronic swellings develop; these consist of mostly fibrous tissue. In others, osteitis of the distal radius or abscessation occurs. Even with protective gear, horses may be reluctant to perform at maximal speed to avoid injury or disruption of gait, or pain caused by interference induces the horse to go off-stride.

Interference within the same limb. Horses can develop interference injuries within a limb when the hoof or shoe hits the ipsilateral elbow. This type of interference sometimes is seen in trotters with high action (excessive carpal flexion) and is common in gaited horses that perform with heavy shoes intended to cause high action of the forelimbs (Fig. 7-2, *B*).

Front foot hitting the ipsilateral hindlimb. Interference in trotters usually involves the toe of a forelimb interfering (hitting) with the dorsal aspect of the ipsilateral hindlimb (see Fig. 7-1). Various names are given to the type of interference based on the location in which the injury occurs. Interference injury at the dorsal aspect of the hind foot or coronary band is called *scalping;* in the pastern region it is called *speedy cutting;* in the metatarsal region (shin) it is called *shin-hitting;* and in the dorsal or medial aspect of the tarsus, it is called *hock-hitting*. Because interference is common in trotters, it is important not to over-interpret signs of pain on palpation. Speedy cutting results in pain over the dorsal aspect of the proximal phalanx that should not be misinterpreted as pain associated with a mid-sagittal fracture.

Front foot hitting the contralateral (the diagonal) hindlimb. Cross-firing, or the striking of the contralateral (diagonal) hindlimb by the front foot, usually occurs only in pacers (Fig. 7-2, C).

Hind Foot Interference

Interference as a result of a hind foot hitting the foot of the ipsilateral forelimb *(forging)* is common and usually does not result in injury. Many horses forge at a trot in hand or while being ridden. In horses with shoes the sound is unavoidable but may not be a sign of a pathological condition. Forging is most common in horses that are trotted in deep footing. Forging may reflect imbalance, lack of strength, incoordination, or poor foot trimming.[5]

Forelimb: Common Abnormalities of Limb Flight

Winging In and Winging Out

Common limb flights observed during lameness examination include winging in and winging out movement of the front feet and are often related to conformation (see Fig. 4-12). Horses that are toed out tend to wing in, whereas those that are toed in tend to wing out. Such abnormalities do not necessarily compromise performance. However, such movement may result in uneven loading of the soft tissue structures and uneven hoof wear, leading to chronic imbalance. Horses that wing in tend to develop interference injuries and wear the medial aspect of the shoe excessively. Horses that wing out tend to develop lateral branch suspensory desmitis and wear the lateral aspect of the shoe excessively.

Lateral Placement of the Foot during Advancement (Abduction)

Horses normally advance the forelimbs straight ahead. When advancing the limb, horses with articular carpal pain, and in some with pain in the proximal metacarpal region, the foot is placed lateral to the expected foot position. This action has been described as *abduction* of the limb, but this term may infer swinging the limb, and horses with carpal lameness seem to place the limb laterally rather than swing the limb. However, ankylosis associated with severe osteoarthritis of the carpus does necessitate swinging the limb during advancement.

Horses with this abnormality of flight almost always have a shortened cranial phase of the stride. They tend to push off with the affected limb from the lateral location, resulting in a wide or peg leg type of motion at walk, and sometimes at trot. With bilateral carpal lameness the horse moves wide bilaterally. Not all horses with carpal pain move wide, and this gait change is seen more often in horses with middle carpal or carpometacarpal joint pain than in those with pain in the antebrachiocarpal joint. Some horses with upper limb lameness may carry the limb wide while walking, although this characteristic is not typical of horse with shoulder pain. A horse with a humeral stress fracture sometimes may travel wide, similar to a horse with carpal lameness, but the latter is more likely. A horse with pain in the lateral aspect of a foot may move wide in the affected limb to reduce load laterally. This characteristic is seen in STB and TB racehorses with subchondral bone trauma or early stress fractures of the distal phalanx in the LF.

Plaiting

The verb *to plait* means to braid or pleat, or to make something by braiding.[3] The term is used to describe horses that walk or trot by placing one foot directly ahead of the other foot. Plaiting in the forelimbs is not nearly as common as in the hindlimbs and usually is the result of base-narrow, toe-out conformation. Old horses (usually broodmares) with severe carpus osteoarthritis and carpus varus limb deformities occasionally may swing the limb laterally and place the foot far enough medially to end up in front of, or lateral to, the opposite foot. Some horses with shoulder region lameness guard the limb and travel very close in front. Plaiting in the forelimbs can be seen in horses with recent fractures of the thoracic dorsal spinous processes at the withers.[5] Horses with neurological disease occasionally plait.

Limb Flight in Horses with Shoulder Region Lameness

I include this section principally because the shoulder often is erroneously incriminated as the source of pain. Horses with moderate to severe lameness of the scapulohumeral joint or bicipital bursa have a marked shortening of the cranial phase of the stride. They also have an unusual motion of the shoulder joint that is difficult to describe. Since the cranial phase of the stride is shortened, during break-over the affected shoulder joint seems to drop or buckle forward, more so than the opposite side (assuming lameness is unilateral). There may be prominent lifting of the head and neck. Limb flight is either straight ahead or somewhat close to the opposite forelimb. However, racehorses with humeral stress fractures may travel wide in front. With mild lameness there are no typical gait characteristics.

Hindlimb: Common Abnormalities of Limb Flight

Stabbing or "Stabby" Hindlimb Gait

A common abnormality of limb flight seen in horses with hindlimb lameness is described as a *stabbing* or "*stabby" gait*. During protraction of the lame hindlimb or limbs the limb travels medially, close to the opposite hindlimb, and then moves laterally during the later portion of the swing phase and is placed lateral to the expected foot placement. This motion results in excessive wear of the lateral or dorsolateral aspects of the shoe. Although this gait often is seen in horses with distal hock joint pain, it can be seen with many other sites of pain from the distal tibia to the foot. Therefore diagnostic analgesia is required to localize the pain. However, horses with the most marked shoe wear consistent with this abnormality of limb flight are most likely to have tarsal

lameness. Exaggerated stabbing hindlimb motion often is seen in horses with neurological disease.

Abduction of the Hindlimbs during Advancement

In some horses with hindlimb lameness the limb is carried forward in a position lateral to the expected position (i.e., abducted). In some horses with this limb flight the limb swings outside the expected line of limb flight, only to strike the ground near the expected position. Lateral swinging of the limb begins immediately after the lame limb leaves the ground. I have observed this abnormality most consistently in horses with stifle lameness, but it also occurs with some other upper limb lameness conditions. Care must be taken when evaluating horses that normally travel wide behind, such as trotters.

I have recognized lateral swinging of the hindlimb most commonly in pacers with articular lesions of the stifle, because the normal gait in these horses is to swing the hindlimb more than would be expected from other horses. Many horses with stifle lameness carry the limb forward lateral to the expected position, but just before impact may actually stab laterally. Therefore the veterinarian must pay close attention to limb flight directly after the lame limb leaves the ground and while it is passing the contralateral limb. Another common characteristic of horses with stifle lameness is a shortened cranial phase of the stride. The stifle joint also may appear unusually prominent and be carried somewhat away from the flank and slightly externally rotated.

Plaiting

Plaiting is more common in the hindlimb than in the forelimb and usually results from lameness rather than faulty conformation, although plaiting can occur in a horse with severe base-narrow conformation. Plaiting can be seen in horses with unilateral or bilateral lameness. In horses with unilateral lameness, it appears that limb flight actually may be altered in both hindlimbs, resulting in both hind feet being placed ahead, or in some horses, lateral to the opposite foot. In horses with severe hindlimb lameness, it appears that the affected foot is being swung around and placed directly in front or lateral to the unaffected foot. Alternatively, the horse may be trying to support most of its weight on the unaffected limb and moves this limb inside to support the lame side. In horses with bilateral lameness, it is equally difficult to determine what exactly is causing the plaiting. The horse may be reluctant to bring either hindlimb along the expected line of flight, leaving the limb medially and forcing the opposite limb to the outside to avoid interference. A horse may swing each hindlimb around the other, ultimately ending placing one foot ahead or lateral to the other. An unusual rocking-type of gait is observed in horses with bilateral hindlimb lameness and plaiting. I have observed plaiting most commonly in horses with osteoarthritis of the coxofemoral joint or pelvic fractures, but I also have seen it in horses with bilateral distal hock joint pain or suspensory desmitis. Plaiting also is observed in some horses with sacroiliac joint pain.[5]

Mechanical Lameness of the Hindlimb and Limb Flight

Mechanical conditions of the hindlimb can cause profound abnormalities of limb flight. These are termed lameness conditions because of the gait abnormality exhibited, although in many horses pain is not characteristic.

Stringhalt

Stringhalt, an ill-defined neuromuscular disorder of the hindlimb, causes mild-to-severe hyperflexion of the tarsus. The condition can be unilateral or bilateral and usually is most obvious at a walk but can also be seen at the trot. In horses with severe stringhalt the dorsal aspect of the hoof comes close to or hits the ventral aspect of the abdomen. Horses may exhibit the clinical signs more prominently during backing or when initially moved after previous standing.

Fibrotic Myopathy

Fibrotic myopathy is characterized by a sudden downward and backward motion of the limb (slapping motion) that occurs during, and restricts the length of, the cranial phase of the stride. Hyperflexion of the hock is not a clinical feature of this gait deficit, but the restriction of the cranial phase of the stride and the slapping motion and sound can be confused with the clinical signs of stringhalt. It is most obvious at the walk.

Upward Fixation of the Patella

Upward fixation of the patella is a classic hindlimb gait deficit and one that displays the function of the stay or reciprocal apparatus. It can be intermittent or permanent and unilateral or bilateral. When the patella is locked in position over the medial trochlear ridge of the femur, the stifle and hock joints are held in extension, whereas the digit is held in partial flexion.

Shivers

Shivers is an ill-defined neuromuscular disease and is most common in Warmbloods and Draft breeds; it can occur unilaterally or bilaterally. Clinical signs usually are most obvious when a horse is backed or first moves from the stall. Horses elevate and abduct the limb, and the limb may actually shiver or shake. The tail often is elevated. Signs may be accentuated if the horse is tense.

Other Hindlimb Gait Deficits

Other unusual unexplained gait deficits affecting one or both hindlimbs are observed occasionally, and they often have characteristics similar to those seen with stringhalt, fibrotic myopathy, upward fixation of the patella, and shivers. However, some distinction usually prevents easy recognition and diagnosis. A gait deficit characterized by marked hindlimb abduction seen most prominently at the walk has been recognized. This is most similar to fibrotic myopathy, since a consistent abduction of the limb is observed, and signs tend to abate when the horse is trotted. It may be related to scarring, abnormal function of the biceps femoris and gluteal muscles, or neurological disease.

Neurological disease can cause many different gait deficits, most commonly recognized in the hindlimbs but also in forelimbs. A complete neurological evaluation usually is not performed during lameness examination unless certain abnormalities are observed. Abnormal or excessive circumduction of the hindlimbs, a bouncy, stabby hindlimb gait noticed when the horse is trotted, knuckling over behind or crouching, stumbling, and lethargy are signs that should prompt further investigation.

EVALUATION OF FOOT PLACEMENT

It is important to critically evaluate foot placement. Ideally, both the front and hind feet should land flat and level on a firm surface. Foot strike patterns change on soft footing. Evaluation of foot strike is most important in horses with lameness localized to the foot, but it can also give clues to other causes of lameness. Abnormal foot placement can be the result of a current lameness problem but may also cause lameness.

In the forelimbs, horses commonly land on the lateral side of the foot first before rocking medially. This can be the result of abnormal conformation or hoof imbalance and can predispose to lameness in the digit and suspensory branch desmitis. Landing abnormalities in the dorsal-to-palmar direction are common but are difficult to recognize unless severe. Horses with profound pain in the toe caused by laminitis or hoof abscessation land heel first, giving a camped-out appearance, and have a shortened caudal phase of the stride. Horses with palmar heel pain may compensate by landing toe first and cause abnormal stress on the dorsal structures of the foot, but this characteristic is difficult to see, except in slow motion.

In the hindlimbs, several patterns or abnormal landing or motion are recognized; not all of which are a cause or the result of lameness. Landing on the toe is commonly considered the result of heel pain, but many horses with severe hindlimb lameness land on the toe. This tendency is particularly prominent when the horse is first moved, and most horses warm out of the lameness. Horses may land on the toe when walking up an incline or at the walk on the flat, but generally place the heel on the ground when trotted. The most consistent lameness I see in horses that land on the toe is distal hock joint pain, but any cause of lameness from the tarsus to the foot can cause a horse to exhibit this abnormal landing pattern. Horses with lameness of the metatarsophalangeal joint region, including osteoarthritis, tenosynovitis of the digital flexor tendon sheath, or desmitis of the accessory ligament of the deep digital flexor tendon, have a tendency to land on the toe. Horses with adhesions within the digital flexor tendon sheath may have severe mechanical restriction that causes toe-first landing. Old Western performance horses with deep digital flexor tendonitis have severe toe-first landing and may stand on the toe and even rise up in the heels during the examination. The condition can be bilateral or unilateral and is difficult to manage. Mild or moderate tendency to land on the toe also has been attributed to stifle lameness.[5]

Abnormal movement of the lower or entire hindlimb occasionally is noted when horses are watched from behind. Horses may place one or both hind feet in an axial position and collapse or break-over the lateral aspect of the fetlock region.[5] Another uncommon hindlimb motion is characterized by excessive rotation of the hindlimb. The horse plants the hind foot and rotates the heel laterally, causing abnormal loading or twisting of the distal limb.[5] Although this movement can lead to lameness, it sometimes is seen in horses that are successful in various sporting endeavors. I have seen this type of hindlimb motion most often in TB racehorses.

ADDITIONAL MOVEMENT DURING LAMENESS EXAMINATION

Further information about the character of lameness often can be obtained by observing the horse as it move in circles and is ridden or driven. Some lameness conditions are apparent only under these circumstances.

Hard and Soft Surfaces

Comparison of movement on hard and soft surfaces is valuable. Foot lameness usually is worse when the horse is trotted on hard surfaces and better on a soft surface, such as grass or sand. Horses with suspensory desmitis or flexor tendonitis are more likely to show lameness on a soft surface. Deep sand may accentuate some lameness conditions, but an extended lameness examination under these conditions could cause proximal suspensory desmitis.[5] A slight downward incline or an uneven, rough surface may make subtle lameness more apparent.[5]

Circling

Lameness often is much more pronounced when a horse is circled. Horses should be circled in both directions: to the left (counterclockwise, LF and LH on the inside) and to the right (clockwise, RF and RH on the inside). Lameness may be more pronounced when circling at either the walk or the trot. In some horses with incomplete fractures, baseline lameness at a trot in straight lines in hand may be subtle or absent. Lameness may be readily apparent during circling, even at the walk. The additional forces of torsion and bending during circling are added to those of compression and tension. In horses with incomplete fractures, such as those involving the proximal or distal phalanges, torsion or bending forces during circling likely cause mild separation of the fracture fragments and exacerbate lameness. In horses with other lameness conditions, exacerbation of lameness may be caused by a change of load on the affected soft tissue structure or bone, redistribution of the forces of compression and tension in a medial to lateral direction, or additional forces of bending and torsion.

From a clinical perspective the force of compression may be dominant to other forces in determining a horse's response to circling, but it is not the only factor. Extension of the limb is also influential.[5] When the lesion is on the outside of the circle and undergoing compression, exacerbation of the lameness occurs. For instance, lameness in horses with medially located lesions of the distal phalanx or of the third carpal bone is worse when the limb is on the outside of the circle and the lesion is being compressed. For some soft tissue injuries, tension forces may be more important. Lameness in horses with proximal suspensory desmitis often is worse with the limb on the outside of the circle, suggesting that tension is important in the expression of lameness. The same observation is not seen in horses with more distally located suspensory desmitis.

Is lameness seen when in the horse while circling the same lameness seen while walking or trotting in straight lines? In most instances, circling exacerbates the primary lameness seen in straight lines. If lameness is subtle or non-existent when the horse is evaluated in a straight line, the lameness seen when circling becomes the baseline lameness. The clinician must recreate the same conditions of circling when evaluating the results of diagnostic analgesic techniques. There is always the possibility that lameness seen while circling may be different from the baseline lameness seen in straight lines. A horse may have a grade 1 RF baseline lameness when it moves in straight lines that increases to 3 when trotted in a circle to the left, but still has grade 1 lameness when trotted to the right. Lameness in straight lines and when trotting to the right is absent after palmar digital analgesia, but still is rated 3 when the limb is on the outside of the circle. The horse has two problems in the RF: palmar foot pain and an additional carpal lameness that becomes evident when the horse is trotted to the left (RF on the outside of the circle). With bilateral forelimb or hindlimb lameness, primary lameness often is seen in a single limb in straight lines, but while circling lameness is seen in whichever limb is on the inside (or outside) of the circle. Circling is useful in exacerbating the primary lameness problem and identifying an additional lameness not previously noted. This additional lameness must be recognized and treated separately from the baseline lameness.

Good correlation usually exists between the cause of lameness seen on the straight and that seen while circling the horse; thus it is helpful to circle the horse to try to exacerbate lameness. Circling can be done at the walk and trot in hand and while lunging or riding the horse. Horses often move more freely and naturally when lunged rather than when being led.[5] However, lunging is not possible in some horses, particularly racehorses, and circling while being led is better than no circling. The surface should be non-slip because horses may be hesitant to move freely on slippery surfaces and shorten or alter the stride even when lameness is not present.[5] Soft footing is best when the horse is first being lunged, since the horse can buck and play without risk of injury. Hard or firm surfaces are best to exacerbate many lameness conditions, but the surface must be non-slip to avoid possible injury.

That lameness of the upper forelimb or hindlimb is worse when the limb is on the outside of the circle is a common misconception. This is true in some horses, but a generalization cannot be made (see following text). Shortening of the cranial phase of the stride may appear more obvious with the limb on

the outside of the circle, but exacerbation of lameness judged by the degree of head nod may not be observed. Another misconception is that horses with lameness of the foot are lamest when the limb is on the inside of the circle. Although a majority (65%) of those with lameness localized to the foot are most lame when the limb is on the inside of a circle, lameness can be worst with the lame limb on the outside of a circle. This depends in part on the location of pain within the foot.

Forelimb

Lameness worsened with limb on the inside of the circle. In many horses, lameness originating from the fetlock region to the foot is worse with the affected limb on the inside of the circle. Comparison of circling on hard and soft surfaces is useful. Baseline lameness associated with foot pain usually is dramatically increased when the horse is circling on a hard surface, but a less obvious response is seen when circling on softer surfaces. However, lameness in horses with medially located lesions can be worse with the limb on the outside of the circle.

Horses with lameness of the metacarpal region vary in response to circling. Lameness in those with lameness related to metacarpal bony injury and distally located lesions in the suspensory ligament or digital flexor tendons tend to be worse with the limb on the inside of the circle, whereas lameness in those with proximal suspensory desmitis is worse with the limb on the outside. Horses with suspensory branch desmitis may show a different response depending on lesion severity and whether the injured branch is undergoing tension or compression.

In my experience, in horses with lameness of the forearm, elbow joint, arm, and shoulder joint region, the lameness tends to be worse with the limb on the inside of the circle, but opinions and experiences do vary.[5] Lameness in some horses with mechanical restriction of movement that dramatically decreases the cranial phase of the stride may be worse with the limb on the outside of the circle.

Lameness worsened with limb on the outside of the circle. Horses with medially based lesions of the lower limb, especially foot lameness, proximal metacarpal lesions (proximal suspensory desmitis or avulsion injury to McIII at the suspensory origin), or carpal pain often are more lame with the limb on the outside of the circle. Horses with lesions in the antebrachiocarpal joint are less consistent in response to circling compared with those with middle carpal joint lesions, since most of the common injuries involve the medial aspect of the latter. Upper limb lameness is accentuated in some horses.[5]

Lameness Improved when Circling

Lameness that appears better on a circle than in straight lines is uncommon. Lameness in horses with medially located lesions in the foot or carpus may improve when the limb is on the inside of a circle. Baseline lameness in horses with middle carpal disease involving the third and radial carpal bones improves with the limb on the inside of the circle. A STB racehorse with grade 2 or 3 RF baseline lameness that increases to grade 3 or 4 when circled to the left, but is only grade 1 or 2 when circled to the right, may have lameness associated with the middle carpal joint, but pain in the medial aspect of the foot also is possible.

A horse with bilateral forelimb lameness (e.g., a horse with grade 3 RF baseline lameness in straight lines) may show grade 3 to 4 RF lameness when trotting to the right, but grade 1 LF lameness when trotting to the left. The primary lameness is in the RF, and lameness is worse when the limb is on the inside of the circle. Circling to the left induced lameness in the LF, masking the RF lameness, because bilateral lameness existed that was not recognized when the horse was trotting in a straight line.

Hindlimb Lameness and Circling

In my experience, baseline lameness in most horses with any hindlimb lameness is worse when the limb is on the inside of the circle. Exceptions do exist, and some have different experiences and thus opinions.[5] Lameness associated with proximal suspensory desmitis often is worse with the affected limb on the outside of the circle, and the horse may stumble or take bad steps. Lameness in some horses with stifle lameness appears worse with the affected limb on the outside of a circle, but in others it appears similar to the left and the right. Many conditions of the stifle involve the medial femorotibial joint, a location that would be compressed with the limb on the outside. Lameness in any horse with a medially located lesion involving the distal hindlimb could be worse with the limb on the outside of the circle. Circling may be useful in exacerbating a primary lameness but generally is not helpful in localizing lameness.

Observation during Riding

Lameness may not be apparent when the horse is evaluated in hand in straight lines and circles but is obvious when the horse is ridden. This lameness becomes the baseline lameness for further investigation. The additional weight of a rider can exacerbate both forelimb and hindlimb lameness. Lameness in horses with primary back pain or those with substantial muscle pain secondary to hindlimb lameness usually is worse when they are ridden. Hindlimb gait restriction can occur in horses with back pain but may be apparent only when a horse is ridden.[5] Problems related to an ill-fitting saddle or girth, behavioral problems, head shaking, abnormal posture or carriage of the head and neck, and refusal to take a lead or bend in certain directions may be evident only when a horse is ridden.

A horse may be easier to control when ridden than when in hand or on the lunge. Performance of specific maneuvers by the horse may accentuate lameness. A collected trot that forces more weight onto the hindlimbs may exacerbate hindlimb lameness. An extended trot may reveal the horse's inability to extend on limb compared with another and reveal lameness that was completely imperceptible under all other circumstances. The primary complaint, such as poor-quality flying changes of lead at canter, may require a riding assessment regardless of whether baseline lameness is evident under other circumstances.

However, it is important for the veterinarian to separate his or her observations from those perceived by the rider. Identification of the lame limb may be difficult for a rider, although he or she may have a very strong opinion. If the veterinarian's observations differ, the rider may be difficult to convince. It is also essential to recognize that bad riding can actually induce a false lameness that is completely unapparent if the horse is ridden well. Nonetheless, an experienced rider, trainer, or driver can be quite helpful in assessing the horse's response to diagnostic analgesia or therapy, particularly in horses with thoracolumbar pain, subtle hindlimb lameness manifested only when ridden, and poor performance related to a musculoskeletal problem. Working regularly with a skilled, experienced, and reliable rider can be very helpful.

Subtle differences in weight distribution of the rider may exacerbate or mask the presence of forelimb and especially hindlimb lameness. When the horse is performing the posting (rising) trot, lameness may be more or less prominent depending on which diagonal the rider is using. In the rising trot the rider sits on either the left or right diagonal. On the left diagonal the rider is sitting when the LF and RH are bearing weight and rising during the swing phase of these limbs. On the right diagonal the rider is sitting when the RF and LH are bearing weight. The correct diagonal is the outside diagonal (i.e., left diagonal on the right [clockwise] rein). Hindlimb lameness often is worse when the rider sits on the diagonal of the lame limb.[5] Therefore if the horse is lame in the RH, the lameness appears and feel worse when the rider sits on the left diagonal.

Horses with hindlimb lameness may try to force or throw the rider to sit on the more comfortable (for both horse and rider) diagonal.[5] A horse with bilateral hindlimb lameness may appear lame in the RH when the rider sits on the left diagonal and lame in the LH when the rider sits on the right diagonal.[5] Forelimb lameness is influenced less by the diagonal on which the rider sits, but a similar pattern exists. RF lameness is worse with the rider sitting on the right diagonal.[5] This difference in lameness expression may be perceptible only by an experienced rider.

Observation of Inclines

Walking or trotting a horse uphill or downhill may exacerbate lameness or identify previously unapparent lameness. Lameness in horses with suspensory desmitis may be worse when they walk uphill or downhill. Lameness associated with palmar heel pain may be worse when the horse walks downhill and the horse may show a tendency to stumble. Horses that tend to stumble or knuckle behind while walking downhill may have loose stifles (inability to maintain the position of the patella, usually caused by lack of muscle tone). Horses with neurological disease usually show more pronounced clinical signs when walking uphill or downhill.

A superficially flat, hard surface may actually slope; this can influence lameness because the horse's feet will be tilted with one side lower than the other. Thus the horse may appear different when trotting away from the observer than when returning.

EVALUATION OF LAMENESS WITH A TREADMILL OR GAIT ANALYSIS

The use of a treadmill for poor performance evaluation is well recognized, and its use in lameness assessment is discussed in detail in Chapter 101. The clinical relevance of lameness apparent only on a treadmill is open to debate. I do not find lameness examinations on a treadmill at high speed particularly useful unless slow-motion videotape is available. I prefer to assess the horse while training or performing. A horse may modify its normal gait on a treadmill. Good correlation was demonstrated between gait regularity in horses exercised on a track and a treadmill, but treadmill strides and steps were shorter, and the swing phase of the stride was reduced.[28] Horses require at least two training sessions on a treadmill before the gait becomes consistent.[29] Stride characteristics of horses galloping on a treadmill change as the slope of the treadmill increases from 0% to 8%; horses reduced the suspension phase to maintain overall stride length.[30]

Gait analysis is discussed in Chapter 22. To date, assessment by an experienced, skilled observer has been more reliable in the identification of the lame limb or limbs than other more sophisticated methods of gait analysis.

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CHAPTER • 8

Manipulation

Mike W. Ross

Flexion or other manipulative tests often are used to induce or exacerbate lameness during lameness or pre-purchase examinations.

INDUCED AND BASELINE LAMENESS

It is important to understand the concept of *induced lameness* and the possible difference between lameness seen at this stage and baseline lameness. During lameness examination, baseline lameness is established before any form of manipulation is performed. This may be difficult if more than one limb is involved, if lameness is subtle or sub-clinical, or if lameness is bilaterally symmetrical, which causes a gait abnormality without overt lameness. Lameness provocation is performed to exacerbate the baseline lameness or to provoke a hidden gait abnormality and attempt to localize the source of pain within a limb or limbs.

Provocative tests create induced lameness that may not have any clinical relevance to the baseline lameness observed during initial movement. These tests are not sensitive or specific, and many times result in false-positive and false-negative findings. Many horses with palmar heel pain respond positively to the "fetlock flexion test" (or lower limb flexion test, see following text) and could erroneously be thought to have lameness of the metacarpophalangeal joint. A racehorse with baseline lameness as the result of a carpal chip fracture may have pre-existing low-grade osteoarthritis of the metacarpophalangeal joint and respond positively to lower limb flexion, but response to carpal flexion may be equivocal. Diagnostic analgesia is essential to localize the source or sources of pain. In the hindlimb, hock flexion, the so-called spavin test, causes many false-positive reactions.

FLEXION TESTS

Flexion tests were first described early in the twentieth century, but information regarding the degree of flexion, force, or duration of the tests was lacking.[1] Variations in technique persist and produce variable responses that can be misleading. There appear to be more false-positive reactions to flexion than there are false negatives, but the latter do occur. Flexion tests are useful during pre-purchase examinations because the horses being examined usually are relatively sound, and the tests are useful at uncovering hidden sources of pain. Flexion tests may be useful in exacerbating lameness, particularly when the primary or baseline lameness is in the region being flexed, but sensitivity is doubtful. Horses judged to be clinically sound underwent a "normal" and then a "firm" lower limb flexion test (fetlock flexion).[2] Of the 50 horses tested, 20 had a positive response to normal flexion, and 10 of these horses were judged to be lame while trotting for about 15 m (50 ft) or more. Forty-nine of 50 horses had a positive response to firm flexion, and 35 of these remained lame for a minimum of 15 m. In this study the force applied was not calibrated, 7 of the 50 horses developed lameness within 60 days of completion of the study, and 24 horses had radiographic abnormalities that could have contributed to a positive response to flexion.[2] Although there may be an explanation for a positive response in some horses, the high percentage of positive results in the study is in agreement with my clinical impression. In a study using the Flextest (Krypton Electronic Engineering NV, Leuven, Belgium), an apparatus designed to control traction force and time during a lower limb flexion test, the optimal force and time for flexion were 100 N and 1 minute, respectively. There was a positive response to flexion in many horses that were considered sound, and a positive response in sound horses was more likely in those in active work than in horses that had been rested or turned out on pasture. Horses were more likely to manifest a positive response to flexion as the force used in the test was increased.[3] A false-positive response to flexion can be observed in clinically normal horses and in those with unimportant low-grade problems. Lameness induced by flexion in these horses may have little clinical relevance.

However, other evidence suggests that a positive response to lower limb flexion in sound horses may be useful to predict future lameness. In a retrospective study, 151 initially sound horses were followed for 6 months. Twenty-one percent of horses with a positive forelimb flexion test result developed lameness in the area being flexed, whereas only 5% of horses with a negative flexion test result subsequently developed lameness. In young Swedish Warmbloods there was a positive correlation between a positive response to flexion and a subsequent insurance claim related to lameness.[4]

Flexion tests lack specificity because it is nearly impossible to flex a single joint without flexing other joints or nearby tissues, particularly in any hindlimb or distal forelimb flexion tests. Elevation of a limb without flexion in severely lame horses may exacerbate the baseline lameness, since horses guard the limb or need to warm-out of the lameness for a number of steps while trotting, thus complicating interpretation. Hindlimb flexion tests are less specific than forelimb tests because the reciprocal apparatus prevents flexion of any joint without concomitant flexion of other joints. Hindlimb flexion tests are useful in exacerbating baseline lameness, but positive responses to individual lower limb and upper limb tests, in my opinion, only localize lameness to the entire hindlimb. I believe that flexion tests are useful in exacerbating lameness, and in some horses it is the baseline or relevant lameness that is being worsened. In general, unless the horse's response is clearly pronounced and different from that of other manipulation, lameness cannot be localized based on response to flexion alone. Diagnostic analgesia should always be used, when possible, to localize pain.

Order, Duration, Force, and Venue during Flexion Tests

Consistency in technique is essential. Although force exerted by individuals varies, the flexion technique of experienced practitioners is sufficient to objectively assess response to

flexion.[5] Response to flexion can and should be compared with the contralateral limb. Ideally the flexion test should be performed in the contralateral sound limb first, before performing the test in the suspect limb, to determine the horse's response. Accurate assessment of response to flexion in the contralateral non-lame limb may not be possible if the horse is severely lame after flexion of the lame limb and lameness persists. In some instances, baseline lameness is actually increased by forcing the horse to stand for the contralateral flexion test, a useful observation seen most commonly in horses with forelimb lameness (see following text).

Duration of flexion is somewhat controversial and may be an individual choice. In a study evaluating lower limb flexion, duration of 1 minute was considered ideal, because normal horses that underwent flexion at 100 N for 1 minute had few false-positive responses.[3] Maintaining firm flexion for 1 minute while performing all flexion tests and repeating the tests in the contralateral limbs can make this portion of the lameness examination time-consuming. On the other hand, if the clinician takes the time to perform these tests, optimum chances of success are desired. A false-positive result is more useful than a false-negative test result. Some clinicians prefer to perform flexion tests with more force but for a shorter duration. This technique works well for lower limb flexion tests. Seldom is it possible to maintain some upper limb manipulative procedures for 1 minute. Thus some latitude is necessary. I believe that duration of flexion of 45 seconds to 1 minute is enough to elicit an accurate response in most horses.

Force used during flexion varies considerably, but excessive force induces lameness in most normal horses. Forces in the range of 100 to 150 N represent a moderate degree of force for lower limb flexion tests. In studies using a dynamometer, the maximum amount of force that could be used without a consistent withdrawal response in normal horses was 150 N.[6] The amount of force also depends on the size of the horse or the joint being flexed. The amount of force used in adult horses cannot be used in foals. Horses with obvious osteoarthritis or articular fracture, or those with substantial soft tissue injury likely to be affected by the flexion test, do not tolerate the same force as horses with more mild conditions. In a study of healthy and injured Thoroughbred (TB) racehorses, a positive correlation existed between decreased range of motion and joint injury.[7] Loss of joint motion was most likely caused by joint capsule fibrosis, but pain associated with increased intra-articular pressure from effusion or flexion also may have limited joint motion.[7] I recommend flexing a joint as much as possible with an amount of force just slightly less than that which consistently causes a withdrawal response.

Proper evaluation of the results of flexion tests requires that the horse be observed while trotting in a straight line on a firm, non-slip surface. Horses usually are trotted in hand, although occasionally a horse's response to flexion is evaluated while it is being ridden. The horse should be trotted immediately after the limb is placed to the ground, with care taken to avoid scaring the horse or providing any excessive encouragement to trot, because many horses will slip initially, gallop off, or balk, all of which necessitate test repetition. If possible, the horse should be trotted away from the examiner for a minimum of 12 to 15 m.

Causes of Pain during Flexion and Positive Flexion Test Results

Forced flexion of a joint can induce pain in many potential sites. Force is being applied to both articular structures and surrounding soft tissues. The tissues on the flexion side of the joint are being compressed, whereas tissues on the extension side are under tension. During flexion, intra-articular pressure and intra-osseous pressure in subchondral bone are increased.[3,7] Stretching or compression of the joint capsule, vascular constriction, and activation of pain receptors in the joints and surrounding soft tissues also can occur during flexion.[3] It is rarely possible to attribute pain on static flexion or during movement after flexion to an individual articular surface. The "fetlock flexion test" is a misnomer because as it is commonly performed, it includes the interphalangeal joints and stresses surrounding soft tissue. Thus the names *lower limb flexion test* or *fetlock region flexion test* are more appropriate.

Positive Responses to Flexion

Positive responses to flexion can be seen with static flexion (see Chapter 6) and when movement follows flexion. A positive flexion test result is defined as obvious lameness or an increase over baseline lameness that is observed for more than three to five strides while the horse trots in a straight line after flexion. A mild response, even in sound horses, often is seen in the first few strides, a finding that should be compared with the contralateral limb. Sound horses warm-out of this mild response quickly. A persistent, one- to two-grade increase over baseline lameness for several steps is a positive response. In horses with hindlimb lameness a marked positive response often is accompanied by reluctance to place the heel on the ground, and the horse may land only on the toe for several strides.

Forelimb Flexion Tests

Lower Limb Flexion Test

The lower limb flexion test often has been equated erroneously with the fetlock flexion test. The fetlock region can be flexed independently of interphalangeal joints (see following text). The lower limb flexion test is the most common test performed in the forelimb and involves placing a hand on the toe and forcing the fetlock and both interphalangeal joints into firm flexion (Fig. 8-1). A positive response to flexion can be observed with any condition of the distal interphalangeal, proximal interphalangeal, and metacarpophalangeal joints; navicular bone or bursa; other causes of palmar heel pain; digital flexor tenosynovitis; any soft tissue problem in the palmar pastern region; and lameness associated with the

Fig. 8-1 The lower limb flexion test often is erroneously called the fetlock flexion test. During the lower limb flexion test the fetlock, proximal interphalangeal, and distal interphalangeal joints are flexed; the palmar pastern and fetlock region soft tissue structures are compressed; and the dorsal structures are stretched.

branches of the suspensory ligament (SL) or proximal sesamoid bones (PSBs). Horses with lesions of the PSBs usually have markedly positive responses to this test. *This test is not specific for lameness of the metacarpophalangeal joint*. I have seen marked responses in horses with navicular disease or osteoarthritis of the interphalangeal joints. However, horses with osteoarthritis, fractures of the metacarpophalangeal joint, or tenosynovitis usually also show a marked positive response. In a recent study of clinically sound horses in which lameness could consistently be induced by flexion with 250 N for 1 minute, lameness was alleviated by intra-articular analgesia of the metacarpophalangeal joint, but not by intra-articular analgesia of the proximal interphalangeal or distal interphalangeal joints or intra-thecal analgesia of the navicular bursa.[8]

The limb should be held as close to the ground as possible, and forced carpal flexion should be avoided (see Fig. 8-1). All soft tissue and bony structures in the palmar aspect of the distal limb are severely compressed, resulting in low specificity for the metacarpophalangeal joint. Some people use a hand as a fulcrum or grab the toe with both hands (Fig. 8-2), but this technique may result in application of excessive force, although it otherwise produces similar results.

Fetlock Flexion Test

The specificity of the lower limb flexion test can be improved by applying force to the metacarpophalangeal joint and avoiding forced flexion of the interphalangeal joints. The fetlock flexion test is performed by placing one hand along the dorsal aspect of the pastern region and one hand along the dorsal aspect of the metacarpal region, while avoiding flexion of the carpus (Fig. 8-3). This test is more difficult to perform because it requires more force and the clinician's effort to maintain a similar degree of flexion. The test is not specific for articular lameness of the metacarpophalangeal joint and horses with soft tissue problems respond positively.

Flexion of Interphalangeal Joints

In my opinion, flexion of either the proximal interphalangeal or distal interphalangeal joint without concomitant flexion of the other, or of the metacarpophalangeal joint, is impossible. Varus or valgus stress can be applied to the interphalangeal joints, and when followed by trotting, this stress can be a suitable provocative test in horses with osteoarthritis or soft tissue injuries of these joints.

Carpal Flexion Test

The carpal flexion test is the most specific of all forelimb flexion tests, and a positive response usually reflects baseline lameness associated with the carpal regions. Few false-positive results occur. A positive response may reflect intra-articular pain, but a positive response also is seen in horses with carpal tenosynovitis, accessory carpal bone fractures, proximally located superficial (SDF) and deep digital flexor (DDF) tendonitis, proximal suspensory desmitis (PSD), or avulsion fracture of the third metacarpal bone (McIII) at the SL insertion. Rarely, a horse with a problem in the scapulohumeral and cubital joints or the antebrachium responds positively. A negative response does not preclude an articular lesion of the carpus, including incomplete fractures or sclerosis of the carpal bones.

The limb is elevated and the carpus is forced into full flexion by pushing the metacarpal region directly underneath the radius (Fig. 8-4). The distal limb can be pulled laterally to place the carpal joints in valgus stress or torsion.

Horses sometimes trot off lame on the contralateral limb after the carpal flexion test is performed. I have seen this most commonly in young Standardbred or TB racehorses with subchondral bone pain in the middle carpal joint and call it the "Ross crossed-extensor phenomenon." I believe that this reflects bilateral lameness, and flexion of the ipsilateral carpus causes less pain than making the horse stand for 1 minute on the contralateral limb. I have observed this response most commonly in horses with bilateral carpal lameness, but exacerbation of contralateral lameness is not restricted to carpal lesions. Dyson[9] has called this a "paradoxical response to flexion" and has observed exacerbation of contralateral lameness in horses with navicular syndrome and distal hock joint pain.

Fig. 8-2 Extreme lower limb flexion can be achieved by using both hands on the toe with the limb cradled between the clinician's legs. With such extreme flexion even normal horses may manifest a positive response.

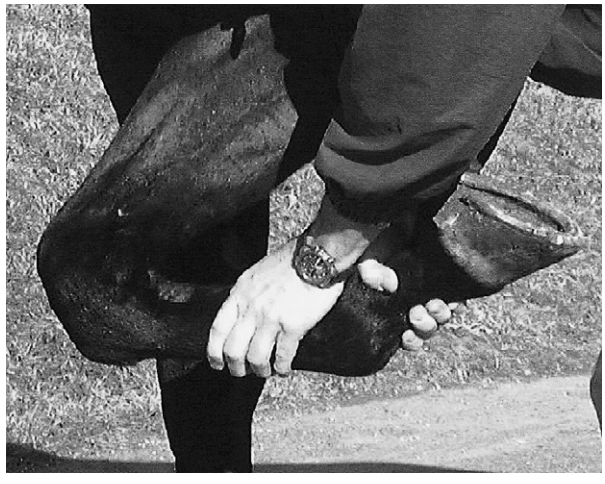

Fig. 8-3 A true fetlock flexion test can be performed by carefully flexing only the fetlock joint. The clinician's hand grasps only the pastern and not the toe of the hoof while avoiding forced flexion of the proximal and distal interphalangeal joints (see Fig. 8-1).

Upper Limb Manipulation

Because of the inverse but simultaneous movement of the elbow and shoulder joints, it is difficult to accurately name the

Fig. 8-4 The carpal flexion test is the most specific of all flexion tests, but it applies concomitant mild flexion of the elbow and shoulder joints. Although false-negative results are possible, a positive carpal flexion test result usually means that lameness originates from the carpal region.

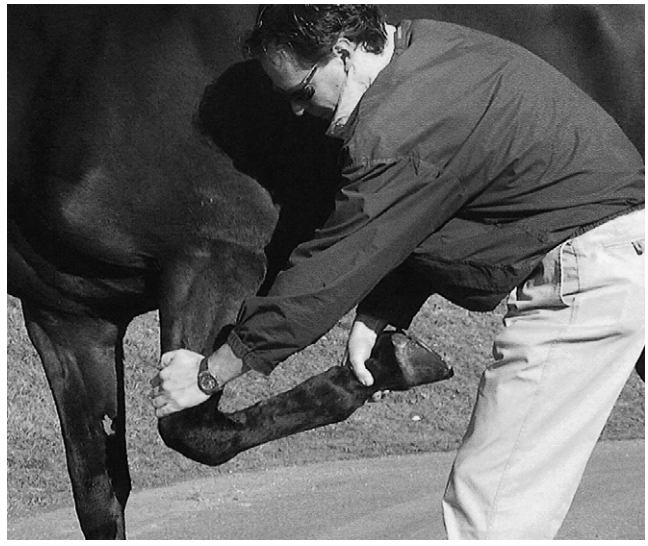

Fig. 8-5 Upper forelimb flexion is performed by grasping the antebrachium and pulling the entire limb caudally and slightly proximally. This maneuver flexes the shoulder joint and extends the elbow joint. Horses with shoulder region lameness often respond positively to this manipulative test.

flexion tests of these joints. For instance, when the limb is pulled in a caudal direction, the shoulder joint is flexed, but the elbow joint is extended. I call this manipulation *upper limb flexion* (Fig. 8-5). This maneuver requires both hands, one hand grasping the pastern region and one grasping the cranial aspect of the antebrachium to force the entire limb in a caudal direction. When the limb is pulled in a cranial direction, resulting in *upper limb extension*, the shoulder joint is extended, but the elbow joint is flexed (Fig. 8-6). Both hands are placed around the pastern region while forcing the entire forelimb into maximal extension. Maintenance of upper limb extension or flexion for even 45 seconds is difficult, so I try to maintain this position for as long as possible and then evaluate the horse while trotting. Many normal horses resist upper limb manipulation, and an alternative is to force the limb into hard flexion or extension in a rhythmical fashion six to eight times and then trot the horse. Even though the entire limb is being manipulated, there are few false-positive test results. However, false-negative test results can occur, probably because of the inability to place either the shoulder or the elbow joints in hard flexion. In my experience, horses with lameness originating from the elbow region are more likely to respond to upper limb extension, whereas those with lameness originating from the shoulder region are more likely to respond to upper limb flexion.

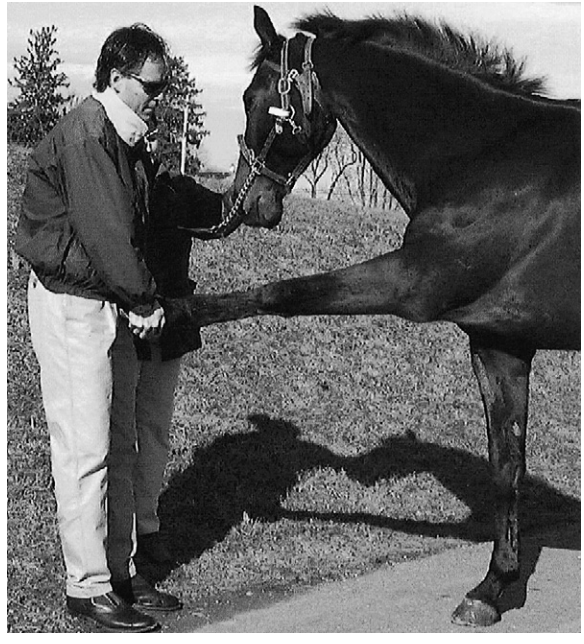

Fig. 8-6 The upper limb extension test is performed by pulling the forelimb out in front of the horse and forcing it proximally. This places the elbow joint in flexion and the shoulder joint in extension. In my experience, lameness of the elbow region is exacerbated by this technique, but occasionally shoulder joint lameness also is worsened.

Hindlimb Flexion Tests

Hindlimb flexion tests are not specific, but they may be useful to exacerbate the baseline lameness or detect hidden sources of potential lameness. I do not believe that hindlimb flexion tests are useful in differentiating the source of lameness in most horses, unless the response is dramatic, and diagnostic analgesia usually is required in all horses.

Lower Limb Flexion Test

The lower limb flexion test is performed similarly to the forelimb flexion test, but with similar force the metatarsophalangeal joint can be flexed more extremely. The veterinarian should try to keep the limb as low as possible to avoid placing hard flexion on the upper limb, although all joints are flexed to a degree. The lower limb flexion test also affects the proximal interphalangeal and distal interphalangeal joints and the surrounding soft tissues (Fig. 8-7). Horses with digital flexor tenosynovitis or DDF tendonitis show a marked response to the lower limb flexion test. False-positive results can occur, but these are less common in a hindlimb than in a forelimb, even in horses in active work. Horses with pain in the upper limb may show a mild or moderate response to lower limb flexion. This test is not specific for pain located in the lower limb, and lameness in horses with stifle pain often is worse after the lower limb flexion test.[9] Horses with subchondral

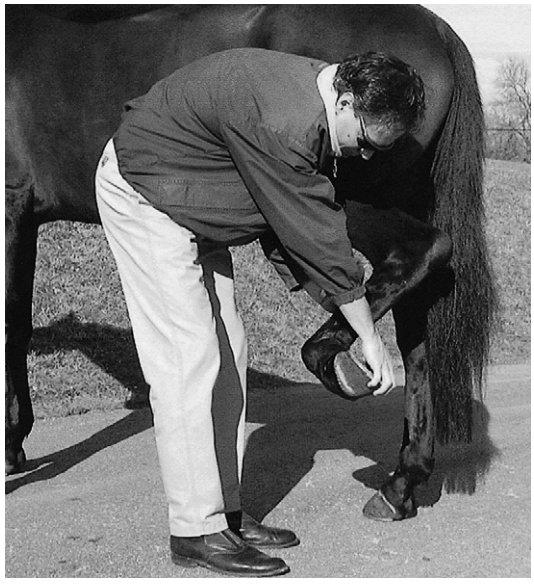

Fig. 8-7 The lower limb flexion test in the hindlimb is performed with the limb as close to the ground as possible. Flexion of one portion of the hindlimb is impossible without flexing the entire limb, a finding that explains many false-positive hindlimb flexion tests.

bone pain from non-adaptive or stress remodeling of the distal third metatarsal bone (MtIII) or those with incomplete fractures of MtIII or incomplete, mid-sagittal fractures of the proximal phalanx may show little response to this test (false-negative result).

Fetlock Flexion and Interphalangeal Joint Tests

The metatarsophalangeal joint region can be flexed independently of the interphalangeal joints in the hindlimb, or the interphalangeal joints can be flexed independently, but these tests are difficult to perform and of limited value.

Upper Limb Flexion Test

The so-called spavin or hock flexion test *is not specific for lameness of the hock*, because the stifle and coxofemoral joints also are stressed hard, and mild flexion of the lower joints is inevitable.

The limb is held in hard flexion for at least 1 minute, but additional time for this test may improve its clinical value (Fig. 8-8). It may be necessary to have an assistant place a hand on the contralateral hip to steady the horse, because proper performance of this test requires that the limb be elevated substantially and the horse may lose its balance. The position of the hands in the metatarsal region is important to consider, since the force required to hold the hindlimb in this position may cause compression and pain in structures along the plantar aspect, potentially contributing to a false-positive response.

Hindlimb Flexion Test

Alternatively, the entire hindlimb can be flexed simultaneously. This test is useless in differentiating potential sources of pain in a limb, but it is quite useful in exacerbating baseline lameness or uncovering occult lameness conditions. The clinician's hands are placed on the toe and the entire limb is held in extreme flexion (Fig. 8-9). An assistant may be necessary to steady the opposite hip while the limb is elevated.

"Hock" Extension Test

Hock extension may be useful in placing selective stress on the hock, independent of the stifle. Forced extension causes tension on the soft tissue structures on the dorsal, medial, and lateral aspects of the hock. Seldom is it possible to perform this test for 1 minute; six to eight attempts at forced extension followed by trotting the horse can be substituted for more lengthy manipulation (Fig. 8-10). False-positive and false-negative responses occur, which are caused mostly by the inelastic reciprocal apparatus. This maneuver can reveal laxity of a damaged fibularis (peroneus) tertius.

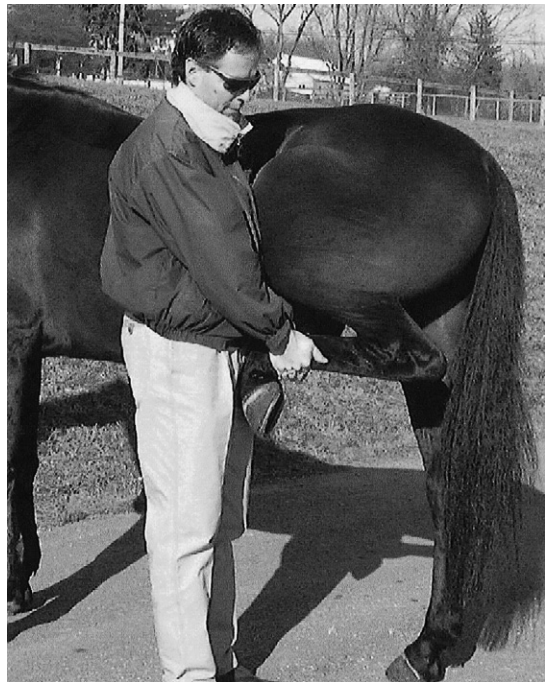

Fig. 8-8 The hindlimb upper limb flexion test is demonstrated. This test has been called the spavin or hock flexion test, but it is not specific for lameness of the hock. The hock and stifle joints are in forced flexion, the lower limb joints are flexed, the metatarsal region is compressed, and a small amount of forced flexion of the coxofemoral joint is induced.

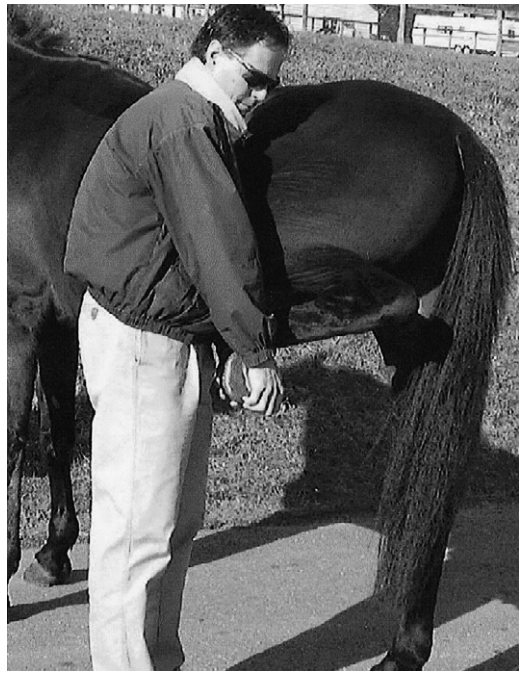

Fig. 8-9 A hindlimb flexion test is a combination of the lower limb flexion and upper limb flexion tests.

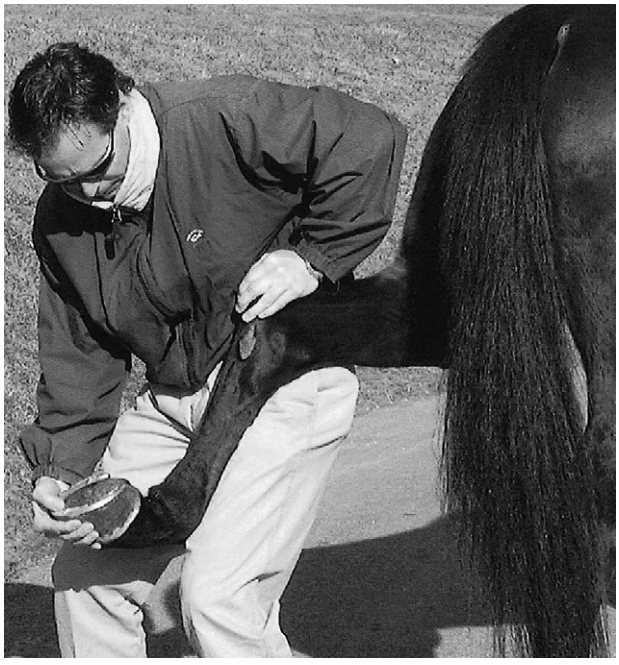

Fig. 8-10 During the hock extension test the clinician forces the hock into extension by pushing down on the calcaneus while pulling up on the distal limb by using both the right arm and left leg. Pain from hock lameness can be exacerbated, but false-positive results from pain in other locations also can occur.

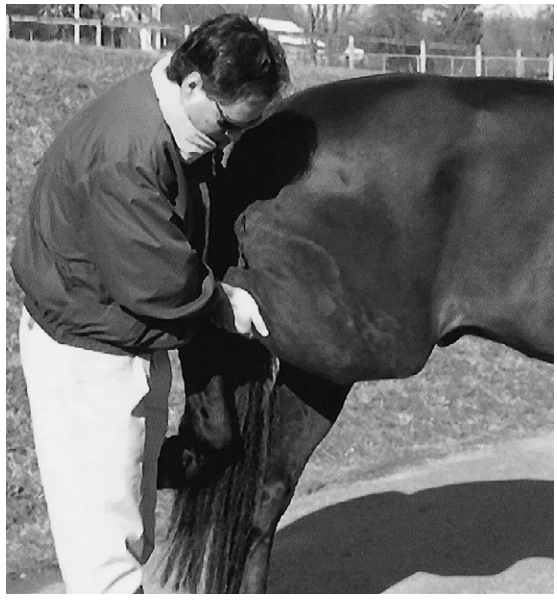

Fig. 8-11 A seldom-used test is the stifle flexion test. This test can be difficult and dangerous to perform in fractious horses. The forced flexion of the stifle joint used in this test attempts to differentiate stifle and hock joint pain.

"Stifle Flexion" Test

A modification of the upper limb flexion test can be used to place hard flexion on the stifle, independent of the hock (Fig. 8-11). This test can be somewhat difficult to perform but may exacerbate lameness in horses with osteoarthritis or other conditions of the stifle. Other proximal limb joints are also in flexion; therefore some false-positive results occur.

DIRECT OR LOCAL PALPATION FOLLOWED BY MOVEMENT

Static palpation, in which the horse's response to compression during palpation while standing is assessed, reveals useful information (see Chapter 6). Additional information can be gained by evaluating movement after palpation, dynamic provocation to induce lameness. Dynamic provocation usually is performed by digital palpation or use of hoof testers. Many horses manifest a positive response during static palpation, but the primary pain is located elsewhere. However, if lameness can be induced or baseline lameness can be increased by one or two grades by deep palpation, then the area may be relevant to the current cause of lameness. False-positive results do occur, but in my opinion, these are less frequent than with most flexion tests. There are few false-negative results.

Digital Compression of a Painful Area

The veterinarian should elevate the limb and compress the painful or otherwise inflamed area for 15 to 30 seconds and then evaluate the horse at a trot in hand. Exacerbation of the baseline lameness by one or more grades is considered a positive response. This procedure is useful in differentiating the cause of lameness in both the forelimb and hindlimb. In the forelimb, I find it useful to compress the dorsal proximal aspect of the proximal phalanx (if a mid-sagittal fracture is suspected), the dorsal cortex of the McIII (for bucked shins or a dorsal cortical fracture), exostoses involving the small metacarpal bones (for splints), the suspensory branches or digital flexor tendons, and the proximal palmar metacarpal region (for PSD or longitudinal or avulsion fracture). In horses with mild tendonitis of the superficial digital flexor tendon (SDFT), baseline lameness usually is mild or non-existent, but obvious lameness after digital compression suggests tendonitis as a substantial problem.

In the hindlimbs, compression of the dorsal proximal aspect of the proximal phalanx can increase lameness from mid-sagittal fracture, but trauma from interference injury (of particular importance in trotters) or other forms can lead to a false-positive response. A *dynamic Churchill test*, compression followed by trotting (see Chapter 6), is useful in the diagnosis of lameness of the proximal metatarsal region and tarsus. In the hindlimb, compression of the proximal aspects of both the second and fourth metatarsal bones puts indirect pressure on the origin of the SL, and a positive response may indicate PSD. Compression of a "curb" followed by trotting may increase lameness.

In some horses with tibial stress fractures, an induced lameness can be seen after deep palpation of the caudal tibial cortex. With the limb elevated, the veterinarian should apply deep pressure to the caudal cortex by wrapping the fingers around the tibia from the medial aspect. Most horses object to this maneuver, but in those with tibial stress fractures, the positive static response is followed by an exacerbation of the baseline lameness.

Axial Skeleton

Application of direct local pressure to many parts of the axial skeleton is difficult, but in some instances this procedure can lead to the detection of pain both statically and while the horse is trotting (see Chapters 6, 94, 98, and 100). In the cervical area, forced lateral bending followed by walking or trotting may exacerbate neurological signs or gait deficits in horses with cervical instability or proliferative changes. Deep palpation over the thoracolumbar spine followed by trotting

can induce hindlimb stiffness or other mild gait abnormalities. Direct and deep palpation over the tubera sacrale and tubera coxae can induce hindlimb lameness in horses with stress fractures or those with chronic lameness as a result of pelvic asymmetry from old fractures. Sacroiliac compression, or manipulation of the sacrum or tail head, can induce hindlimb stiffness or lameness in horses with injuries in these areas.

INDUCED LAMENESS AFTER HOOF TESTER EXAMINATION

The hoof testers are applied in a suspected area for 15 to 30 seconds and the horse is evaluated for lameness while trotting. False-positive test results are quite common, but a marked difference between limbs can be an important clinical sign. Shoes and pad combinations may preclude complete hoof tester examination of the sole, so I often apply pressure across the heels. I have found this position to yield the most useful information in horses with palmar heel pain from most causes, but it also induces a positive response in horses with non-specific foot pain (sore feet). Most normal horses object to firm pressure placed across the heels using hoof testers, and mild lameness on the initial few steps is common, but severe lameness after this test is a useful indication that the foot is the source of baseline lameness.

THE WEDGE TEST

The *wedge test* is a form of manipulation similar to the flexion or other varus or valgus stress tests, but it is used specifically to evaluate the digit and associated soft tissues. The wedge can be used to dramatically change the dorsal-to-palmar (heel) or medial-to-lateral hoof angles. Collateral ligaments, joint capsules, subchondral bone and articular surfaces, and surrounding soft tissues can be stretched or compressed when the horse stands on the wedge. Changes in hoof angles of this magnitude can greatly change the stress placed on the deep digital flexor tendon (DDFT), SDFT, and SL. Raising the heel reduces stress on the DDFT but increases stress on the SL. Raising the toe reduces stress on the SL but increases stress on the DDFT, navicular bone, and associated ligaments and bursa. The number of tissues affected by the wedge accounts for the lack of specificity of this test, and it likely accounts for many false-positive results. The wedge is placed in the desired position and the horse is made to stand in this position for 30 to 60 seconds with the contralateral limb elevated (Fig. 8-12). The horse is then trotted in a straight line on a firm surface. The test can be used in any limb but is performed most commonly in the forelimbs. In some horses, it is difficult to attain the desired duration regardless of whether they are lame. The horse's response to simply standing on the wedge may not give an accurate indication of how lame it will be when it is trotted. In horses content to stand in such an abnormal position, a dramatic lameness may be seen at the trot. Horses with navicular syndrome or sore feet from many causes of palmar heel pain are most likely to manifest a positive response. In my experience, the direction of the wedge that elicits the most positive response from horses with palmar heel pain is with the apex (low end) directed medially (see Fig. 8-12). This substantial change in the medial-to-lateral hoof angle is likely to cause stretching of the suspensory apparatus of the navicular bone and collateral ligaments of the distal interphalangeal joint or compression on articular structures. Horses with palmar heel pain may show severe lameness, but diagnostic analgesia is required to confirm the foot as a source of pain. Horses with injuries of the DDFT, SDFT, and SL may show a milder response.

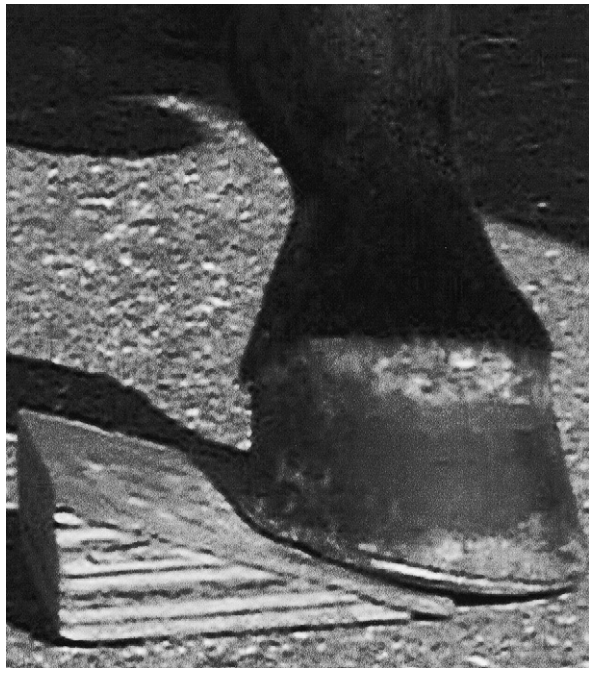

Fig. 8-12 A 15° to 20° wedge can be used to manipulate the joints and soft tissue structures of the digit. The most consistent response is elicited by directing the apex (low end) of the wedge medially (as shown). The wedge also can be used to raise the heel and toe. (Wedge courtesy Norman Ducharme, Ithaca, New York.)

VARUS OR VALGUS STRESS TESTS

Evaluation for lameness after placing varus or valgus stress on an individual joint may incriminate this area as a potential source of pain and is used most commonly in the stifle. To perform the stifle valgus stress test, the clinician's shoulder (or hand) is used as a fulcrum against the distal femur, and the distal limb is pulled laterally several times before the horse is trotted (see Fig. 6-24). False-positive results can be obtained because the entire distal extremity is manipulated during this test. Valgus or varus stress tests can be used in many joints in the distal limb, particularly the interphalangeal joints.

Patellar manipulation followed by trotting (see Fig. 6-26) may be helpful but can be difficult to perform when horses resist forced proximal movement of the patella (frequently, the veterinarian's wrist is forced into hyperextension). Although cranial and caudal drawer tests can be used to exacerbate stifle lameness, I have not found them particularly helpful, and they are dangerous to perform.

FLEXION TESTS AND DIAGNOSTIC ANALGESIA

I do not generally recommend combining the results of flexion tests and diagnostic analgesia. I often hear that baseline lameness abated after a block, but the horse still had positive flexion test results. My usual comment is, "Why bother to flex the horse if baseline lameness has been abolished?" Flexion tests induce lameness that may be unrelated to the baseline lameness. Thus it is not unusual that a horse might have residual lameness after flexion, even if the baseline lameness has been eliminated.[4] I usually do not recommend further investigation once baseline lameness has been eliminated.

If baseline lameness is not obvious, but a low-grade gait deficit is present, or if a horse has bilaterally symmetrical lameness, flexion tests or other forms of manipulation or

provocation may be the only way of "seeing" lameness. In this instance, induced lameness from manipulation can be assumed to be the baseline lameness, and diagnostic analgesia can proceed. All involved parties should be well informed about the potential for misdiagnosis, but in certain circumstances this pathway may lead to a successful diagnosis.

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CHAPTER • 9

Applied Anatomy of the Musculoskeletal System

Matt Durham and Sue J. Dyson

It is beyond the scope of this book to describe all aspects of musculoskeletal anatomy in depth, yet a detailed knowledge of anatomy is fundamental to a lameness diagnostician, as highlighted in the chapters on observation and palpation (Chapters 5 and 6). Some aspects of anatomy are considered in depth in individual chapters dealing with conditions of specific areas. This chapter considers some philosophical aspects of the importance of anatomical knowledge and describes some basic principles. It also provides illustrations that we hope will help the reader to understand better the three-dimensional aspects of anatomy.

Accurate interpretation of what we see and feel during an examination requires knowledge of what structures we are looking at and palpating. For example, a swelling is noted over the dorsal aspect of the carpus. Is the swelling diffuse and possibly related to a hygroma, periarticular edema or cellulitis, or is there a discrete swelling, horizontally oriented, reflecting distention of the middle carpal joint? Or is it a longitudinal swelling reflecting distention of the common digital extensor tendon sheath or the tendon sheath of extensor carpi radialis? If the swelling is longitudinal, are any compressions in the swelling caused by normal retinaculum or adhesions within the sheath (Fig. 9-1)? If we examine the sheath by ultrasonography, is the echogenic band extending from the sheath wall to the enclosed tendon normal mesotendon, or is it an adhesion? If diffuse swelling is present around the dorsal aspect of the carpus associated with lameness, how can we tell if the middle carpal joint capsule is distended? We need to know that there is a palmar outpouching of the middle carpal joint on the palmarolateral aspect of the carpus, just distal to the accessory carpal bone. Thus during visual inspection and palpation the clinician should be constantly asking "what

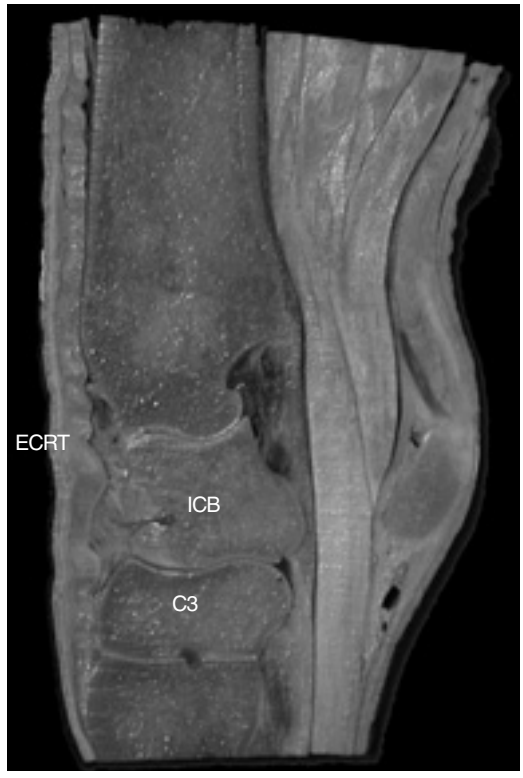

Fig. 9-1 Sagittal anatomical section through the carpus, transecting the extensor carpi radialis tendon *(ECRT)*. *ICB,* Intermediate carpal bone; *C3*, third carpal bone.

provocation may be the only way of "seeing" lameness. In this instance, induced lameness from manipulation can be assumed to be the baseline lameness, and diagnostic analgesia can proceed. All involved parties should be well informed about the potential for misdiagnosis, but in certain circumstances this pathway may lead to a successful diagnosis.

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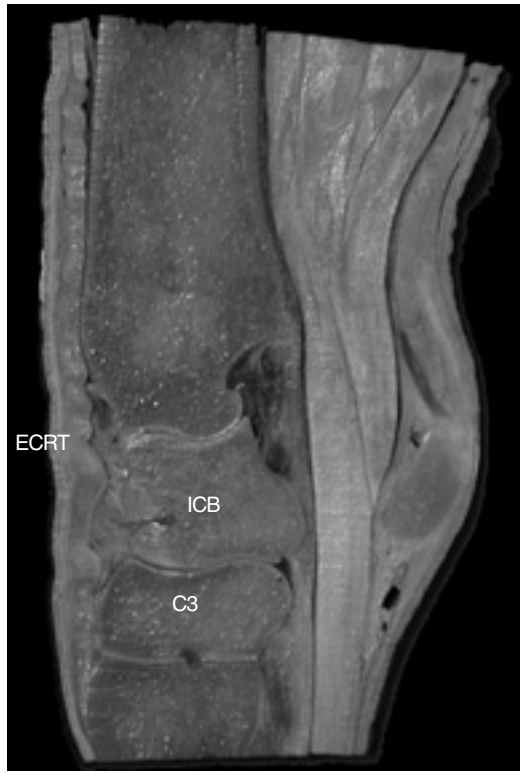

Fig. 9-1 Sagittal anatomical section through the carpus, transecting the extensor carpi radialis tendon *(ECRT)*. *ICB,* Intermediate carpal bone; *C3*, third carpal bone.

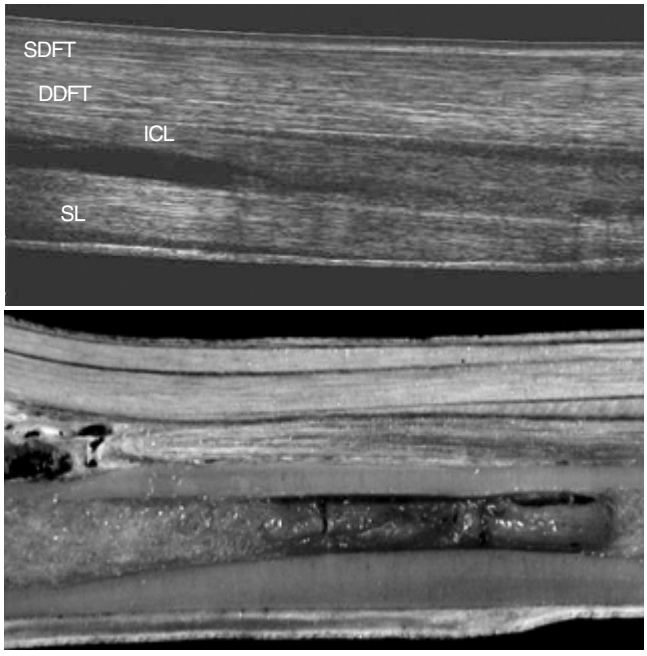

Fig. 9-2 Sagittal views of the palmar metacarpal region. Proximal is to the right. **A,** FreeStyle Extended Imaging (Sequoia model, Acuson Co., Mountain View, CA) ultrasonographic image of the palmar metacarpal region. *SDFT,* Superficial digital flexor tendon; *DDFT,* deep digital flexor tendon; *ICL*, accessory ligament of the DDFT (inferior check ligament); *SL*, suspensory ligament. **B,** Corresponding anatomical section.

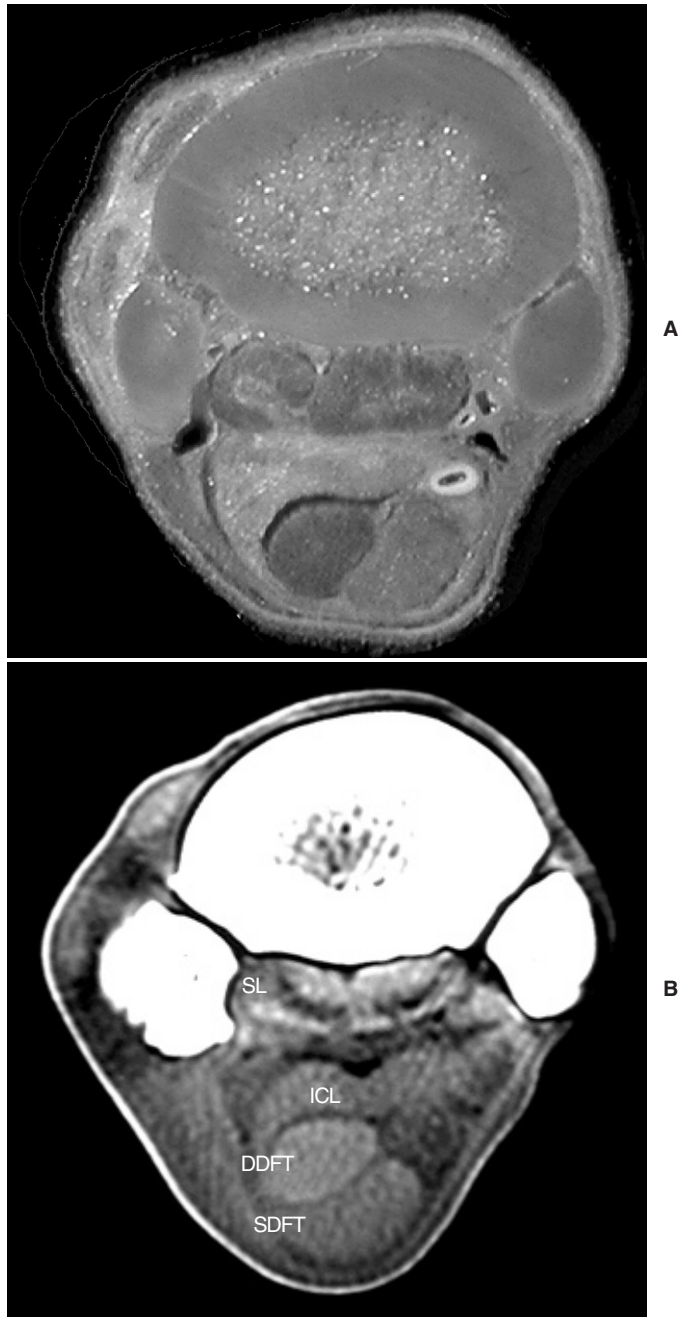

Fig. 9-3 Transverse sections through the proximal metacarpal region. Dorsal is to the top and lateral is to the left. *SL*, Suspensory ligament; *ICL*, accessory ligament of the deep digital flexor tendon (inferior check ligament); *DDFT,* deep digital flexor tendon; *SDFT,* superficial digital flexor tendon. **A,** Anatomical specimen. **B,** Computed tomographic scan using soft tissue windowing. (Courtesy Alamo Pintado Equine Medical Center, Los Olivos, California.)

structure am I seeing or palpating, what are its functions, and what would be the consequences of loss of function?" If it has abnormal contour or size, is this the result of swelling of that structure or an adjacent or underlying structure? Having established what structure is abnormal, the clinician then must consider the best imaging modality. If it is a tendonous or ligamentous structure, ultrasonography probably will provide the most information, but we must remember that it has bony attachments, and damage at those attachments might best be assessed by either radiography or nuclear scintigraphy. So we need to know not only what each structure is, but also the structures to which it is attached.

During visual inspection and palpation, we also need to think logically. We know that the superficial and deep digital flexor tendons (SDFT, DDFT), the accessory ligament of the DDFT (ALDDFT), and the suspensory ligament (SL) lie on the palmar aspect of the third metacarpal bone (Fig. 9-2). Swelling confined to just the medial aspect of the metacarpal region is far more likely to reflect direct trauma to the medial aspect of the limb than sprain or strain of any of the ligamentous or tendonous structures. We need to know that the proximal aspect of the SL lies between the bases (heads) of the second (McII) and fourth (McIV) metacarpal bones and therefore is inaccessible to direct palpation, and that desmitis often may be present without discernible soft tissue swelling (Fig. 9-3).

We must be aware of anatomy to realize the possible consequences of trauma to an area. The paucity of soft tissues over the cranial aspect of the stifle makes the patella and the tibial tuberosity vulnerable to direct trauma, hence the risk of fracture after hitting a fixed fence. The lack of soft tissues also means that if the horse hits a thorn hedge, the possibility of a thorn penetrating the femoropatellar joint capsule, resulting in contamination and infection, is quite high. We also need to think how structures move relative to one another while the horse is in motion. If a steeplechase horse sustains an interference injury on the palmar aspect of the metacarpal region while galloping, the position of the skin laceration probably will not coincide with the level of the laceration in the SDFT (Fig. 9-4). We also need to know the relative positions of the laceration and the digital flexor tendon sheath (DFTS) to be aware of the likelihood that the sheath may have been traumatized, and thus the risk of infectious tenosynovitis. Faced with a contaminated wound on the dorsal aspect of a hind fetlock and severe lameness, and the possibility of infection of

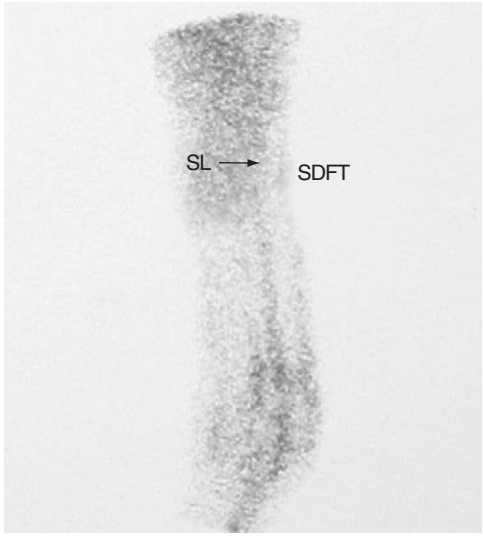

Fig. 9-4 Lateral scintigraphic image of the metacarpal region acquired before the end of the vascular phase and at the beginning of the pool phase. This jumper had a history of low-grade chronic proximal suspensory ligament *(SL)* desmitis, and mild diffuse superficial digital flexor *(SDFT)* tendonitis. An acute interference injury to the mid-metacarpal region was evident on the skin. Note the proximal location of the acute injury to the SDFT. The linear area of uptake between the SL and SDFT is vascular artifact related to the time of acquisition. (Courtesy Alamo Pintado Equine Medical Center, Los Olivos, California.)

the metatarsophalangeal joint, we need to know where to expect to see distention of the plantar pouch of the joint capsule and to know that this site is safely accessible for arthrocentesis.

A fundamental principle of lameness investigation is the identification of the source or sources of pain. Although this may be possible through detailed clinical examination, in many instances it is essential to perform diagnostic analgesia (see Chapter 10). A detailed knowledge of the anatomy of nerves, joint capsules and the various out-pouchings, tendon sheaths, and bursae is fundamental to safe, accurate performance of perineural and intra-synovial injections.

Given the knowledge of the close relationship among the distal interphalangeal joint capsule, the distal sesamoidean impar ligament, the collateral ligaments of the navicular bone and the distal phalanx, and the close proximity of branches of the palmar digital nerve, it is not surprising that intra-articular analgesia is not specific and that other structures can be affected, especially if interpretation of the response is delayed or an excessively large volume of local anaesthetic solution is used. Knowledge that the medial and lateral femorotibial joints do not normally communicate and that the cruciate ligaments usually are extra-articular structures is crucial for an understanding of why these joint compartments must be injected separately, and why the response to intra-articular analgesia may be both incomplete and delayed if a cruciate ligament is damaged.

Knowledge of functional neuroanatomy also is important for interpretation of specific gait abnormalities. Inability to bear weight on a hindlimb after general anesthesia may be due to myopathy, but in the absence of marked pain and distress, it is more likely that the horse has lost extensor function and is unable to extend any of the hindlimb joints because of femoral nerve paresis. Loss of ability to extend the elbow may be due to loss of triceps function associated with a fracture of the olecranon but may also be due to radial nerve paresis.

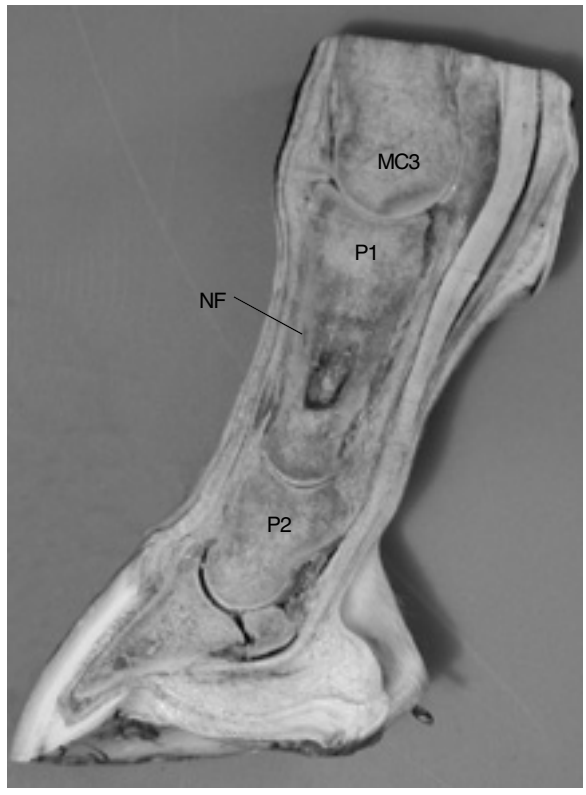

Fig. 9-5 Sagittal anatomical section through the pastern demonstrating a common location for the nutrient foramen *(NF)* entering the proximal phalanx *(P1)*. *MC3*, Third metacarpal bone; *P2*, middle phalanx.

Vascular anatomy is important because many nerves lie close to vessels. With superficial nerves and vessels, identification of the vessel may facilitate palpation of the nerve and thus aid accurate perineural injection. Avoiding penetration of the vessel and causing hematoma formation also is desirable. With regard to deeper nerves the veterinarian may benefit by knowing that the needle must be in close proximity to the nerve if blood appears in the needle hub. This information can be helpful when performing perineural analgesia of the deep branch of the fibular nerve.

Assessment of digital pulse amplitudes is an integral part of palpation. Increased pulse amplitude usually signifies a site of inflammation at, or distal to, the region of palpation, especially in association with inflammatory conditions of the foot, such as subsolar abscessation or laminitis. Palpation of the pulse in the dorsal metatarsal artery and assessment of saphenous vein filling can be helpful in the evaluation of a horse with suspected aortoiliac thrombosis.

Knowledge of the sites of major vessels is important when considering the consequences of major laceration to an area and possible avascular areas, and in planning a surgical approach to an area. All bones have one or more nutrient foramina through which major vessels enter. These usually are in standard locations (Fig. 9-5). Knowledge of these sites is critical for accurate radiological interpretation because a nutrient foramen appears as a radiolucent area, which should not be confused with a pathological lesion. The position of these intra-osseous vessels also has important consequences in considering repair of major long bone fractures.

Thermography relies on the detection of surface heat and is obviously greatly influenced by the position of superficial vessels. Interpretation may be misleading without knowledge of location. Thus it should be absolutely clear that anatomy is

a dynamic subject and is not merely a function of knowing the origins and insertions of numerous structures.

We also need to know some fundamentals of biomechanics. What is the biomechanical function of the SL? What are the implications of loss of function? For example, how may function be altered by a change in foot angle after application of a heel wedge? How is load in the distal limb joints affected by mediolateral foot imbalance? If the accessory ligament of the SDFT is cut (superior check desmotomy), how does this alter the function of not only the SDFT but also other tendonous and ligamentous structures? Does consequent overload of the SL predispose to an increased risk of suspensory desmitis? When orthopedic surgery is being considered, which is the tension side of the bone, to which a dynamic compression plate should be applied to take advantage of the tension band principle?

In more general terms, how will lameness in the left hindlimb alter forces in the other limbs, and does this vary with the gait? Given the reciprocal apparatus of the hindlimb and the inability to flex and extend the limb joints independently, it is not surprising that the gait characteristics of hindlimb lameness are so similar, irrespective of the source of pain causing lameness. Understanding the reciprocal apparatus, in addition to the results of loss of its function (e.g., after damage to the fibularis tertius) are hugely important for an understanding of hindlimb lameness.

After the source of pain causing lameness is isolated, then it is necessary to establish what is causing pain; this requires one of a number of imaging modalities: radiography, ultrasonography, nuclear scintigraphy, magnetic resonance imaging (MRI), computed tomography (CT), and exploratory arthroscopy, bursoscopy, or tenoscopy. Accurate interpretation of any of these techniques requires specialist anatomical knowledge. With radiographic images, various structures are superimposed, resulting in potentially confusing radiolucent lines that can mimic a fracture (e.g., in the relatively complex carpus and tarsus). A frog shadow superimposed over the navicular bone may mimic a fracture. We must be cognizant of anatomical variations, for example, the shape and size of the crena of the distal phalanx. We have to know how best to image a specific anatomical location, such as the sustentaculum tali of the calcaneus (fibular tarsal bone) using a skyline projection. To interpret the significance of periosteal or entheseous new bone, detailed knowledge of the soft tissue structures that do (or do not) attach in that area is vital. Particularly in the fetlock and pastern areas, numerous ligamentous structures have discrete areas of attachment (Fig. 9-6).

Radiography requires the awareness that we are looking at a three-dimensional structure in two dimensions, and thus images of the area must be obtained from several different angles. With ultrasonography, and more particularly with MRI and CT, structures can be imaged in three dimensions; this requires detailed knowledge of the shape, size, and relationships between structures. In the proximal metatarsal region the DDFT lies more medial than the SDFT and SL, and thus these structures cannot be imaged adequately by ultrason-

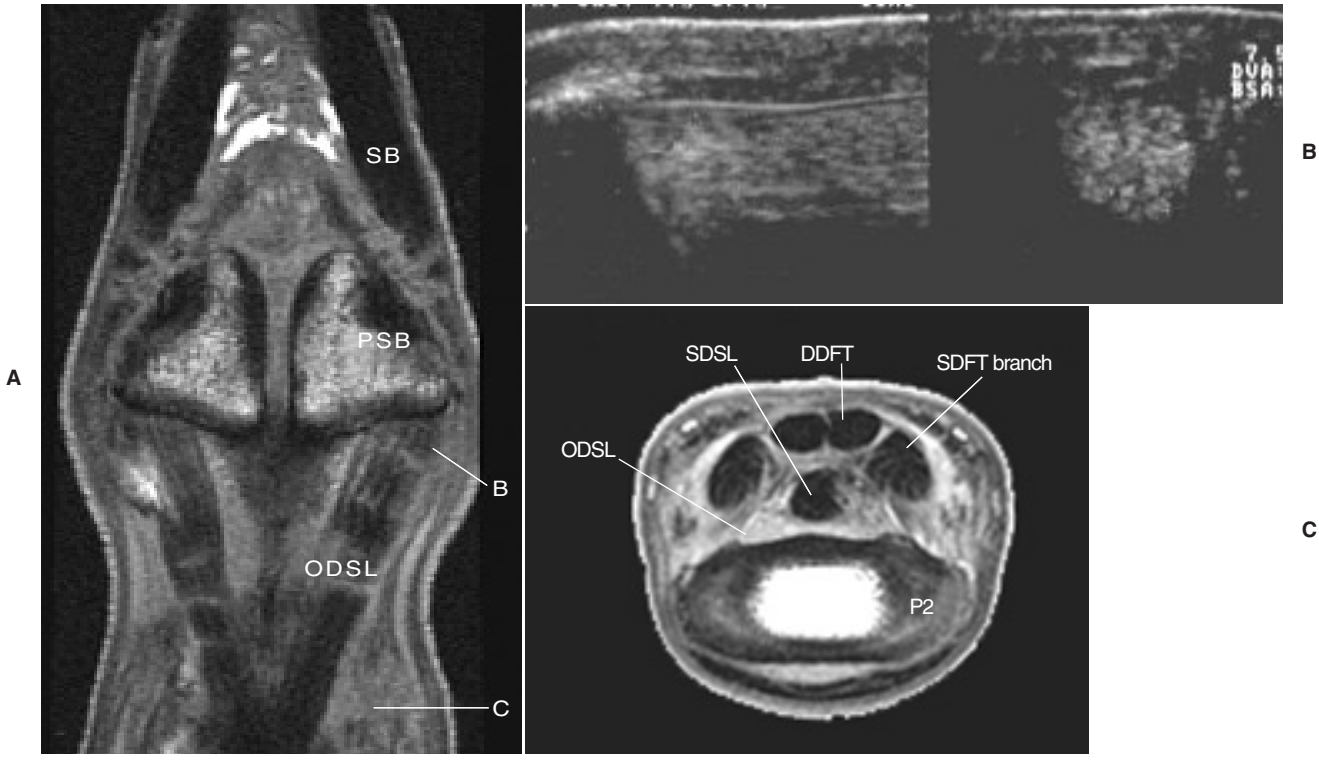

Fig. 9-6 The oblique distal sesamoidean ligaments. **A,** Frontal magnetic resonance imaging (MRI) scan of the pastern showing the origins and insertions of the oblique distal sesamoidean ligaments *(ODSL)*. *SB*, Suspensory branch; *PSB*, proximal sesamoid bone. (Courtesy Alexia L. McKnight, University of Pennsylvania, Philadelphia, Pennsylvania.) **B,** Frontal *(left)* and transverse *(right)* ultrasonographic images of the ODSL obtained at point *B* in part **A.** Proximal and dorsal are to the left. **C,** Transverse MRI scan obtained at point C in part **A.** *DDFT*, Deep digital flexot tendon; *SDFT*, superficial digital flexor tendon; *SDSL*, straight distal sesamoidean ligament; *P2*, middle phalanx.

ography at the same time from the plantar aspect of the limb (Fig. 9-7). The transducer must be moved to a plantaromedial site to evaluate the DDFT in its entirety. A large vessel on the plantarolateral aspect of the SL can cause shadowing artifacts in the SL.

The internal architecture of the joint becomes important during exploratory arthroscopy. What are the normal variations in cartilage thickness? Where do you expect to see a synovial fossa? Which parts of the synovial membrane are usually more vascular? Is it normal that the cranial cruciate ligament can be seen without synovial covering from the medial femoral tibial joint?

A textbook of this type cannot possibly provide detailed descriptions of all aspects of anatomy, functional anatomy, and biomechanics, nor answer all of the questions posed earlier in this chapter. It is hoped that this overview will stimulate readers to have a thirst for more knowledge of these subjects, in the knowledge of their huge importance.

Lameness clinicians are encouraged to acquire a set of boiled-out bones for reference and perform detailed dissections of cadaver limbs to improve knowledge of anatomy. Practicing nerve block techniques on cadaver limbs is very important for inexperienced clinicians or those performing a new block for the first time. If a lame horse must be humanely destroyed, clinicians should take the opportunity, whenever possible, to perform a post-mortem examination to correlate clinical findings with the actual lesions and revise anatomy at the same time. Each time a dissection is performed, new anatomical detail becomes apparent that had previously been missed.

The remainder of this chapter provides some basic definitions of anatomical terms used elsewhere in the book, describes the reciprocal apparatus of the forelimb and hindlimb, and presents correlative illustrations of anatomical specimens and images of those areas to assist in the understanding of three-dimensional anatomy.

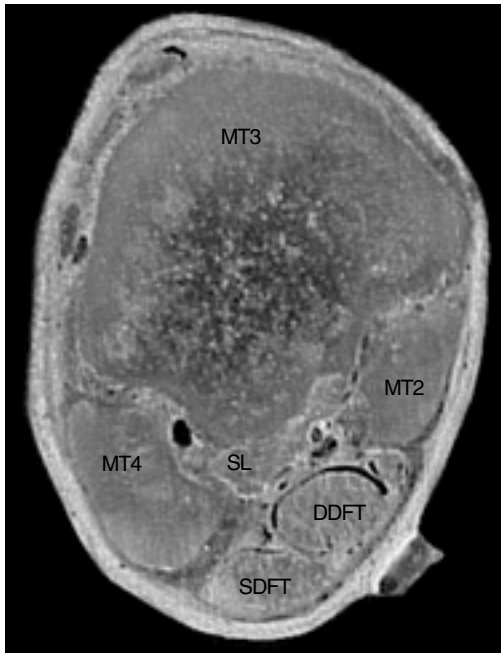

Fig. 9-7 Transverse anatomical section of the proximal metatarsal region demonstrating the lateral position of the superficial digital flexor tendon *(SDFT)* relative to the deep digital flexor tendon *(DDFT)*. Lateral is to the left and dorsal to the top of the image. This arrangement is the opposite of that seen in the forelimb (compare with Fig. 9-3). *SL*, Suspensory ligament; *MT2*, *MT3*, and *MT4*, second, third, and fourth metatarsal bones, respectively.

The Language of Anatomy

The system described in the *Nomina Anatomica Veterinaria* (NAV) according to the guidelines of the International Committee on Veterinary Anatomical Nomenclature has been used so that anatomical terminology is universal. English translations of NAV terms have been used whenever possible according to these guidelines.

Forces

The interaction of anatomical structures allows for the conversion of chemical energy into purposeful movement. It is often useful to think of complex anatomical structures in terms of interactions between simplified structural units. The interaction of forces within these anatomical units dictates the abilities and the potential weaknesses of the equine athlete. In simple terms, the stresses acting on the body are compression, tension, shear, torsion, and bending.

Compression is the force applied between two points to move them together. Examples of compression are seen in joints, such as within the middle carpal joint at the interface between the radial and third carpal bones, or that sustained by the digital cushion between the sole, frog, and the distal phalanx. Compression also is sustained within most bones, such as the third carpal bone, or the dorsal cortex of the third metacarpal bone (McIII).

Tension is the force that tends to stretch or elongate a structure. Examples of tension are most obvious in tendons and ligaments, but bones such as the olecranon or within the palmar cortex of McIII also sustain tensile strain.

Shear is a stress at the interface between two structures moving in opposite directions. Examples of shear are seen in the femoropatellar and tarsocrural joints, within bone, and within the hoof capsule.

Torsion is the stress produced when a twisting motion is applied to an object. Examples of torsional strain are seen within joints, such as the distal hock joints, or within individual bones, such as McIII.

Bending is a combination of compression on one side of a structure and tension on the other side. Structures submitted to bending are long bones such as McIII, where the dorsal cortex is submitted to compression, whereas the palmar cortex is submitted to tension.

Specialized Structures

Synovial Structures

Synovial bursae, tendon sheaths, and joints have a similar function and generally similar structure. All are sacs containing synovial fluid produced by the lining of the sac. In simple terms, synovial structures facilitate the movement between independent structures by providing a hydraulic cushion of viscous fluid that limits the effects of friction to help dissipate compressive and shear forces. (For a more complete discussion on synovial structures, see Chapters 63 and 80.)

A diarthrosis is a mobile joint containing a synovial membrane. This membrane is flexible enough to allow for movement of the joint. The synovial fluid lubricates, hydraulically equalizes pressure between cartilage plates, and nourishes the articular cartilage.

A synovial sheath is a sac that completely surrounds a tendon, forming a synovial lining on the surface of the tendon and the lining of the sheath. The synovial reflection between these visceral and parietal layers is termed the *mesotendon*. This structure is similar to the mesentery in the abdominal cavity. Nerve and blood supply to the tendon is found within the mesotendon. In areas of great mobility within the synovial sheaths, the nerve and blood supply to the tendons is through

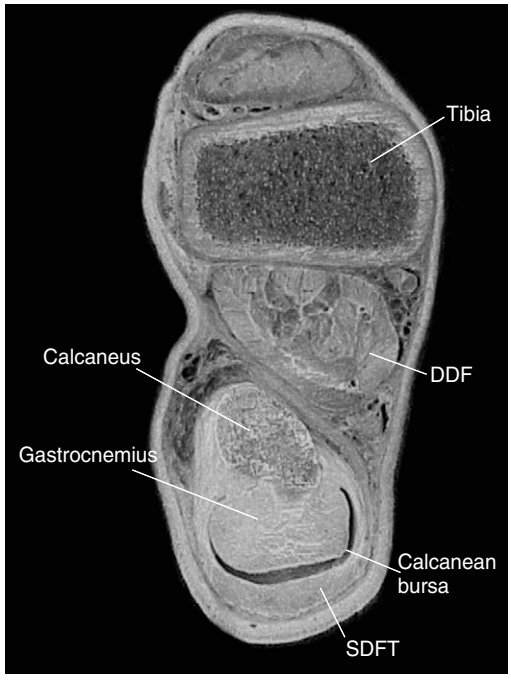

Fig. 9-8 Transverse anatomical section through distal tibia and proximal calcaneus demonstrating the distinct calcanean bursa bounded by the collateral ligaments of the superficial digital flexor tendon *(SDFT)*. *DDF,* Deep digital flexor muscle.

a vinculum, which is a modified mesotendon in the form of a narrow band connecting visceral and parietal layers.

A synovial bursa is a simple sac lying between a tendon or muscle and an adjacent bony prominence. A bursa does not surround the tendon but acts as a cushion at the interface where pressure is concentrated (Fig. 9-8).

Intercalated Bones

Intercalated bones are bones that arise within tendons or ligaments allowing for the interface between the tendonous structure and the underlying bone at an area of focal pressure, typically at the level of a joint. The interface between these bones is within a synovial sac. The navicular bone, proximal sesamoid bones (PSBs), and patella are intercalated bones. These bones allow for smooth movement and dissipation of focal pressure between the tendon or ligament and the underlying joint (Fig. 9-9).

Fibrocartilaginous Structures

In general terms, there are four functional arrangements of fibrocartilage: interarticular, connecting, circumferential, and stratiform.

Interarticular fibrocartilage. *Menisci* are fibrocartilaginous structures located between the articular cartilages of a diarthrosis. Menisci are not directly attached to the joint surfaces but are held in place by ligaments immediately adjacent to the articular surfaces. They provide congruency between the condyles, allow for a greater range of movement of the joint, and absorb concussion. Menisci are found in the stifle and temporomandibular joints of the horse.

Connecting fibrocartilage. A *symphysis* is a fibrocartilaginous joint that allows minimal movement. The pelvic symphysis and intersternebral and intervertebral joints are examples of fibrocartilaginous joints.

Circumferential fibrocartilage. In the coxofemoral joint the acetabular lip (labrum acetabulare) is a fibrocartilaginous ring extending the articular surface in a firm, semi-flexible manner. The transverse acetabular ligament is the portion of

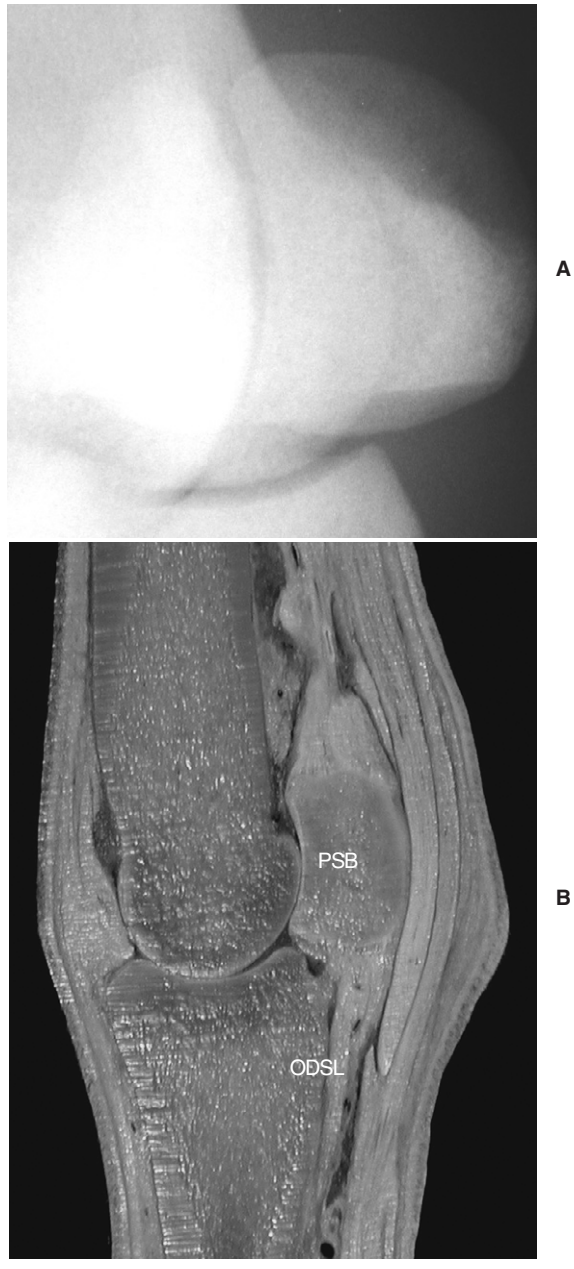

Fig. 9-9 **A,** Oblique radiographic projection of a normal proximal sesamoid bone *(PSB)*. **B,** Parasagittal anatomical section through suspensory branch, PSB, and oblique distal sesamoidean ligament *(ODSL)*.

the labrum crossing the acetabular notch. The glenoid labrum seen in other species is a poorly developed fibrous band in the shoulder of the horse.

Stratiform fibrocartilage. Stratiform fibrocartilages arise within ligamentous structures at an interface with high focal pressure between soft tissue and bone, either within a ligament or as an extension of a bony surface. These structures are similar to intercalated bones in that they typically provide rigidity to help dissipate compressive forces, but the moderate elasticity allows for some flexibility of the structures.

The parapatellar fibrocartilage on the medial aspect of the patella, portions of the biceps brachii tendon of origin within the intertubercular (bicipital) bursa, the manica flexoria, and portions of the DDFT adjacent to the proximal aspect of the middle phalanx are examples of stratiform cartilage formation

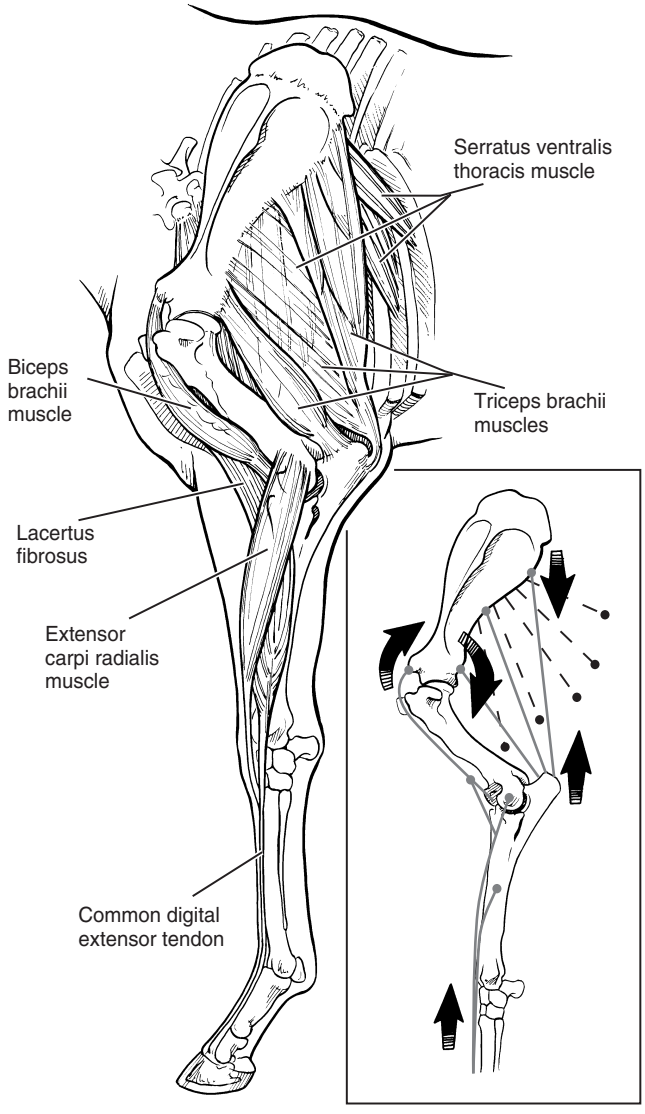

Fig. 9-10 The passive stay apparatus of the forelimb.

within tendonous structures. The proximal, middle, and distal scuta are stratiform fibrocartilaginous structures associated with the intersesamoidean ligament, the palmar aspect of the middle phalanx, and the collateral ligaments of the navicular bone, respectively. These structures serve as semi-rigid pulleys primarily for the DDFT.

Passive Stay Apparatus

Distal Limb

The horse is uniquely equipped to be able to stand at rest while expending minimal muscular effort. In the forelimb and hindlimb the fetlock is prevented from over-extension by a combination of structures providing passive resistance. The suspensory apparatus is the main contributor, forming a sling that maintains the fetlock in extension. In addition, the SDFT, DDFT, and the associated accessory (check) ligaments (in the forelimb) act as tension bands providing passive support. The suspensory apparatus consists primarily of the SL and branches, PSBs, and distal sesamoidean ligaments. The intercalated PSBs provide a broad face at the point where focal pressure is high at the palmar/plantar aspect of the fetlock joint, enabling the ligamentous tension band to support the fetlock. Dorsal branches of the SL join with the common/long digital extensor tendon, helping to stabilize the dorsal aspect of the digit. The axial and abaxial palmar/plantar ligaments of the proximal interphalangeal joint, the SDFT branches, and the straight distal sesamoidean ligament support the palmar/plantar aspect of the proximal interphalangeal joint. The navicular bone and its suspensory apparatus, in combination with the distal sesamoidean impar ligament, stabilize the palmar/plantar aspect of the distal interphalangeal joint.

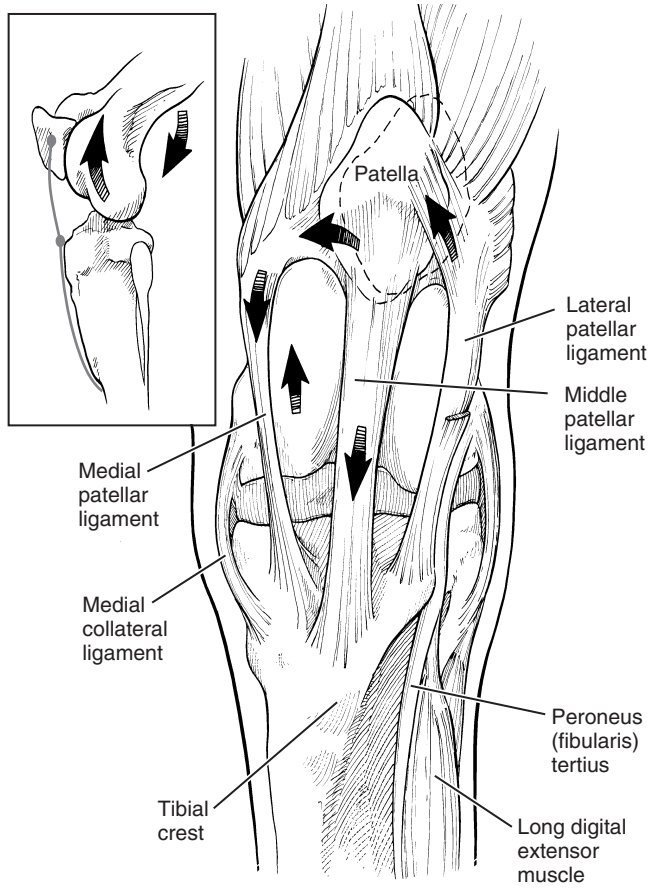

Fig. 9-11 The patellar locking mechanism.

Forelimb

In the forelimb the fibrous portion of the serratus ventralis thoracis acts as a sling suspending the thorax from the forelimb by its attachment to the scapula. The downward force applied by the serratus ventralis on the caudal aspect of the scapula causes slight flexion of the scapulohumeral joint, applying tension to the biceps brachii. A fibrous band of the biceps brachii extends from the supraglenoid tubercle of the scapula and continues as the lacertus fibrosus, which joins with the extensor carpi radialis to passively extend the carpus. Minimal muscular effort by the triceps on the olecranon maintains the elbow in extension (Fig. 9-10).

Hindlimb

The stifle is maintained in extension by the patellar locking mechanism with minimal muscular effort. Slight muscular effort by the quadriceps and tensor fascia lata rotates the patella medially, where the cartilaginous process of the patella is caught caudal to the large prominence of the medial trochlear ridge of the femur. Slight relaxation of the quadriceps as a whole allows slight flexion of the stifle, which "locks" the patella in place by applying tension primarily to the medial and middle patellar ligaments (Fig. 9-11). When the stifle is extended, the hock is passively extended by the superficial digital flexor and the fibrous component of the lateral head of the gastrocnemius muscles, which extend from the femur to the tuber calcis.

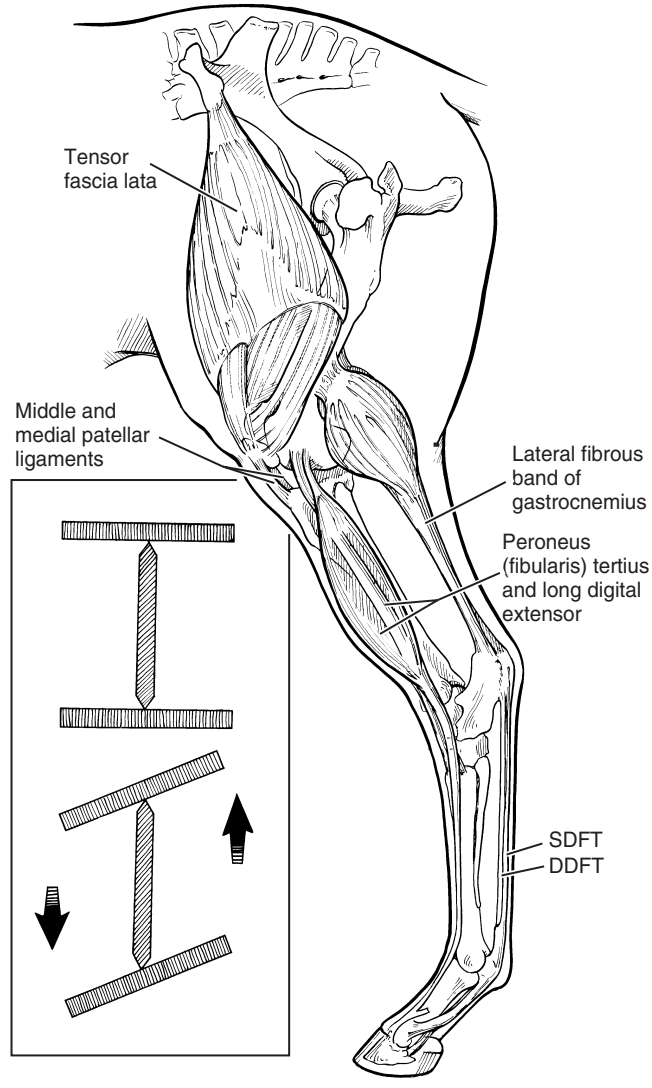

Fig. 9-12 The reciprocal apparatus. *SDFT,* Superficial digital flexor tendon; *DDFT,* deep digital flexor tendon.

The reciprocal apparatus forces the hock to flex and extend in unison with the stifle. The reciprocal apparatus transfers mechanical energy to the distal limb from the massive muscular structures of the upper limb without adding mass to the lower limb. The superficial digital flexor and the fibrous portion of the gastrocnemius serve as the caudal component of the reciprocal apparatus, along with the long plantar ligament, which acts as a tension band to make the calcaneus, distal tarsus, and metatarsal region a single functional lever arm. The fibularis (peroneus) tertius serves as the cranial component of the reciprocal apparatus, extending from the femur to the dorsal and lateral aspects of the tarsus (Fig. 9-12). Although the fibularis tertius is important as part of the reciprocal apparatus, it is not essential for function of the passive stay apparatus, as its function is flexion of the tarsus.

A second reciprocal mechanism has been described for the lower limb of the hind limb, where the fetlock and digit are flexed at the same time as the stifle and hock. The long digital extensor tendon and DDFT were the dorsal and plantar components suggested, but the SDFT probably also contributes.

Three-Dimensional Anatomy

Major advances in lameness diagnosis are being made with the assistance of advanced imaging techniques. Radiography, nuclear scintigraphy, and ultrasonography are well established, whereas CT and MRI are growing in importance. CT and MRI in particular require a detailed knowledge of three-dimensional anatomy. It is beyond the scope of this text to provide detailed correlative images of the entire musculoskeletal system. Figs. 9-13 through 9-18 give a flavor of what is possible. Figs. 9-13 through 9-16 highlight the complex anatomy of the navicular bone region, showing the close relationship between the collateral ligaments of the navicular bone, the distal sesamoidean impar ligament, the DDFT and the navicular bursa and distal interphalangeal joint capsule. Figs. 9-17 and 9-18 demonstrate the relationship between some aspects of the complex anatomy of the carpal region.

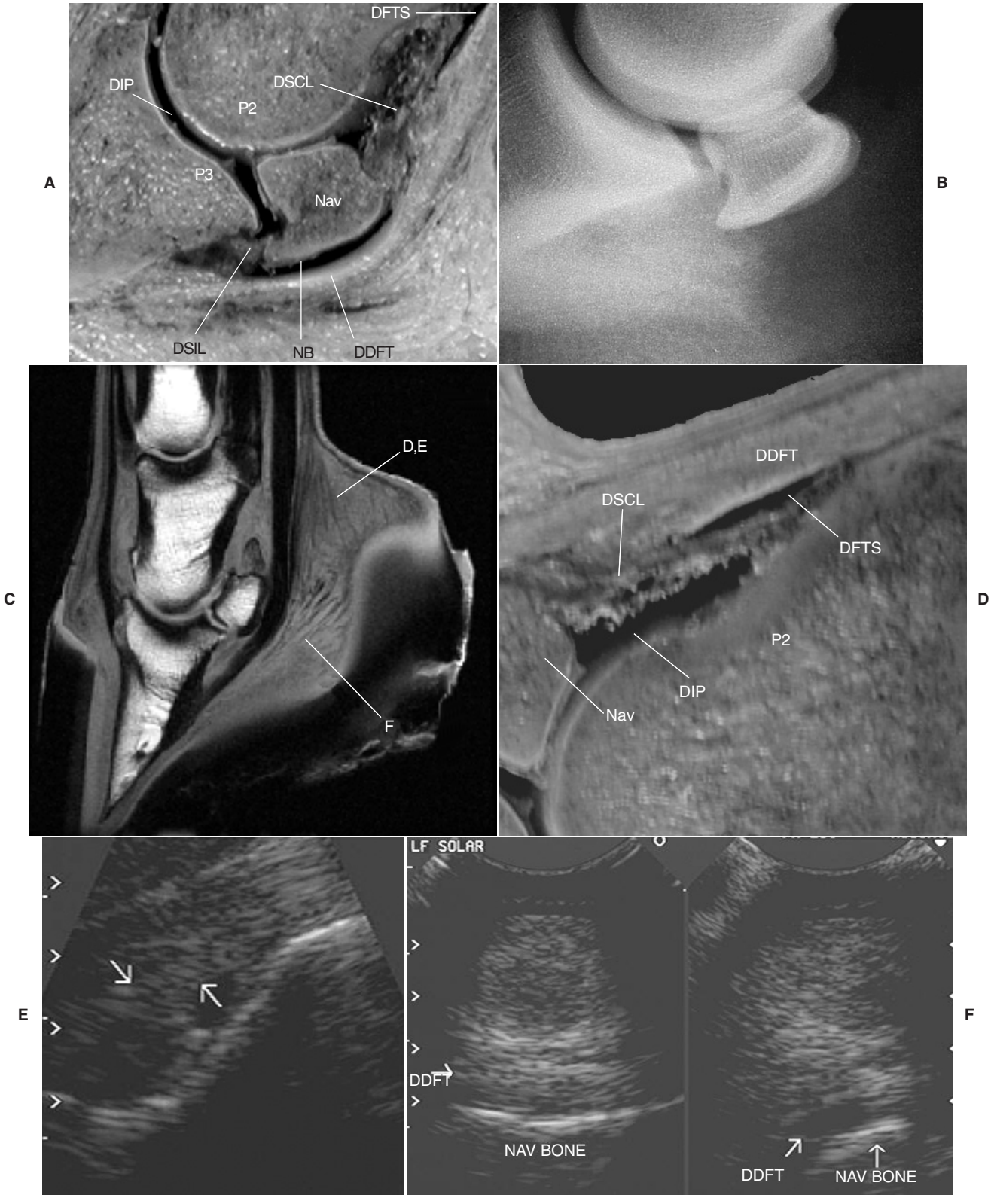

Fig. 9-13 Comparisons of the lateral view of the navicular bone and its relationship to neighboring structures. **A,** Sagittal anatomical section showing the digital flexor tendon sheath *(DFTS)*, navicular bursa *(NB)*, and distal interphalangeal *(DIP)* joint surrounding the navicular bone. *DSIL*, Distal sesamoidean impar ligament; *DSCL*, distal sesamoidean collateral ligament (axial union forming fibrous portion of T ligament); *P2*, middle phalanx; *Nav*, navicular bone; *P3*, distal phalanx. **B,** Lateral radiographic projection centered on the navicular bone. **C,** Sagittal magnetic resonance imaging scan of the foot. (Courtesy Alexia L. McKnight, University of Pennsylvania, Philadelphia, Pennsylvania.) **D** and **E,** Sagittal anatomical section and corresponding ultrasonographic image of the palmar aspect of the distal pastern obtained at points *D* and *E* on part **C.** Proximal is to the right. The *arrows* outline the distal sesamoidean collateral ligament. **F,** Frontal *(left)* and sagittal *(right)* ultrasonographic images obtained through the frog at point *F* in part **C.** The hypoechoic appearance to the portion of the deep digital flexor tendon *(DDFT)* is due to the off-incidence artifact because the fibers are not perpendicular to the line of the ultrasound beam. Lateral and proximal are to the right.

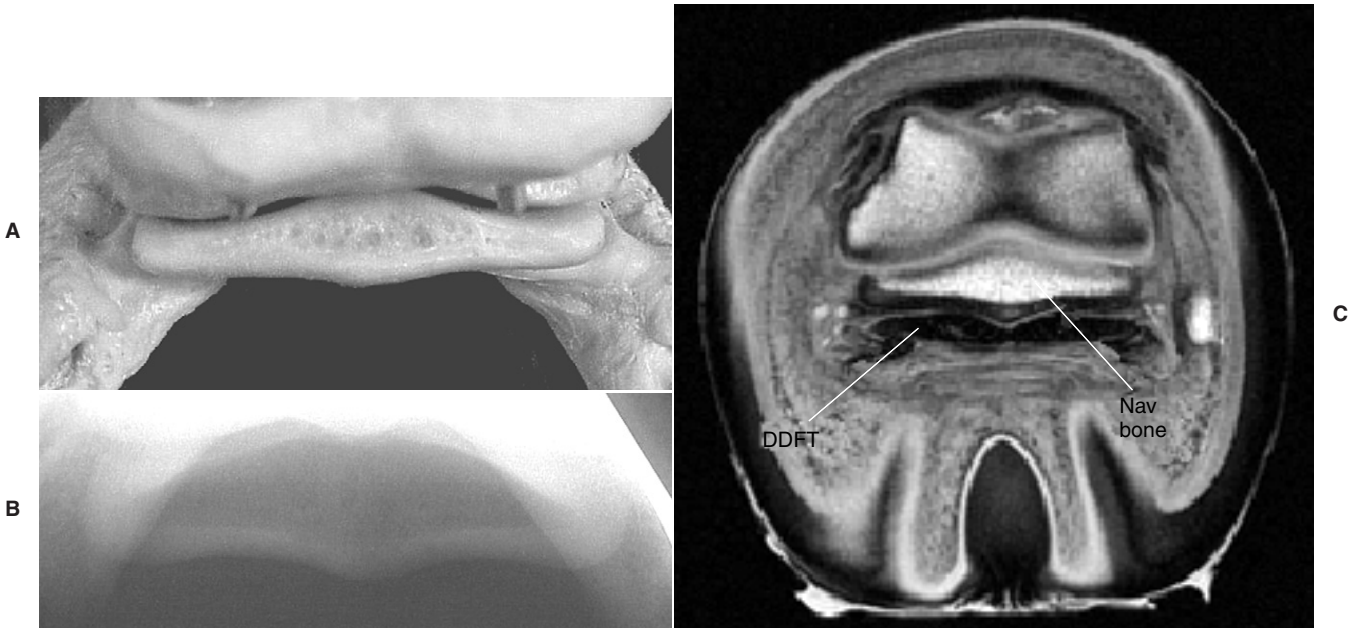

Fig. 9-14 Transverse sections through the navicular bone. **A,** Anatomical specimen, palmar view. **B,** Palmaroproximal-palmarodistal oblique radiographic projection of a normal navicular bone. **C,** Transverse magnetic resonance imaging scan. The deep digital flexor tendon *(DDFT)* is nearly as broad at this point as the navicular bone. (Courtesy Alexia L. McKnight, University of Pennsylvania, Philadelphia, Pennsylvania.)

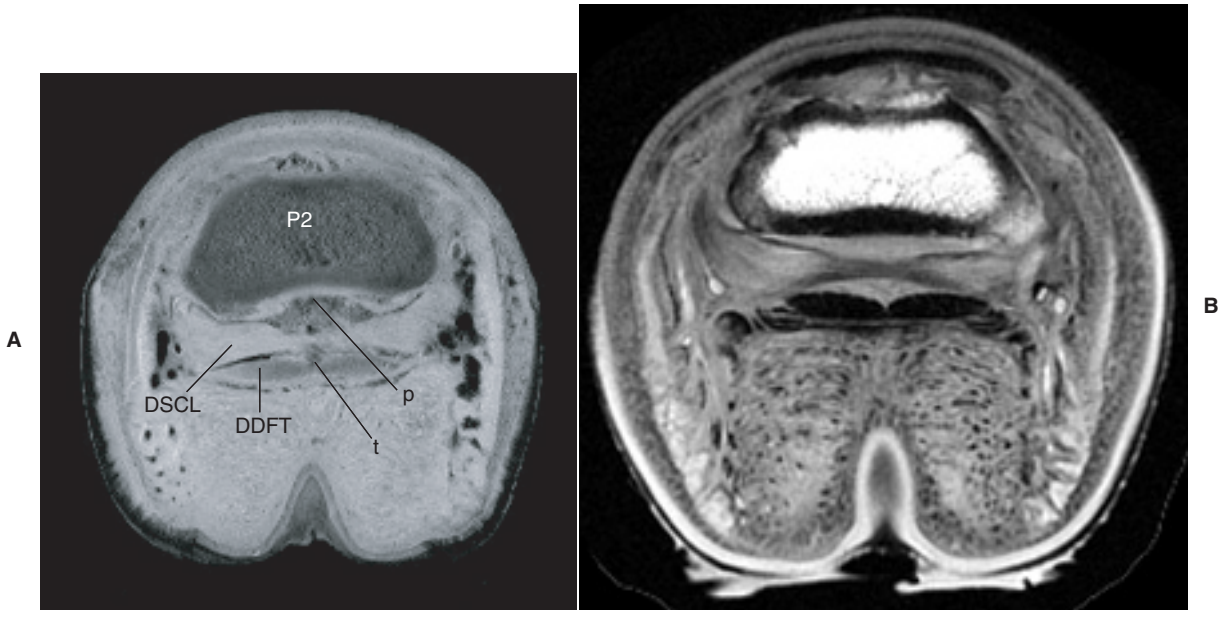

Fig. 9-15 Transverse sections through the foot at the level of the distal sesamoidean collateral ligaments *(DSCL)*. **A,** Anatomical section showing the attachments of the DSCL to the deep digital flexor tendon *(DDFT)* marked at point *t*, and to the middle phalanx *(P2)* at point *p*. These attachments form the so-called T ligament, which forms the boundaries between the navicular bursa, distal interphalangeal joint, and digital flexor tendon sheath. **B,** Corresponding magnetic resonance imaging scan. (Courtesy Alexia L. McKnight, University of Pennsylvania, Philadelphia, Pennsylvania.)

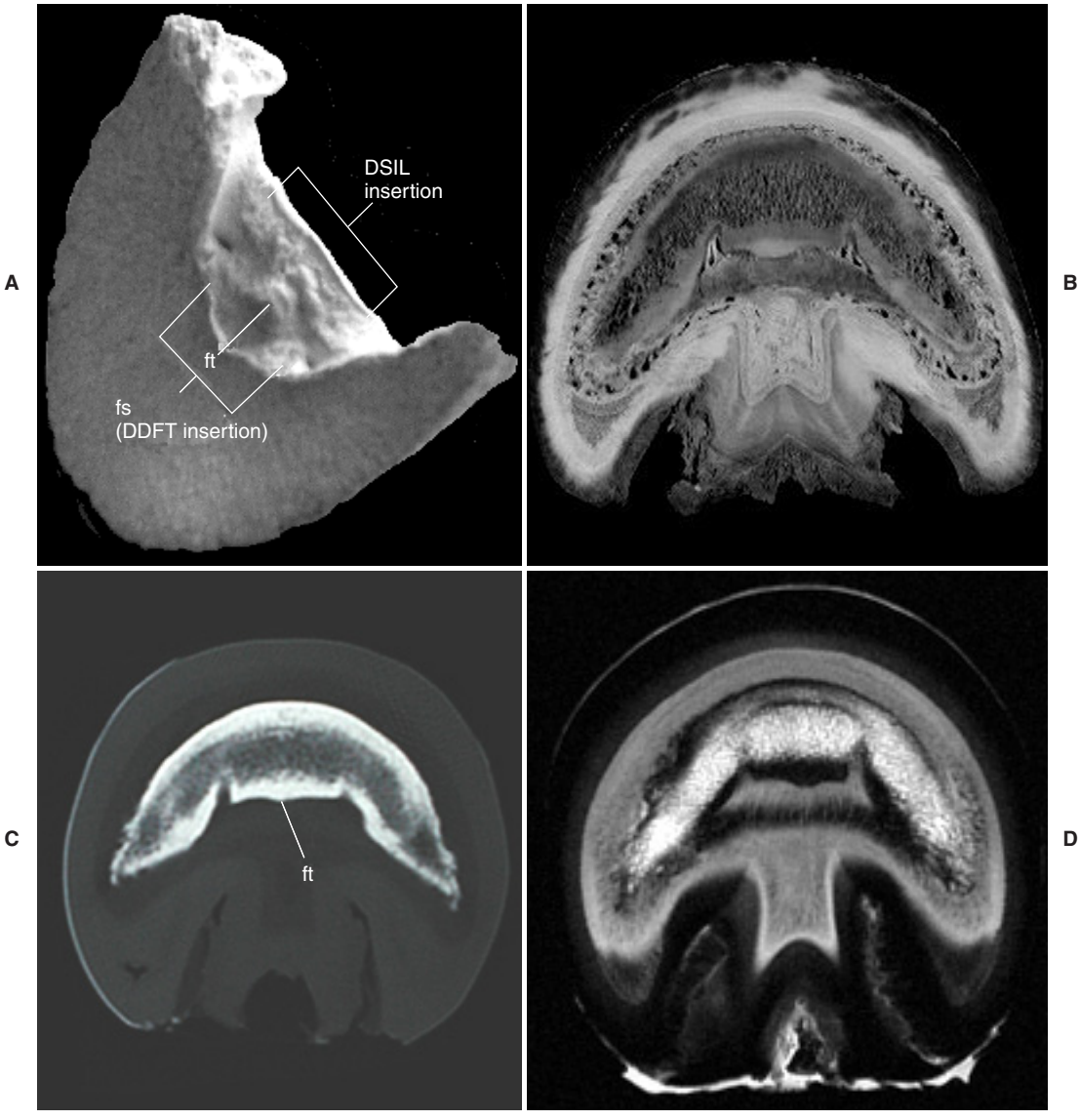

Fig. 9-16 The insertions of the deep digital flexor tendon *(DDFT)* and distal sesamoidean impar ligament *(DSIL)*. **A,** Isolated distal phalanx solar view, showing the point of insertion of the DDFT on the flexor surface. The flexor tubercle (see *ft* on part **C***)* is relatively smaller than in other species but should be recognized as a normal structure as seen on computed tomographic (CT) imaging. **B,** Transverse anatomical section through the insertion of the DDFT. This slice is slightly distal to the site of insertion of the distal sesamoidean impar ligament. **C,** Transverse CT scan showing a normal flexor tubercle *(ft)*. Avulsions here are difficult to demonstrate radiographically. Nuclear scintigraphy and ultrasonography can be helpful, but this area is best imaged using CT or magnetic resonance imaging (MRI). (Courtesy Alamo Pintado Equine Medical Center, Los Olivos, California). **D,** Transverse MRI scan. (Courtesy Alexia L. McKnight, University of Pennsylvania, Philadelphia, Pennsylvania.)

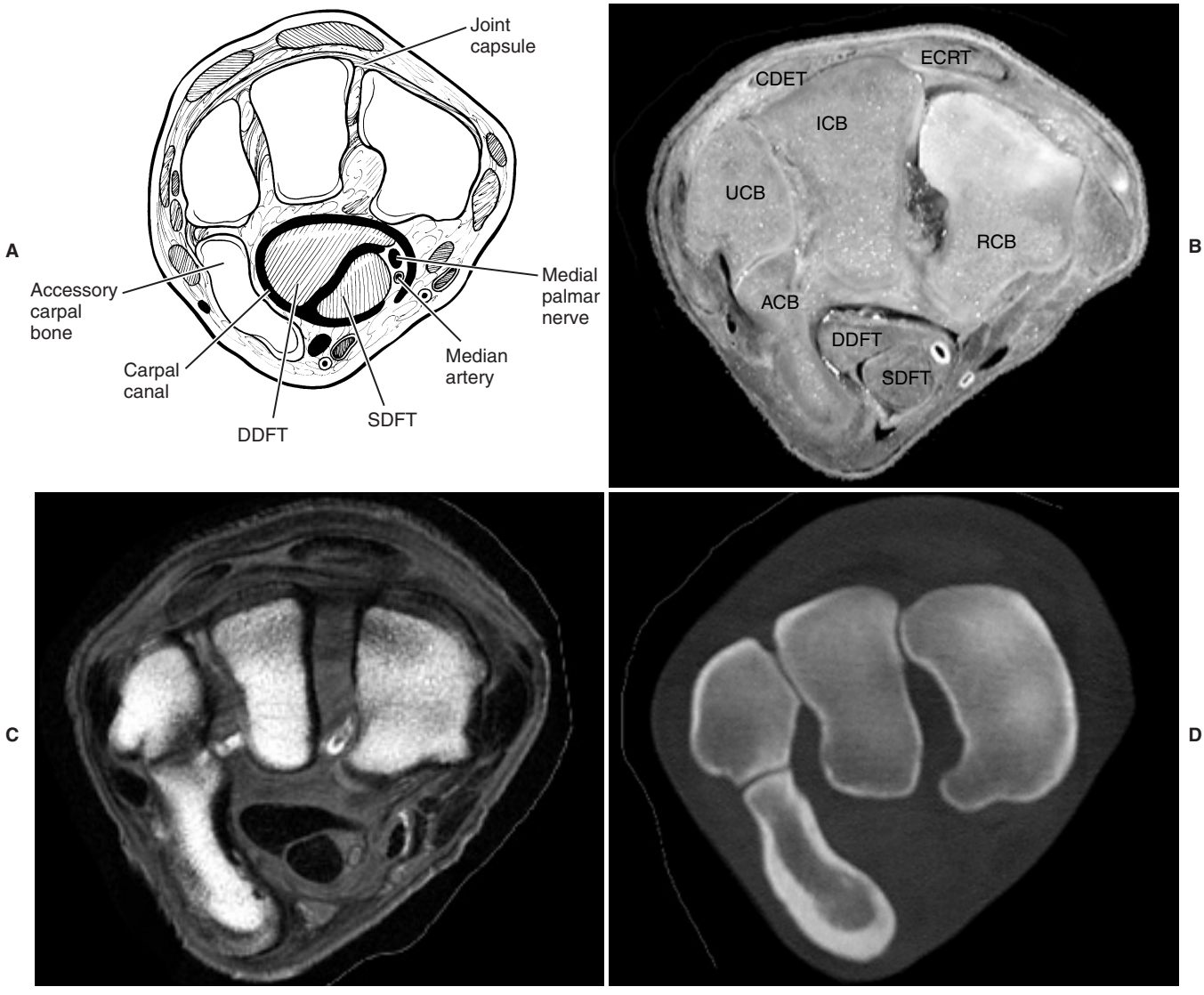

Fig. 9-17 Transverse slices through the proximal row of carpal bones. All images are oriented with dorsal to the top and lateral to the left. **A,** Diagram of carpal bones. **B,** Anatomical section. **C,** Computed tomographic scan. (Courtesy Alamo Pintado Equine Medical Center, Los Olivos, California.) **D,** Magnetic resonance imaging scan. (Courtesy Alexia L McKnight, University of Pennsylvania, Philadelphia, Pennsylvania.) *ACB,* Accessory carpal bone; *DDFT,* deep digital flexor tendon; *ICB* intermediate carpal bone; *RCB,* radial carpal bone; *SDFT,* superficial digital flexor tendon; *UCB,* ulnar carpal bone; C*DET,* common digital extensor tendon; *ECRT,* extensor carpi radialis tendon.

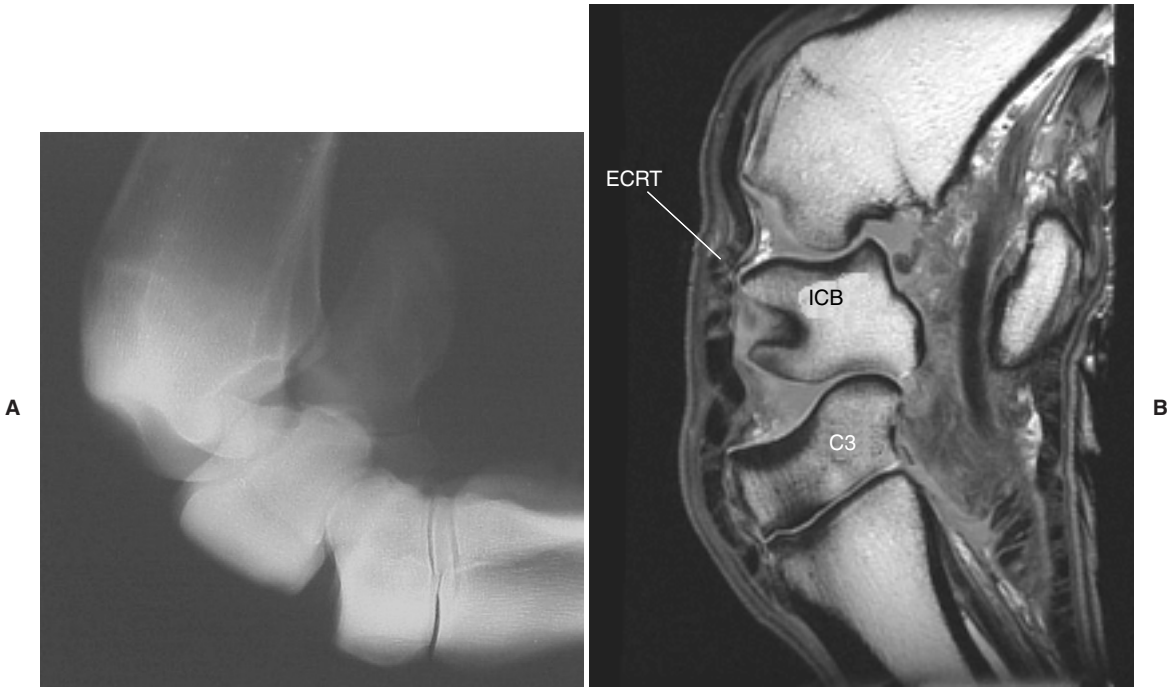

Fig. 9-18 Lateral views of the carpus. Compare with Fig. 9-1. **A,** Flexed lateral radiograph. **B.** Flexed sagittal magnetic resonance imaging scan through the extensor carpi radialis tendon *(ECRT)*. *ICB,* Intermediate carpal bone; C3, third carpal bone. (Courtesy Alexia L McKnight, University of Pennsylvania, Philadelphia, Pennsylvania.).

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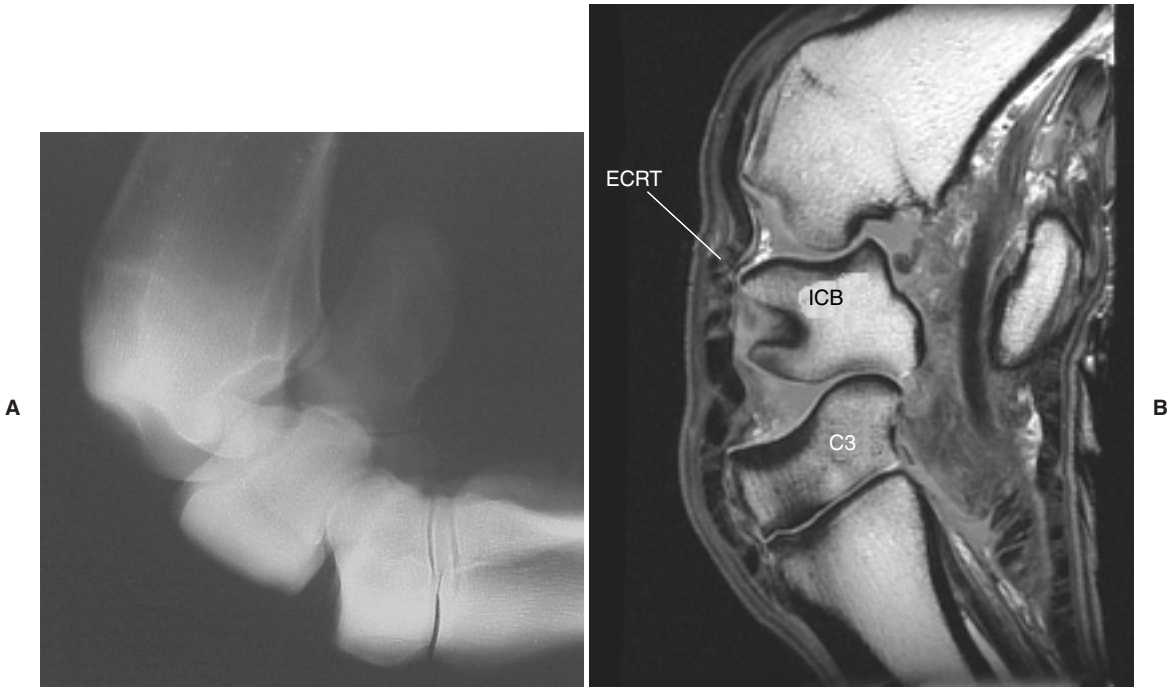
CHAPTER • 10

Diagnostic Analgesia

Lance H. Bassage II and Mike W. Ross

Despite the many technological advances in equine sports medicine over the past two decades, diagnostic analgesia arguably remains the most valuable tool in the equine clinician's arsenal to localize lameness. Although the technique requires a thorough understanding of anatomy, basic technical skill, and clinical experience, the equipment and expense are minimal. In addition, diagnostic analgesia can be performed on site, with the outcome immediately obvious. In this way, any lingering concern that a suspected "shoulder problem" exists is convincingly erased when the response to local analgesia of the digit is observed. This chapter reviews the various perineural, intrasynovial, and local (regional) infiltration techniques for application of local analgesia in the diagnosis of lameness in horses.



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LOCAL ANESTHETICS: PHARMACOLOGY AND TISSUE INTERACTIONS

Pain is transmitted specifically in the small, lightly myelinated, A-delta and non-myelinated C nerve fibers.[1] All commonly used local anesthetics, regardless of the specific molecular structure, share the same basic mechanism of action, specifically, the ability to block or inhibit nociceptive nerve conduction by preventing the increase in membrane permeability to sodium ions.[2] These agents consist of a lipophilic and a hydrophilic group, connected by an intermediate chain containing a carbonyl group of an amide or ester linkage, and have traditionally been categorized as either amide- or ester-type local anesthetics.[3] Common local anesthetics used in horses—2% solutions of lidocaine, mepivacaine, and bupivacaine—are of the amide type.

Compared with most local anesthetics, lidocaine and mepivacaine are considered relatively fast-acting and have a reported duration of action of 1½ to 3 hours and 2 to 3 hours, respectively. In contrast, bupivacaine is intermediate in onset but has a much longer duration of action (3 to 6 hours).[4] Bupivacaine is most suited for providing therapeutic rather than diagnostic analgesia. The results in clinical practice vary, because in *severely* lame horses the degree and duration of local analgesia are decreased, regardless of the agent used.

When local anesthetics are injected, tissue damage can occur but is extremely rare.[3,4] Soft tissue swelling occurs occasionally and is likely caused by needle trauma or hematoma formation and not from a direct drug-tissue interaction. We suggest that alcohol and a clean wrap be applied to the injection sites when the diagnostic evaluation is complete to prevent or minimize swelling at injection sites. Cellulitis or other forms of infection are rare potential complications.

Acute synovitis, or *flare*, is a rare complication that can occur after intrasynovial (most commonly intra-articular) injection of anesthetics. Synovitis from intrasynovial injection of local anesthetics is much less common than from injection of other medications. Mepivacaine is less irritating than lidocaine when administered intra-articularly, but we have not recognized this difference.[3] However, Dyson reported that lidocaine may be considerably more irritating than mepivacaine, and clinical data documenting differences was used successfully in the licensing of mepivacaine in the United Kingdom.[5] Like cellulitis after perineural injections of local anesthetics, infectious synovitis is a rare but possible sequela. To mitigate the possibility of contaminated solution, we use a new vial of local anesthetic solution when performing intrasynovial analgesic procedures.

Systemic side effects from diagnostic analgesic techniques are exceedingly rare. Cardiovascular or central nervous system signs, including muscle fasciculation, ataxia, and collapse, were reported.[3] Systemic intoxication would require a dose much higher than is commonly used, even for an extensive diagnostic evaluation. For example, the maximum single infiltration dose of lidocaine that can be safely administered to a 500-kg horse is about 6.0 g, or 300 ml of a 2% solution.[6]

Strategy, Methodology, and Other Considerations

A few basic principles must be followed to ensure success. A thorough working knowledge of regional anatomy is required. Even for seasoned veterans a review of anatomy may be required before performing less common techniques. A most important principle when performing perineural analgesia is to *start distally in the limb and work proximally* (Figs. 10-1 to 10-4). If possible, sequential blocks from distal to proximal should always be used, but in certain circumstances a different strategy can be successful. Sequential blocking requires a fair amount of time, and in certain horses, selective intra-articular or local blocks can be performed without following this "golden rule." However, in most situations, blocking a large portion of the distal limb at a proximally located site may preclude accurate determination of the source of lameness and may require an additional visit to perform additional diagnostic procedures.

It is important to test the efficacy of a perineural block before reevaluating the horse's degree of lameness. If any question exists, the block should be repeated rather than assuming deep pain has been abolished, when skin sensitivity persists. If a horse shows *partial* improvement only minutes after injection, an additional few minutes should be allowed to achieve complete analgesia before proceeding with the next block. Alternatively, the block can be repeated. In so doing, the clinician minimizes the potential for misinterpretation and the tendency to ascribe the residual lameness to a "second problem" that does not exist.

During this portion of the examination, we are attempting to eliminate baseline rather than induced lameness, and care must be taken when adopting the practice of "blocking out a positive flexion test" (see Chapter 8). Once baseline lameness has been eliminated, we rarely perform additional flexion tests or attempt to eliminate all induced lameness.

How is the efficacy of the block assessed? Several methods are available, but the following points should be considered. Individual horses react differently to noxious stimuli applied to the skin. Therefore it is helpful to test the contralateral (unblocked) limb to establish the horse's baseline response to the test. Similarly, covering a horse's eye or feigning a few gestures with an instrument (pen tip, hemostatic forceps) without actually contacting the skin can help differentiate between a random or anticipatory response by an apprehensive horse and a true painful response. Positioning oneself on the contralateral side of the patient when testing for sensation also can help in making this determination. The clinician should avoid using sharp instruments that can penetrate the skin and cause hemorrhage, a situation not well understood by a concerned horse owner. Hemostatic forceps, used to pinch the skin, are ideal, because they are blunt and appear to consistently induce an appropriate amount of pain. Forceps are only useful in assessing superficial or skin sensation, however.

Perineural blocks must be assessed for the amelioration of deep and not just superficial pain. To assess whether deep pain in the hoof has been ameliorated after palmar digital analgesia or other techniques, hoof testers can usually be applied with enough force to cause a painful response, even in the most stoic of horses. Physical strength of the operator must be considered. Extreme or hard joint flexion (combined with varus or valgus stress) can be used to assess whether deep pain has been abolished in more proximal locations. In some instances, however, it is impossible to avoid contacting the skin proximal to the site of anesthetic administration, leading the clinician to assume that the block has not worked. The application of firm digital pressure in the blocked area may be a viable alternative to flexion or manipulation to help avoid these potentially confounding factors.

It is important to understand that the region of the limb that is *actually* desensitized may, in fact, differ from that which the clinician has *intended*.[7] Proximal diffusion of local anesthetic solution appears to be the most likely cause, but other intangible factors may play a role. Using a small volume of anesthetic (1 to 5 ml for most perineural blocks) can minimize but not abolish this phenomenon. To further minimize the potential for diffusion of anesthetic, the horse should be reevaluated no more than 10 minutes after the injection (exceptions apply in certain situations).

Complete analgesia, and thus 100% improvement in lameness score, is the goal when performing diagnostic analgesia, but in many horses this level of pain relief is never achieved. Improvement in degree of lameness greater than 70% to 80%

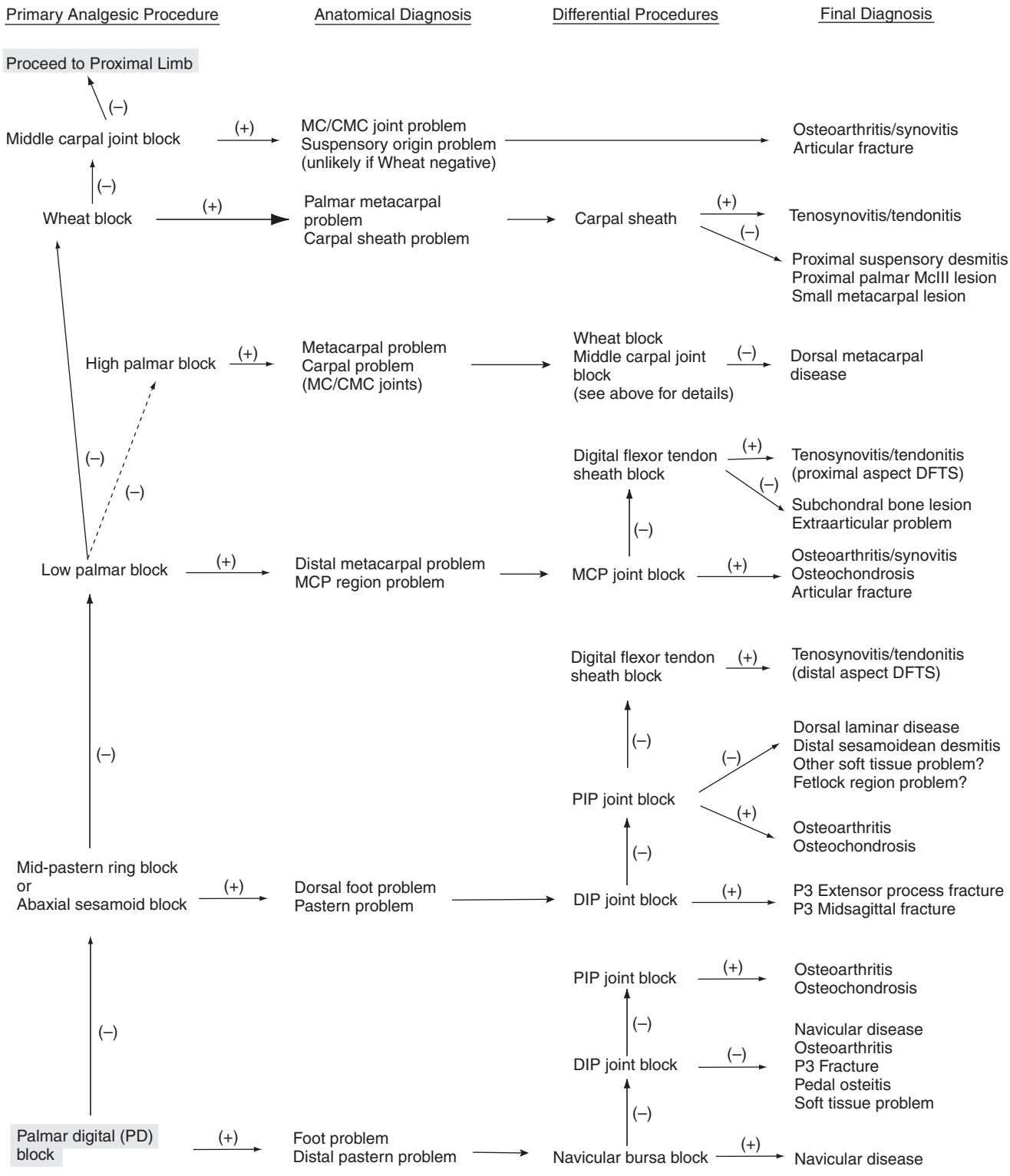

Fig. 10-1 Blocking strategy in the forelimb: foot to carpus. *CMC,* Carpometacarpal; *DFTS,* digital flexor tendon sheath; *McIII,* third metacarpal bone; *MC,* middle carpal; *MCP,* metacarpophalangeal; *PIP,* proximal interphalangeal; *Mc3,* third metacarpal bone; *P3,* distal phalanx.

after most perineural or intra-articular techniques should be considered a positive response in most horses. The quintessential response is the horse "switching lameness" to the contralateral limb, indicating that now, pain arising from the opposite limb is greater than that originating from the baseline lameness. However, complete response may not occur, and the clinician must decide when to stop sequential blocks or when the horse has "blocked out." The clinician hopes for an obvious difference in lameness score when the horse is blocked, but in some horses, serial improvement occurs with each successive block, a situation that makes assessing the primary source of lameness difficult.

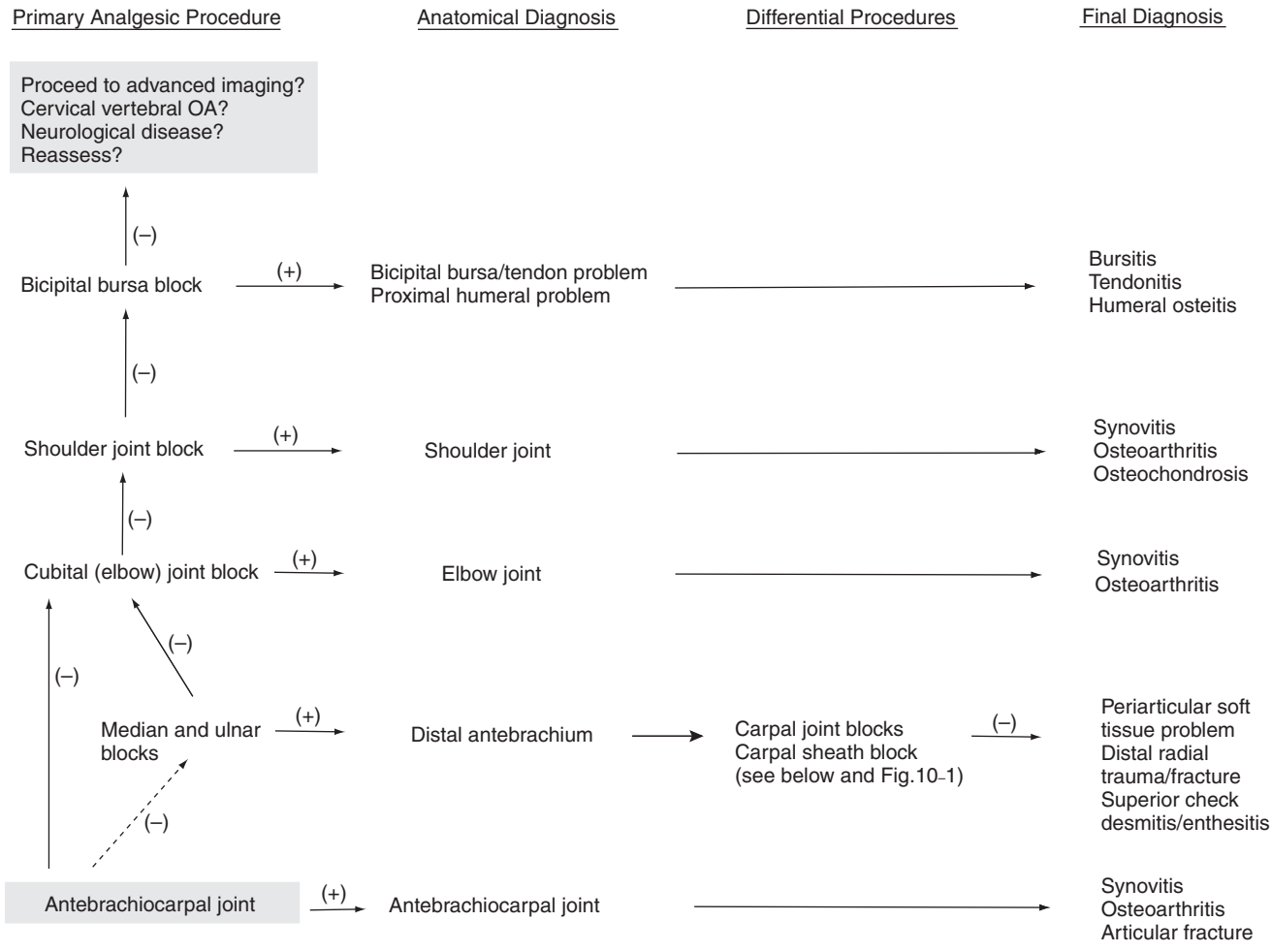

Fig. 10-2 Blocking strategy in the forelimb: antebrachium to shoulder joint, *OA*, Osteoarthritis.

Incomplete response to local analgesics in some horses may be explained by the fact that chronic pain, particularly deep bone pain, may remain resistant to complete analgesia when perineural techniques are used. For example, horses with laminitis tend to remain lame despite blocking many times at the appropriate level. Mechanical gait deficits do not improve after diagnostic analgesia because pain is minimal. Horses may continue to show lameness even with pain abolition, a situation that appears to be caused by habit. These horses tend to show mild residual lameness initially, only to warm out of it quickly during examination. Other factors affecting response to diagnostic analgesia include individual variation in neuroanatomy, the intermittent nature of certain lameness conditions, and the inherent difficulty in assessing and abolishing pain in horses with subtle lameness.[8] Articular lesions may not be desensitized by intra-articular analgesia.

Sensory innervation of joints is complex and involves three classes of neurons that transmit information from four receptor types, each of which has a specific distribution throughout the joint.[9-12] Articular pain can arise from several sources, including the synovium (inflammation, effusion), fibrous joint capsule (increased intra-articular pressure), articular and periarticular ligaments, periosteum, and subchondral bone (injury, osseous vascular engorgement).[8,10,13] Other than small branches in the perichondrium, articular cartilage is devoid of innervation. In osteoarthritic joints, however, erosion channels, formed in the calcified layer of cartilage, are invaded by subchondral vasculature.[10] Putative nociceptive neurotransmitters were identified in these areas, and therefore it is plausible that in horses with advanced osteoarthritis, pain could be emanating from the deep cartilage layers.[14,15]

On occasion, lameness from an articular lesion abates after perineural analgesia but shows minimal or only partial response after intra-articular analgesia. In some horses, this can be explained by the fact that pain is originating from articular and periarticular tissues.[8] Subchondral bone pain—caused by stress or non-adaptive remodeling, cystic or erosive lesions, incomplete fractures, and osteoarthritis—is inconsistently abolished by intra-articular analgesia. In fact, subchondral bone pain is abolished much more consistently by perineural techniques. Subchondral bone receives innervation from endosteal branches of peripheral nerves that enter the medullary cavity through the nutrient foramen.[8,9,16,17] Intra-articularly administered local anesthetic solutions may not penetrate subchondral bone sufficiently to completely block these nerves. This shortcoming is presumably even more likely in situations in which the overlying articular cartilage is intact.

Unfortunately, intra-articular analgesia, although easier to perform, inconsistently abolishes pain from many of the common articular problems. This fact, however, is either overlooked or misunderstood by many practitioners. Whenever possible, perineural analgesia should be performed, particularly in the distal limbs, because this type of analgesia more consistently abolishes pain from all aspects of the joint and surrounding soft tissue structures.

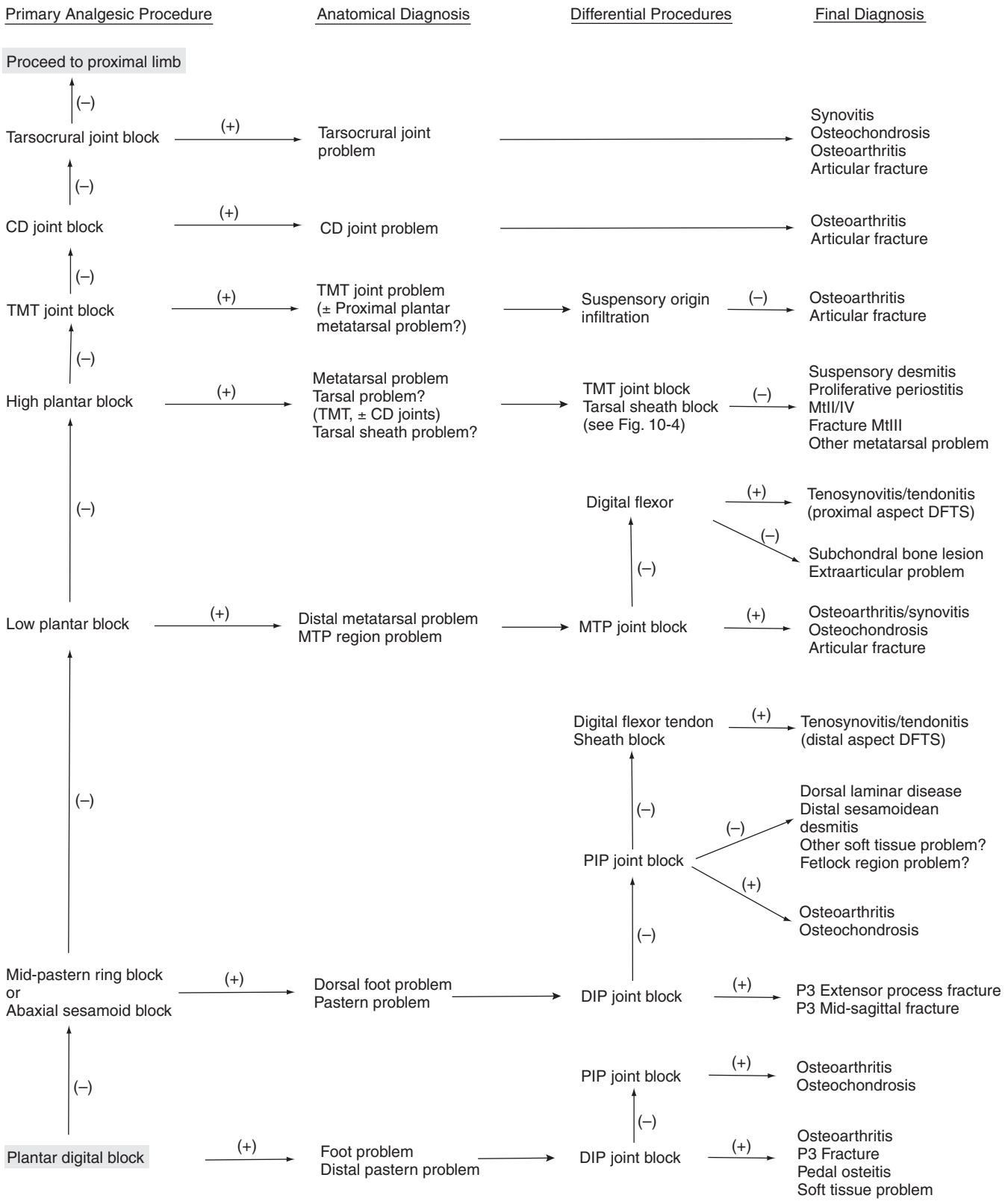

Fig. 10-3 Blocking strategy in the hindlimb: foot to hock joint. *CD,* Centrodistal; *DFTS,* digital flexor tendon sheath; *DIP,* distal interphalangeal; *MtII, MtIII, MtIV,* second, third, and fourth metatarsal bones, respectively; *MTP,* metatarsophalangeal; *P3,* proximal phalanx; *PIP,* proximal interphalangeal; *TMT,* tarsometatarsal.

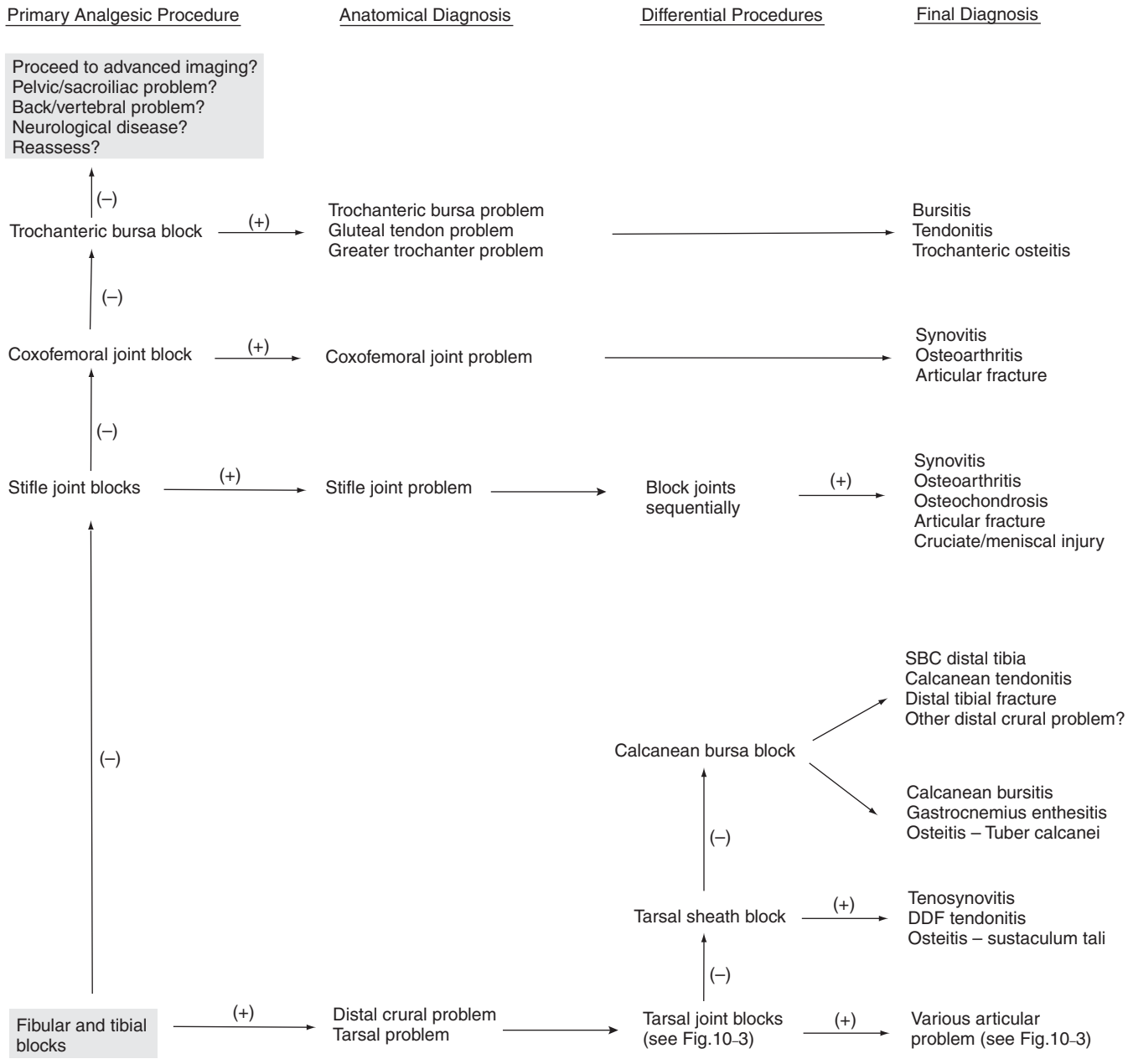

Fig. 10-4 Blocking strategy in the hindlimb: crus to coxofemoral joint. *DDF,* Deep digital flexor; *SBC,* subchondral bone cyst.

Perception of Diagnostic Analgesia by Laypersons

One of the intangible factors that can complicate the lameness examination is the layperson's perception of diagnostic analgesia or nerve blocking. In many instances the opportunity for an owner or trainer to observe the outcome of diagnostic analgesia provides the concrete evidence that finally convinces him or her of the diagnosis. The classic example is the acute shoulder injury that is actually chronic navicular disease. However, for many reasons, misunderstanding about diagnostic analgesia can lead to frustration for everyone involved. Many laypersons are not fully able to recognize the baseline lameness and therefore may not be capable of seeing that the horse's lameness improves after the block. Another difficulty is trying to explain why lameness in a horse with an articular problem is better after a perineural block but no better when local anesthetic solution is placed directly into a joint. Similarly, many laypersons do not understand why a horse with an articular lameness may "block sound" but does not respond satisfactorily to therapeutic injection. This finding that a horse blocks sound but does not inject sound is quite common in young racehorses with subchondral bone pain. Most experienced practitioners have learned to deal with these issues, but the new graduate may need fortitude and ingenuity when explaining the results of diagnostic analgesia is difficult.

Role of Chemical Restraint

Whenever possible, use of physical (nose or shoulder twitch) rather than chemical restraint is best when performing diagnostic analgesia. This is particularly important in horses with low-grade lameness. The analgesic properties of α_2-agonists (xylazine, detomidine) and synthetic opiates (e.g., butorphanol) are well recognized and may lead to false-positive

results. Ataxia after sedation can complicate lameness interpretation. However, in some horses mild sedation or tranquilization may be necessary to perform diagnostic analgesia and may improve the clinician's ability to evaluate the baseline lameness. Acetylpromazine (0.02 to 0.04 mg/kg intravenously) can calm a highly strung horse and facilitate the lameness examination. Extra care must be taken when performing hindlimb procedures, and the safety of everyone involved and the horse must be considered. In horses with moderate or severe lameness, xylazine (0.15 to 0.30 mg/kg intravenously) may not interfere appreciably with lameness interpretation. Similarly, extremely fractious horses can be sedated with an α_2-agonist, which then is reversed with the prescribed α_2-antagonist (e.g., yohimbine) before reevaluation. Alternatively, sedation can simply be allowed to wear off before reevaluating the horse, but diffusion of anesthetic may occur or the effect may wear off, both of which may potentially cause misinterpretation of results.

Patient Preparation

Before perineural analgesia is performed, the skin and hair should be cleaned of any gross debris such as mud, bedding, feces, or poultice. Clipping usually is not necessary unless the hair coat is long and prohibits accurate palpation of anatomical landmarks or adequate cleaning of the site. The site should then be scrubbed with an antiseptic, such as povidone-iodine or chlorhexidine, using clean gauze sponges or cotton. If the clinician has any concern about inadvertent penetration of a synovial cavity, a 5-minute aseptic preparation should be performed. This is followed by isopropyl alcohol administration over the site using cotton or gauze sponges.

Aseptic preparation should always be performed before any intrasynovial injection. Considerable debate and variation exists among clinicians regarding the need to clip the hair over the site. Some clinicians always clip the hair, whereas others never do. Still others shave the hair in a small area directly over the injection site. The results of a recent study indicated no significant difference in the number of post-scrub colony-forming units (bacterial flora) between clipped and unclipped skin over the distal interphalangeal (DIP) and carpal joints.[18] Nonetheless, we still clip the hair over all proposed intrasynovial injection sites before undertaking a 5-minute aseptic preparation. The only time we deviate from this policy is when we are specifically asked not to clip the hair, a situation that arises in some sport horses actively competing, in claiming horses, or in those being sold.

Similar variation among clinicians exists regarding wearing of sterile latex gloves when performing an intrasynovial injection. However, we recommend wearing sterile gloves during these procedures. Science aside, clipping hair and wearing sterile gloves project a positive impression to all in attendance.

How does, or should, the practitioner attempt perineural or intrasynovial analgesia in a horse with contact or chemical dermatitis (scurf) over the proposed injection site? A superficial wound or abrasion with a localized infection presents a similar quandary. For obvious reasons, these areas are difficult, if not impossible, to clean effectively. If possible, an alternative site, away from the area of dermatitis, should be used. If not, then the procedure should be delayed until the skin condition (or wound) has resolved. In many instances, dermatitis can be treated with topical medications (medicated sweats such as nitrofurazone-dimethylsulfoxide) for a few days to facilitate resolution of the problem.

Injection Techniques

Perineural injections are typically performed using needles ranging in size from 25 gauge, 1.6 cm (⅝ inch) to 20 gauge, 2.5 or 4 cm (1 to 1½ inches). Small needles cause less pain but carry the risk of breaking off within tissues if the horse kicks out or otherwise misbehaves. For this reason, we recommend using 18-or 19-gauge needles for injections or blocks within the proximal metatarsal or plantar tarsal regions. In the distal limb the needle is inserted subcutaneously directly over and parallel to the nerve. We generally direct the needle proximally rather than distally, although this portion of the procedure differs among clinicians. The needle is inserted before the syringe is attached. To avoid excessive manipulation once the needle is inserted, a slip-type syringe hub is preferred. Syringes with screw-on hubs can be difficult to attach, requiring additional manipulation in a sometimes fractious horse, and are not generally used. However, when dense tissue requires that additional force be used for injection, the seal between the hub and the needle can be broken, a complication minimized by using a screw-on hub (see the following discussion of lateral palmar block).

Volume of local anesthetic solution varies, but for a majority of blocks in the distal limb 1 to 5 ml is injected at each site. Larger volumes are used to perform the median/ulnar and fibular (peroneal)/tibial techniques and when infiltrating the proximal palmar (plantar) metacarpal (metatarsal) region. After injection, we briefly massage the sites with gauze sponges or clean cotton soaked in alcohol. Skin sensation and deep pain are assessed between 5 and 10 minutes after injection. More time is allowed under certain circumstances (see specific comments throughout the chapter). At the completion of the examination an alcohol wrap should be applied to minimize swelling, a common sequela resulting from local irritation and bleeding from nearby vessels.

For "ring" blocks, circumferential subcutaneous infiltration of local anesthetic solution, and other local or regional infiltration techniques, we most commonly use 18- to 22-gauge, 4-cm needles. To perform a ring block, the needle is inserted perpendicular to the long axis of the limb, and local anesthetic solution is injected as the needle is advanced, leaving a clearly visible wheal or subcutaneous bleb in most locations. The needle then is reinserted at the leading edge of this wheal, a practice that minimizes the number of injections and the horse's discomfort. However, most horses object to needle insertion even when it is performed well within the bleb. The injection is continued around the limb in this manner. For most ring blocks in the distal limb, 10 to 15 ml of local anesthetic solution is used, but larger volumes may be preferred for surgical procedures. Ring blocks can be done as a substitute for or in combination with perineural injections (see the specific blocking techniques discussed in the chapter). However, simply placing anesthetic in a subcutaneous location is not a substitute for the preferred approach, direct perineural injection.

To block a local area such as a splint or curb, the needle is typically inserted in one or two locations, and local anesthetic solution is deposited in a fan-shaped pattern. As with the perineural analgesia, the sites are massaged briefly and the horse is reevaluated in 5 to 10 minutes.

Intrasynovial injections typically are performed using needles ranging in size from 22 gauge, 2.5-cm to 18 gauge, 4-cm. If marked effusion is present, drainage of synovial fluid is advised, either by allowing the fluid to drip from the hub of the needle or by aspirating with a sterile syringe before proceeding with injection. We prefer the former procedure unless fluid analysis is necessary. The manipulation required to attach the syringe may cause the horse discomfort and potentially dislodge the needle but, if successful, may hasten withdrawal of synovial fluid. Brief evaluation of the color and viscosity of synovial fluid can shed some light on the disease process within and is expected practice among most racehorse trainers. Volume of local anesthetic solution varies considerably between synovial cavities, but the clinician should keep in mind that small volumes might contribute to a false-negative result. False-negative results are common in horses with

severe osteoarthritis, and larger volumes of local anesthetic solution should be used. We routinely spray or wipe antiseptic solution over the injection site. After the examination a light bandage is applied over the injection sites from the metacarpophalangeal/metatarsophalangeal joint, distally. Initial reevaluation is done 5 to 10 minutes after injection. Additional evaluations may be necessary depending on the response during the initial time period. General practice is to have the horse walked in hand or with a rider after perineural or intrasynovial analgesia is administered, a procedure thought to hasten local distribution of anesthetic solution and potentially improve success. Excessive diffusion of local anesthetic solution is a potential drawback to this practice, particularly with techniques such as distal interphalangeal or middle carpal analgesia (see the following discussion), although it would be a complication difficult to quantify.

Another issue to consider when performing diagnostic analgesia is whether riding or driving a horse after blocks have been performed is safe. In general, riding on the flat or driving a horse at slow speed after any of the common blocks are performed is safe. Stumbling or knuckling can be a concern after upper limb perineural techniques, such as the median/ulnar and fibular (peroneal)/tibial techniques. Common sense should prevail, however, with regard to the horse and rider negotiating fences or performing at high speed. Horses at risk of lameness from stress or incomplete fracture are candidates for imaging before evaluation at speed after diagnostic analgesic techniques have been performed.

PERINEURAL ANALGESIA IN THE FORELIMB

Palmar Digital Analgesia

Palmar digital analgesia (or palmar digital block) is the most common diagnostic analgesic procedure performed. The medial and lateral digital neurovascular bundles, consisting, in a dorsal to palmar direction, of the digital vein, artery, and nerve, course in an abaxial location to the digital flexor tendons. With the exception of small breeds or Draft horses with exceptionally long-haired pasterns (feathers), the palmar digital nerve is easily palpable between the proximal sesamoid bones and the cartilages of the foot. The palmar digital block can be performed with the horse in a standing position or with the limb held off the ground. We prefer the latter. If held by an assistant, the limb should be grasped in the mid-metacarpal region, with the fetlock and digit hanging in neutral position. The palmar digital nerve is easily palpated in this extended position. Alternatively, the clinician performing the block can hold the limb, a technique that requires practice. The clinician can stand facing backward with a hand grasping the mid-pastern region or can stand behind the limb and clutch the hoof between both legs.

A 25-gauge, 1.6-cm needle is inserted subcutaneously, directly over the nerve, just proximal to the cartilages of the foot (Fig. 10-5). One of us (L.H.B.) directs the needle in a distal direction, whereas the other (M.W.R.) directs the needle in a proximal direction to avoid deeper penetration or laceration of digital vessels if the horse withdraws the limb. Alternatively, a 22-gauge, 4-cm needle can be inserted on the palmar midline in the mid-pastern region, and local anesthetic solution is then infiltrated in a V-shaped pattern. This modification of the PD block is quite difficult to perform in the hindlimb, but when done in the forelimb, provides maximal analgesia to the bulbs of the heel and minimizes the potential of depositing local anesthetic solution dorsal to the nerve. Loss of skin sensation in the midline between the bulbs of the heels should be assessed, because this area seems most recalcitrant to palmar digital analgesia. Deep pain is assessed using hoof testers.

Traditionally the PD block was felt consistently to desensitize only the palmar (plantar) one third to one half of the foot.[19] However, in clinical practice, this block desensitizes 70% to 80% of the foot. Most of the distal interphalangeal joint is affected, with the exception of the proximodorsal aspect. Horses with fractures of the extensor process of the distal phalanx may show partial improvement after palmar digital analgesia, however. Our clinical observations have been substantiated in a recent study. Setscrews were placed near the medial and lateral aspects of the toe to simulate pain from the sole. Lameness in these horses was abolished using palmar digital analgesia performed just proximal to the heel bulbs.[20]

Classically, most horses that responded positively to palmar digital analgesia were thought to have navicular syndrome, but this block desensitizes many lameness conditions within and outside the hoof capsule (Table 10-1). This is an important and common misconception. Lameness in horses with mid-sagittal fracture of the proximal phalanx or other conditions involving the fetlock joints can be abolished using palmar digital analgesia.[7] Although using small volumes of anesthetic solution and performing the block just above the cartilages of the foot may help to minimize the area of analgesia, these procedures do not prevent inadvertent diagnosis in some horses. Diffusion of anesthetic solution is the most likely explanation, and even a small volume can readily spread in a proximal direction, but the normal anatomy of the digit prevents distal placement of anesthetic solution (Fig. 10-6).

The concept that palmar digital analgesia abolishes lameness in an area considerably more than the palmar (plantar) one third of the foot appears to be difficult for many to accept. Although results of studies are widely published and this finding has been emphasized at international meetings, most veterinary students still graduate today armed with this common misconception. Diffusion of anesthetic solution easily explains why lameness conditions in the proximal aspect of the pastern or fetlock regions are desensitized by palmar digital analgesia. But what about the innervation of the hoof itself? Skeptics should consider the anatomy of the palmar digital nerve. Most practitioners have severed the palmar digital nerve while performing neurectomy. Can the clinicians recall any instance of having identified a large dorsal branch, or for that matter, any branching of the nerve at all? The lack of nerve branches in the mid-pastern region is circumstantial evidence that important innervation to the structures located dorsally within the hoof capsule occurs farther proximally (ill-defined dorsal branches) or after the nerve courses deep to the collateral cartilages of the foot. It makes little sense that ill-defined dorsal branches would innervate the dorsal two thirds of the foot, leaving the robust palmar digital nerve to innervate only the palmar one third.

Accurately quantifying the contribution of the palmar digital nerve to the innervation of the foot or, for that matter, the exact percentage of structures desensitized by palmar digital analgesia may be impossible. Clinical experience will undoubtedly convince practitioners of the broad nature of palmar digital analgesia. Finally, it is imperative to develop expertise in diagnostic imaging of the entire digit, because the many lameness conditions affected by palmar digital analgesia require detective-like differential diagnostic skills.

Mid-Pastern Ring Block

Traditionally the diagnostic blocks performed after palmar digital analgesia are the basisesamoid or abaxial sesamoid techniques. The basisesamoid block provides little additional information compared with palmar digital analgesia, unless, of course, the dorsal branch, originating from the digital nerve at the level of the proximal sesamoid bone, is blocked. If, however, the dorsal branch is blocked, then the basisesamoid block is in reality an abaxial sesamoid block. For this reason, we rarely

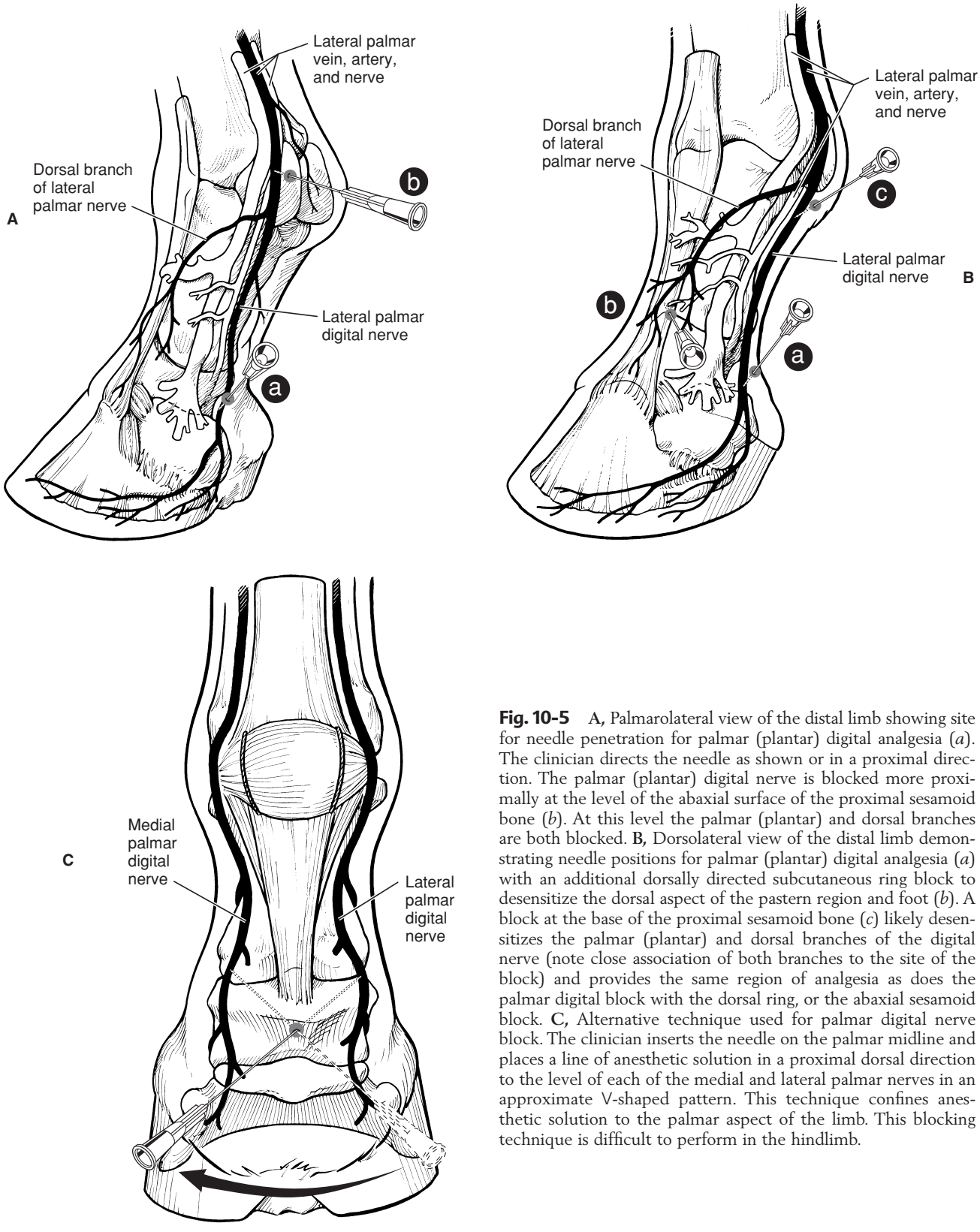

Fig. 10-5 **A,** Palmarolateral view of the distal limb showing site for needle penetration for palmar (plantar) digital analgesia (*a*). The clinician directs the needle as shown or in a proximal direction. The palmar (plantar) digital nerve is blocked more proximally at the level of the abaxial surface of the proximal sesamoid bone (*b*). At this level the palmar (plantar) and dorsal branches are both blocked. **B,** Dorsolateral view of the distal limb demonstrating needle positions for palmar (plantar) digital analgesia (*a*) with an additional dorsally directed subcutaneous ring block to desensitize the dorsal aspect of the pastern region and foot (*b*). A block at the base of the proximal sesamoid bone (*c*) likely desensitizes the palmar (plantar) and dorsal branches of the digital nerve (note close association of both branches to the site of the block) and provides the same region of analgesia as does the palmar digital block with the dorsal ring, or the abaxial sesamoid block. **C,** Alternative technique used for palmar digital nerve block. The clinician inserts the needle on the palmar midline and places a line of anesthetic solution in a proximal dorsal direction to the level of each of the medial and lateral palmar nerves in an approximate V-shaped pattern. This technique confines anesthetic solution to the palmar aspect of the limb. This blocking technique is difficult to perform in the hindlimb.

perform the basisesamoid block. When performing the abaxial sesamoidean technique in racehorses or, for that matter, any sport horse with a propensity to develop lameness of the metacarpophalangeal or metatarsophalangeal joints, the veterinarian runs the risk of an additional misdiagnosis. When local anesthetic solution is deposited in a location abaxial to the proximal sesamoid bones, pain from the metacarpophalangeal or metatarsophalangeal joints can be inadvertently blocked, explained most likely because of anesthetic diffusion, leading the clinician to assume the horse has a problem in the foot or

Table • **10-1**

Differential Diagnostic Analgesia of the Equine Foot

DISEASE	PALMAR DIGITAL NERVE BLOCK	DISTAL INTERPHALANGEAL JOINT BLOCK	NAVICULAR BURSA BLOCK
Navicular disease	+	±	+
Synovitis DIP joint	+	+	–
Osteoarthritis DIP joint	+	+	–
Subchondral bone DIP joint	+	±	–
P3 Fracture (wing)	+	+	–
P3 Fracture (midsagittal)	±	+	–
Extensor process fracture (P3)	±	+	–
Pedal osteitis	+	±	–
Subsolar abscess	+	±	–
Solar pain (heel/quarter)	+	±	–
Solar pain (toe)	+	±	–
DDF tendonitis	+	–	–
DDF enthesitis (P3 insertion)	+	±	–
Sheared heels	+	–	–
Quittor	+	–	–
Laminitis (toe)	–	–	–
Laminitis (quarter/heel)	+	±	–
Toe crack	–	–	–
Quarter crack/heel crack	+	±	–
Distal sesamoidean desmitis	±	–	–
PIP joint problem	±	–	–
DFTS problem	±	–	–
P2 Fracture	±	±	–
P1 Fracture	±	–	–

DIP, Distal interphalangeal; *P3,* distal phalanx; *DDF,* deep digital flexor; *PIP,* proximal interphalangeal; *DFTS,* digital flexor tendon sheath; *P2,* middle phalanx; *P1,* proximal phalanx.

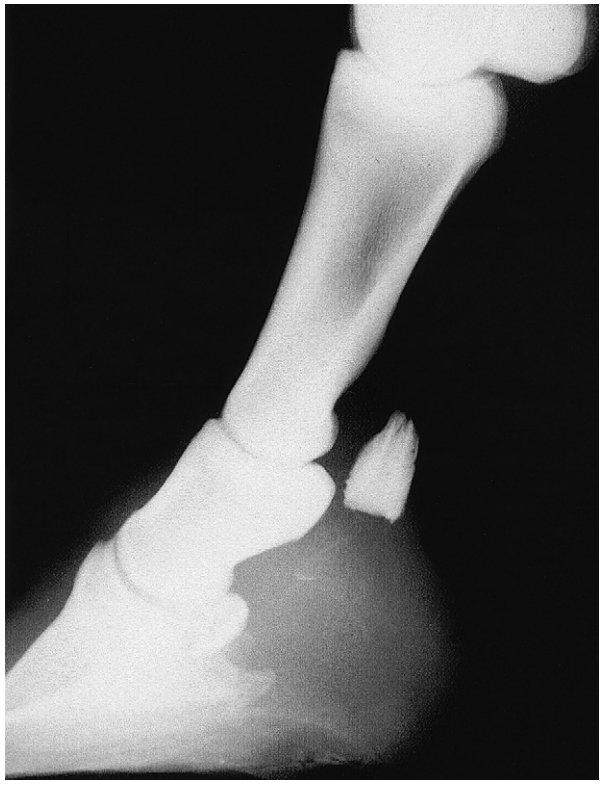

Fig. 10-6 Radiograph showing palmar digital anesthesia performed with positive contrast material. The clinician performed palmar digital anesthesia as far distal as possible, but the injection site is still at the level of the proximal interphalangeal joint, explaining why palmar digital anesthesia desensitizes most of the foot and the pastern region in some horses.

digit, but in reality the pain originated from these joints. For these reasons, we prefer to use a blocking sequence as follows: palmar digital nerve, followed by a dorsally directed subcutaneous ring block, followed by the low palmar or plantar block.

The mid-pastern ring block affects the dorsal branches of the digital nerves and desensitizes any remaining areas of the foot and pastern region that were not affected by palmar digital analgesia. In most horses, this includes the dorsal 20% of the foot (dorsal laminar and extensor process regions of the distal phalanx), and dorsal pastern region (middle phalanx, proximal interphalangeal joint, and distal portions of the proximal phalanx). Although desirable, performing the dorsal ring block just above the cartilages of the foot usually is not possible. Instead the block is performed at the level of the mid-pastern region.

A 20- to 22-gauge, 4-cm needle is used to deposit subcutaneously 10 to 12 ml of local anesthetic solution, beginning near the injection site used for palmar digital analgesia over the lateral neurovascular bundle and continuing dorsally and medially, ending over the medial neurovascular bundle (see Fig. 10-5). Resistance to needle advancement and injection of local anesthetic solution will invariably be encountered dorsally, if the block is done just proximal to the coronary band, because of the dense tissue (proximal interphalangeal joint capsule, extensor branches of the suspensory ligament, and extensor tendons). Performing the block in the mid-pastern region minimizes this problem and mitigates the potential for inadvertent penetration of the proximal interphalangeal joint.

Abaxial Sesamoid Block

Desensitizing the medial and lateral palmar nerves at the level of the proximal sesamoid bones is commonly referred to as the abaxial sesamoid block but may provide the same information as the basisesamoid block, if the dorsal branch of the palmar digital nerve is blocked. To avoid redundancy, we rarely perform the basisesamoid technique before progressing to the

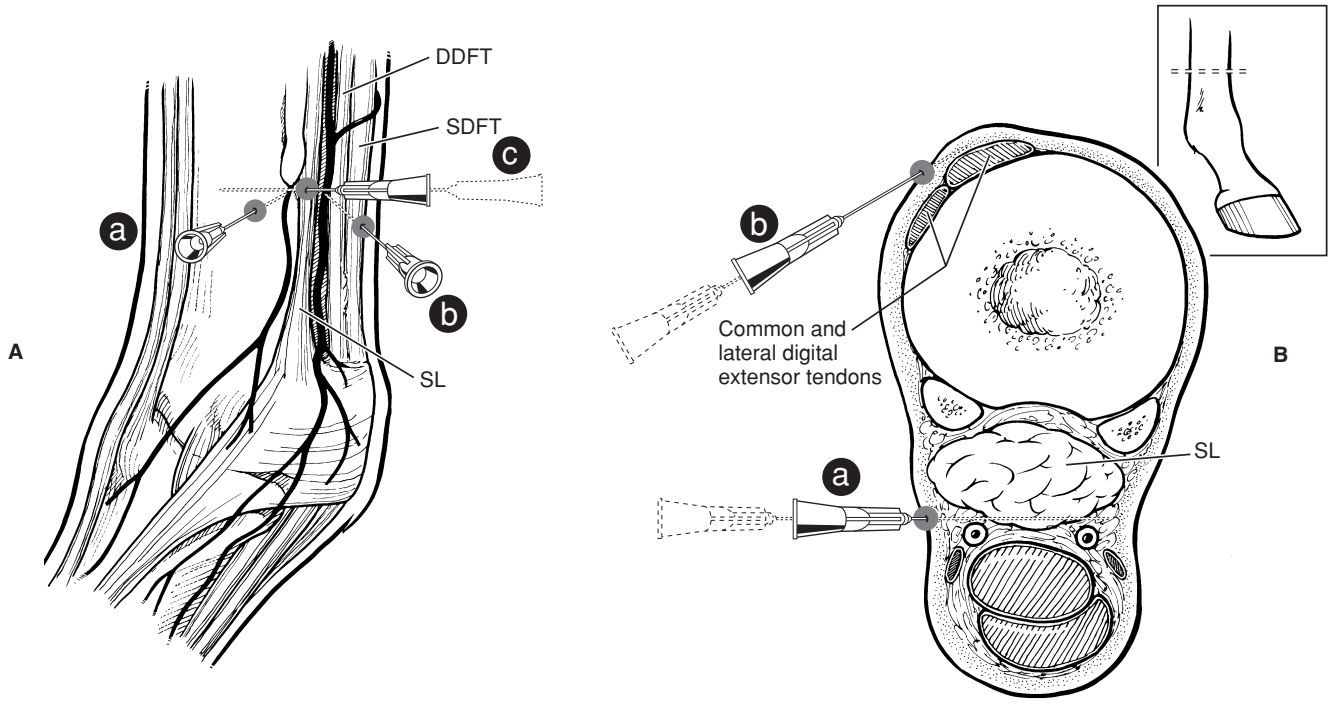

Fig. 10-7 **A,** This lateral view shows needles positioned for a low palmar (plantar) nerve block. The clinician inserts a needle (*a*) just distal to the distal aspect of the fourth metacarpal or metatarsal bone and directs it axially to block the lateral palmar (plantar) metacarpal (metatarsal) nerve. The clinician then inserts a needle (*b*) between the suspensory ligament (*SL*) and deep digital flexor tendon (*DDFT*) to block the lateral palmar (plantar) nerve. The clinician repeats the two injections on the medial side. A subcutaneous ring block from the first injection site around to the dorsal midline (*c*) completely abolishes skin sensation. **B,** Transverse view of the distal left metacarpal region demonstrating an alternative technique for low palmar (plantar) analgesia. The clinician inserts a needle (*a*) in a lateral-to-medial direction between the deep digital flexor tendon and the SL to block the lateral and medial palmar (plantar) nerves. The palmar (plantar) metacarpal (metatarsal) nerves are blocked as depicted in **A** (not shown in this diagram), which also shows the subcutaneous ring block. The clinician inserts a needle (*b*) in a lateral-to-medial direction dorsal to the digital extensor tendons to block the dorsal metatarsal nerves of the hindlimb.

abaxial sesamoid block (see previous comments). A block done at this level essentially provides analgesia of the entire digit, because the block is performed at the level of or just proximal to the origin of the dorsal branch of the palmar digital nerve. Response to this block may vary, however. Some horses retain skin sensation in the dorsoproximal aspect of the pastern region. In others, pain arising from lesions involving the fetlock joint or peri-articular tissues is abolished. In part, these phenomena can be explained by proximal diffusion of anesthetic, affecting the palmar digital nerves proximal to the fetlock joint. Branches of the palmar digital nerves supplying the proximal sesamoid bones, the sesamoidean nerves, could easily be blocked using an analgesic technique in this abaxial position.[21]

The abaxial sesamoid block can be performed in the standing horse or with the limb held by the clinician or an assistant. The palmar digital nerve can easily be palpated over the rigid proximal sesamoid bones and in fact is in its most superficial position in this location. A 25-gauge, 1.6-cm needle, directed in a proximal or distal direction, and typically 1 to 3 ml of local anesthetic solution is used for the medial and lateral injection. Deep pain is assessed by hard flexion of the interphalangeal joints.

Low Palmar Analgesia

Analgesia of the metacarpophalangeal joint region and distal limb is induced using the low palmar block or low palmar analgesia (low four-point). This technique blocks the medial and lateral palmar nerves and the medial and lateral palmar metacarpal nerves. In the forelimb a subcutaneous, dorsally directed ring block and block of the dorsal branch of the ulnar nerve completely abolishes skin sensation. Disagreement exists about whether abolishing skin sensation is necessary when performing perineural techniques. Abolition of skin sensation does not necessarily mean deep pain is abolished, which is particularly relevant when a nerve responsible for skin sensation is blocked independently from nerves contributing to deep pain sensation, as in the case of the low palmar technique. When using these techniques for diagnostic purposes, it may be best to avoid blocking nerves that only contribute skin sensation, thus minimizing the number of needle insertions. For therapeutic interventions, however, these nerves need to be blocked.

The low palmar block is performed at the level of the distal end (button) of the second and fourth metacarpal bones (splint bones), with the limb in a standing position or held off the ground (Fig. 10-7). A 20- or 22-gauge needle is used to inject 1.5 to 5 ml of local anesthetic solution at each injection site. To block the palmar metacarpal nerves, the needle is inserted perpendicular to the skin, just distal to the end of the splint bones, to a depth of 1 to 2 cm. It is important to deposit anesthetic solution deep in the injection site, rather than simply in a subcutaneous location. While continuously injecting local anesthetic solution, the needle is slowly withdrawn, leaving a visible bleb in the subcutaneous space. To block the medial and lateral palmar nerves, the needle is inserted subcutaneously, in the palmar aspect of the space between the

suspensory ligament and deep digital flexor tendon (DDFT) at the level of or slightly more proximal to the distal end of the splint bone. To improve the accuracy of the injection, using a fan-shaped injection technique is helpful. To complete this block, local anesthetic solution is placed in the subcutaneous tissues from the bleb at the distal end of the splint bone to the dorsal midline.

Alternatively, some clinicians prefer to use a longer needle first to deposit local anesthetic solution over the palmar metacarpal (metatarsal) nerves. The needle is then pushed subcutaneously to deposit local anesthetic solution over the palmar nerves (see Fig. 10-7). When performing this modification, incompletely blocking the palmar metacarpal (metatarsal) nerves or lacerating the digital vessels is possible. The lateral and medial palmar nerves can be blocked using only the lateral injection site by advancing the needle in a medial direction, palmar to the DDFT. Although each of these modifications may theoretically decrease the number of injections needed to perform this technique, they have the disadvantages of potential hemorrhage and incomplete analgesia.

High Palmar Block

To provide analgesia to the metacarpal region, the high palmar block (high four-point, sub-carpal block) is the most common technique, but a modified block (lateral palmar or Wheat block) can be performed. Inadvertent penetration of the carpometacarpal joint is a potential complication with the high palmar block. A similar complication can occur in the hindlimb but is less frequent (see the following discussion). Inadvertent penetration of the carpometacarpal joint occurred in 17% of specimens, in which a conventional high palmar block was performed, because of extensive distopalmar outpouchings (Figs. 10-8 and 10-9). However, when the high palmar block was performed within 2.5 cm of the carpometacarpal joint, inadvertent penetration of this joint occurred in 67% of specimens. The carpometacarpal joint always communicates with the middle carpal joint, and therefore penetration of the carpometacarpal joint during high palmar analgesia would lead the clinician to diagnose a metacarpal problem, when in reality, the authentic lameness condition exists in the carpus. Moving the injection site in a distal direction decreases the possibility of entering the carpometacarpal joint but also narrows the scope of the technique. Two ways around this likely complication are these: first, the clinician could perform middle carpal analgesia before performing high palmar analgesia; second, the clinician could perform a lateral palmar block in lieu of the conventional high palmar technique. In an experimental study, it was unlikely to enter the carpal joints inadvertently when performing the lateral palmar block, although in every specimen, local anesthetic solution would have entered the carpal canal.[22] Unless the clinician is familiar with the lateral palmar block, the most straightforward approach to reduce the possibility of misdiagnosis in this region is to perform middle carpal analgesia before proceeding to the high palmar block. When local anesthetic solution is placed in the middle carpal joint, not only is the carpometacarpal joint blocked, but also the possibility exists of providing local analgesia to the proximal palmar metacarpal region. Using this approach, abolishing pain associated with proximal suspensory attachment avulsion injury (desmitis, fracture), stress remodeling, and longitudinal fracture is possible (see Chapter 38). The palmar metacarpal nerves and suspensory branches from the lateral palmar nerve are closely associated with the distopalmar outpouchings of the carpometacarpal joint, and anesthetic diffusion from this area could explain in part this clinical finding (Fig. 10-10).

It is important for the clinician to understand that interpretation of analgesic techniques in the proximal palmar metacarpal region or carpus can be somewhat complex.

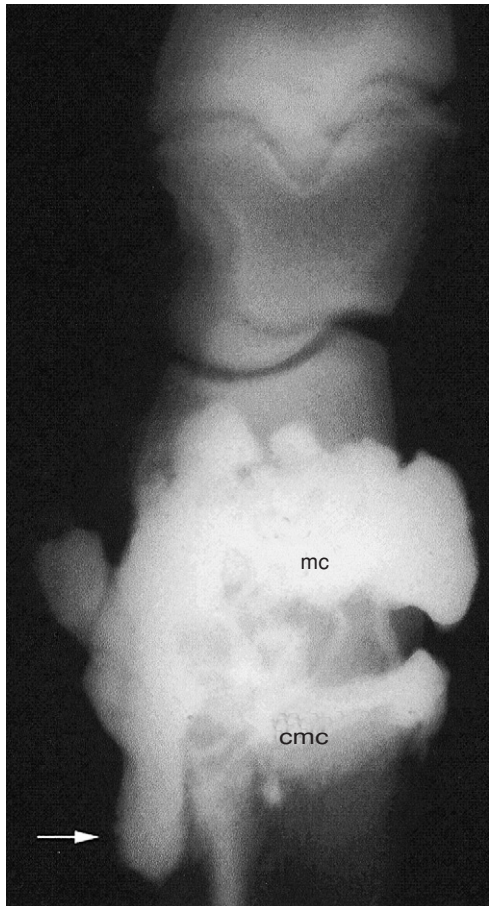

Fig. 10-8 Positive contrast arthrogram of the middle carpal (*mc*) and carpometacarpal (*cmc*) joints (dorsal is to the right). Contrast material injected into the middle carpal joint flows freely distally into the carpometacarpal joint and fills the extensive distopalmar outpouchings of the joint *(arrow)*.

Correct diagnosis is always the key, and comprehensive evaluation using multiple imaging modalities is a must in differentiating lameness in this region. From the clinical perspective, one is more likely to assume incorrectly that one is dealing with a carpal problem, when the authentic lameness condition resides in the proximal palmar metacarpal region, than vice versa. Numerous techniques are used to perform high palmar analgesia; some provide partial and others provide complete analgesia to the metacarpal region. For complete analgesia, blocking the following nerves is necessary: the medial and lateral palmar nerves, the medial and lateral palmar metacarpal nerves, the suspensory branches, and nerves providing skin sensation along the dorsum (dorsal branch of ulnar nerve and musculocutaneous nerve). To block these nerves effectively, one must use a site close to the carpometacarpal joint, at the level where the splint bones begin to taper (Fig. 10-11). If the block is done at a lower level, the region of the suspensory attachment will be missed. A 20- or 22-gauge needle at least 2.5 cm long is necessary to reach the palmar metacarpal nerves in this location. The needle is inserted axial to the splint bones just abaxial to the suspensory ligament and then guided to the palmar cortex of the third metacarpal bone (McIII). Five milliliters of local anesthetic solution is deposited, first deep within the tissues, and continued as the needle is withdrawn, ending with a bleb in the subcutaneous tissues. To block the medial and lateral palmar nerves between the suspensory ligament and DDFT, a smaller gauge needle can be used to deposit 3 to 5 ml of local anes-

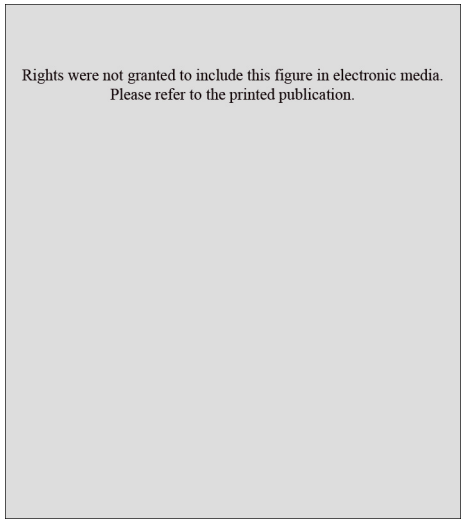

Fig. 10-9 Liquid acrylic injected into the middle carpal joint and allowed to harden created this specimen showing the lateral (*A*) and medial (*B*) distopalmar outpouchings of the carpometacarpal joint. Secondary fingerlike outpouchings ramify in the proximal palmar metacarpal region.

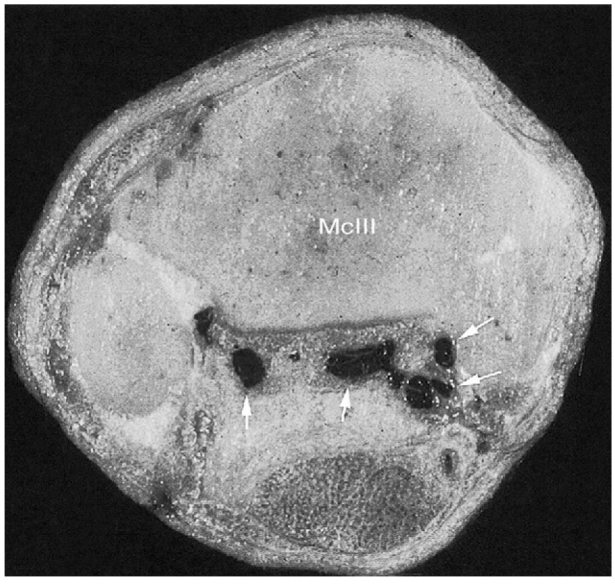

Fig. 10-10 Transverse section of the proximal metacarpal region just distal to the carpometacarpal joint after latex injection into the middle carpal joint showing primary and secondary distopalmar outpouchings of the carpometacarpal joint (*dark areas, arrows*) interdigitating with the proximal aspect of the suspensory ligament (dorsal is up; lateral is left). This anatomical arrangement explains inadvertent analgesia of the carpus and proximal palmar metacarpal region during high palmar and middle carpal analgesia, respectively. *McIII*, Third metacarpal bone.

thetic solution at each of two sites. To complete this block, a circumferential subcutaneous ring block is performed to abolish skin sensation dorsally. Alternatively, the subcutaneous nerves can be blocked on either side of the common digital extensor tendon, but small zones of sensation may persist using this technique. It is necessary to complete the dorsal portion of this block to provide complete analgesia when performing procedures in the dorsal metacarpal region, such as laceration repair or standing osteostixis.

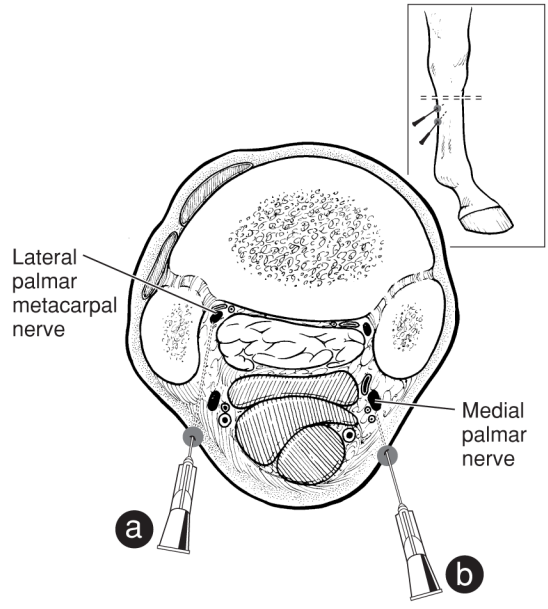

Fig. 10-11 Transverse view of the left metacarpal region showing the technique for high palmar analgesia. The clinician inserts a needle (*a*) axial to each second and fourth metacarpal bone and uses two separate injections (*b*) to block the medial and lateral palmar nerves. The location of the high palmar technique appears in the lateral view (*inset*).

A modification of the high palmar block is performed by locally infiltrating the suspensory origin from a lateral injection site in a fan-shaped pattern. This procedure, along with one specifically to block the medial and lateral palmar metacarpal nerves, improves specificity of this complex block, because only pain from a limited number of structures is eliminated. The medial and lateral palmar nerves can also be blocked from a single lateral injection site.

Lateral Palmar Block

An alternative method of providing analgesia to the metacarpal region is to perform what is known as the *lateral palmar* (high two-point) or *Wheat block*.[23] To provide complete analgesia, however, combining this block with an independent injection over the medial palmar nerve and with a dorsal subcutaneous ring block is necessary. Originally proposed as an alternative method for analgesia of the suspensory ligament origin, this technique involves blocking the lateral palmar nerve just distal to the accessory carpal bone (Fig. 10-12). The lateral palmar nerve is formed as the median and deep ulnar nerves join, proximal to the accessory carpal bone (see Fig. 10-12).[24] At the level of the block, just distal to the accessory carpal bone, the lateral palmar nerve is blocked before it branches to form the medial and lateral palmar metacarpal nerve, the suspensory branches, and continues distally (see Fig. 10-12). The high two-point block is completed with the separate but concurrent block of the medial palmar nerve.

This technique has at least three advantages compared with conventional high palmar analgesia. Inadvertently penetrating the distopalmar outpouchings of the carpometacarpal joint is virtually impossible, although local anesthetic solution will likely enter the carpal canal.[22] Lateral palmar analgesia requires fewer needle penetrations than does conventional high palmar analgesia. Finally, only a small volume of local anesthetic solution is necessary to desensitize a number of nerves

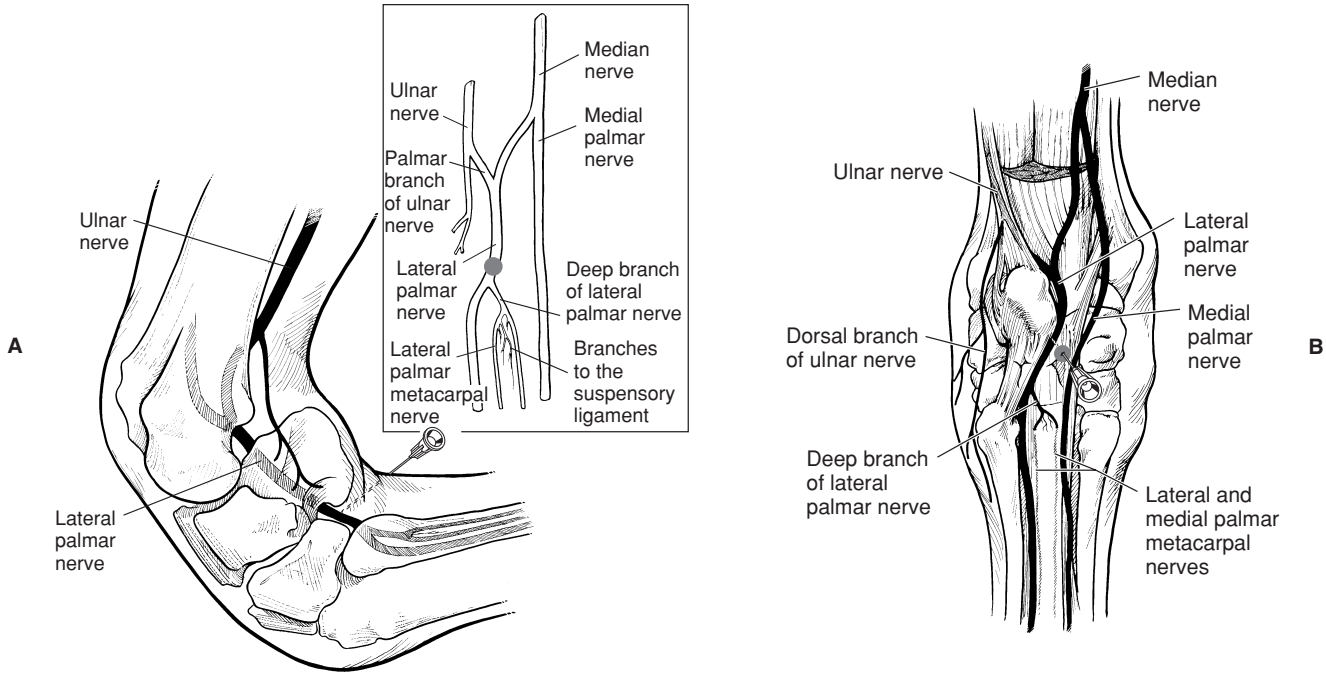

Fig. 10-12 **A,** This diagram of the left carpus in a flexed position shows the location of the lateral palmar nerve block and parent nerves (*inset*) contributing to the origin of the lateral palmar and other important nerves. **B,** Palmar view of the limb showing nerves in situ and the site for needle penetration for lateral palmar nerve block.

and the origin of the suspensory ligament. Pain associated with the carpal canal is abolished, however.

The lateral palmar block can be performed in the standing position or with the limb held off the ground, with the carpus in 90° of flexion. The nerve cannot be palpated because it courses in the accessorial-metacarpal ligament, dense connective tissue distal to the accessory carpal bone. A 25-gauge, 1.6-cm needle is inserted to the hub, perpendicular to the skin, just distal to the accessory carpal bone, and 5 ml of local anesthetic solution is deposited within this dense tissue. Injection can be difficult to perform, and breaking the seal between the needle and syringe is common, so a screw-type hub should be used. The medial palmar nerve is then blocked as described previously. If desired, a dorsal, circumferential subcutaneous ring block provides complete analgesia to the dorsum.

Median, Ulnar, and Medial Cutaneous Antebrachial Block

Although not frequently performed, analgesia of the distal antebrachium and carpus can be induced by blocking the median, ulnar, and medial cutaneous antebrachial (musculocutaneous) nerves.[19] Because the latter nerve supplies only skin sensation, for diagnostic purposes it does not need to be included in the technique. In our practices, this block is most commonly performed to facilitate lavage of the carpal joints or carpal canal or to perform regional limb perfusion of antibiotics in standing horses. We generally default to intrasynovial analgesia in these structures, however. The median, ulnar, and medial cutaneous antebrachial nerve block is occasionally useful in diagnosing subchondral carpal bone pain or lameness involving the carpal canal. Although the prevalence of lameness in the distal antebrachium is low, this block can be used to diagnose distal radial bone cysts or enthesitis at the origin of the accessory ligament of the superficial digital flexor tendon (SDFT) (superior check ligament). This block can be used to eliminate the entire distal limb as a potential source of pain. Alternatively, this block can be used alone to eliminate pain distal to the injection site, or the median and ulnar nerves can be blocked independently to improve specificity of the technique.

The ulnar nerve is blocked about 10 cm proximal to the accessory carpal bone on the caudal aspect of the antebrachium (Fig. 10-13). A 20- or 22-gauge, 4-cm needle is inserted to the hub, perpendicular to the skin, in the groove between the flexor carpi ulnaris and the ulnaris lateralis muscles. Needle contact with the ulnar nerve may cause the horse to strike forward.[5] Ten milliliters of local anesthetic solution is injected as the needle is slowly withdrawn. Skin sensitivity along the lateral aspect of the limb from the carpus to the metacarpophalangeal joint will be eliminated.[19]

The median nerve is blocked 5 cm distal to the cubital (elbow) joint on the medial aspect of the antebrachium. At this level, the nerve lies along the caudal aspect of the radius, just cranial to the flexor carpi radialis muscle. A 20- or 22-gauge, 4-cm needle is inserted to the hub, in a lateral direction, along the caudal aspect of the radius, just distal to the superficial pectoral muscle, and 10 ml of local anesthetic solution is used (see Fig. 10-13). Rarely in large horses, a 9-cm (3½-inch) spinal needle may be necessary to reach the median nerve. Often the needle hits the median nerve, a useful indicator that the tip is in the proper location.[5] In any event the needle should be kept close to or against the caudal cortex of the radius to avoid inadvertent puncture of the median artery or vein, which lies caudal to the nerve.[19,24] To facilitate these deep injections, the skin can be first desensitized by using a small volume of local anesthetic solution. A more distal injection site for the median nerve may eliminate the possibility of inadvertently eliminating elbow joint pain using the suggested approach.[5]

Finally (for therapeutic applications), to block the cranial and caudal branches of the medial cutaneous antebrachial (musculocutaneous) nerve, 3 ml of local anesthetic solution is injected, subcutaneously, on the cranial and caudal aspect of the accessory cephalic and cephalic veins, about halfway between the carpus and elbow (see Fig. 10-13).[19] Alterna-

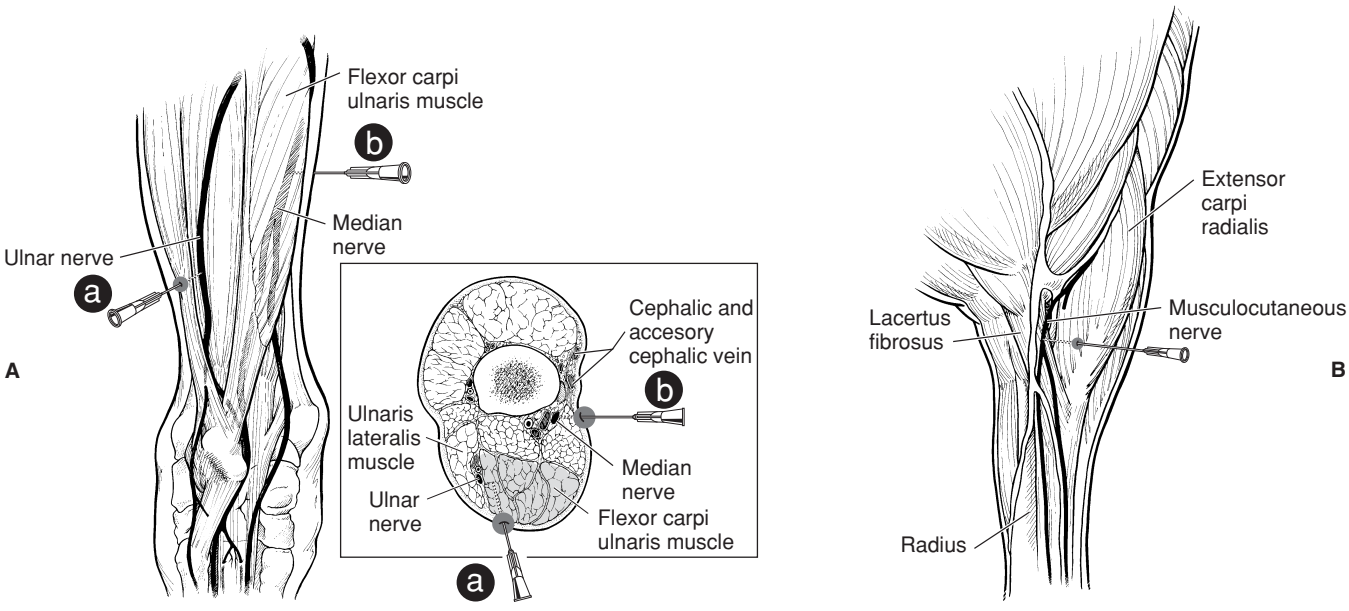

Fig. 10-13 **A,** This caudal view of the left antebrachium shows the sites of needle insertion for the median and ulnar nerve blocks. A needle placed between the ulnaris lateralis and flexor carpi ulnaris muscles (*a*), about 10 cm proximal to the accessory carpal bone, blocks the ulnar nerve. A needle inserted along the caudal aspect of the radius about 10 cm distal to the elbow joint (*b*) blocks the median nerve. The *inset* shows the orientation between the radius, median artery, vein, and nerve at the site of needle insertion (*b*) and shows the orientation of the needle for the ulnar nerve block (*a*), which is performed distally. **B**, This medial view of the proximal left antebrachium shows the technique for a musculocutaneous nerve block. The nerve is blocked as it crosses the lacertus fibrosus on the cranial aspect of the proximal antebrachium. This block abolishes skin sensation on the medial and dorsal aspects of the antebrachium.

tively, this nerve can be blocked before it branches, as it courses across the lacertus fibrosus. At this location, the nerve is easily palpable in most horses. A third method to completely abolish skin sensation is using a circumferential subcutaneous ring block, a technique that can effectively block all four cutaneous antebrachial nerves but requires a large volume of local anesthetic solution.

INTRA-ARTICULAR ANALGESIA IN THE FORELIMB

Distal Interphalangeal Joint

The assumption is that analgesia of the DIP joint is specific for intra-articular pain, but clinical experience and the results of recent clinical and anatomical investigations have convinced us otherwise (see Fig. 10-1). Of great clinical interest is the comparative accuracy of analgesia of the DIP joint and navicular (podotrochlear) bursa in the diagnosis of navicular syndrome. Overall, analgesia of the navicular bursa is likely the most specific technique to diagnose navicular syndrome. However, analgesia of the distal interphalangeal joint lacks specificity for intra-articular pain and in fact can eliminate pain associated with many conditions of the foot.[25,26] For instance, using high-performance liquid chromatography to study the effects of 8 ml of mepivacaine injected into the distal interphalangeal joint, there was local anesthetic solution in the synovium of the navicular bursa in all horses and in the medullary cavity of the navicular bones in 40% of horses.[27] Anatomical studies showed that nociceptive neurofibers are present in the dorsal and palmar aspects of the collateral sesamoidean ligaments, within the distal sesamoidean impar ligament, and directly innervating the navicular bone, in the periarticular connective tissues of the distal interphalangeal joint and proximal intramedullary portions of the distal phalanx.[28,29] The close anatomical relationship between these structures and the palmar digital neurovascular bundles to the distal interphalangeal joint capsule makes them susceptible to desensitization by local anesthetics injected into the DIP joint.[29]

In a recent study using a setscrew model to create solar pain at the toe, distal interphalangeal intra-articular analgesia abolished lameness, leading to the conclusion that pain in distant sites can be abolished using this technique.[20] Therefore a positive response to distal interphalangeal intra-articular analgesia could mean lameness is caused by an articular problem, navicular syndrome, or for that matter, solar pain. Close juxtaposition between the palmar synovial extensions of the distal interphalangeal joint and digital nerves at this level was theorized as the reason that these nerves were blocked, secondary to diffusion of local anesthetic solution from the joint.[20] Therefore a protocol to examine a lame horse no longer than 5 minutes after intra-articular analgesia of the distal interphalangeal joint may minimize diffusion and improve accuracy. Because diffusion of local anesthetic solution may be hastened by moving the horse, some clinicians prefer the horse to stand until the results of the block are evaluted.[5]

Traditionally, arthrocentesis of the distal interphalangeal joint has been performed in the dorsal pouch, either medial or lateral to the common digital extensor (CDE) tendon. A 20-gauge, 2.5- to 4-cm needle is inserted about 1.5 cm proximal to the coronary band, abaxial to the CDE tendon, and directed in a distal and axial direction (Fig. 10-14). An easier approach, however, is to insert the needle, angled just slightly distal from horizontal, on the dorsal midline, *through the CDE*. Synovial fluid is consistently obtained using this approach. To open up the dorsal aspect of the distal interphalangeal joint,

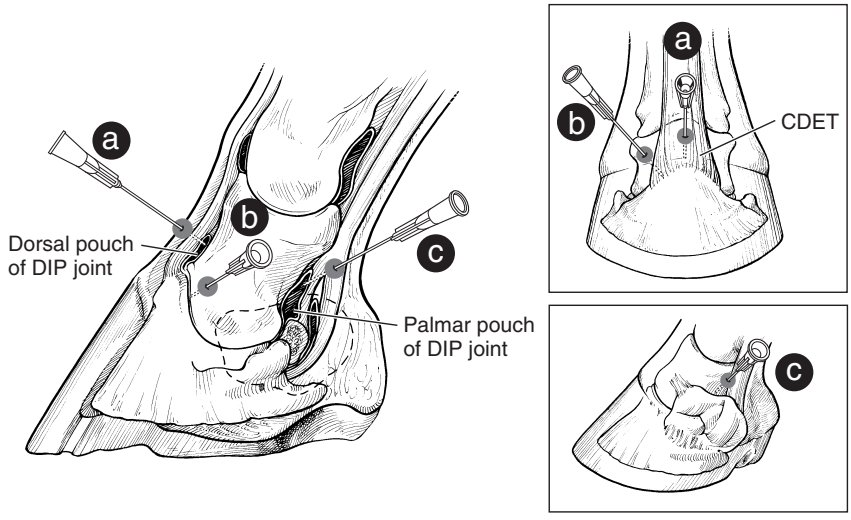

Fig. 10-14 A, Lateral view of the foot showing our preferred approach for arthrocentesis of the digital interphalangeal joint using a dorsal midline needle insertion site (*a*) and directing the needle slightly distally through the common (long) digital extensor tendon. Alternatively, the clinician approaches the digital interphalangeal joint using a site medial or lateral to the extensor tendon (*b*). The top inset shows the needle positions from the dorsal aspect. The clinician may use a palmar (plantar) approach by positioning the needle between the distal palmar (plantar) border of the middle phalanx and a palpable notch in the proximal border of the cartilage of the foot. The clinician directs the needle (*c*) in a palmaroproximolateral to dorsodistomedial direction. The lower inset shows the notch into which the needle is inserted. *DIP,* Distal interphalangeal; *CDET,* common (long) digital extensor tendon.

the limb should be positioned slightly ahead of the contralateral limb, and the horse should be in a standing position. Five to 10 ml of local anesthetic solution has been used traditionally, but a maximum of 6 ml may prevent leakage from the joint. The horse is examined after 5 minutes.

Alternatively, a lateral approach to the distal interphalangeal joint can be used (see Fig. 10-14). Landmarks include the distal palmar border of the middle phalanx dorsally and the palpable notch in the proximal border of the lateral cartilage of the foot distally. A 4-cm needle is inserted laterally and directed in a dorsodistomedial direction. This technique, however, is less reliable than the dorsal approach, because contrast material entered exclusively the distal interphalangeal joint in only 13 of 20 specimens and in 7 specimens inadvertently entered the navicular bursa or digital flexor tendon sheath.[30]

Proximal Interphalangeal Joint

Arthrocentesis of the proximal interphalangeal joint is most commonly performed in the dorsal pouch. Effusion is rarely present even in horses with severe lameness, a situation that makes arthrocentesis challenging. The injection site is just lateral (or medial) to the CDE tendon at a level of or just distal to the distal, palmar process of the proximal phalanx, located and easily palpable on the distopalmar aspect of this bone. With the horse in the standing position, a 20-gauge, 2.5-cm needle is directed slightly distally and medially (using the dorsolateral approach) and inserted until articular cartilage is encountered (Fig. 10-15). Although a desirable sign, synovial fluid appearing in the hub of the needle is an unusual occurrence. Five to 10 ml of local anesthetic solution is injected, and the horse is examined after 5 minutes.

Alternatively, the proximal interphalangeal joint can be approached using the proximal palmar pouch, from the lateral aspect. The injection location is a V-shaped notch, located dorsal to the neurovascular bundle and between the distal palmar process of the proximal phalanx the insertion of the lateral branch of the SDFT (see Fig. 10-15). The limb is held off the ground with the digit in flexion, and a 2.5- or 4-cm needle is directed distomedially (and slightly dorsally), at an angle of about 30° from the transverse plane, until fluid is collected (generally at a depth of 2 to 3 cm).[31] Advantages of this compared with the dorsal approach include less needle manipulation, a larger injection volume, and more frequent recovery of synovial fluid. Diffusion into palmar soft tissue structures could confound interpretation of results, however.[5]

Metacarpophalangeal Joint

Four sites commonly used for arthrocentesis of the metacarpophalangeal joint include the dorsal, proximopalmar, distopalmar, and approach through the collateral ligament of the proximal sesamoid bone. The two most commonly used, the dorsal and proximopalmar sites, have potential disadvantages compared with the less commonly used sites. The dorsal pouch can be prominent in horses with effusion, but inadvertently stabbing articular cartilage repeatedly is common using this approach. The proximopalmar pouch or recess is large and easily identified, but prominent synovial villi often occlude the needle end, complicating retrieval of synovial fluid, even in horses with severe effusion. Hemorrhage associated with large intra-capsular vessels is also a common complication with the proximopalmar approach. The palmar pouch is located dorsal to the suspensory branch, palmar to McIII, proximal to the collateral sesamoidean ligament, and distal to the bell of the splint bone (Fig. 10-16). Arthrocentesis using the proximopalmar approach can be performed with the limb in the standing position or being held. An 18- to 22-gauge, 2.5- to 4-cm needle is inserted in the center of the pouch and directed slightly distally in the frontal plane, until synovial fluid is recovered (Fig. 10-17). It may be necessary to aspirate synovial fluid if the joint capsule is not distended.

Dorsally, arthrocentesis is performed medial or lateral to the CDE tendon (see Fig. 10-17). With the limb in a standing or

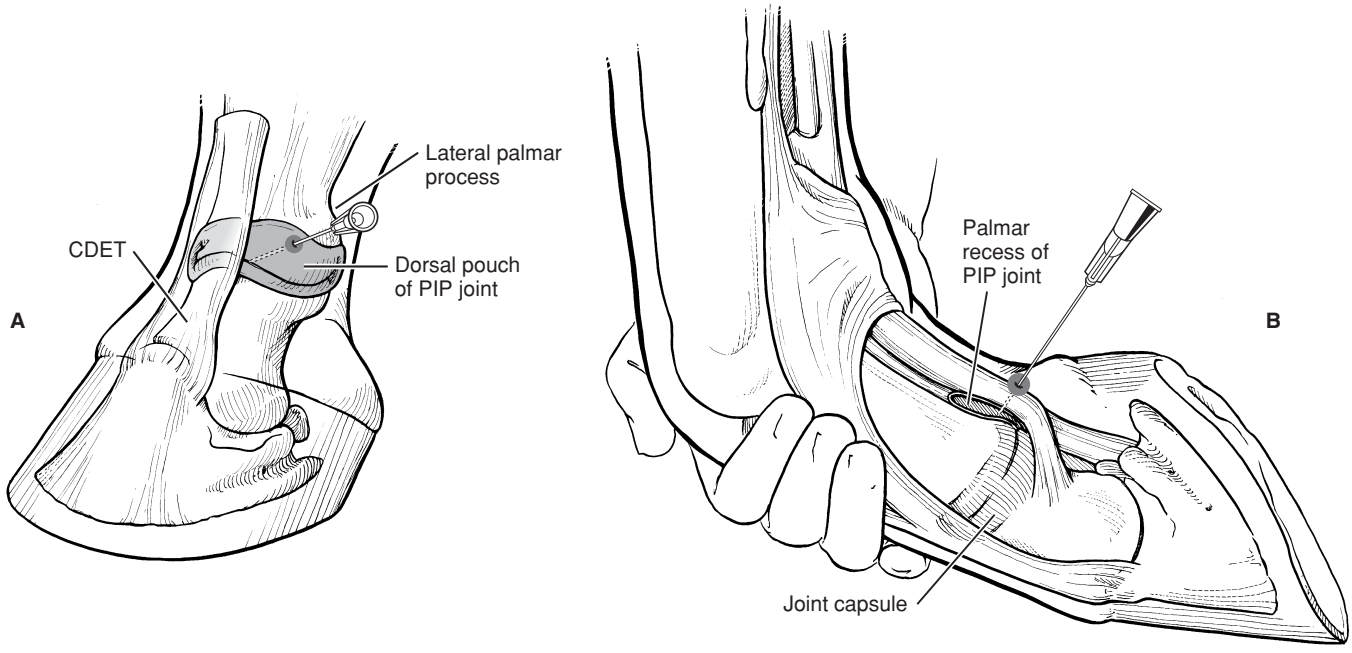

Fig. 10-15 **A,** Dorsolateral view of the digit showing the site for arthrocentesis of the dorsal pouch of the proximal interphalangeal joint. The clinician inserts the needle just abaxial to the digital extensor tendon at a site level with the palpable distal palmar (plantar) process of the proximal phalanx. **B,** Flexed lateral view of the digit indicating the site for arthrocentesis of the palmar (plantar) aspect of the proximal interphalangeal joint. The clinician inserts the needle into the V-shaped notch formed by the distal palmar (plantar) aspect of the proximal phalanx dorsally, the bony eminence associated with the attachment of the lateral collateral ligament to the distal aspect of the proximal phalanx and proximal aspect of the middle phalanx distally, and the insertion of the lateral branch of the superficial digital flexor tendon palmaro- (plantaro) distally. The clinician directs the needle distomedially (in a slightly dorsal direction) at an angle of about 30° from the transverse plane until fluid appears. *CDET,* Common (long) digital extensor tendon; *PIP,* proximal interphalangeal.

flexed position, the clinician can insert a needle in the distal aspect of the palmar pouch, through the collateral sesamoidean ligament, a less common but effective approach for arthrocentesis of the metacarpophalangeal joint. The technique is more easily performed with the joint held in flexion. This approach for arthrocentesis was shown to be associated with less subcutaneous and synovial inflammation than was the proximopalmar approach.[32]

Under most circumstances we prefer to perform arthrocentesis of the metacarpophalangeal joint using the distopalmar approach. The injection site is in a small but reliable recess bounded by a triad of structures. Just proximal to the readily palpable proximal, palmar process of the proximal phalanx is a distinct depression. The dorsal aspect of the proximal sesamoid bone and the palmar condyle of McIII complete the triad but are not readily palpable. The injection site is *dorsal* to the neurovascular bundle. Synovial fluid is consistently retrieved because the injection site is in the most distal aspect of the joint, and hemorrhage is rare. A large volume of fluid can be collected, if desired, because this area is devoid of the large synovial villi that complicate the proximopalmar approach. With the horse in a standing position, a 20-gauge, 2.5-cm needle is inserted, parallel to the ground, in a dorsomedial direction until fluid is obtained (see Fig. 10-17). The needle can be advanced to the hub, but the joint is quite superficial in this location. This technique can also readily be performed with the limb being held in a flexed position.

Ten milliliters of local anesthetic solution is injected, and the horse is reexamined in 5 to 10 minutes. In horses with subchondral bone pain, additional time may be necessary, but perineural analgesia may be necessary to abolish lameness in

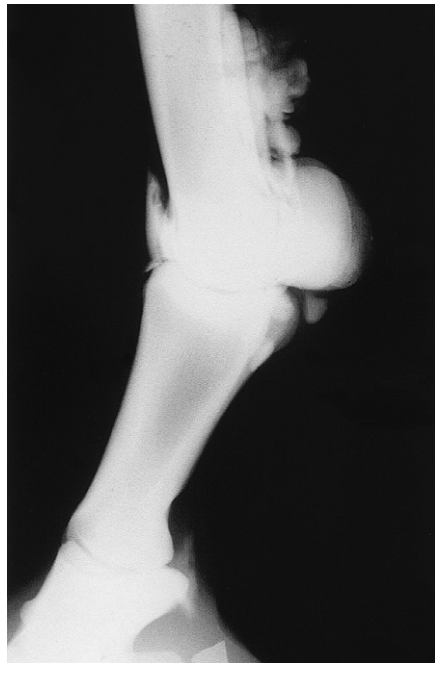

Fig. 10-16 Positive contrast arthrogram of the metacarpophalangeal joint showing the extensive nature of the palmar pouch that extends proximally to the level of the distal end of the splint bones. The distopalmar outpouchings are reliable sites for retrieval of synovial fluid and injection.

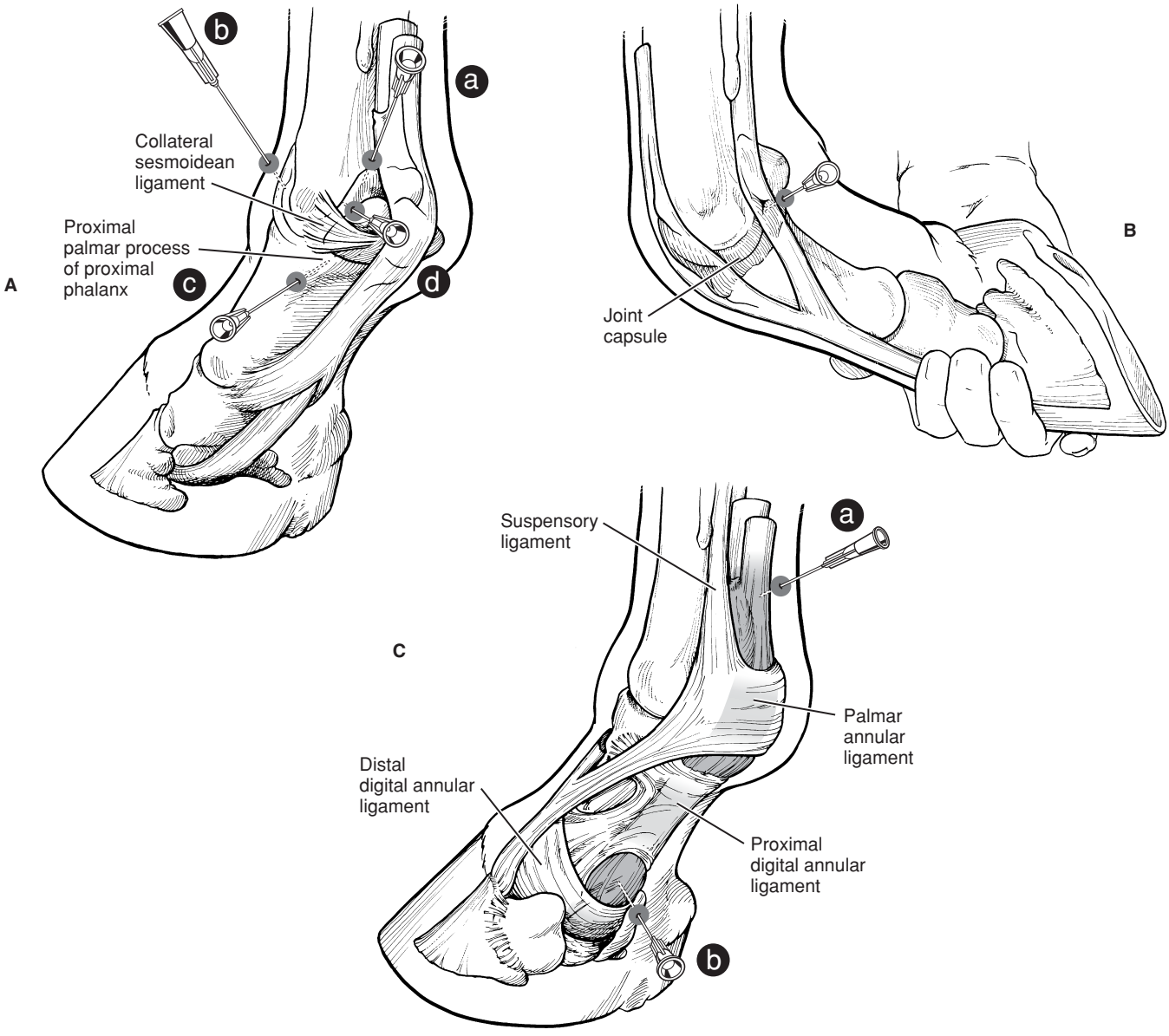

Fig. 10-17 **A,** Palmaro (plantaro) lateral view of the left metacarpophalangeal (metatarsophalangeal) joint and digit showing the sites for arthrocentesis of the proximal palmar (plantar) pouch (*a*), the dorsal pouch (*b*), the distal palmar (plantar) pouch (*c*), and the palmar (plantar) pouch through the collateral ligament of the proximal sesamoid bone *(d)*. **B,** Our preferred site for metacarpophalangeal (metatarsophalangeal) joint arthrocentesis, the distopalmar approach, using a site just proximal to the palmar (plantar) process of the proximal phalanx, is easily located in the standing or flexed position. **C,** Palmaro (plantaro) lateral view of the digit indicating sites for synoviocentesis of the proximal (*a*) and distal (*b*) aspects of the digital flexor tendon sheath. Proximally, the clinician inserts the needle proximal to the palmar (plantar) annular ligament, and distally inserts the needle on the palmar (plantar) midline into an outpouching of the digital flexor tendon sheath between the proximal and distal digital annular ligaments.

these horses. Diffusion of local anesthetic solution may account for partial improvement in lameness in horses with suspensory branch desmitis or sesamoiditis. Therefore timely evaluation of horses after metacarpophalangeal analgesia is necessary.

Carpal Joints

Arthrocentesis of the middle carpal or antebrachiocarpal joints is one of the easiest and most straightforward of all joint injection techniques. With the carpus in flexion, injection sites are easily identified, and large portals exist through which to access the joints. Portals can be found either medial to the extensor carpi radialis tendon or between the ECR and the CDE tendons (Fig. 10-18). The middle carpal and carpometacarpal joints always communicate, but a communication between the middle carpal and antebrachiocarpal joints rarely exists. A communication between the middle carpal joint and carpal sheath rarely is encountered clinically but

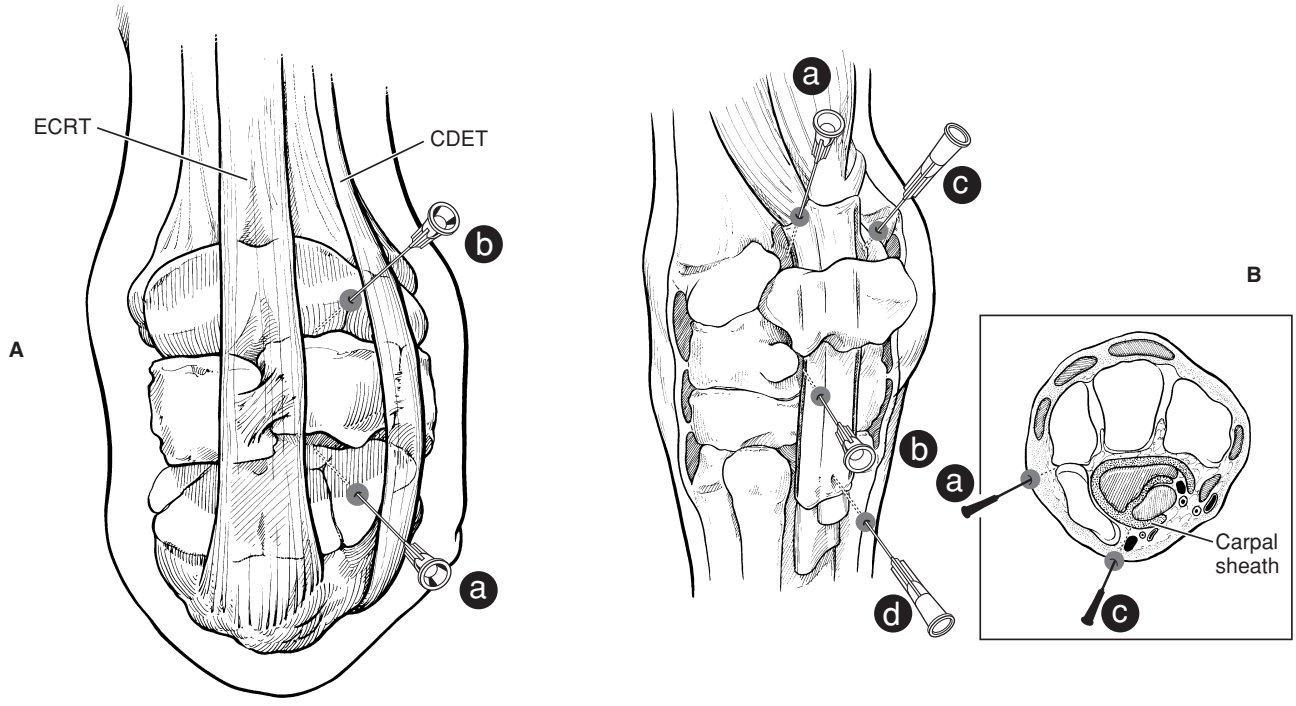

Fig. 10-18 **A,** Dorsal view of the left carpus in a flexed position showing the sites for arthrocentesis of the middle carpal (*a*) and antebrachiocarpal (*b*) joints. Needles are usually positioned between the extensor carpi radialis and common digital extensor tendons (as shown) but sites for injection of both joint cavities located medial to the extensor carpi radialis tendon can be used. **B,** Lateral view of the left carpus demonstrating sites for arthrocentesis of the proximal palmar pouch of the antebrachiocarpal joint (*a*), the palmarolateral pouch of the middle carpal joint (*b*), and the proximal (*c*) and distal (*d*) pouches of the distended carpal sheath. The inset shows the relative needle positions to enter the palmar pouch of the antebrachiocarpal joint and the carpal sheath. *CDET,* Common (long) digital extensor tendon; *ECRT,* extensor carpi radialis tendon.

was not seen in a study using cadaver limbs. Analgesia of the middle carpal and antebrachiocarpal joints should be performed separately to differentiate lameness between these independent cavities. Distopalmar outpouchings of the carpometacarpal joint complicate interpretation of analgesic techniques, because these extend a mean distance of 2.5 cm distal to the carpometacarpal articulation and are closely associated with the suspensory ligament origin and the palmar metacarpal nerves (see Figs. 10-8 to 10-10).[33] Careful differential analgesic techniques and comprehensive imaging are necessary for accurate diagnosis of lameness in the carpal and proximal metacarpal regions.

Typically, a 20-gauge, 2.5-cm needle is used to inject 5 to 10 ml of local anesthetic solution into the middle carpal and antebrachiocarpal joints. If the skin can be prepared aseptically on the dorsal aspect, the injections are most commonly performed with the joint in 90° to 120° of flexion. The clinician can maintain flexion, but having an assistant hold the limb securely is easier.

If the dorsal aspect of the carpus cannot be prepared aseptically, as occurs commonly in racehorses with chemically induced dermatitis (scurf), or if an additional site is needed for thorough lavage, the palmarolateral pouch of the middle carpal and antebrachiocarpal joints can be used. The palmar pouch of the antebrachiocarpal joint is bounded by the lateral digital extensor tendon dorsally and the ulnaris lateralis tendon palmarly. In horses with substantial effusion, this pouch is easily identified but must be differentiated from the lateral outpouching of the carpal sheath. Arthrocentesis can be performed either proximally or distally in the palmar pouch (see Fig. 10-18). The distal injection site is located in a shallow recess between the distal lateral radius (ulna) and the ulnar carpal bone, just distal to the V-shaped convergence of the lateral digital extensor and ulnaris lateralis tendons. With the horse in a standing position, a 20-gauge, 2.5-cm needle is inserted perpendicular to the skin and advanced until synovial fluid is recovered.

The palmar pouch of the middle carpal joint is similarly accessed in a shallow depression between the ulnar and fourth carpal bones, located 2 to 2.5 cm distal to the recess palpated to access the antebrachiocarpal in the palmar aspect (see Fig. 10-18). The shallow depression in the middle carpal joint is difficult to palpate, but in horses with severe effusion an outpouching of the joint is palpable. This approach is done with the limb in a standing position, decreases the potential for iatrogenic cartilage injury, and is less dangerous to the clinician because the procedure is performed on the side, rather than in front of the limb.[34] The injection is more difficult and less commonly used, however.

Cubital (Elbow) Joint

Two sites are used for arthrocentesis of the elbow joint. The cranial pouch is accessed at the level of the radiohumeral articulation, just cranial to the lateral collateral ligament. The lateral collateral ligament courses between the palpable lateral tuberosity of the radius and the lateral epicondyle of the humerus (Fig. 10-19). An 18- or 20-gauge, 6- to 9-cm needle is directed medially and slightly caudally to a depth of 5 to 6 cm, beginning in the adult horse, about 3.5 cm proximal to the lateral tuberosity of the radius and 2.5 cm cranial to the

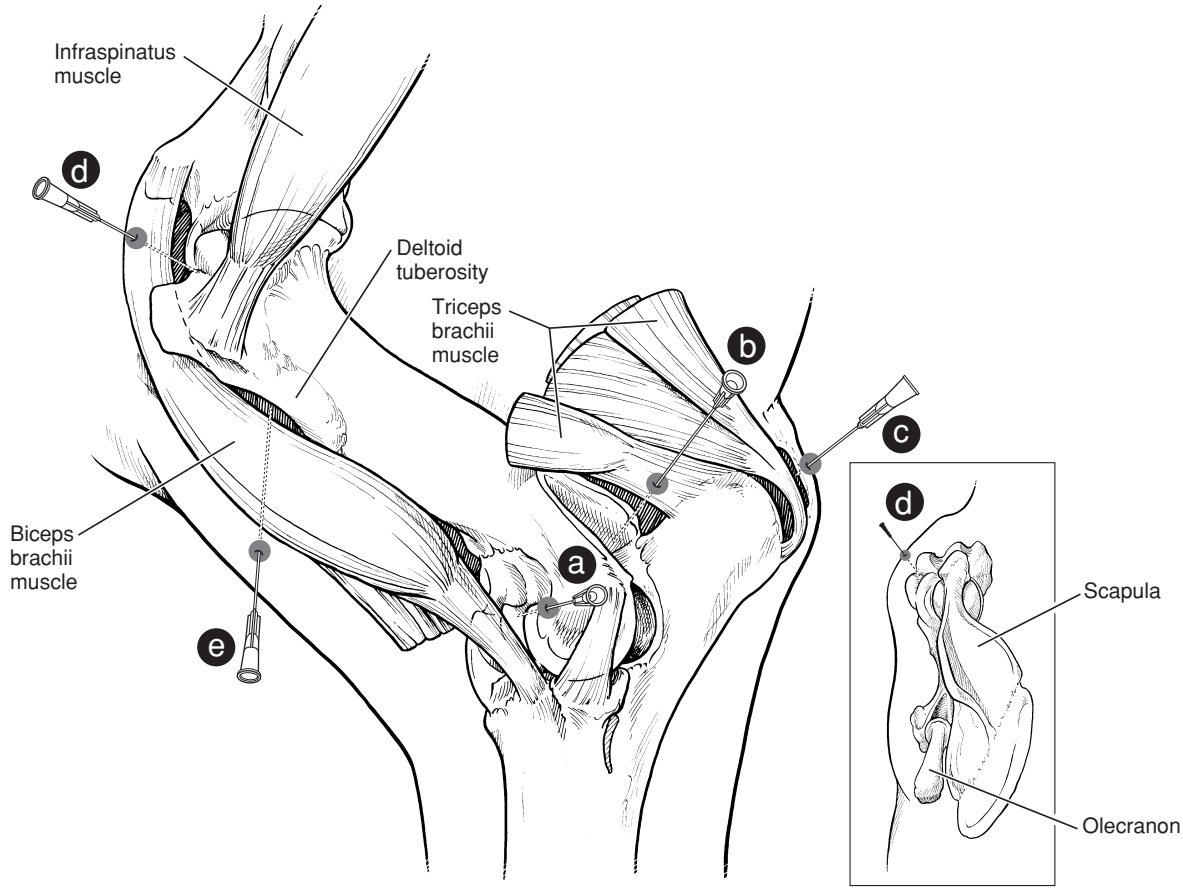

Fig. 10-19 Lateral view of the left elbow and shoulder regions. For arthrocentesis of the cranial pouch of the elbow (cubital) joint, the clinician directs the needle (*a*) medially and slightly caudally to a depth of about 5 to 6 cm at a point about 3.5 cm proximal to the lateral tuberosity of the radius and 2.5 cm cranial to the lateral collateral ligament. Arthrocentesis of the proximal, caudal pouch (*b*) is performed at a site in the palpable depression between the cranial aspect of the olecranon and the caudal aspect of the lateral epicondyle of the humerus. One injection site is for the rarely performed technique of synoviocentesis of the olecranon bursa (*c*). Proximally is the site for arthrocentesis of the scapulohumeral joint (*d*). A needle is inserted cranial to the infraspinatus tendon in the notch between the cranial and caudal eminences of the greater tubercle of the humerus and advanced in a caudomedial direction, roughly parallel to the ground and about 45° to the long axis of the body (*inset*). For the bicipital bursa (*e*), the clinician inserts the needle at a point about 4 cm proximal to the palpable distal aspect of the deltoid tuberosity of the humerus (or alternatively, a point about 3 to 4 cm distal and 6 to 7 cm caudal to the palpable aspect of the cranial process of the greater tubercle) and directs it proximally and medially, and in some patients slightly cranially.

lateral collateral ligament.[19] To account for differences in horse size, the injection site is generally located two thirds of the distance between the humeral epicondyle and the lateral tuberosity of the radius. This block may be more easily performed closer to the lateral collateral ligament, and at this site the joint is penetrated in a more superficial location.[5] Before injection, every effort should be made to verify the needle is actually in the joint. Peri-articular deposition of anesthetic solution in this location can induce temporary radial nerve dysfunction, and horses may lose the ability to extend the carpus and digit.[35] Twenty to 25 ml of local anesthetic solution is used. An older approach relied on injection of local anesthetic solution into the ulnaris lateralis bursa, once universally thought to communicate with the elbow joint. The frequency of communication between the elbow joint and ulnaris lateralis bursa was determined to be 37.5%, and therefore this approach is no longer recommended.[36]

We prefer to perform arthrocentesis in the proximolateral aspect of the caudal pouch in the palpable depression cranial to the olecranon process and caudal to the lateral epicondyle of the humerus. In most horses the site of needle penetration is 3 to 3.5 cm caudal to the lateral epicondyle (see Fig. 10-19). In small horses and ponies, an 18- or 20-gauge, 4-cm needle is sufficient, but in large horses a 9-cm spinal needle is often necessary. The needle is advanced for 5 to 7 cm in a distal, slightly cranial, and medial direction until synovial fluid is recovered.

Elimination of skin sensitivity at the site of needle insertion by depositing a small volume of local anesthetic solution may facilitate elbow arthrocentesis, because multiple attempts

may be necessary. Synovial fluid is consistently retrievable from the joint with proper needle positioning.

Scapulohumeral (Shoulder) Joint

The shoulder joint is frequently blamed for lameness in many horses but, based on the results of diagnostic analgesia, is an uncommon source of lameness. Arthrocentesis of this joint is most commonly performed at a site between the cranial and caudal prominences of the greater tubercle of the humerus, just cranial to the infraspinatus tendon. This tendon is easily palpated in most horses and serves as the primary landmark. Firm, careful palpation between the cranial and caudal prominences reveals a depression or notch, which is the point of needle insertion (see Fig. 10-19). Identification of landmarks is easier in horses with muscle atrophy resulting from chronic lameness.

In most horses an 18- to 20-gauge, 9-cm spinal needle is preferred, although using the entire length is not necessary. Elimination of skin sensitivity is usually not necessary. The needle is inserted in a caudomedial direction (about 45° from lateral), and directed slightly distal. Attaching a syringe to aspirate synovial fluid is sometimes necessary, because in joints with minimal effusion, confirming intra-articular position of the needle may be difficult. Twenty-five to 30 ml of local anesthetic solution is used and the horse is assessed 10 and 30 minutes after injection, because severe pain associated with osteochondrosis may resolve slowly. Analgesia of the suprascapular nerve and subsequent supraspinatus and infraspinatus muscle paralysis was reported following attempts at intra-articular shoulder analgesia.[35] This complication is rare, in our experience, and may result from anesthetic diffusion to nerves of the brachial plexus.[5] Trauma from multiple needle insertions or injection of large volumes of anesthetic solution (>30 ml) may increase the likelihood of this complication. In fact, some have recommended using only 8 to 10 ml, but false-negative results from the block would likely occur.[35] Our opinion is that the most likely cause of this rare complication is malposition of the needle or iatrogenic trauma. Rarely, a communication between the bicipital bursa and the shoulder joint occurs.[37] Thinking a horse has shoulder joint pain is possible then, but in reality the diagnosis is bicipital bursitis or tendonitis. Rather than a communication between the structures, the most likely explanation is inadvertent penetration of the bicipital bursa from a misdirected needle.

ANALGESIA OF FORELIMB BURSAE AND TENDON SHEATHS

In most instances, analgesia of bursae and tendon sheaths is achieved using perineural techniques, but in some horses, selective intrasynovial analgesia is indicated. Pain sensation from bursae and sheaths is likely complex, and lameness after intra-bursal or intrathecal (within a sheath) analgesia may improve but not completely resolve. In fact, horses with severe lameness resulting from bursitis or tenosynovitis often have other associated soft tissue damage, a fact that explains partial improvement after intrasynovial analgesia. Extra time is usually given, after blocking, to reassess the horse's clinical signs.

Podotrochlear (Navicular) Bursa

A palmar midline approach is most commonly used for analgesia of the navicular bursa. Because needle position is difficult to assess and fluid recovery varies, radiographs should be used to confirm proper needle position. Positioning the foot on a wooden block can minimize the problems of manipulation at the bulbs of the heel and helps to maintain aseptic technique. Subcutaneous deposition of a small volume (1 to 2 ml) of local anesthetic solution can improve patient compliance during this procedure. An 18- to 20-gauge, 9-cm spinal needle is inserted on the palmar midline, just proximal to the hairline, and directed parallel to the sole until the needle contacts bone (Fig. 10-20).[23,25,38] Others describe a similar approach, although they direct the needle parallel to the coronary band.[19,39,40] Because redirection of the needle proximally or distally often is necessary, these approaches differ little. Success depends most on personal experience, but radiographs can be critical in confirming successful entry into the navicular bursa. The direction in which the needle is inserted is often dictated by the shape of the horse's foot, and the projected position of the navicular bone.[5] Plotting and marking the navicular position on the hoof wall can be helpful in determining needle direction and depth of insertion. The navicular position is located at a site 1 cm distal to and halfway between the dorsal and palmar aspects of the coronary band.[41,42] However, with experience and after identifying the navicular position, the procedure can be done blindly. In most horses the flexor surface of the navicular bone will be contacted at a depth of 4 to 5 cm. The needle is likely improperly positioned if resistance is encountered at a depth of less than 3 to 4 cm or if the needle can be advanced more than 6 to 7 cm. Spontaneous retrieval of synovial fluid is rare and usually indicates that the needle is in the distal interphalangeal joint capsule or the digital flexor tendon sheath (DFTS). To avoid penetrating these structures, the needle should be placed in the middle of the flexor surface of the navicular bone.[5] Three to 5 ml of local anesthetic solution is used. If navicular syndrome is suspected, some clinicians combine injection of local anesthetic solution with a corticosteroid. Alternatively, navicular bursography can be performed in combination with diagnostic analgesia by adding 1 to 2 ml of sterile, iodinated contrast material (see Chapter 30). Because in the standing horse the navicular bursa is under compression by the DDFT, suspending the foot and having the foot in partial flexion during actual injection are useful. Without using radiographs, being confident of accurate needle placement is difficult.

A proximal, palmar injection technique has been described. A needle is inserted into the deepest part of the hollow between the heel bulbs and advanced dorsodistally, about 30° from horizontal, until contact with the bone is made.[38,43] A lateral (or medial) approach is also described. A needle is inserted just proximal to the cartilage of the foot, between the neurovascular bundle and the DFTS and directed axially, distally, and slightly dorsally until contact with bone is made (see Fig. 10-20).[38,40,44] Recently, these five techniques were compared in an in vitro study. The most reliable technique was determined to be the distal palmar approach, with the needle being directed to the navicular position and the limb in a non–weight-bearing position.[45]

Digital Flexor Tendon Sheath

Two sites for intrasynovial injection of the DFTS are just proximal to the palmar annular ligament (PAL) or in the palmar aspect of the pastern region in an outpouching of the sheath located between the proximal and distal digital annular ligaments (see Fig. 10-17). Effusion facilitates identification of these sites, and rarely would an intrathecal injection be contemplated without the presence of effusion. Proximal to the PAL, villous hypertrophy of synovial membrane can complicate the procedure, because even with severe distention of the sheath, synovial fluid may be difficult to retrieve. For this reason, we favor the palmar pastern approach. In some horses, the sheath appears to be compartmentalized and distended proximal to the PAL, but not below, and therefore injections are easier to perform

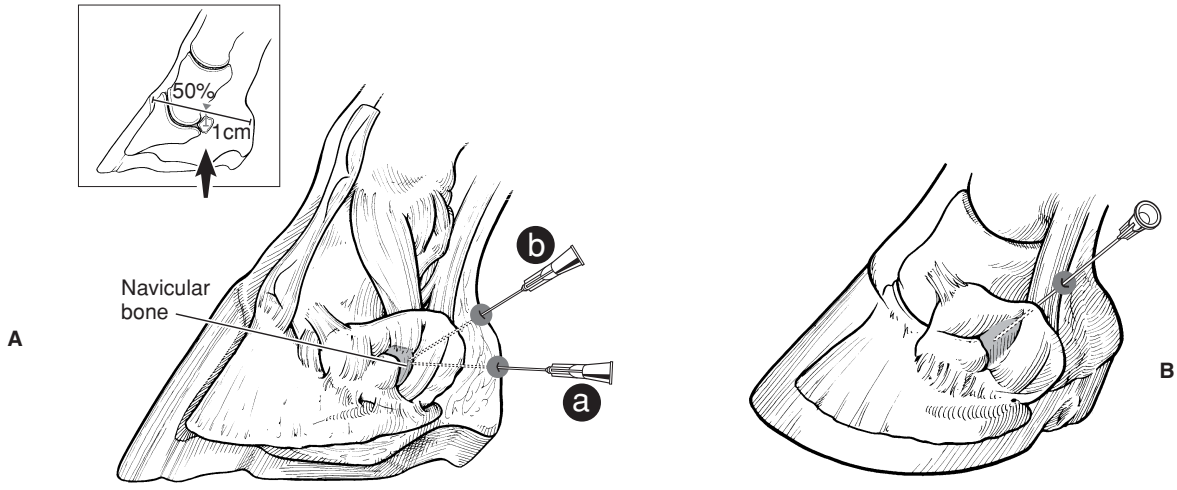

Fig. 10-20 **A,** Lateral view showing two techniques for synoviocentesis of the navicular bursa of the foot. In the palmar (plantar) approach (*a*) the needle is placed just proximal to the hairline between the bulbs of the heels and inserted to the navicular bursa using the navicular position as a guide. The navicular position (*arrow*) is located by determining the point on the outside of the hoof wall that is 50% of the distance from the dorsal to the palmar (plantar) extent of the coronary band and 1 cm below (*inset*). An approach slightly more proximal (*b*) requires placing the needle in the depression between the heel bulbs and advancing the needle in a dorsodistal direction, about 30° from horizontal toward the navicular position. **B,** Palmaro (plantaro) lateral view of the digit showing the lateral approach for synoviocentesis of the navicular bursa. The needle is inserted just proximal to the cartilage of the foot between the digital neurovascular bundle and the digital flexor tendon sheath and directed axially, distally, and slightly dorsally.

proximally. A 20-gauge, 2.5-cm needle and 10 to 15 ml of local anesthetic solution are used.

Alternatives to these approaches for intrasynovial injection of the DFTS are described. The procedure can be performed at a site just distal to the proximal sesamoid bones, between the distal aspect of the PAL and the proximal aspect of the proximal digital annular ligament (see Fig. 10-17). Recently the palmar axial sesamoidean approach was described. With the metacarpophalangeal joint held in flexion (225° angle between McIII and the proximal phalanx), a 20-gauge, 2.5-cm needle is inserted at an angle of 45°, 3 mm axial to the palmar border of the proximal sesamoid bone (mid-body) and just palmar to the neurovascular bundle. Putative advantages included more reliable access to the DFTS when effusion is absent and reduced time required for successful entry.[46]

Carpal Sheath

The carpal sheath (carpal flexor sheath) envelops the SDFT and DDFT in the carpal canal. Distention of the carpal sheath is most easily recognized laterally, just proximal to the accessory carpal bone, between the lateral digital extensor and ulnaris lateralis tendons (see Fig. 10-18). Effusion of the carpal sheath must be differentiated from that of the antebrachiocarpal joint. Concurrent distention of the dorsal aspect of the antebrachiocarpal joint or distention of the distal aspect of the carpal sheath (lateral or medial, distal to the flexor retinaculum on the palmar aspect of the metacarpal region) are signs that are helpful in determining this. Ultrasonographic evaluation or positive contrast radiography can be useful adjunct diagnostic techniques. Synoviocentesis can be performed either in the proximal or distal aspect of the sheath, using a 20-gauge, 2.5-cm needle and 10 to 15 ml of local anesthetic.

Olecranon Bursa

This technique is mentioned to be complete, but we have never found an indication to perform analgesia of this bursa (see Fig. 10-19). If distended, this bursa could be entered using the same techniques described for other bursae. Rarely, local analgesia over implants used to repair olecranon process fractures is necessary to investigate whether implant removal is indicated.

Bicipital Bursa

Bicipital bursitis and shoulder lameness are frequently diagnosed but in reality are uncommon causes of lameness, if the clinician religiously adheres to the principles of diagnostic analgesia. However, bicipital bursitis and tendonitis, and proximal humeral osteitis, fractures, or osseous cyst-like lesions can cause lameness, and are diagnosed using analgesia of the bicipital bursa. The bicipital bursa is located between the greater and lesser tubercles of the humerus and the overlying tendon of origin of the biceps brachii muscle. Synoviocentesis of the bicipital bursa is routinely performed from a lateral approach, but if severe effusion exists, the bursa can be accessed medially. The injection site is located just cranial to the humerus, 4 cm proximal to the distal aspect of the deltoid tuberosity.[38] Alternatively the site can be located by finding a point 3 to 4 cm distal and 6 to 7 cm caudal to the cranial process of the greater tubercle (see Fig. 10-19).[19] Subcutaneous infiltration of local anesthetic solution at the site can be used but is rarely needed. An 18-gauge, 9-cm needle is directed in a proximal, medial, and slightly cranial direction and can be "walked off" (shaft of the needle in contact with the bone) the cranial cortex of the humerus. A change in resistance is felt, and synovial fluid may be seen in the needle hub or can be aspirated. Ten to 20 ml of local anesthetic solution is used. If injection is difficult, synovial fluid cannot be retrieved, and retrieving

anesthetic solution already injected is not possible, the bursa likely has not been entered. An alternative is to use an ultrasound-guided technique.

PERINEURAL ANALGESIA IN THE HINDLIMB

Perineural analgesia in the distal hindlimb is similar to that described for the forelimb. Minor differences in innervation and anatomy must be taken into consideration, however. Technical differences in whether, or how, the limb is held and other intangible differences exist. Most clinicians are not as familiar, or frankly as comfortable, with performing hindlimb analgesic techniques, and this is particularly true with perineural analgesia. It takes a dedicated lameness detective to be enthusiastic about hindlimb analgesia, particularly in fractious or highly strung horses. Obviously, safety for the veterinarian and assistants is paramount, and physical and chemical restraint become important. Performing intra-articular analgesia is far easier, but the clinician must keep in mind that perineural techniques are much more effective in abolishing subchondral bone pain. Therefore false-negative results will likely be obtained if one is limited to only intra-articular procedures. We generally recommend that most perineural techniques distal to the tarsus be performed with the limb held off the ground by an experienced assistant, but personal preference can of course prevail. In some instances, such as when performing plantar digital analgesia, the anatomy is much easier to identify when the limb is bearing weight.

To limit the number of hindlimb injections in horses that lack clinical signs referable to the digit, starting with the low plantar block may be reasonable, in lieu of performing sequential blocks starting with palmar digital analgesia. Of course, performing blocks distal to this site at another time may be necessary if baseline lameness is discovered using this approach. The clinician should take care when testing the efficacy of hindlimb blocks, and using a pole or similar device may be safer than using forceps.[5] Complete abolition of skin sensation in the hindlimb is less likely than in the forelimb, because the distribution of cutaneous innervation varies.

Plantar Digital Analgesia

This analgesic technique is essentially the same as in the forelimb (see Fig. 10-5). The prevalence of lameness abolished by palmar digital analgesia in the hindlimb is considerably lower than in the forelimb but is not zero, and therefore this block should still represent a good starting point for a horse with undiagnosed lameness. Because of the reciprocal apparatus, the digit is constantly flexed when the limb is held off the ground, and this block can be slightly more difficult to perform in this position.

Dorsal Ring Block of the Pastern

The section on the dorsal ring block of the pastern in the forelimb describes this technique (see Fig. 10-5). This block requires several needle insertions and can be difficult to perform if the limb is held off the ground, because the dorsal aspect of the pastern is constantly flexed, making subcutaneous injection difficult.

Basisesamoid and Abaxial Sesamoid Blocks

Basisesamoid and abaxial sesamoid blocks present no essential differences between the forelimbs and hindlimbs (see Fig. 10-5). Our philosophical points about the basisesamoid block (see forelimb) hold true in the hindlimb as well. The abaxial sesamoid block is avoided, if possible, in racehorses, because of the high prevalence of lameness involving the metatarsophalangeal joint may lead to inadvertent misdiagnosis (a positive response to the block will be interpreted as lameness in the foot, when in reality lameness involves the metatarsophalangeal joint).

Low Plantar Block

Analgesia of the metatarsophalangeal joint region is achieved using the low plantar block, a procedure similar to the low palmar block (see Fig. 10-7). This block is one of the most overlooked but most useful of all perineural techniques. It is essential to block the medial and lateral plantar, the medial and lateral plantar metatarsal, and the dorsal metatarsal nerves. Anecdotal information suggests that some practitioners may not include the plantar metatarsal nerves when performing this block. The plantar metatarsal nerves supply innervation to the subchondral bone of the distal third metatarsal bone (MtIII), and to provide analgesia to this important area, these nerves need to be blocked. In fact a modification of this technique can be used in horses suspected of having subchondral, non-adaptive remodeling of MtIII, a common diagnosis in the Standardbred and Thoroughbred racehorse (see Chapters 108 and 109). A positive response to an independent block of the lateral plantar metatarsal nerve can help establish this syndrome as the cause of lameness.

The only difference between the low palmar and low plantar blocks involves the dorsal aspect of the limb (see Fig. 10-7). Skin sensation laterally and medially is retained after this block, unless a circumferential, subcutaneous ring block is used. In the forelimb, it is only necessary to use subcutaneous infiltration to the dorsal midline. Alternatively, the dorsal metatarsal nerves can be blocked individually.

High Plantar Nerve Block

The high plantar or sub-tarsal block is one of the most important but often overlooked perineural analgesic procedures in the horse. This block is used to diagnose suspensory desmitis, arguably one of the most important lameness conditions in the hindlimb. However, suspensory desmitis can be a catchall diagnosis in some horses with occult hindlimb lameness, and the high plantar block must be done to confirm the authentic location of pain. The tarsometatarsal joint has distoplantar outpouchings (similar to but less extensive than the distopalmar outpouchings of the carpometacarpal joint) that may complicate tarsometatarsal intra-articular or high plantar analgesic techniques (Fig. 10-21). However, this is certainly less of a problem in the hindlimb than in the forelimb. For example, inadvertent penetration of the tarsometatarsal joint occurred in only 5% of limbs in which high plantar analgesia was performed, at a level of 1.5 cm distal to the tarsometatarsal joint. However, contrast material was found in the tarsal sheath in 40% of limbs, adding yet another dimension to this already somewhat difficult blocking technique. False-negative results have been attributed to inadvertent injection into blood or lymphatic vessels.[47] The clinician should take care in preparing the limb for this procedure and interpreting the results. It is possible, although not likely, that when performing a high plantar block, local anesthetic solution could be inadvertently placed in the tarsometatarsal joint. A good chance also exists, however, of inducing analgesia of the tarsal sheath. Comprehensive evaluation using multiple imaging modalities is needed when attempting to differentiate causes of lameness in this important area.

The medial and lateral plantar and the medial and lateral plantar metatarsal nerves are blocked, and a circumferential dorsal ring block provides complete analgesia to the metatarsal region. Blocking only the plantar metatarsal nerves can abolish pain associated with the suspensory ligament. Most clinicians do not include the dorsal ring block, but it is necessary to do so to eliminate lameness resulting from injury of the dorsal cortex of MtIII or to suture lacerations in this

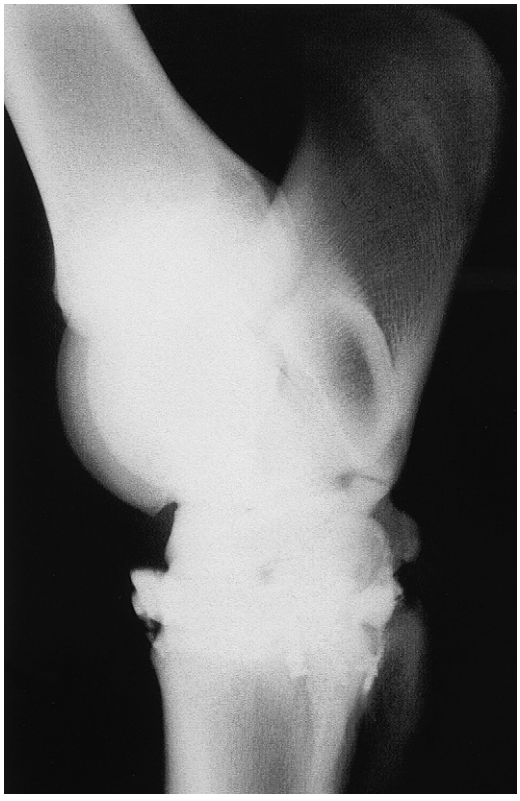

Fig. 10-21 Positive contrast arthrogram of the tarsometatarsal joint showing short, distoplantar outpouchings extending distally toward the origin of the suspensory ligament. Inadvertent penetration of these pouches occurs during subtarsal or high plantar analgesic techniques.

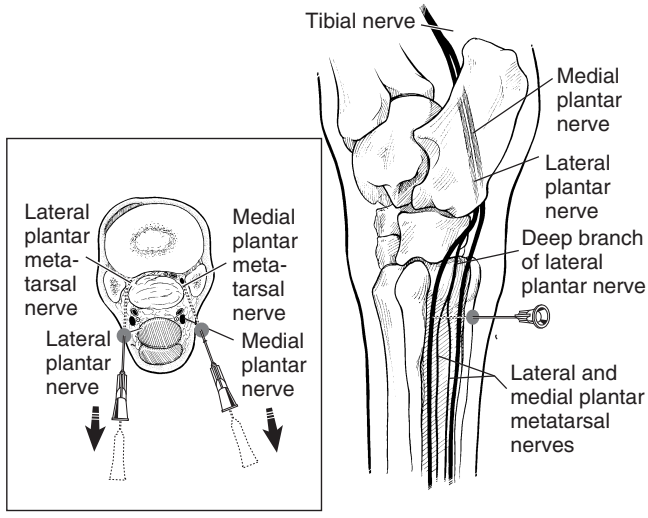

Fig. 10-22 The high plantar block is performed at a level 4 cm distal to the proximal aspect of fourth metatarsal bone and on the medial side 3 cm distal to the proximal aspect of second metatarsal bone. The needles are inserted axial to the respective splint bone and advanced deep to contact the plantar surface of the third metatarsal bone. Anesthetic solution is deposited in this location to block the lateral (medial) plantar metatarsal nerves and in a more superficial position as the needle is withdrawn blocks the lateral (medial) plantar nerves (*inset*).

area. This block is performed most commonly and safely with the limb held off the ground. Although uncommon to rare, needle breakage is a complication during high plantar analgesia, and for this reason we prefer to use needles no smaller than an 18- to 20-gauge and 4 cm long. At this level on the plantar aspect of the limb, it is impossible to palpate nerves, and unlike the high palmar block, only one injection site exists for each, on the medial and lateral aspects of the limb. The needle is placed just distal to the tarsometatarsal joint and axial to the fourth metatarsal bone and inserted until contact is made with MtIII (Fig. 10-22). A minimum of 5 ml of local anesthetic solution is deposited at this deep location, and an additional 5 ml is deposited as the needle is withdrawn, leaving a definite bleb in the subcutaneous tissues. Some clinicians prefer lower volumes of local anesthetic solution. Additional local anesthetic solution can be used without risk, and a common modification is flooding the origin of the suspensory ligament with an additional 5 to 10 ml of local anesthetic solution. The procedure is then repeated medially, and the needle is inserted axial to the second metatarsal bone. To complete the block, a circumferential subcutaneous ring block is performed. The clinician must take care not to lacerate the dorsal metatarsal artery or the saphenous vein during this procedure.

Fibular (Peroneal) and Tibial Nerve Block

Analgesia of the distal crus and tarsus or entire distal hindlimb is induced using the fibular and tibial nerve blocks. These blocks are used most commonly in horses with distal hock joint pain, in which intra-articular analgesia is difficult or impossible to perform. The fibular and tibial nerve blocks, when completed successfully, are more effective in eliminating pain from the complex hock joint than is intra-articular analgesia. The fibular and tibial nerve blocks also are useful in eliminating pain associated with subchondral trauma of the distal tibia and talus, distally located tibial stress fractures, the tarsal sheath, the distal aspect of the common calcaneal tendon, the calcaneal bursa, and the plantar aspect of the hock. The clinician should keep in mind that if the high plantar block has not already been performed, the fibular and tibial nerve blocks eliminate pain associated with proximal suspensory desmitis.

The deep fibular nerve is blocked at a site located laterally, 10 cm proximal to the point of the hock (tuber calcanei), in the groove between the long and lateral digital extensor muscles (Fig. 10-23). In this groove the superficial fibular nerve is easily palpated and can be rolled against the fascia of the crus. An 18- to 22-gauge, 4-cm needle is inserted to the hub or until it contacts the lateral tibial cortex, and 10 to 15 ml of local anesthetic solution is injected, beginning deep and continuing as the needle is withdrawn. The needle can be re-directed in a fan-shaped pattern if desired to ensure complete block of the deep branch of the fibular nerve. Seeing blood in the needle hub is common, a reliable sign of accurate needle placement, because the cranial tibial vein and artery are located close to the deep peroneal nerve.[48] Performing the tibial block first is therefore preferable, as is warning the client that blood may appear.[5] The superficial fibular nerve is blocked as the needle is withdrawn from deep within the injection site. Additional local anesthetic solution (5 to 10 ml) is placed in this subcutaneous location.

The tibial nerve is blocked at a site 10 cm proximal to the tuber calcanei, cranial to the common calcaneal tendon, and caudal to the deep digital flexor tendon (see Fig. 10-21). The nerve can be palpated as a firm cord-like structure with the limb in a flexed position. For this reason, performing this block is easier with the leg not bearing weight. Although the tibial nerve is slightly more superficial, medially, the injection can be performed either medially or laterally. A 20-gauge, 2.5-cm needle is inserted laterally, and 15 ml of local anes-

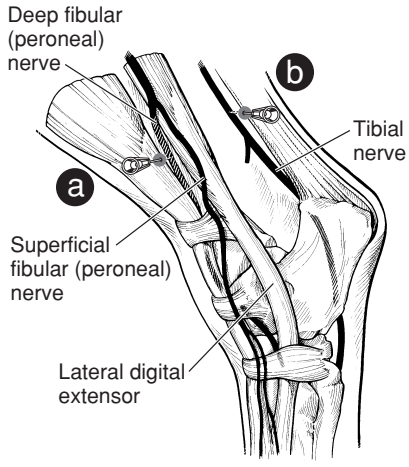

Fig. 10-23 Lateral view of the left crus and tarsus showing the fibular (peroneal) and tibial nerve block. The deep and superficial fibular nerves are blocked by finding the groove between the long and lateral digital extensor muscles, 10 cm proximal to the tarsus, in which the superficial fibular nerve is palpable. The needle (*a*) is advanced deep to block the deep branch and withdrawn to a more superficial position to deposit local anesthetic solution subcutaneously to block the superficial branch. The tibial nerve block (*b*) is performed by palpating the nerve just cranial to the common calcaneal tendon (from either a lateral or medial approach) in a location about 10 cm proximal to the tuber calcanei.

thetic solution is injected over the nerve. The needle tip should be palpated under the skin, medially, to ensure the proper depth of penetration. Local anesthetic solution can be placed using a fan-shaped injection technique, but the horse will object if the tibial nerve is penetrated. Although deep pain will be abolished if these nerves are successfully blocked, superficial sensation persists on the medial aspect and occasionally in the caudal (plantar) aspect of the limb. To use the fibular and tibial nerve blocks therapeutically, it is necessary to perform a circumferential subcutaneous ring block to completely abolish skin sensation. After the fibular and tibial nerve blocks, paradoxically, pre-existing toe drag may persist or increase, despite resolution of weight-bearing lameness.[5] Some horses stumble or knuckle, indicating loss of extensor muscle function, but this is not common and certainly not a necessary sign to suggest complete analgesia has been obtained. However, exercising a horse at speed or over fences should be avoided. Because of nerve size and depth, we suggest that additional time be given, as much as 20 to 30 minutes, to evaluate the effect of this block before reaching a final conclusion. Dyson has recognized improvement in horses up to 1 hour after blocking and warns that proceeding with a stifle block too soon leads to false-positive results.[5] The fibular and tibial nerve blocks are not commonly performed in practice, at least in the United States, and at best may result in only 50% to 80% improvement in lameness score, particularly in those horses with severe distal hock joint pain. Allowing more time for maximal response and taking a realistic approach to the percent improvement expected are warranted when using the fibular and tibial nerve blocks. Intrasynovial analgesic techniques are certainly more specific than are the fibular and tibial nerve blocks, and although the fibular and tibial nerve blocks have limitations, the lameness diagnostician should become familiar and comfortable with this procedure. Proficiency in performing these blocks is a must for accurate diagnosis of hindlimb lameness. Performing the fibular and tibial components can independently improve specificity of the fibular and tibial nerve blocks.

INTRA-ARTICULAR ANALGESIA IN THE HINDLIMB

Analgesia of the distal interphalangeal and proximal interphalangeal joints in the hindlimb is exactly the same as that described for the forelimb. Analgesia of the metatarsophalangeal (metatarsophalangeal) joint is the same as that described for the metacarpophalangeal joint. Perineural analgesic techniques should be used, whenever possible, because subchondral pain is more completely abolished using these techniques, and false-negative results are less likely.

Tarsus

Tarsometatarsal Joint

The most reliable site for arthrocentesis of the tarsometatarsal joint is a lateral approach, just proximal to the fourth metatarsal bone. At this site is a subtle but consistent depression that can reliably be palpated. A 20-gauge, 2.5-cm needle is inserted in a craniomedial and slightly distal direction (Fig. 10-24). The needle can usually be inserted to the hub, but occasionally it hits articular cartilage. Synovial fluid is consistently retrieved, but we find it interesting that even in horses without lameness of the tarsometatarsal joint, the fluid is generally watery, lacking what is thought to be normal viscosity. In most horses, 4 to 8 ml of local anesthetic solution can be injected without encountering elevated intra-articular pressures and patient discomfort. Anecdotal reports of a subtle pop or sudden decrease in pressure have been attributed to communication between the tarsometatarsal and centrodistal (distal intertarsal) joints. In reality, this most often results from rupture of the tarsometatarsal joint capsule and subsequent deposition of anesthetic solution (or medication) extra-articularly into the tarsal space and not the centrodistal joint. We recommend using no more than 4 to 8 ml of local anesthetic solution or injecting only that amount of local anesthetic solution necessary to develop moderate intra-articular resistance to avoid inadvertent deposition into the intertarsal space. An alternative site for tarsometatarsal arthrocentesis is a medial approach, similar to that described for the centrodistal joint.

The issue of communication between the distal tarsal joints is important from diagnostic and therapeutic standpoints. Studies have shown that the tarsometatarsal and centrodistal joints communicate in 8% to 35% of normal horses.[47,49,50] Communication between the tarsometatarsal joint (and presumably the centrodistal joint) and the talocalcaneal-centroquatral (proximal intertarsal) and tarsocrural joints was shown to be about 4% in an in vivo study, after injection of latex in the tarsometatarsal joint.[50] A common misconception is that a single injection into the tarsometatarsal joint also provides analgesia or treats the centrodistal joint. In addition, some clinicians preferentially inject a large volume of anesthetic solution, hoping to block or medicate the tarsometatarsal and centrodistal joints. However, based on the low communication rate, the clinician should consider *the tarsometatarsal and centrodistal joints to be separate synovial cavities*. Because the tarsometatarsal joint has distoplantar outpouchings, abolishing pain associated with the proximal suspensory attachment or lesions involving the proximal aspect of the third metatarsal bone is also possible when performing tarsometatarsal analgesia. Accurate differential diagnosis for pain involving the lower hock joints and proximal metatarsus depends on careful interpretation of response to diagnostic analgesia and evaluation of ancillary images.

Centrodistal Joint

Compared with the tarsometatarsal joint, arthrocentesis of the centrodistal joint is relatively difficult. The centrodistal joint is small, and in fact inserting a needle any larger than 22 to 25 gauge into this joint is difficult, even in horses with

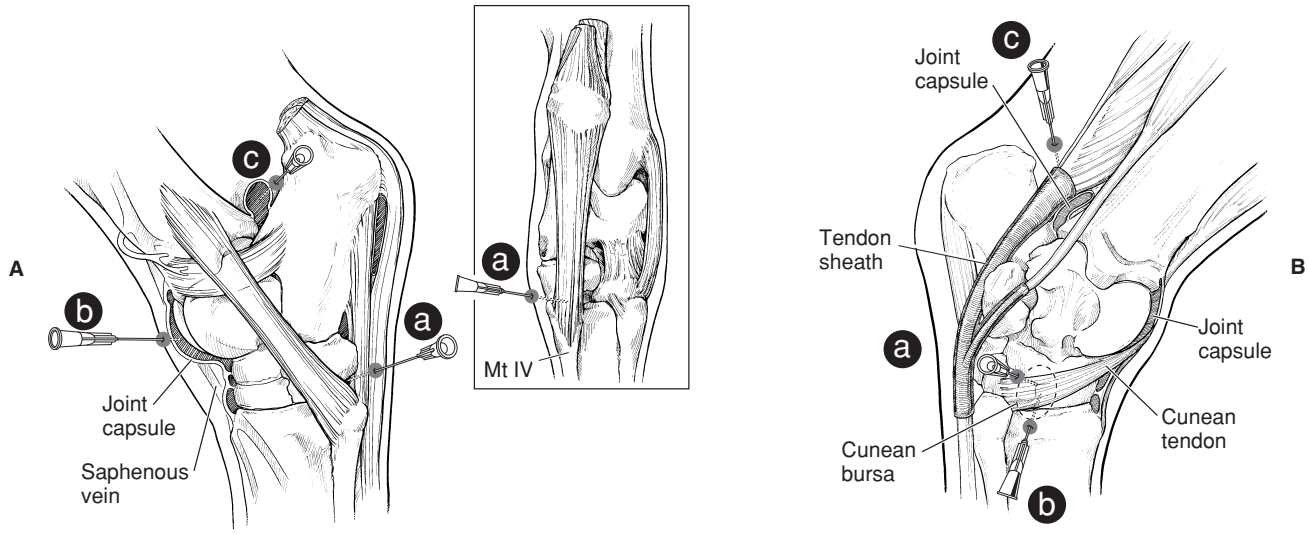

Fig. 10-24 **A,** Lateral and plantar (*inset*) views of the left tarsus showing sites for tarsal arthrocentesis. The tarsometatarsal joint is entered by locating the depression just proximal to the proximal aspect of the fourth metatarsal bone (*Mt IV*) and inserting a needle (*a*) in the plantar aspect of this depression, directing it dorsomedially. The dorsomedial pouch of the tarsocrural joint (*b*) is entered either just lateral or medial to the dorsal branch of the saphenous vein or, alternatively, using the plantarolateral pouch (*c*). **B,** Medial view of the left tarsus. The centrodistal joint is entered by placing the needle (*a*) in the depression formed between the fused first and second tarsal bones, the third tarsal bone, and the central tarsal bone, which is at the proximal edge or just slightly distal to the proximal edge of the cunean tendon. The cunean bursa (*dotted ellipse*) is entered by locating the distal border of the cunean tendon and inserting the needle (*b*) under the tendon from the distal aspect or placing it directly through the tendon. The distended tarsal sheath (*c*) can be entered proximal, just caudal to the tarsocrural joint capsule or distal (not shown) to the tarsus.

normal width of joint space. We have tried several alternative sites, including dorsomedial and dorsolateral approaches. Anecdotal reports suggest the dorsomedial approach, about 1 cm distal to the distal end of the medial trochlear ridge, is a consistent, reliable injection site, but we often enter the proximal intertarsal joint from this approach. An outpouching of the centrodistal joint exists dorsolaterally, but the perforating tarsal artery precludes use of this site in vivo. For these reasons, we use a medial approach at the distal aspect of, or through, the cunean tendon (medial tendon of insertion of the cranialis tibialis muscle), a structure that can be readily palpated. Using the fingertip, the distal edge of the cunean tendon is moved proximally to reveal an ill-defined concavity, the articulation of the fused first and second tarsal bones, with the third and central tarsal bones (see Fig. 10-24). This depression is sometimes located in a slightly more proximal location. This injection technique is one of the few commonly performed by standing on the opposite side of the horse. A skin bleb is useful because in most horses inserting a needle directly into the joint is difficult, and multiple attempts may be necessary. A 22- to 25-gauge, 2.5-cm needle is inserted directly in a lateral direction, horizontally, roughly parallel to the central and third tarsal articulation, perpendicular to the skin. Slight redirection of the needle may be necessary, and in many horses joint fluid is not obtained. If the needle can be inserted to a depth of 1 to 1.5 cm, it is likely properly positioned even if synovial fluid cannot be retrieved. Fluid retrieved in a more superficial location likely indicates penetration of the cunean bursa. In horses in which diagnostic information or therapeutic injection is critical and any question of needle placement exists, radiographs are warranted.

A maximum of 4 to 5 ml can be injected. If a larger volume can be comfortably injected, the needle tip is likely in the tarsal space or in the proximal intertarsal joint, or a communication with the tarsometatarsal joint exists. If the injection is difficult to perform, the needle is likely malpositioned in the subcutaneous tissues, or the needle tip is touching articular cartilage. Most clinicians attempt injection of the centrodistal joint *after* first injecting the tarsometatarsal joint, and in some instances, medication or local anesthetic solution readily flow from the needle. The typical response is, "There must be a communication between the tarsometatarsal and centrodistal joints." However, this clinical finding most often results from inadvertent penetration of the distended medial pouch of the tarsometatarsal joint. Fluid accumulation in the tarsal space from the tarsometatarsal joint injection can cause the same result, if the needle enters the tarsal space rather than the centrodistal joint space.

In horses with advanced osteoarthritis or even in horses with early distal hock joint pain, it may be difficult or impossible to be confident that intra-articular analgesia has been achieved. An alternative approach to provide tarsal analgesia is first to perform sequential, intra-articular analgesia of the tarsometatarsal and tarsocrural joints, and then to perform the fibular and tibial nerve blocks if lameness persists. If lameness abates after the fibular and tibial nerve blocks, a presumptive diagnosis of centrodistal joint pain can be made, assuming other sources of pain abolished by this block can be ruled out.

Tarsocrural Joint

Arthrocentesis of the tarsocrural joint is straightforward and easy compared to some joints, because of extensive and multiple dorsal and plantar outpouchings. In horses with moderate to severe effusion, identifying four distinct outpouchings—the dorsolateral, dorsomedial, plantarolateral, and plantaromedial pouches—is easy. The clinician must keep in mind that the tarsocrural and proximal intertarsal joints communicate through a large fenestration at the dorsal aspect of the joints in adult horses, although in weanlings and yearlings

the fenestration cannot be seen during arthroscopic examination. Any one of the tarsocrural joint pouches can be used, but the most common site of entry is on either side of the saphenous vein, in the dorsomedial pouch (see Fig. 10-24). This particular site is preferred in horses without obvious effusion. An alternative site is the plantarolateral pouch. The most consistent site to use is the distal aspect of the dorsomedial pouch, just distal to the medial malleolus of the tibia and medial to the saphenous vein. An 18- to 20-gauge, 2.5- or 4-cm needle is used to deposit 20 to 30 ml of local anesthetic solution into the tarsocrural joint. In horses with severe osteoarthritis of the tarsocrural joint or those with subchondral bone pain, as much as 30 to 50 ml of local anesthetic solution is necessary to abolish pain. In these horses, a false-negative result is common if only 10 to 20 ml of local anesthetic solution is used.

The plantar pouches can be useful alternative sites for arthrocentesis, if the dorsomedial pouch is unsuitable, as sometimes occurs with a wound, swelling associated with trauma of the fibularis (peroneus) tertius, or superficial dermatitis. The plantar pouches must be differentiated from distention of the tarsal sheath or other forms of thoroughpin. Although the dorsal and plantar pouches freely communicate, anatomically, flushing from one aspect of the tarsocrural to the other when the horse is in a weight-bearing position may be difficult. Fluid flow between articular surfaces and joint spaces under collateral ligaments is likely restricted when horses are in a weight-bearing position. This same phenomena occurs in other joint spaces.

Stifle Joint

The three compartments of the equine stifle joint are the medial femorotibial, lateral femorotibial, and femoropatellar joint compartments. Most consider that the femoropatellar and medial femorotibial joints communicate in almost all horses and that the lateral femorotibial compartment is solitary, but recent anatomical studies have shed new light on this time-honored concept. The frequency of communication between the medial femorotibial and femoropatellar compartments was found to be 60% to 74% in normal horses, when the injection was performed from the femoropatellar compartment.[51,52] The frequency of communication was higher (80%) when the injection was performed in the medial femorotibial compartment.[51] It is important to realize, however, *that the medial femorotibial and femoropatellar compartments did not communicate in all horses*. Inconsistency in communication depending on which compartment was injected was attributed to directionality in the normal foramen or slit between the two compartments (flow easier *from* the medial femorotibial *to* the femoropatellar compartment).

The time-honored assumption that the lateral femorotibial joint is a solitary compartment was also challenged. The lateral femorotibial joint communicated with the femoropatellar joint in 3% to 18% of horses but was indeed solitary in the majority of normal horses[51,52] Communication may be more frequent after trauma and certainly after arthroscopic surgical procedures.

We recommend that each compartment of the stifle joint be injected independently, either sequentially or simultaneously, to avoid confusing results during stifle analgesia. The variable degree of communication will obviously cause some degree of uncertainty in diagnosis. The same principle is recommended for therapy as well. Needle insertion in the stifle joints is complicated by a natural tendency of horses to react inappropriately to manipulation compared with other areas of the limbs. Horses seem to object to simple palpation of the stifle and may become fractious during arthrocentesis. To avoid excessive manipulation during injection, we have found it useful to attach an extension set to the needle, a procedure that obviates the need to touch the needle or skin when attaching the syringe. If necessary, the extension set may be useful for many diagnostic procedures, particularly in the hindlimbs. In general, 20 to 30 ml of local anesthetic solution is used in each of the medial femorotibial, lateral femorotibial, and femoropatellar compartments. A common misconception is that long needles are needed to perform arthrocentesis of the stifle joint compartments. In fact, some racehorse trainers will insist that "the long needles, Doc" are necessary to achieve success in medicating the femoropatellar joint. If arthrocentesis is performed with the limb in a weight-bearing position, the joint capsules can easily be penetrated with needles no longer than 4 cm. In the flexed position, using a spinal needle when performing femoropatellar arthrocentesis is necessary. We prefer to have the horse in a weight-bearing position, with the limb slightly ahead of the contralateral limb, a position that allows the clinician to palpate landmarks readily without undue tension on patellar and collateral ligaments.

Arthrocentesis of the medial femorotibial joint is performed at a site located just caudal to the medial patellar ligament, cranial to the medial collateral ligament, and 1 to 2 cm proximal to the medial tibial plateau (Fig. 10-25). In the normal horse a distinct depression occurs at this location, but in horses with effusion, a considerable bulge in the joint capsule can be present. An 18-gauge, 4-cm needle is inserted perpendicular to the skin and can be redirected or rotated if synovial fluid is not immediately retrieved. A common mistake is to insert the needle too far distally, and in this position the needle tip enters ligaments or the medial meniscus.

Arthrocentesis of the lateral femorotibial joint is more challenging than for the other two compartments, because the lateral joint pouch is small and located deep within tissue. The site is caudal to the long digital extensor tendon and cranial to the lateral collateral ligament, just proximal to the lateral tibial plateau (see Fig. 10-25). These landmarks are easily palpated, but distention of the joint capsule is not, in contrast to the medial femorotibial joint. An 18-gauge, 4-cm needle is inserted horizontally and directed in a slight caudomedial direction. Retrieval of synovial fluid varies, and redirecting or rotating the needle is often necessary. An alternate site can be used, located caudal to the lateral patellar ligament and cranial to the long digital extensor tendon, and just proximal to the tibial plateau.

Arthrocentesis of the femoropatellar joint is most commonly performed at a sub-patellar site and either lateral or medial to the middle patellar ligament. The joint capsule can be easily palpated even in most normal horses, if the horse is in a weight-bearing position. With the horse in a weight-bearing position, an 18-gauge, 4-cm needle is inserted perpendicular to the skin, or directed slightly proximally, until joint fluid is obtained or the needle tip contacts articular cartilage of the distal femur (see Fig. 10-25). The clinician does not need to angle the needle sharply proximally using this technique. What is sometimes frustrating is that even in horses with obvious femoropatellar effusion, a steady flow of synovial fluid cannot be obtained, and attempting aspiration of fluid with a syringe is seldom helpful, because synovial villi readily plug the needle, making aspiration impossible. Some clinicians perform femoropatellar arthrocentesis with the limb in a non–weight-bearing position, in which case a 9-cm spinal needle is used and the needle is directed severely proximally, between the patella and distal femur. An alternative lateral approach to the femoropatellar joint was described.[53] An 18-gauge, 4-cm needle is inserted into the lateral cul-de-sac of the femoropatellar compartment, located about 5 cm proximal to the lateral tibial plateau, caudal to the lateral patellar ligament and the lateral trochlear ridge of the femur. The needle is directed perpendicular to the long axis of the femur until bone is contacted (about 1.5 to 2 cm in most

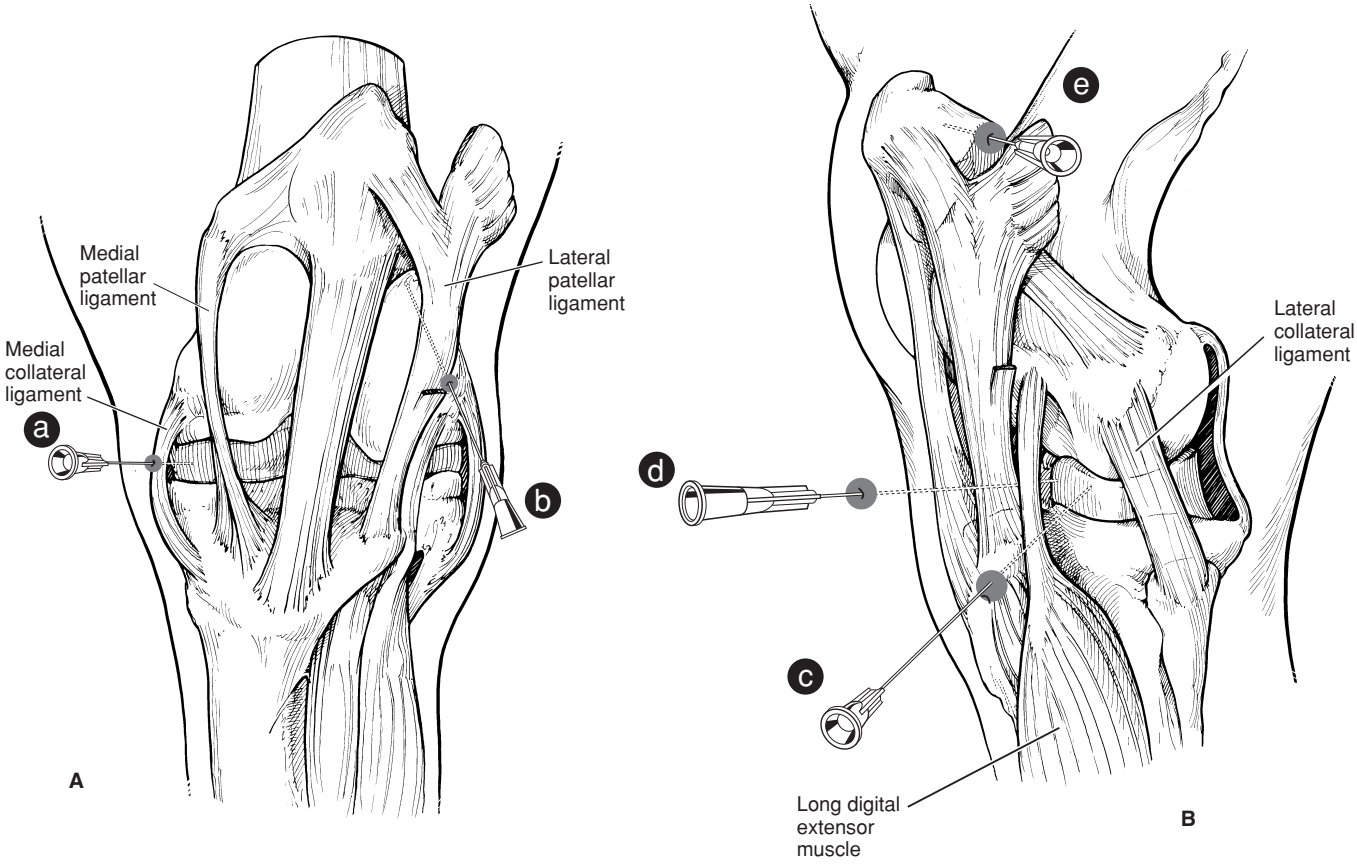

Fig. 10-25 **A,** Cranial view of the left stifle. The medial femorotibial joint (*a*) is approached from a site between the medial patellar and medial collateral ligaments, about 2 cm proximal to the proximal aspect of the tibia. The femoropatellar joint (*b*) most commonly is injected either between the lateral and middle patellar ligaments or between the middle and medial patellar ligaments (not shown). The needle is directed proximally in this subpatellar position. **B,** Lateral view of the left stifle. The lateral femorotibial joint can be approached by placing the needle caudal to the long digital extensor tendon and cranial to the lateral collateral ligament (*c*) or inserting it between the lateral patellar ligament and the cranial edge of the long digital extensor tendon (*d*). An alternative site for arthrocentesis of the femoropatellar joint (*e*) can be used by passing the needle through the lateral femoropatellar ligament.

horses) and then withdrawn slightly until synovial fluid is collected. Proposed advantages of this approach are a reduced potential for iatrogenic injury to the articular cartilage and more reliable recovery of synovial fluid compared to the subpatellar approach.[54]

Coxofemoral (Hip) Joint

Although the coxofemoral joint is relatively large and the landmarks for needle insertion are consistent, injection is considered to be a daunting task. Few of us perform this injection technique on a regular basis, and depth of penetration makes accurate needle placement difficult. An 18-gauge, 15-cm (6-inch) spinal needle is adequate for all but the largest of draft horses. A needle of this length should be inserted carefully, and if the horse is moving or fractious, it may be necessary to provide sedation. The site is in the angle formed between the long caudal and short cranial processes of the greater trochanter of the femur (Figs. 10-26 and 10-27). This site can be difficult to palpate in heavily muscled horses, and ultrasonographic evaluation can be useful to identify the injection site. The most difficult landmark to palpate consistently, but an important one nonetheless, is the cranial process. The site is *between* the two processes and not *caudal* to the trochanter.

Before the needle is inserted, blocking the injection site may be useful. Because the shaft of the needle is handled, sterile gloves are recommended. Needle direction is important. The needle is inserted in a slightly craniomedial direction and slightly distally and directed just dorsal to the femoral neck, until the joint capsule is penetrated. In most horses, a subtle pop can be felt as this occurs. "Walking" the needle off the femoral neck may be useful, using the bone as a guide to the coxofemoral joint. In most adult light breed horses, this occurs within 3 to 5 cm of the hub of the needle. Synovial fluid is reliably retrieved from the coxofemoral joint, spontaneously or by aspiration. A large volume of local anesthetic solution should not be injected if synovial fluid is not readily obtained, but injecting a small volume and attempting retrieval with a syringe is useful. It is possible inadvertently to inject local anesthetic solution around the sciatic nerve, causing temporary paresis, if the needle is caudally malpositioned, and therefore anesthetic solution should not be injected if any doubt exists that the needle is correctly positioned. Twenty-five to 30 ml of local anesthetic solution is used. Most patients are evaluated in 20 to 30 minutes, but in horses with fractures of the acetabulum the clinician should expect only 50% improvement in lameness score, and improvement may be short lasting (15 to 30 minutes).

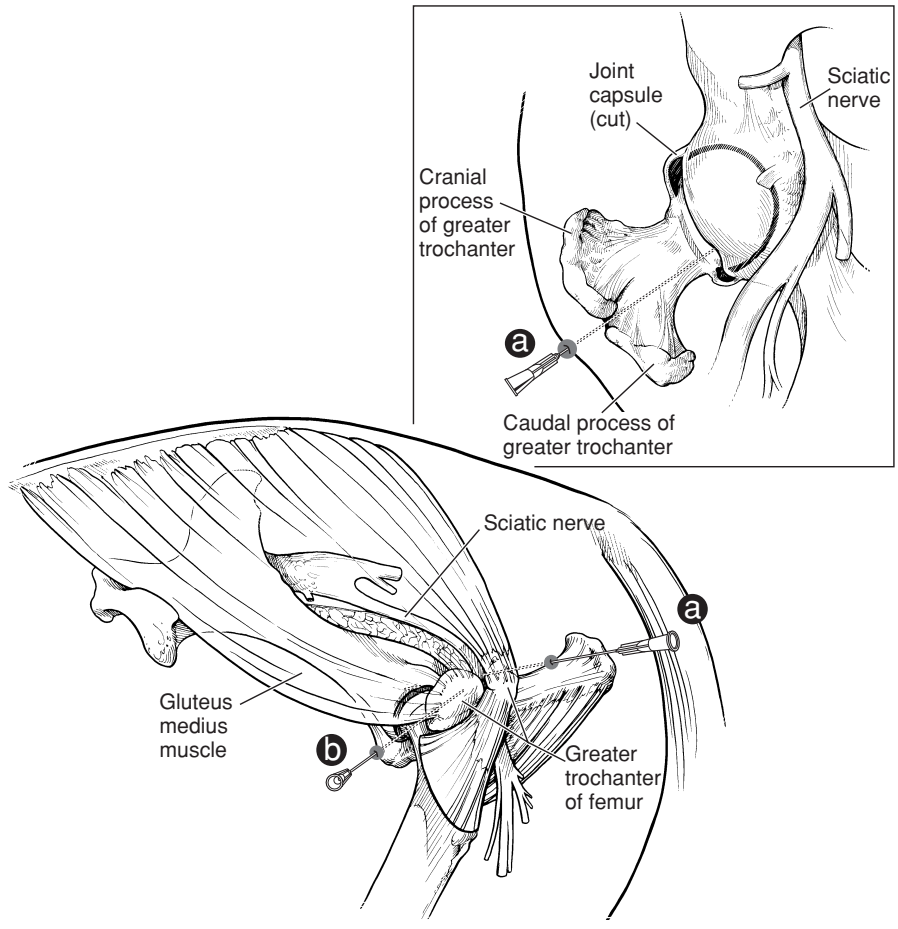

Fig. 10-26 Lateral and dorsal (*inset*) views of the left coxofemoral joint. Arthrocentesis of the coxofemoral joint is performed by inserting the needle (*a*) in the angle formed between the caudal and cranial processes of the greater trochanter of the femur. The needle is inserted slightly cranially, distally, and medially just dorsal to the shaft of the femoral neck (*inset*). This view (*b*) shows the seldom used diagnostic technique of synoviocentesis of the trochanteric bursa.

ANALGESIA OF HINDLIMB BURSAE AND TENDON SHEATHS

Analgesia of the navicular bursa and digital flexor sheath in the hindlimb is the same as in the forelimb.

Cunean Bursa

Occasionally, injecting the cunean bursa is necessary to assess the role of the cunean bursa and tendon in horses with distal hock joint pain, to perform cunean tenectomy, or to medicate the structure. The cunean bursa is seldom the sole source of distal hock joint pain, but can play a role, so analgesia or medication of this structure is sometimes combined with other injections.

The cunean bursa is between the distal tarsal bones and the medial branch of the cranialis tibialis tendon (called the *jack tendon* or *cord*) but is seldom palpable (see Fig. 10-24). The distal aspect of the cunean tendon is usually easily palpated, however, by starting at the distal aspect of the hock and sliding the fingertip in a proximal direction. Retrieving synovial fluid is unusual but possible, but during injection the clear outline of the bursa can be seen as it distends. A 20- to 22-gauge needle is inserted deep to the distal edge of the cunean tendon and directed in a proximal direction, and 3 to 5 ml of local anesthetic solution is injected. We prefer this approach, but alternatively the needle can be inserted perpendicular to

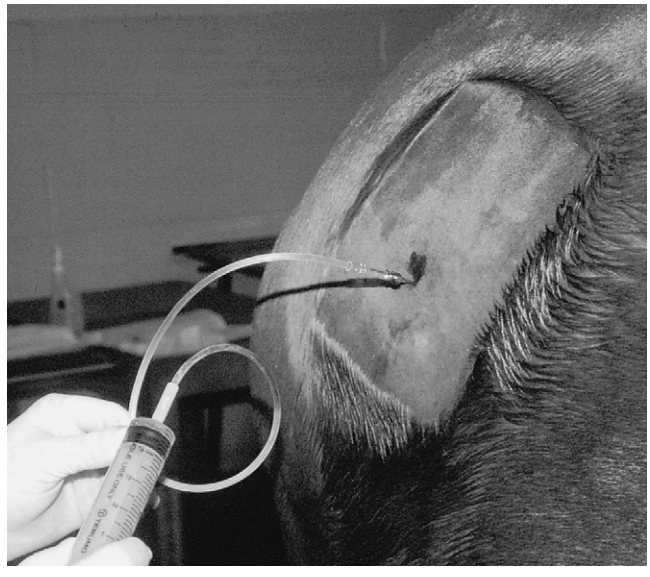

Fig. 10-27 Arthrocentesis of the right coxofemoral joint. Using an extension set between needle and syringe, a technique that reduces the amount of manipulation necessary during the procedure, facilitates arthrocentesis of this and other joints.

the skin and directly through the tendon itself until bone is contacted.

Tarsal Sheath

Analgesia of the tarsal sheath is performed to confirm the structure as a source of lameness associated with traumatic and infectious tenosynovitis (although the response may be limited in face of infection) and various osseous lesions, such as those involving the sustentaculum tali, or unusual exostoses (osteochondroma). The tarsal sheath surrounds the deep digital flexor tendon from a point approximately level with the tuber calcanei and extends to a point 2 to 3 cm distal to the tarsometatarsal joint. Distention of the tarsal sheath is commonly called thoroughpin, but occasionally, thoroughpin appears as fluid swelling proximal to the tarsus that does not involve the tarsal sheath. The deep digital flexor tendon is located medial to the calcaneus as it crosses the sustentaculum tali. The heavy tarsal retinaculum medially and the calcaneus laterally restrict outpouching of the tarsal sheath to the proximal and distal aspects. The clinician should take care to differentiate tarsal sheath effusion from distention of the plantar pouches of the tarsocrural joint. A 20-gauge, 2.5-cm needle is used to inject 10 to 15 ml of local anesthetic solution.

Calcaneal Bursa

Indications for analgesia of the calcaneal bursa include traumatic and infectious bursitis, tendonitis of the gastrocnemius or SDFT at this level, and osseous lesions of the tuber calcanei. The bursa is located between the SDFT and tuber calcanei. Proximal to the tuber calcanei, the bursa is interposed between the SDFT and the gastrocnemius tendon. When distended, an unusual clinical finding, the bursa is palpable as medial and lateral outpouchings just proximal to the tuber calcanei. Smaller outpouchings are often discernable just distal to the tuber calcanei but are inconsistent. The bursa can be accessed for injection at any of these outpouchings. A 20- to 22-gauge, 2.5- or 4-cm needle is used to inject 10 ml of local anesthetic solution, after retrieving fluid for analysis if indicated (see Fig. 10-24). Pain may take 20 to 30 minutes to abate in horses with osseous lesions or with severe lameness.

Trochanteric Bursa

Seldom does an indication exist to block the trochanteric bursa, although injections in this region are commonly performed to manage bursitis and muscle pain (see Chapter 48). The trochanteric bursa is located between the tendon of insertion of the gluteus accessorius muscle and the cranial process of the greater trochanter of the femur (see Fig. 10-26; see also Chapter 48). In normal horses, this bursa is small and likely has minimal synovial fluid. Synoviocentesis is performed using an 18- to 20-gauge, 4-cm needle, although in larger, more heavily muscled horses, a longer needle may be necessary. The needle is inserted perpendicular to the skin, directly over the cranial aspect of the greater trochanter until contact with bone is made. We have had difficulty retrieving fluid even in lame horses that have a positive response to analgesia. Generally, 5 to 10 ml of local anesthetic solution is injected until pressure is felt. If local anesthetic solution can be aspirated, the needle was likely in the bursa, but if not, the injection was likely performed in the surrounding tissues.

LOCAL INFILTRATION IN THE FORELIMB AND HINDLIMB

Local infiltration of local anesthetic solution in painful soft tissues or over painful bony swellings can be performed at any location, although some areas deserve special mention. Any localized area of pain, into which a needle can be inserted safely, is fair game for local analgesia.

The clinician must be aware, however, that local infiltration may not provide total analgesia to the region, mostly because the entire nerve supply to the region cannot be blocked. Incomplete analgesia is common in horses with bony lesions, such as bucked shins, because deep pain from the cortex of McIII is difficult if not impossible to eliminate using subcutaneous infiltration of local anesthetic solution. In most instances, perineural analgesia for this particular condition is preferred. Local infiltration is performed in many horses in lieu of perineural technique, or in horses in which perineural analgesia has localized pain to a general region, but conflicting or numerous clinical problems exist. An advantage of local infiltration is that proprioception is not lost, and horses can be moved at speed for re-evaluation after this form of analgesia. Efficacy can be assessed by deep, direct digital palpation, to confirm that the previously identified source of pain was eliminated by local analgesia.

Splints

A common suspected cause of lameness in many horses are exostoses associated most commonly with the second (McII) and fourth (McIV) metacarpal or metatarsal bones (MtII, MtIV) or in combination with the McIII or MtIII. A 20- to 22-gauge, 2.5- to 4-cm needle is used to deposit 5 ml of local anesthetic solution, subcutaneously, over the painful exostosis. The needle is slid directly alongside the proliferative lesion, between skin and bone. For splints involving McII/MtII and McIV/MtIV, it is important to deposit local anesthetic solution abaxial and axial (between the suspensory ligament and splint bones) to the lesion. In some horses proliferative changes involve only the axial aspect of McII/MtII or McIV/MtIV (blind splints), and it is critical to block in this location. In others with primary proliferation between McII/MtII, McIV/MtIV, and McIII/MtIII, subcutaneous injection will suffice. When local anesthetic solution is infiltrated on the axial aspect of the splint bones, the palmar/plantar metacarpal/metatarsal nerves are likely blocked, making it possible to abolish pain from a more distal site and leading to misinterpretation of results.

Suspensory Ligament Origin

Local infiltration or flooding the palmar/plantar metacarpus/metatarsus at the origin of the suspensory ligament is often done in lieu of perineural analgesia, as described previously. This is also referred to by some as sub-tarsal or sub-carpal blocks. An 18- to 22-gauge, 2.5- to 4-cm needle can be used to distribute 5 to 15 ml of local anesthetic solution in a fan-shaped pattern, usually from a lateral injection site just axial to McIV/MtIV. It is important to use adequate restraint and have the limb in a flexed position when performing this technique. In the hindlimb an 18- to 19-gauge needle should be used to minimize the potential for needle breakage, should the horse kick during the procedure.

False-positive results, attributed to inadvertent analgesia of palmar/plantar metacarpal/metatarsal nerves, penetration of the distal outpouchings of the carpometacarpal and tarsometatarsal joints, or penetration of the tarsal sheath can occur.[22,47] Compared with high palmar analgesia, the incidence of inadvertent injection of the distal palmar outpouchings of the carpometacarpal joint was highest when local infiltration of the suspensory origin was performed.[22]

Curb

Curb, the term used for swelling of the distal, plantar aspect of the tarsus, is a complex condition involving superficial digital flexor tendonitis, long plantar desmitis, subcutaneous swelling, or various combinations of these soft tissue injuries

(see Chapter 79). Local infiltration can partially abolish pain associated with curb and usually involves depositing local anesthetic solution subcutaneously. Completely blocking deep pain associated with the long plantar ligament or SDFT is not possible without using the fibular and tibial nerve blocks. A tibial nerve block may be more specific.[5] A 20-gauge, 2.5- to 4-cm needle is used to inject 15 to 20 ml of local anesthetic solution with the limb in a flexed position. Adequate restraint and the help of an assistant are mandatory. Local anesthetic solution is infiltrated subcutaneously along the plantar, medial, and lateral aspects of the swelling, but deep injection into or between the SDFT and long plantar ligament is avoided. The medial injection is most comfortably and safely performed by standing on the opposite side of the horse.

Dorsal Spinous Process Impingement

This local infiltration technique is performed when attempting to confirm or rule out lameness or poor performance associated with back pain caused by impingement or other pain originating from the dorsal spinous processes of the thoracolumbar vertebrae.[55] The horse is usually evaluated under saddle or in harness or on a lunge line, because lameness associated with this condition may be subtle and only manifested under these conditions. The hair along the dorsal midline is clipped, and the site or sites are prepared aseptically. We prefer to use 22-gauge, 9-cm spinal needles, although in most instances shorter needles can easily reach the tops of the dorsal spinous processes. Needles are inserted on the dorsal midline and directed ventrally to the dorsal spinous processes or the inter-spinous space. Markers placed after scintigraphic or radiographic examination are helpful to determine the precise location for blocking or to administer medication. The interspinous space can be located by redirecting the needle in a cranial or caudal direction. If impingement of the dorsal spinous processes exists, it may be impossible to infiltrate between them, but placing local anesthetic solution around the processes is satisfactory.[5] Seven to 10 ml (per site) of local anesthetic solution is deposited as the needle is slowly withdrawn, and the horse is reevaluated 10 to 15 minutes later.

Orthopedic Implants

Occasionally, pain associated with orthopedic implants is suspected to cause lameness. This is most commonly seen in horses after distal McIII/MtIII condylar fracture repair but can occur after repair of proximal phalanx or olecranon process fractures. Low-grade lameness is most common. Differentiating pain arising from negative interaction of implants with bone or surrounding soft tissue is nearly impossible based on the results of any diagnostic analgesia technique, because innervation to the joint or surrounding tissues is complex. Local anesthetic solution can be injected around screw heads, next to pins and wires or bone plates, and the horse is then re-evaluated. Because lameness is often subtle, improvement is often difficult to judge. A combination of clinical findings and those from ancillary diagnostic techniques is used to determine the role of implant pain.

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CHAPTER • 11

Neurological Examination and Neurological Conditions Causing Gait Deficits

Jill Beech and William V. Bernard

Differentiating neurological gait deficits from lameness can sometimes be a dilemma for the clinician. Many repeated examinations and ancillary testing may be necessary, and even then experienced clinicians may give varied opinions about the same horse. Lack of definitive diagnostic tests to identify the origin of subtle gait changes, which in some horses may be perceived only by a rider or driver and not visible, promote diagnoses that are based purely on opinions and individual prejudices. This chapter discusses the examination of the horse with gait deficits caused by disease of either the spinal cord, the most frequently documented cause of neurological gait deficits, or peripheral nerves. The chapter does not consider neurological syndromes characterized by signs of brain dysfunction, such as vestibular, cerebral, and cerebellar disorders. We also refer readers to a recent review on the equine spinal cord.[1]

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DIAGNOSIS

History is important but depends not only on asking the appropriate questions but also on many uncontrollable factors, such as how closely, impartially, and astutely the horse has been observed. The time of onset of signs and rate of progression, whether the gait deficit waxes and wanes or is affected by exercise or rest, whether one or more limbs are affected, whether the affected limb varies, what the horse was doing before onset of signs occurred, whether exercise or management has changed, whether the horse has been moved geographically, whether signs occurred after transport, what medications may have been given and any observed effects, and whether other horses on the farm or in the stable have had any recent illnesses or fever should be determined. It is also important to know if other horses on the same farm have similar clinical signs. For instance, a history of fever, respiratory disease, or abortions in horses in contact with the patient would make one suspect equine herpes virus I (EHVI) infection.

Clinical Examination

The clinician should observe whether the horse displays cranial nerve dysfunction; muscle hypertrophy, atrophy, or asymmetry; muscle trembling; abnormal hoof wear; or abnormal posture. Muscle atrophy may be due to disease of the ventral horn cells of the gray matter of the spinal cord, peripheral nerve, or the muscle itself; it also can occur with disuse. Palpation can reveal abnormalities such as altered skin temperature, sweating, muscle fasciculations, abnormal sensitivity, or soreness. The horse should be observed on a flat surface, at a walk and trot, and in straight and curving lines. The horse should be evaluated on a surface that allows detection of abnormal hoof flight and placement, toe dragging, or excessive force when landing. The sound of the feet landing should be noted for consistency and loudness. Hard surfaces also may enhance abnormal hyperflexion in horses with stringhalt. Evaluation on a soft surface may be necessary if the horse is unstable or if the clinician is trying to determine whether the horse could have sore feet. Any abnormal head or neck movement associated with limb movement should be noted. It is important to permit normal neck and head movement when the horse is being led. The person leading the horse should hold the horse as loosely as is safely possible. Collapsing or sinking on a limb, knuckling, hyperflexion, spasticity, hesitance in any part of the stride, dragging of a toe, landing excessively hard, leaning to one side, or failing to track straight can indicate a neurological deficit. Various manipulations are used to diagnose whether proprioceptive or motor deficits exist and to localize the lesion. While being led, the horse should be evaluated while stopping and starting from a walk and trot, backing up, circling tightly in both directions, walking while sideway traction is applied and released on the tail, being pushed sideways from a standstill, and walking with its head elevated. Some clinicians also evaluate repositioning of the foot after placing the horse's hoof in an abnormal position. We do not find this particularly helpful because a horse's disposition, age, training, and distractions can affect its response. A horse with a normal gait may stand with its feet placed in an abnormal position for what seems an abnormally long time. Some clinicians also use wheelbarrowing and hopping reactions.[1] We do not use these tests in mature horses because we believe responses may be inconsistent and difficult to evaluate accurately and safely. Observation of the horse walking or trotting up and down inclines can be helpful in revealing whether the horse "knows where its limbs are" (proprioception) and can adjust limb movement appropriately. It may be helpful to observe the horse while it is being ridden or lunged, and in some horses while it is loose in an enclosure. Watching the horse stop and start, turn, back up, and maintain its balance during many postural maneuvers allows detection of neurological deficits that may not be obvious when the horse is being led. Are errors in range of movement of the limb (dysmetria) apparent? Is the horse extending its limbs to the full extent, or is the range decreased (hypometria)? Does the horse lift its limbs excessively high (hypermetria)? Is spasticity or stiffness of movement apparent? In some horses it is necessary to observe the horse performing its usual activity, providing it is capable. However, gait deficits may be much less apparent at speed than when the horse is walking or trotting slowly. Basically, the clinician is trying to determine whether the horse moves symmetrically and smoothly with normal stride length and height of foot flight appropriate to the breed and use, whether it appears strong and consistently places its feet in the appropriate positions, and whether it moves in balanced harmonious fashion. It is sometimes difficult to determine whether certain postural or gait changes are due to pain or weakness, or associated with motor or proprioceptive deficits. Is the horse flexing its hindlimbs excessively and holding its croup more ventrally and flexed because of pain or weakness? If the horse is shifting weight between the hind feet, is it because of weakness, as seen for example in lower motor neuron disease, or because of pain? When both limbs are affected, manipulation of a limb to try to localize pain may not be possible. Gaited horses can be extremely difficult to evaluate, especially if one is unfamiliar with the specific gaits. Conformation also can confound interpretation of clinical signs. It may be necessary to observe the horse on many occasions and compare its gait before and after exercise. Is the deficit consistent, or does it vary? If it worsens with exercise, is it because of pain or inability to compensate for a neurological deficit as the horse tires? Perineural analgesia may be helpful. Is there palpable evidence of muscle cramping with exercise or an increase in creatine kinase (CK) level, indicating rhabdomyolysis? A variable gait deficit and inconsistent alterations in foot flight or placement are more likely to represent a neurological deficit than lameness; single limb lameness may vary in intensity but usually remains similar in character. Painful and neurological conditions could coexist but may be difficult to differentiate even with use of commonly used analgesics such as phenylbutazone.

If the horse buckles in a limb, especially on turns, is easily pulled sideways by the tail when standing or walking, or trembles its limb, weakness of the extensor muscle groups should be suspected. When the flexor muscles are weak, the horse is unable to lift its limb normally, and the toe may be worn from dragging. Pushing the horse sideways or trying to pull on the halter and tail simultaneously can reveal weakness. If the horse is weak or has pain in one limb, it is not able to bear weight normally when the contralateral hoof is lifted from the ground. Neck flexion sideways and vertically should be evaluated for ease and range of movement. Skin sensation and the cutaneous trunci reflex and cervical reflexes should be evaluated. Tapping the trunk should elicit contraction of the cutaneous trunci muscle. Abnormalities can delineate a thoracic spinal cord lesion, because afferent input is through the dorsal thoracic nerves and cranially through the spinal cord white matter, and the efferent pathway involves the cranial thoracic motor neurons in the first thoracic and eighth cervical segments and the lateral thoracic nerve. Hypalgesia of the cutaneous trunci as assessed by response to a two-pinch test with a hemostat is rare and occurs only with severe thoracic spinal cord disease.[1] Lack of a cervicofacial reflex (failure of the facial muscles to twitch when the ipsilateral side of the proximal neck is tapped) can suggest a lesion in the cervical cord or a branch of cranial nerve VII. If tapping the side of the neck fails to elicit contraction of the cutaneous coli muscle, a cervical cord lesion could exist. If any abnormal response to skin stimulation is detected, the test should be repeated because

the horse's disposition can influence its responses. Limb reflexes usually are not used, although patellar reflexes can be elicited in horses. We do not consider the thoracolaryngeal reflex (slap test) to be helpful. Response is inconsistent in horses with cervical spinal cord lesions and may be absent in normal horses. Blindfolding the horse usually is not part of our routine neurological examination unless vestibular disease is suspected. A complete physical examination should always be conducted. In some horses with hindlimb gait deficits, palpation per rectum of the pelvic bones, lumbar region, distal aorta, and iliac vessels may be necessary. Simple observation may not differentiate hindlimb weakness caused by spinal cord disease from that caused by partial aortoiliac thrombosis. Horses that do not "feel right" to the rider yet show no obvious deficits to the observer whether observed saddled or in hand are problematic. It may be necessary to observe a horse from the jog cart or carriage if the gait deficit about which a driver complains is not visible to the bystander. In attempting to differentiate between a musculoskeletal and neurological condition causing a gait deficit in a limb, diagnostic analgesia may be necessary. Obviously, this does not help differentiate pain from lameness emanating from a lesion proximal to the coxofemoral or scapulohumeral joints. A course of non-steroidal anti-inflammatory drugs (such as moderate doses of phenylbutazone for days or even several weeks) may be helpful in determining whether a gait deficit is caused by pain.

Hematology and Serology

In most horses serum chemistry screens and hematological tests are not particularly helpful; however, in horses with a gait deficit caused by an underlying muscle disease, evaluation of aspartate aminotransferase (AST) and CK levels may be helpful. Stage of training, exercise pattern, and whether the blood specimen is obtained after exercise preceded by a day of rest must be considered in evaluation of enzyme levels. If a horse consistently has abnormally elevated enzyme levels, the horse has rhabdomyolysis, and the clinician must decide whether the condition is causing or contributing to the horse's abnormal gait. Plasma CK and AST levels do not increase simply because of muscle atrophy; rhabdomyolysis must occur to increase the enzyme levels in the blood (see Chapter 84). Elevated plasma concentrations of CK and AST in horses that are not being exercised suggest a primary muscle disorder, such as (but not limited to) polysaccharide storage myopathy (see Chapter 84). An elevation in white blood cell count and fibrinogen level indicates inflammation. In our experience, elevation in fibrinogen level is a more consistent indicator of inflammation in the adult horse than is elevation in white blood cell count.

If clinical signs suggest equine lower motor neuron disease, serum levels of vitamin E (α-tocopherol) should be measured because levels consistently have been low unless the horse has been given supplements.[2] Low vitamin E levels are not specific for equine lower motor neuron disease. Tocopherol concentrations can decrease during winter when horses lack access to green pasture.[3] Daily variations in plasma levels may occur.[4] Low levels also have been reported in clinically normal horses[5-7] and in one horse with chronic gastrointestinal disease.[8] The laboratory that performs the test should be contacted for any specific requirements for submission of samples and to ensure they have an established normal range for vitamin E levels.

Serological testing for antibodies to various infectious agents may be indicated. In EHVI infection, detection of an increase in antibody titer is considered diagnostic of the disease. A horse that shows signs of neurological disease secondary to EHVI should have an elevated serum antibody titer, and single high titers have been the basis for initial diagnosis in individual horses. Recent vaccination confounds interpretation. Rarely, high titers may be measured in horses with no history of recent vaccination and no obvious clinical signs of EHVI infection.

Antibody titers for *Borrelia burgdorferi*, the cause of Lyme disease, sometimes are measured in serum from horses with ill-defined gait deficits. High titers, or rising titers, have been used as a basis for treatment of the disease. A positive titer, however, does not mean the horse has active disease. Because of the geographical variation in exposure to *B. burgdorferi*, titers may vary greatly. Serological surveys in the United States have demonstrated positive test results in 1% of samples from non-endemic areas and up to 68% in endemic areas.[9-11] Reports of horses "responding" to treatment exist,[12,13] but to date we are unaware of any horses with Lyme disease in in which neurological deficits mimic primary lameness. Currently the importance of Lyme disease as a cause of equine gait deficits is unclear.

Serological testing for the presence of antibodies to *Sarcocytis neurona* can be used only to indicate exposure to the organism. Serological surveys in certain areas of the United States have shown that a high percentage of horses have positive antibody titers. A positive test result does not mean the horse has equine protozoal myelitis (EPM). A negative test result could theoretically occur in horses with peracute disease or perhaps in severely immunocompromised animals. However, a negative test result usually indicates that disease caused by *S. neurona* is highly unlikely. The test result also could be negative in a horse with signs of EPM if another protozoan, such as *Neospora*, causes the spinal cord lesions. In a U.S. study of several hundred horses with neurological disease, test sensitivity was 89%, but specificity was only 71%, because 30% of horses with other neurological diseases also had antibodies to *S. neurona*. Although the positive predictive value was only 72% in horses with neurological diseases, the negative predictive value was almost 90%, indicating that a negative test result is useful in this population.[14] In one study of 44 horses on a farm sampled for more than 1 year, all horses were seropositive for at least 50 weeks yet showed no neurological signs.[15]

Cerebrospinal Fluid Aspiration and Analysis

Cerebrospinal fluid (CSF) can be obtained from either the atlanto-occipital or the lumbosacral space. The advantage of lumbosacral centesis is that it can be performed in the standing sedated horse, whereas atlanto-occipital centesis requires general anesthesia. Fluid from the atlanto-occipital site is considered easier to obtain and not as likely to be contaminated with blood. The atlanto-occipital site is identified by palpating the cranial edge of the wings of the atlas. The hair is clipped and the site prepared aseptically. Atlanto-occipital centesis is performed at the intersection of the median plane and a line drawn across the cranial edge of the wings of the atlas. In the adult horse a 9-cm (3½-inch), 18- or 20-gauge spinal needle is directed toward the horse's lower lip with the head held in a flexed position. It is important that the needle remain on the midline as it is advanced, because otherwise it will be too far lateral to enter the sub-arachnoid space. The needle is initially inserted to a depth of approximately 2.5 cm (1 inch) and then gradually advanced. While the needle is gradually advanced to the sub-arachnoid space, it should be held carefully to prevent penetrating the spinal cord when advancing through the atlanto-occipital membrane and the dura mater. Usually a "pop" is felt as the needle advances through the dura; however, this finding is not consistent and the stylette should be frequently removed to observe for flow of CSF. CSF usually flows from the needle once the sub-arachnoid space is entered; however, once a substantial depth has been reached (about 5 to 8 cm [2 to 3 inches] in the average-size horse),

some clinicians advise gentle and frequent aspiration with a small syringe.

In preparation for aspiration from the lumbosacral space the type and degree of restraint is guided by the horse's behavior, the horse's stability, and the clinician's personal preference. A nose twitch, stocks, sedation, or a combination of physical and chemical restraint are options. We prefer to use light sedation with xylazine, sometimes combined with butorphanol. However, lumbosacral CSF pressure can be transiently decreased up to 15 minutes after administration of a high dose of xylazine (1.1 mg/kg intravenously).[16] The puncture site for lumbosacral centesis is identified by combining several landmarks, realizing that individual variation exists. A line drawn between the caudal edge of the tuber coxae and the intersection with the midline can be used to locate the lumbosacral space. The lumbosacral space is bordered cranially by the caudal edge of the sixth lumbar vertebra, caudally by the cranial edge of the sacrum, and laterally by the medial rim of the tubera sacrale. The dorsal spinous process of the last lumber vertebra is lower than the dorsal spinous process of the fifth lumbar vertebra. The V formed by the medial rim of the tubera sacrale is one of the more useful landmarks, and the appropriate site for puncture is within this V. The site should be prepared aseptically, and local anesthetic solution is placed subcutaneously. A small skin stab incision is usually made. The needle is inserted on the midline, at the depression palpated just caudal to the last lumbar vertebra, in the middle of the V formed by the tubera sacrale. A 15-cm, 18-gauge spinal needle is generally adequate for a horse that is 16 hands or less. A 20-cm needle may be necessary in a horse greater than 16 to 17 hands. While the clinician advances the needle, it is critical to remain on the midline. The needle can be advanced until a pop indicates it is advancing through the dura or until the patient responds as the needle stimulates nervous tissue. These responses can be unreliable and occasionally dangerous for the horse, handler, and the individual performing the tap. Because horses can react unpredictably (including rearing, bolting, collapsing, or kicking), it is safer to advance the needle gradually until it is near the spinal canal, approximately 12.5 cm (5 inches) in a 15- to 16-hand horse. Once the needle is near the canal, it should be advanced slowly with repeated frequent removal of the stylette and aspiration with a small syringe. The horse may move its tail when the dura is penetrated, but usually minimal reaction occurs. If fluid is obtained, but the amount is small, the needle can be rotated 180°. Jugular vein compression for at least 10 seconds (Queckenstedt's test) is thought to elevate intra-cranial CSF pressure and aid fluid collection, provided flow is not obstructed. If a hemorrhagic sample is thought to be from iatrogenic causes, the syringe can be changed frequently until subsequent aliquots are clear. If fluid is not obtained on the first attempt, the needle is withdrawn and the procedure is repeated slightly cranial or caudal to the original location. CSF samples should be placed in sterile tubes and rapidly processed after collection.

Normal CSF is clear and colorless, and red discoloration indicates hemorrhage. However, normal fluid can sometimes appear mildly hazy when grossly examined, especially in a tube with ethylenediamine tetraacetic acid. Hemorrhage can be iatrogenic or caused by underlying disease. Fluid may appear clear even with red blood cell contamination, and studies indicate that subjective evaluation of spinal fluid is sensitive in detecting blood only when the red blood cells number more than 1200/μl.[17,18] Centrifugation of a bloody sample should produce a clear fluid with a pellet of red blood cells on the bottom of the sample tube. If hemorrhage occurred before collection and lysis of cells has occurred, the supernatant may be slightly pink or xanthrochromic (orange/yellow or yellow). Lysis of red blood cells reportedly can occur within 1 to 4 hours.[19] Xanthochromic CSF results from red blood cell breakdown products (bilirubin) and suggests hemorrhage or vasculitis. A centrifuged xanthochromic sample does not become clear. Turbid CSF may appear with hypercellularity or epidural fat contamination. The latter is not uncommon with lumbosacral aspirates. Formulas used to differentiate between white cell or protein elevations caused by iatrogenic blood contamination of CSF versus pathological increases have been shown to be unreliable. Contamination with a few thousand red blood cells results in minimal increase in white blood cell count or protein content.[18]

The normal reported range for leukocyte counts has been variable; usually a range of 0 to 6/μl is cited,[20] but higher values have been reported.[21,22] Diversity in techniques can account for different values in normal CSF. Undiluted fluid can be assayed in a hemocytometer, or acidified crystal violet can be added to accentuate the cells.[20] It is important that equine reference values be determined in the laboratory the practitioner uses. As previously stated, the cell quality rapidly deteriorates in CSF, and samples for cytological testing should be processed rapidly or a portion fixed in 40% ethanol if processing must be delayed. For morphological and differential evaluation, cytocentrifugation or filtration through a glass fiber membrane filter is the preferred method of processing spinal fluid. In our experience, cell and differential counts are often normal in horses with spinal cord disease. Small lymphocytes and monocytes are normally seen. Neutrophils may be seen with blood contamination or inflammation. Eosinophilia is rarely seen in equine CSF but could occur secondary to parasite migration. Rarely, eosinophils have been seen in samples from horses with protozoal encephalomyelitis,[21] but frequently spinal fluid from horses with EPM is normal. A relative neutrophilia, with or without an increase in cell count, indicates inflammation, and intracellular bacteria may be seen in horses with bacterial meningitis.

Reported values for protein content of CSF vary considerably between laboratories, probably because of diversity in measurement techniques. A range of 10 to 120 mg/dl is generally acceptable, although some authors consider 100 to 105 mg/dl the high end of normal range.[20,23] Protein may increase because of vascular leakage (vasculitis), inflammatory lesions, trauma, iatrogenic blood contamination, or intrathecal globulin production. High-resolution protein electrophoresis of CSF has been reported in a small number of horses, but its value as a diagnostic test remains to be determined. Compared with normal horses (n = 18), horses with cervical cord compression (n = 14) often had a decreased β fraction and post-β peaks.[24] However, divergent findings have been reported. Because CK is abundant in neural tissue (and in skeletal tissue and cardiac muscle) and is a large macromolecule that does not cross the blood-brain barrier, measurement was suggested to be a sensitive index of central nervous system lesions. Horses with EPM were reported to frequently have increased CSF CK concentrations, unlike horses with cervical vertebral malformation.[25] However, another study showed the sensitivity and specificity of CSF CK activity are inadequate for diagnostic use. Also, CSF simultaneously collected from the atlanto-occipital and lumbosacral sites had disparate values for CK activity, which was not associated with site or other CSF parameters. Contamination of CSF with either epidural fat or dura, which is possible during collection, increases CK activity.[26]

Albumin is the predominant protein in normal CSF. Elevated albumin concentration can indicate hemorrhage or altered blood-brain barrier integrity. To eliminate serum albumin as a source of increased CSF protein and albumin, the following albumin quotient (AQ) has been suggested[27]:

$$AQ = \frac{\text{CSF albumin}}{\text{Serum albumin}} \times 100$$

The AQ cited for normal equine CSF 1.4 ± 0.04,[23,27] and it was suggested that an increase above reference range indicated blood contamination during sample collection or compromise of the blood-brain barrier. The immunoglobulin G (IgG) index

$$\frac{\text{CSF IgG concentration}}{\text{Serum IgG concentration}} \div \frac{\text{CSF albumin concentration}}{\text{Serum albumin concentration}}$$

was suggested to be useful for differentiating intrathecal IgG production from an increase secondary to blood contamination or increased blood-brain barrier permeability. Normal reference range has been reported to be 0.14 to 0.24.[27] However, we do not consider these to be specific, and a recent study has shown that blood contamination can increase the IgG index without a concomitant change in AQ.[18]

Although CSF cell count, cytological examination, and total protein often do not represent the extent or type of spinal cord or brain tissue disease, when abnormal, the values can be useful. For example, in CNS disease caused by EHVI infection the fluid may be xanthochromic with a high protein level but normal cell count. This disassociation between elevation in protein level and normal cell count may help differentiate EHVI infection from EPM. Also, xanthochromic CSF indicates an alteration in the blood-brain barrier and could explain false-positive CSF immunoblot findings for *S. neurona* in a horse with positive serological test results. Unfortunately, except for EHVI infections and meningitis, CSF analysis with currently available tests frequently is not helpful in diagnosing spinal cord disease in horses.

In the United States the frequency of performing CSF aspirates increased with the introduction of a Western immunoblot test for detecting *S. neurona* antibodies. Although limited data are available, the specificity and sensitivity of the immunoblot test on CSF from horses with clinical signs consistent with EPM were reported to be approximately 90%.[14] However, positive test results have been found in clinically normal horses and in horses with neuropathological lesions other than EPM. Even minute amounts of contamination of CSF with blood can cause the test result to be positive in a horse with high serum antibody levels.[19] When CSF was contaminated by even minute amounts of strongly immunoreactive blood (10^{-3} μL of blood/ml of CSF), the fluid was falsely positive even though the AQ was normal.[18] This small amount of blood contamination is grossly undetectable and can correlate with as little as eight red blood cells per microliter of CSF. Also, blood contamination, without increasing the AQ, can increase the IgG index. The IgG index is not specific for intrathecal IgG production. Although the red blood cell count may be a more sensitive indicator of blood contamination than the AQ, it does not correlate with the amount of antibody contamination. Minute amounts of highly immunoreactive blood may have a greater impact on CSF Western blot analysis than a greater amount of contamination with blood with low immunoreactivity.[18] Any compromise to the blood-brain barrier regardless of cause allows antibodies to leak into the CSF from the serum, causing a false-positive test result. Although the test for immunoblot *S. neurona* antibodies was reported to have 85% positive predictive value in a study of horses with neurological disease,[14] in the general equine population the test has poor positive predictive value. Many normal horses have positive antibody test results. In contrast the negative predictive value for the test is high. With what is currently known, interpretation of positive Western blot results must be made with caution. Negative Western blot tests are generally useful to rule out EPM.

Polymerase chain reaction (PCR) testing detects DNA of infectious organisms and has been applied to CSF. Its value in the diagnosis of EPM is controversial, especially when positive results have been reported on CSF samples that were negative for *S. neurona* antibodies and from horses that did not exhibit overt neurological deficits. We do not find PCR testing for the diagnosis of EPM useful. PCR technique on CSF has been useful in diagnosing neuroborreliosis in a horse. Similar to some human cases, the PCR test result was positive yet the CSF had a negative antibody titer.[12]

Radiography

The use of radiographs in evaluating traumatic or infectious injuries, congenital lesions, and developmental malformations of the spinal column is limited by the size of the patient. Radiographs are useful in diagnosing congenital abnormalities of vertebrae, narrowing of intervertebral disk spaces, stenosis of the cervical spinal canal, osteoarthritic changes, osteomyelitis or osseous cysts, vertebral neoplasia, malalignment, and fractures. However, in most mature horses, except for the cervical spine, anesthesia may be required for adequate radiographs of the spine. Computed tomography (CT) and magnetic resonance imaging have tremendous potential for evaluating the equine central nervous system but also are limited by the size of the horse. At present, except in foals, CT is available only for evaluating the head and cranial–mid-cervical regions. The primary use of radiography in evaluating horses with neurological disease is localization of cervical vertebral lesions or cervical vertebral malformation and diagnosis of cervical compressive myelopathy or stenotic myelopathy (see Chapter 62).

Survey radiography is useful in the diagnosis of cervical vertebral malformation and cord compression but can be misleading. Standing lateral radiographic views of the cervical vertebrae are routinely evaluated to detect vertebral malformation and to measure spinal canal diameter and can suggest the likelihood of cervical compressive myelopathy.[1,29,30] In horses with cervical compressive myelopathy, malformations that characteristically may be identified include flare of the caudal epiphysis of the vertebral body (vertebral endplate remodeling), caudal extension of the dorsal laminae, vertebral non-alignment, and osteoarthritis of articular facets. Remodeling of the articular processes of the caudal cervical vertebrae is a common malformation identified in horses with cervical compressive myelopathy and in horses not affected with cervical compressive myelopathy. Interpretation of changes is more difficult in older horses, because obvious changes may be seen radiographically, without impingement on the spinal canal. Subjective evaluation of articular facet abnormalities can result in a false-positive diagnosis of cervical compressive myelopathy. Identification of characteristic vertebral malformations supports, but does not confirm, the diagnosis of cervical compressive myelopathy, and subjective evaluation of radiographic malformation does not reliably differentiate between horses affected or not affected with cervical compressive myelopathy. Objective assessment of vertebral canal diameter is a more reliable indicator of cervical compressive myelopathy than the subjective evaluation of vertebral malformation. The minimum sagittal diameter (MSD) is the first described method of assessment of canal diameter based on lateral cervical radiographs.[29] Determination of canal diameter using the sagittal ratio improves on the original measurements by adjusting for magnification and providing a more accurate adjustment for body size.[30] The sagittal ratio measurements were developed using a population of affected (confirmed by myelogram or histopathological studies) versus non-affected horses.[30] The sagittal ratio is determined by dividing the MSD by the width of the corresponding vertebral body. Although a sagittal ratio percent at any cervical vertebra from the third to seventh cervical vertebrae less than 50% is a strong predictor of spinal cord compression, a few horses with no pathological evidence of spinal cord compression have had sagittal ratios of less than 50%. Recently intervertebral measurements of canal diameters were shown to improve diagnosis of cord compression, and addition of intervertebral sagittal ratio measurements was recommended to increase accuracy of plain radiographs.[1]

A semi-quantitative scoring system for evaluating cervical radiographs in horses younger than 1 year of age has been published. This scoring system used neurological examination alone to determine affected versus non-affected foals and combined subjective determination of radiographic vertebral malformation and objective determination of canal diameter.[29] Vertebral canal stenosis is determined by measurement of intervertebral and intravertebral MSD. Dividing the MSD by the length of the vertebral body corrects for magnification. Malformation is determined by the subjective assessment of five categories. The most discriminating factors in the semi-quantitative scoring system in differentiating affected from non-affected foals are canal stenosis and the angle between adjacent vertebrae. The disadvantage of the semi-quantitative scoring system is the inclusion of subjective determinations.

Myelographic examination is advised to obtain the best evidence of compression.[1,31-33] Myelograms also can demonstrate compression from soft tissue masses, which are not evident on radiographs, and suggest transverse compression. However, myelography may not be definitive and occasionally is misleading. A study to evaluate myelography critically and compare the results with necropsy findings in a large number of horses has not been done. A diagnosis of cord compression is assumed if a 50% reduction in the width of the dorsal dye column exists. However, the diagnostic criterion of 50% decrease in width of the dorsal dye column is not well documented[32] and has been found in horses with no histological evidence of cord compression at the site of dye column decrease. Iohexol is currently the preferred contrast medium for myelography. It is important that the owner understands the advantages and disadvantages (including risks) of a myelogram before the procedure is undertaken.

Electromyography and Nerve Conduction Studies

Recording electrical activity of muscles can indicate whether evidence of denervation or a myopathy exists, although the distinction is not always clear-cut. Electromyographic examination in the early stages of disease or injury may be normal. Certain abnormal patterns can indicate denervation. However, depending on the specific areas to be examined, electromyography may require anesthesia or heavy sedation. It may be helpful in identifying abnormal muscles and indirectly the affected nerves. In a standing, awake horse, spontaneous muscle movement can hinder interpretation.

Values for sensory and motor nerve conduction velocities in horses and ponies have been reported.[34-37] Differences in speed of conduction occur in different nerves and horses' sensory nerve conduction velocities are slower than those of ponies.[37] However, similar motor nerve conduction velocities have been reported for the median and radial nerves of ponies and horses.[35] Location of the segment being measured may be important, because distal tapering of nerves may be associated with slower velocity. Skin temperature significantly affects nerve conduction velocity,[37] and variability in technique can alter findings. Slower motor nerve conduction velocities were reported in horses older than 18 years of age.[34] The procedure usually requires the horse be anesthetized and, similar to electromyography, should be performed by a skilled person. The technique mainly has been used in research.

Nuclear Scintigraphy

Nuclear scintigraphy has been helpful in identifying lesions in the thoracic and lumbar spinal column and pelvic areas not readily evaluated by radiography. It also has been used to evaluate vertebral changes identified radiographically, to determine whether active bone change has occurred. It has revealed hairline fractures and other unsuspected bone lesions in the appendicular skeleton as the cause of gait deficits, which sometimes had been suspected to be caused by spinal cord disease. Scintigraphic imaging from both sides of the horse can differentiate which side may have a lesion. The role of nuclear scintigraphy in diagnosing equine spinal cord disease is limited.

Ultrasonography

Ultrasonography has been used to diagnose aortoiliac thrombosis and to identify soft tissue masses near the spine or deep within muscles. It has also revealed bony proliferation or fractures of the pelvis in horses with obscure gait deficits, which were originally suspected to be due to spinal cord disease.

Virus Isolation

If horses die or are euthanized with neurological signs thought to be caused by viral disease, the spinal cord, brain, or both should be sent for virus isolation. In horses with acute disease, nasal swabs and whole blood samples can be collected.

Immunohistochemistry and Polymerase Chain Reaction Testing

Immunohistochemistry and PCR testing can be used to detect the antigen of certain infectious organisms and are applied most commonly to tissues collected at necropsy but can also be used on affected tissues obtained by biopsy.

SPECIFIC DISEASES AND SYNDROMES

Equine Protozoal Myelitis

EPM was first reported in 1974[38-41] and appeared to be the same condition originally reported as segmental myelitis of unknown cause.[42] It is caused by infection with *S. neurona*. EPM currently appears to be limited to the Western hemisphere. It is particularly of concern in the United States, where in some regions a high percentage of horses are infected. The actual number of horses confirmed as having neurological disease from EPM is much lower than the actual number of horses infected, but the disease does have a substantial and serious impact. EPM has not been confirmed in horses younger than 6 months of age, although antibodies were detected in serum from a 2-month-old foal.[43] A recent comprehensive review of this disease should be consulted for details.[44] *Neospora* species have been identified as a cause of EPM in horses from the western United States.[45-48] CSF testing was positive for *S. neurona* antibodies by Western blot test, and no antemortem features distinguished *Neospora* infection from *Sarcocystis* infection.

The disease caused by *S. neurona* tends to occur in warm, temperate, non-arid areas with resident opossums. The horse is a "dead-end" host and the disease is not contagious. The life cycle is not completely understood, although opossums have been identified as the definitive host. The proportion of infected horses that show clinical signs is low. This disease can cause gait deficits affecting one or all limbs and may be difficult or impossible to differentiate from musculoskeletal or other neurological diseases. Signs ascribed to EPM by veterinarians in the United States have been seen in horses in the United Kingdom, where horses have no known exposure to the organism.[1] Infected horses and horses with confirmed EPM seen in Europe, Asia, or South Africa have been imported from the Western hemisphere.[44] Horses frequently show asymmetrical deficits and may have focal or multifocal muscle atrophy or cranial nerve deficits. Horses may have profound or mild motor or proprioceptive gait deficits, and onset of signs can be acute or chronic, with slow or rapid progression. It may be difficult or impossible to differentiate subtle neurological deficits from those caused by subtle lameness or musculoskeletal pain. Behavior may change. Focal sweating

may occur. Diagnosis is based on clinical signs and history, by eliminating other potential causes by radiography and other diagnostic tests, and by testing of serum or CSF for antibodies to *S. neurona*. No definitive antemortem test exists, although absence of serum antibodies to *S. neurona* makes it highly unlikely that a horse has EPM. If a horse demonstrates classic signs (e.g., asymmetrical motor deficits and muscle atrophy in the hindlimbs, asymmetrical motor deficits in one or more limbs, a limb deficit combined with cranial nerve deficits not deemed caused by peripheral nerve trauma) and has no other organ dysfunction, we would treat the horse for EPM if it has been in the United States and serological findings are positive. We would forgo CSF testing for reasons outlined earlier.

To date, drugs used to treat EPM have been a combination of trimethoprim sulfa (sulfadiazine or sulfamethoxazole) and pyrimethamine, or sulfas and pyrimethamine, diclazuril, toltrazuril, and nitazoxanide. Because no definitive antemortem test exists to confirm the disease, evaluation of response to therapy is problematic, especially because the clinical syndrome as treated is so variable and often poorly defined. To date, no treatment trials of experimental infections have been reported. Confounding assessment of drug response is the fact that experimentally infected horses develop clinical signs that decrease over time, despite receiving no treatment.[49] Numbers of organisms ingested, virulence factors, and the horse's own immune status (which depends on heredity, previous exposure to *S. neurona*, stresses such as transport and parturition, lack of adequate nutrition, and other factors) all presumably can affect development of and recovery from the disease. In the United States the most widely used drug combination is one of the sulfa drugs and pyrimethamine. Because pyrimethamine reaches higher concentrations in the CSF and neural tissue, it is considered superior to trimethoprim. The usual dosage regimen is 20 mg/kg of sulfadiazine once or twice daily and 1 mg/kg of pyrimethamine once daily, both by mouth for at least 2 to 3 months. Diarrhea occasionally occurs in horses treated with trimethoprim-sulfamethoxazole, and anemia and leukopenia have been observed in some horses receiving 1 mg/kg twice daily of pyrimethamine with sulfas. Whether horses require such a prolonged course of treatment or continued high levels of pyrimethamine is unknown. Earlier treatment regimens used a lower dose, but to our knowledge no observations comparing dosages have been reported. A syndrome of bone marrow aplasia and hypoplasia, renal nephrosis or hypoplasia, and epithelial dysplasia was reported in three foals born from mares given sulfonamides, trimethoprim, pyrimethamine, vitamin E, and folic acid during gestation. The authors of that report suggested that administration of the folic acid reduced absorption of active folic acid and combined with the folic acid inhibitors (trimethoprim and pyrimethamine) induced folic acid deficiency and lesions in the foals.[50] We do not routinely add supplements for horses being treated with trimethoprim or pyrimethamine, but if sequential blood tests indicate anemia or leukopenia, the horse should be given folinic acid, a form of bioactive tetrahydrofolate. Folic acid should not be used because it is poorly absorbed in the horse, conversion to its active form is prevented by the dihydrofolate reductase inhibitors pyrimethamine and trimethoprim, and it can competitively decrease absorption of the active form of folic acid.[44,50]

Diclazuril, a coccidiostat, has anti–*S. neurona* activity in cell cultures infected with *S. neurona*[51] and has been used to treat horses with suspected EPM.[52] It is absorbed quickly after feeding. Dosage and therapeutic efficacy is being evaluated. Toltrazuril, like diclazuril, is a triazine-based anti-coccidial drug. Because the drug has good lipid solubility and oral absorption and is absorbed into the CSF, it has potential for treating EPM.[53] Ponazuril, a metabolite of toltrazuril, has in vitro activity against *S. neurona*,[54] and the latter appeared to have favorable clinical results in a multicenter treatment study.[44]

Nitazoxanide kills *S. neurona* in cell cultures and has been tested in a field trial. Safety studies showed lethargy at twice the recommended dose and illness and death at four times the recommended dose. In seven horses with clinical signs compatible with EPM and positive immunoblot results for *S. neurona* antibodies in the CSF, clinical signs improved in six horses by the end of the trial (85 to 140 days).[55] Clinical signs recurred in two horses when treatment was stopped, but signs improved when treatment was re-initiated. Another report described two horses with a diagnosis of EPM that improved after 28 to 42 days of treatment with 50 mg/kg nitazoxanide once daily.[56] Anorexia and depression were reported as side effects.[56] The CSF remained positive for *S. neurona* antibodies. Until more information is available about this drug, we do not recommend its use.

To our knowledge, no evidence shows that concurrent use of immune stimulants, oral anti-oxidants, and anti-inflammatory drugs has any beneficial effect. The use of corticosteroids is controversial, because some clinicians claim corticosteroid administration can exacerbate infection. Severity of neurological signs in horses infected with *S. neurona* reportedly was increased by corticosteroids,[57] but in another study of induced disease, signs were less severe in horses given corticosteroids.[58]

Providing an accurate prognosis is difficult, given the inherent diagnostic problems. Some horses that recover or respond to treatment may not have EPM, and others may recover spontaneously. Economic factors influence duration of treatment and time allowed for convalescence. Even when a severely affected horse improves dramatically, if recovery of function is not complete, a return to previous performance levels is not possible. Signs also may recur in the same horse; whether this is caused by recrudescence of infection or re-infection is unknown. We usually give a guarded prognosis for full recovery of horses showing moderate gait deficits compatible with EPM.

Because the exact life cycle and natural intermediate hosts are unknown, definitive recommendations for control of the disease are difficult. Because the opossum is the definitive host and sheds sporocysts, which the horse ingests, fecal contamination of feedstuffs or water sources by this animal should be prevented. The role of other intermediate mammalian hosts is unclear. The efficacy of a recently introduced vaccine remains to be determined.

Cervical Spinal Cord Compression

Cervical vertebral malformations of various types have been described as the cause of cord compression and neurological signs.[1,59,60] Occasionally it may be difficult to decide if a horse is mildly affected with cervical cord compression or is bilaterally lame in the hindlimbs. Mildly affected horses may show only a slightly stiff, stabbing gait at a walk and trot, only mild circumduction of the outside hindlimb when turning, and equivocal hindlimb dysfunction at a canter. Horses with bilateral osteochondrosis dissecans of the hocks or stifles may show similar signs but usually also have joint capsule distention. Thorough lameness and neurological examinations and radiographs are needed. With more severe compression, the gait deficits increase. Circumduction may be severe, and the horse may strike the distal limb with the opposite hoof, causing hair loss or wounds from interference. A horse may lose balance or fall, especially when backing up or turning. If the caudal cervical spinal cord is compressed, thoracic limb motor deficits and hypometria, frequently asymmetrical, may occur. The horse may severely scuff or drag its toes and have abnormal hoof wear. Occasionally, substantial bony proliferation at the

synovial articular facets can result in neck stiffness and decreased ability to turn in one direction. Cervical muscle atrophy is rare but can occur if the nerves or lower motor neurons are affected. An affected horse usually lacks hindlimb impulsion and may have a somewhat stiff, bouncy canter. The horse frequently is imprecise when stopping, and the hindquarters may sway or bounce. When compression of the cranial cervical spinal cord occurs, the horse may hold its neck and head higher than normal, in an extended position, and in horses with severe clinical signs all limbs may be affected. Signs may occur suddenly or have a more gradual onset, and progression is variable.

Various vertebral abnormalities have been reported in young horses, but clinical signs can be delayed, even when radiographs reveal chronic lesions. We suspect that trauma may cause a pre-existing lesion to become clinically relevant. If a horse with vertebral malformation falls, acute spinal cord compression can occur. Acute cervical spinal cord compression caused by trauma can cause tetraparesis or recumbency, but signs may be delayed in the initial stages after injury and may become apparent only when muscle spasms subside, the unstable fracture displaces, or progressive hemorrhaging is present. In the neck the occipito-atlantoaxial and caudal cervical regions are predilection sites for spinal cord injury.[1] Synovial cysts may also cause severe sudden signs of spinal cord compression, often asymmetrical and sometimes intermittent.[1] The diagnosis of synovial cysts is usually made at necropsy.

Diagnosis of cervical cord compression is based on radiography and myelography. Numerous types of vertebral abnormalities have been described. Management depends on the nature of the lesion, severity of clinical signs, intended use of the horse, and financial considerations. Horses affected with cervical vertebral malformation and cord compression at less than a year of age may improve when exercise and energy intake are restricted.[61] Although no controlled studies of a paced diet and restricted exercise program have been conducted, clinical experience supports its use in young horses with radiographic evidence of cervical vertebral malformation.[1,61] This treatment is not helpful for young horses with very severe stenosis, for defects such as occipito-atlantoaxial or other cranial cervical malformations, or for older horses. Prognosis with conservative management is poor. Surgical fusion of vertebrae is indicated in some horses and has been used successfully.[60,62,63] This subject is discussed in Chapter 62.

Equine Degenerative Myeloencephalopathy and Neuroaxonal Dystrophy

Horses mildly affected with equine degenerative myeloencephalopathy and neuroaxonal dystrophy may be misdiagnosed as being lame. Clinical signs may be somewhat similar to those of cervical spinal cord compression. Because no definitive antemortem test exists, clinical diagnosis is based on clinical signs, sometimes supported by the presence of other affected horses on the same farm or in the same family.

Equine degenerative myelopathy is thought to be a vitamin E deficiency, with a likely genetic predisposition.[64,65] Neuroaxonal dystrophy appears to have a genetic basis in Morgan horses.[66] Various breeds and also Przewalski's horses and Zebras can be affected, and no geographical restriction is apparent. When horses are affected at a young age (i.e., < 6 to 12 months old), signs are more severe and progressive than when signs are first noted in horses 2 years old or older. However, because signs can be mild and only slowly progressive, owners may not be aware of the abnormality. When a severely affected horse is identified on an individual farm, other more mildly affected horses are often found on the same premises or among relatives. Signs tend to be most noticeable in the hindlimbs. Affected horses usually lift the hind feet too high and slap them down on the ground and frequently lift the hoof toward the midline and then place it more laterally. The gait is jerky and asynchronous and sometimes ataxic, with excessive sideways sway of the hindquarters. Interference may occur, with a hind hoof hitting the opposite hind fetlock or pastern region. The horse may have a jerky foot placement when stopping and may pivot on the hindlimbs when turning. Severely affected horses may show forelimb ataxia and weakness of all limbs. The gait lacks impulsion. Occasionally, middle-aged horses are examined because of inability to perform at collected gaits with impulsion and precision. No musculoskeletal cause is found, but the hindlimb gait is characteristic of mild equine degenerative myeloencephalopathy or neuroaxonal dystrophy. Mildly affected mature horses appear to function without substantial progression of signs. No ancillary diagnostic test confirms the disease. Vitamin E supplementation (5000 to 6000 units by mouth daily) has been used to treat affected horses, with some, but not total, improvement reported.[67] Horses at risk for the disease should be given vitamin E supplements. Supplementation on farms with a number of affected horses was associated with a subsequent decrease in the incidence of disease.[64] Mares and foals should have access to grass pasture, because lack of access to green pasture has been identified as a risk factor.

Equine Lower Motor Neuron Disease

Equine lower motor neuron disease has been diagnosed in many countries.[68] Older horses and those lacking access to green pasture appear to be at risk to develop equine lower motor neuron disease. The disease is thought to be caused by deficiency of antioxidant activity in the central nervous system, leading to degeneration and loss of lower motor neurons in the brainstem and spinal cord.[69] Affected horses lose muscle mass and have generalized muscle trembling, which may be more severe in the triceps and quadriceps and is exacerbated by transport. Other clinical signs include stiffness, shifting of weight between the hindlimbs, standing with all feet excessively under the body, excessive sweating, holding the tail elevated and trembling, long periods of recumbency, and sometimes excessively low head carriage. Trembling disappears when the horse lies down. Although the gait may be choppy, the horse has no lameness or ataxia. Horses move better than they stand, and therefore the condition is unlikely to be confused with lameness. Muscle atrophy may be profound. Ophthalmoscopic examination of horses with chronic equine lower motor neuron disease may reveal abnormal pigment deposition in the tapetum with a horizontal band of pigment at the tapetal-nontapetal junction.[70] Diagnosis is based on clinical signs and low serum vitamin E concentrations in unsupplemented horses or biopsy of the sacrocaudalis dorsalis medialis (dorsolateral coccygeal) muscle. Affected horses that have not been given vitamin E usually have serum vitamin E concentrations less than or equal to 1 μg/ml.[71] Biopsy of a branch of the spinal accessory nerve, which had a high specificity and sensitivity in diagnosis of equine lower motor neuron disease, has been replaced by the muscle biopsy, which is technically much easier, can be performed in the standing horse and has a similar diagnostic specificity and sensitivity.[72] However, false-positive test results can occur in horses that have had "tail blocks."[72] The dorsolateral coccygeal muscle is ideal for biopsy because it contains a high percent of type I oxidative fibers, which are the main muscle fibers affected in the disease. Other muscles with a high proportion of type I fibers are not accessible for biopsy. Most limb muscles have high percentages of type II fibers and are not suitable for diagnosis of the disease.

Oral vitamin E supplementation (6000 to 10,000 units by mouth daily) improves horses, and green pasture is also helpful. However, athletic ability may remain impaired.[69]

Equine Herpes Virus I Infection

Neurological disease caused by EHVI can occur in individual horses or as an outbreak. Ataxia is variable but usually symmetrical. The hindlimbs are more severely affected, and recumbency can occur. Signs occur acutely and usually stabilize within 24 to 48 hours. Because this condition is unlikely to be confused with lameness, it is not discussed further and readers are referred to a review.[1]

Miscellaneous Diseases of the Spinal Cord

Spinal cord disease from migrating parasites could manifest as an asymmetrical gait deficit. Incidence appears to vary geographically, and clinical signs reflect the path of migration. Antemortem diagnosis is usually not possible, although eosinophilia in the CSF supports the diagnosis. Various parasites including *Setaria* species, *Halicephalobus (Micronema) deletrix*, *Hypoderma*, and *Strongylus* species have been identified. Treatment includes anti-parasitic and anti-inflammatory drugs.

Vertebral osteomyelitis, neoplasia, and diskospondylitis are rare causes of spinal cord disease. Signs reflect location of the lesion, which may be confirmed by radiography or scintigraphy. CSF may reflect the disease condition if it extends through the dura. Traumatically induced diskospondylitis has been described and may be difficult to differentiate from bacterial diskospondylitis.[1,73] Spinal cord traumas may occur directly or from instability of intervertebral joints. External trauma can affect any horse, and clinical signs reflect the site of the lesions. Three predilection sites for injury are the occipito-atlantoaxial region, the caudal cervical region (the fifth cervical to the first thoracic vetebrae) and mid-thoracolumbar region.[1] Clinical signs may initially be mild or peracute, and some horses develop severe progressive signs. It is often not possible to perform an adequate or accurate neurological examination or form a prognosis on an acutely injured horse. Initial treatment includes first aid care, sedation if needed, and the administration of analgesics, anti-inflammatory drugs, and mannitol. Radiographs can be useful, depending on site of injury and size of the horse. Repeated neurological evaluations are used for prognosis.

Peripheral Nerve Injuries

Except for stringhalt and radial nerve injury, peripheral nerve diseases affecting the gait are rarely diagnosed. Suprascapular nerve injury *by itself* does not alter the gait but results in atrophy of the supraspinatus and infraspinatus muscles ("sweeney").[74-76] However, injury to the nerve usually occurs with more general trauma to the region such as a collision or fall. This type of injury frequently can lead to damage to other nerves of the limb and soft tissue structures. If other nerve roots of the brachial plexus are simultaneously damaged, the shoulder joint may be unstable and subluxate laterally. The horse may circumduct the limb during protraction. Rest and anti-inflammatory drugs are usually used. Several surgical procedures have been advocated for suprascapular nerve injury.[77]

Radial nerve paresis or paralysis is recognized, usually secondary to trauma. Horses with radial nerve paralysis cannot flex the shoulder joint or extend the elbow, knee, fetlock, or interphalangeal joints. The dorsum of the toe rests on the ground, and the elbow is dropped. Severely affected horses have difficulty rising and often collapse on the limb if it bears weight. More mildly affected horses may advance the leg by flinging or jerking it forward from the shoulder. Evaluation of skin sensation may not be helpful. Atrophy of the triceps and other limb extensor muscles occurs after 2 weeks, and denervation potentials can be found on electromyographic examination 3 to 4 weeks, or sooner, after radial nerve injury.[77,78] Because of the difficulty of knowing whether the gait deficits are due solely to radial nerve injury or muscle damage, an accurate prognosis can be difficult in horses with acute clinical signs. Signs of radial paralysis occurring after recumbency or general anesthesia are probably caused by ischemic myopathy, with possible ischemic neuropraxia, and these horses generally recover. Prognosis depends on the cause and extent of radial nerve injury, neither of which may be identified. Prognosis is obviously better in horses that are less severely affected and those that show early signs of improvement. However, some severely affected horses completely recover. Prognosis is worse if rapid severe atrophy of extensor muscle occurs. Signs of radial nerve dysfunction can also occur from lesions in the caudal cervical and cranial thoracic ventral gray matter, but other signs of spinal cord disease usually coexist, especially in EPM. Physical therapy, including splinting to avoid flexor deformity, is very important, and electrical stimulation of muscles may also help prevent atrophy. Irreversible fibrosis and contracture are likely without intervention.

Lesions in the nerves supplying the flexor muscles of the thoracic limb are extremely rare, although signs of dysfunction can accompany brachial plexus or spinal cord lesions.[75] If the ulnar nerve is sectioned, the horse may move its foot in a jerking fashion with decreased flexion of the fetlock and carpal joints. When the median nerve is cut, the horse drags the toe because of decreased flexion of the fetlock and carpus. Hypalgesia of the medial pastern occurs, whereas with ulnar neurectomy, hypalgesia of the lateral metacarpal region occurs.[75,77] After neurectomy of the proximal musculocutaneous nerve, the horse drags its toe because of decreased elbow flexion. Since natural disease syndromes affecting these nerves are not described, prognosis is difficult because gait deficits improve with time after neurectomy.[77]

If the femoral nerve is damaged, the horse cannot extend its stifle and rests the leg in a flexed position. The hip is lower than the opposite limb, and the horse cannot support weight normally or at all when walking. When both limbs are affected, the horse will appear crouched and have great difficulty rising. The patellar reflex is absent or depressed, and with time the quadriceps muscles atrophy. Damage to the nerve has occurred during anesthesia with horses positioned in dorsal or lateral recumbency (usually with the affected limb having been positioned uppermost) or after over-extension of the limb, pelvic or femoral fractures, or in association with space-occupying masses impinging on the nerve.[77,79,80] Lesions in the spinal cord ventral gray matter or nerve roots at L5 or L6 lumbar vertebrae can also cause signs of femoral paralysis. Complete neurological evaluation to detect other deficits may be difficult if signs of femoral nerve paralysis are severe. Because the condition is rare and rhabdomyolysis and postoperative myopathy can mimic the signs of femoral nerve damage, giving a prognosis is difficult. Anti-inflammatory drugs are usually used. Most horses with post-anesthetic femoral nerve paresis make a complete recovery.[80]

Signs of paresis or paralysis of the sciatic nerve can occur in horses with pelvic fractures, with deep muscle injections in foals, or with spinal cord lesions affecting the ventral gray matter or nerve roots of the fifth lumbar to third sacral nerves. Signs reflect flexor muscle weakness. The horse can support weight on the limb if the hoof is placed flat on the ground under the pelvis. Otherwise, the horse stands with the hock and stifle extended and the dorsum of the hoof on the ground behind it. When the horse walks, it drags or jerks the limb forward. With time all muscles distal to the stifle and those of the caudal thigh atrophy. The prognosis is very poor if the nerve is severed. If the fibular (peroneal) nerve is damaged (usually because of blunt trauma), the horse cannot extend the fetlock and interphalangeal joints or flex the tarsus normally. At rest it stands with the hoof behind it, resting on its dorsal surface. If the hoof is placed flat on the ground under

the horse, the horse can support weight. When the horse moves, it drags the foot cranially, then jerks it caudally, sliding it on the ground. Skin sensation is decreased over the front and lateral aspects of the tarsus and metatarsal region. With time muscle atrophy in the craniolateral crus can occur. Treatment involves support and protection of the distal limb. Electrical stimulation of muscles might help prevent muscle atrophy. Many horses recover with time. Gait had returned to virtually normal within 3 months of experimental transection of the fibular nerve.[77] Tibial nerve injury is uncommonly diagnosed, but a stringhalt-like gait has been described. When walking, the horse over-flexes the limb and drops the foot straight to the ground when it reaches the end of the cranial phase of the stride. The gastrocnemius muscle reportedly atrophies. The horse stands with the fetlock flexed or partly knuckled, the tarsus flexed, and the hip lower than that of the unaffected leg.[79] Obturator nerve damage, which can occur after foaling, results in signs varying from abduction or circumduction and stiffness of the affected limb when walking to paraplegia. Prognosis depends on severity of signs, whether both limbs are affected, and whether adequate supportive care can be provided.

Stringhalt is easily recognized with its exaggerated flexion of the hock, which can result in a bizarre hopping, jerking, and propulsive gait when both hindlimbs are affected (see Chapter 49). Horses may be so severely affected that they "freeze" in the abnormal position or are very reluctant or unable to move. They may strike the ventral abdomen with the hoof. The gait usually is worse when the horse is walking on a hard surface and when it is anxious or frightened. Horses with mild signs may show the exaggerated hock flexion only when backing, turning, or during the first few strides after walking from a standstill. Atrophy of the distal limb muscles may occur in horses with chronic stringhalt. Spasticity, toe scuffing, and stumbling of the thoracic limbs and left laryngeal hemiplegia have been described in some affected horses.[81,82] A distal axonopathy of peripheral nerves has been described.[83,84] The condition can be sporadic or occur in outbreaks. The cause frequently is unknown, especially when only one horse is affected. Outbreaks have been associated with particular pastures, and mycotoxins are a suspected cause.[81,82] Lathyrism can also be a cause. Phenytoin and baclofen have been used with some success to decrease clinical signs.[85,86] Tenectomy of the lateral digital tendon has also been used. The course is variable and some horses recover spontaneously. However, because it is difficult to predict which horses will recover, the prognosis is guarded, especially in horses with severe clinical signs or in those which are and not associated with a pasture outbreak.

Shivers is somewhat similar to stringhalt, and the origin and pathogenesis are unknown (see Chapter 49). Affected horses tremble one or both pelvic limbs, primarily when backing up or lifting a hoof, and they elevate the tail. Some affected horses cannot stand to have the hooves trimmed, even though the hindlimb gaits are relatively normal and otherwise functional. The clinical course seems variable, and the disease is thought to be progressive, at least in Draft breeds.[77] However, we have seen affected horses remain relatively static and functional, although hoof care can be difficult because of the inability to stand for the farrier. A group of horses exists with mild hindlimb deficits resembling a combination of stringhalt and shivers. Although they may continue to be functional for riding, the gait disability impairs the dressage performance. The cause usually is undiagnosed.

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CHAPTER • 12

Unexplained Lameness

Sue J. Dyson

Lameness diagnosis is a never-ending challenge, even for an experienced clinician, since despite a logical and thorough investigation it still may prove difficult to reach a satisfactory conclusion. This chapter discusses some of the reasons that a definitive diagnosis may remain elusive. In some horses it may be possible to isolate the source of pain reasonably accurately, but it may not be possible to determine the cause of pain. In other horses the source of pain cannot be determined (see Chapter 100).

FALSE-NEGATIVE RESPONSES TO LOCAL ANALGESIC TECHNIQUES

A false-negative response to local analgesic techniques may occur for a variety of reasons, including the following:

- Inaccurate injection
- Inadequate time for the local anesthetic solution to be effective
- Failure to appreciate improvement in the lameness
- Very severe pain
- Failure to alleviate subchondral bone pain after intra-articular injection
- Extra-articular pain
- Aberrant nerve supply
- Failure, in an unshod horse, to appreciate the extent of foot soreness contributing to lameness
- Failure to appreciate the degree of lameness fluctuatation within an examination period

The following are common case examples:

1. It is misleading to conclude that pain does not arise from the centrodistal and tarsometatarsal joints and the central and third tarsal bones after a negative response to intra-articular analgesia of the centrodistal and tarsometatarsal joints. Intra-articular analgesia has only a limited ability to alleviate subchondral bone pain. In moderate to advanced osteoarthritis, subchondral bone pain is a significant contributor to pain. Hock pain may be missed if it is concluded that a negative response to intraarticular analgesia precludes the existence of hock pain and if there is a failure to desensitize the hock region using regional analgesia. It should also be recognized that perineural analgesia of the fibular and tibial nerves may only result in partial improvement in lameness associated with moderate to severe osteoarthritis of the centrodistal and or tarsometatarsal joints.
2. Laminitis, a fracture of either the distal phalanx or the navicular bone, and a subsolar abscess are all common causes of severe foot pain. Desensitization of the foot by perineural analgesia of the palmar (plantar) nerves at the level of the proximal sesamoid bones may have a negligible effect or result in only mild improvement in lameness. Foot pain in the hindlimb is often more difficult to alleviate than in a forelimb. If clinical signs point to foot pain, but apparent desensitisation of the foot fails to markedly alter the lameness, further investigation of the foot should be performed with other means (e.g., radiographic examination).

   The horse in Fig. 12-1 was admitted with suspected back pain, but showed obvious blateral forelimb lameness, with right forelimb lameness predominating. Digital pulse amplitudes were increased in the right forelimb, but there was no response to hoof testers. Nonetheless, the horse appeared clinically to have foot

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CHAPTER • 12

Unexplained Lameness

Sue J. Dyson

Lameness diagnosis is a never-ending challenge, even for an experienced clinician, since despite a logical and thorough investigation it still may prove difficult to reach a satisfactory conclusion. This chapter discusses some of the reasons that a definitive diagnosis may remain elusive. In some horses it may be possible to isolate the source of pain reasonably accurately, but it may not be possible to determine the cause of pain. In other horses the source of pain cannot be determined (see Chapter 100).

FALSE-NEGATIVE RESPONSES TO LOCAL ANALGESIC TECHNIQUES

A false-negative response to local analgesic techniques may occur for a variety of reasons, including the following:

- Inaccurate injection
- Inadequate time for the local anesthetic solution to be effective
- Failure to appreciate improvement in the lameness
- Very severe pain
- Failure to alleviate subchondral bone pain after intra-articular injection
- Extra-articular pain
- Aberrant nerve supply
- Failure, in an unshod horse, to appreciate the extent of foot soreness contributing to lameness
- Failure to appreciate the degree of lameness fluctuatation within an examination period

The following are common case examples:

1. It is misleading to conclude that pain does not arise from the centrodistal and tarsometatarsal joints and the central and third tarsal bones after a negative response to intra-articular analgesia of the centrodistal and tarsometatarsal joints. Intra-articular analgesia has only a limited ability to alleviate subchondral bone pain. In moderate to advanced osteoarthritis, subchondral bone pain is a significant contributor to pain. Hock pain may be missed if it is concluded that a negative response to intraarticular analgesia precludes the existence of hock pain and if there is a failure to desensitize the hock region using regional analgesia. It should also be recognized that perineural analgesia of the fibular and tibial nerves may only result in partial improvement in lameness associated with moderate to severe osteoarthritis of the centrodistal and or tarsometatarsal joints.
2. Laminitis, a fracture of either the distal phalanx or the navicular bone, and a subsolar abscess are all common causes of severe foot pain. Desensitization of the foot by perineural analgesia of the palmar (plantar) nerves at the level of the proximal sesamoid bones may have a negligible effect or result in only mild improvement in lameness. Foot pain in the hindlimb is often more difficult to alleviate than in a forelimb. If clinical signs point to foot pain, but apparent desensitisation of the foot fails to markedly alter the lameness, further investigation of the foot should be performed with other means (e.g., radiographic examination).

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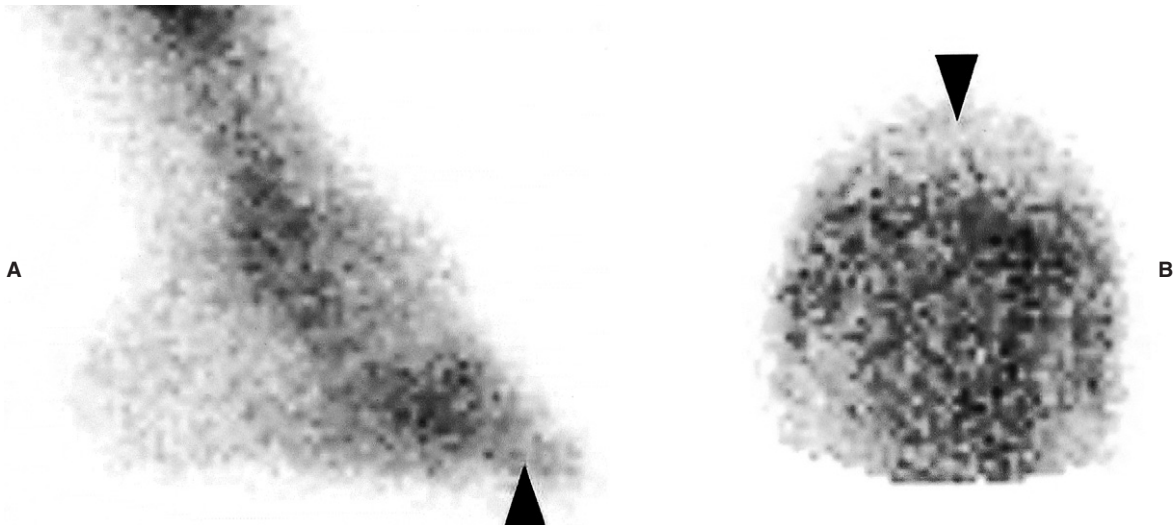

Fig. 12-1 **A,** Lateral and **B,** solar scintigraphic images of the front feet of a horse with suspected back pain but clinical signs of laminitis. There is reduced uptake of the radiopharmaceutical in the toe region *(arrows)*. The horse showed no improvement in lameness after apparent desensitization of the lamer right front foot by palmar (abaxial sesamoid) nerve blocks, despite the firm focal pressure applied with artery forceps around the coronary band. However, the horse responded well to symptomatic treatment for laminitis.

pain typical of laminitis. Apparent desensitization of the foot, performed to convince the owner that the horse had foot pain, produced absolutely no change in the lameness. The horse responded rapidly to treatment for laminitis.

3. Inadvertent intrasynovial injection may result in misleading results. For example, accidental injection into the tarsal sheath when attempting to deposit local anesthetic solution around the plantar metatarsal nerves distal to the hock may result in proximal suspensory lesions being missed.
4. Failure to allow sufficient time for local anesthetic solution to be effective may result in a false-negative response in some circumstances. Premature assessment of the response to intra-articular analgesia of the femorotibial joints may result in a false-negative result in association with a cruciate ligament injury. These ligaments have an extrasynovial location and it may take up to an hour after injection for cruciate ligament pain to be significantly improved.

Failure to allow sufficient time may in some circumstances result in a false-positive response and then confusion. The tibial and fibular nerves are relatively large, and it takes time for the local anesthetic solution to diffuse into them and take effect. This time requirement, combined with the deep location of the deep fibular nerve and thus difficulty in precisely locating the site for injection, may result in a response delayed for up to an hour after injection. Testing the efficacy of these blocks through evaluation of cutaneous sensation is unreliable. If the response is deemed to be negative after 30 minutes, and intra-articular analgesia of the compartments of the stifle is then performed and the lameness improves, it may be wrongly inferred that pain originated in the stifle. However, the improvement in lameness may reflect alleviation of pain arising from the hock region. Much wasted time and money may then be spent trying to establish a cause of stifle pain.

Blocking each compartment of the stifle joint separately (e.g., the medial femorotibial joint), may not result in substantial clinical improvement in the lameness, despite the presence of stifle pain. A considerably better response is frequently seen after blocking the medial and lateral femorotibial joints and the femoropatellar joint in combination.

The importance of the clinical examination and repeated observations of a horse cannot be overemphasised. Each clinician has to learn how much to trust nerve blocks. This depends on experience and the frequency of performing blocks. An inexperienced clinican is far more likely to encounter false-negative responses. The results of nerve blocks must be compared with the clinical signs, and if the interpretation is doubtful, the block should be repeated or the area desensitized with a different technique. The clinician must develop experience in the interpretation of improvement in lameness compared with complete alleviation of pain and lameness. This contrast depends to some extent on the degree of the baseline lameness and whether the forelimbs or hindlimbs are involved.

Failure to Perform the Appropriate Nerve Blocks

Failure to perform nerve blocks in a logical and complete sequence can lead to confusion. If the response to a low 6-point block in a hindlimb is negative and is followed by a positive response to tibial and fibular nerve blocks, the clinician may conclude that pain arose from the hock. Lesions of the proximal aspect of the suspensory ligament (SL) may be completely overlooked.

Blocking the Wrong Limb

Failure to appreciate that a head nod reflects hindlimb lameness and is not always a sign of forelimb lameness may result in a blocking a forelimb with negative results, when the primary source of pain is in the ipsilateral hindlimb.

Sources of Pain that Cannot Be Desensitized by Nerve Blocks

Many regions of the limbs proximal to the carpus and tarsus cannot be satisfactorily desensitized. In young horses, stress fractures are now well-recognized causes of lameness that in many circumstances cannot be blocked out. In young or older horses a fracture of the deltoid tuberosity of the humerus

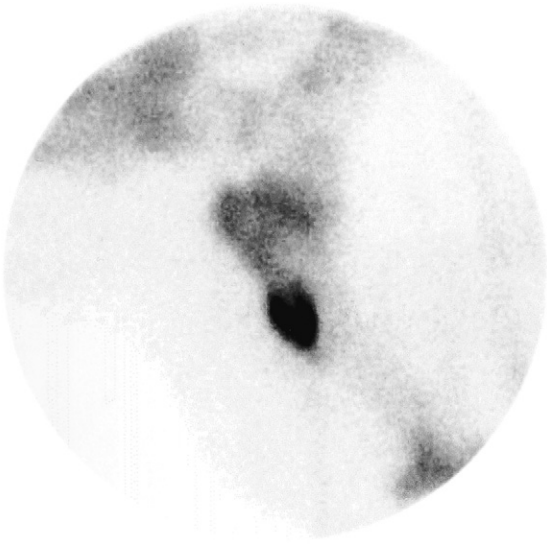

Fig. 12-2 Lateral scintigraphic image of the shoulder region of a 6-year-old Warmblood with acute onset of moderate right forelimb lameness. Note the marked focal increased uptake of the radiopharmaceutical in the region of the deltoid tuberosity of the humerus. There was a slightly displaced fracture of the deltoid tuberosity of the humerus, which healed satisfactorily with conservative management.

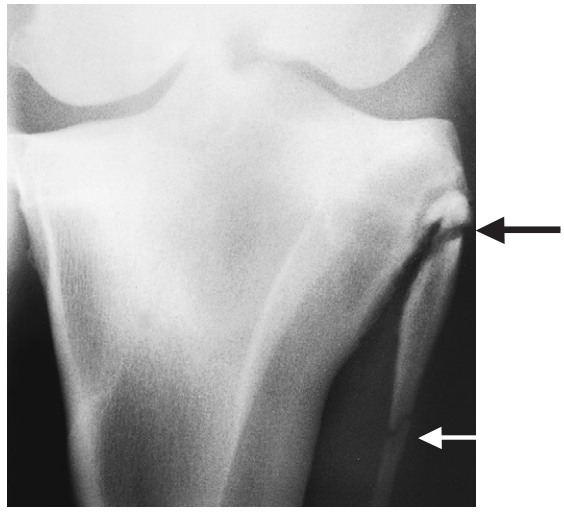

Fig. 12-3 Caudocranial view of the left stifle of a general purpose riding horse with recent-onset, episodic, and transient severe left hindlimb lameness. There is a fracture of the proximal aspect of the fibula *(large arrow)*. The lucent line separating the different centers of ossification further distally *(small arrow)* should not be confused as a fracture. There was little evidence of bony union after 6 weeks, but after 12 weeks the fracture healed satisfactorily.

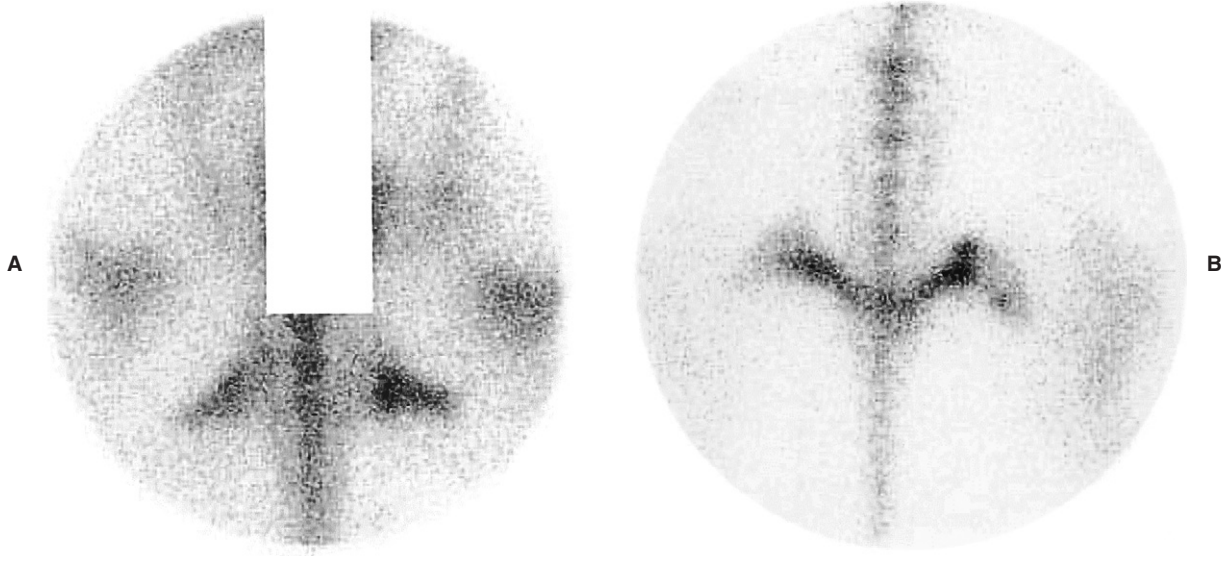

Fig. 12-4 **A,** Dorsal oblique and **B,** caudal scintigraphic image of the tubera ischii of an 8-year-old Grand prix show jumper with loss of hindlimb power and a tendency to jump to the right. There is increased uptake of the radiopharmaceutical in the right tuber ischium and a change of contour compatible with a fracture.

(Fig. 12-2), the proximal aspect of the fibula (Fig. 12-3), the third trochanter of the femur, and the tuber ischium (Fig. 12-4) are all causes of lameness that are unaffected by nerve blocks.

Muscle injuries, such as tearing or fibrosis of brachiocephalicus or the pectoral muscles, may have no localizing signs (see page 141). Associated lameness cannot be influenced by nerve blocks.

POTENTIALLY CONFUSING RESPONSES TO LOCAL ANALGESIC TECHNIQUES

Improvement without complete alleviation of lameness after perineural analgesia is not always easy to interpret. It may reflect failure to completely alleviate pain from a single source, or it may be due to additional sources of pain. Sometimes lameness improves with each successive block

(e.g., palmar digital, palmar [abaxial sesamoid], low 4-point, and subcarpal nerve blocks). However, the lameness is not associated with any detectable radiographic, ultrasonographic, or scintigraphic abnormalities. Sometimes further useful information can be obtained by performing intra-articular analgesia of the interphalangeal and metacarpophalangeal joints, but if the response is negative the diagnosis remains inconclusive.

Isolation of pain to a region but failure to define the cause is particularly frustrating. For example, intra-articular analgesia of the femorotibial joints may be positive, but no radiographic or ultrasonographic abnormalities may be detectable. Nuclear scintigraphy may reveal a generalized increased uptake of the radiopharmaceutical in the distal femur and proximal tibia compared with the contralateral limb. Medication of the joints may result in no improvement. Exploratory arthroscopy may reveal minor findings (e.g., mild fibrillation of the cranial meniscal ligaments) of questionable significance, but evaluation of all the joint surfaces and meniscal cartilages is impossible. The definitive diagnosis for the cause of pain remains elusive.

The importance of subchondral bone pain as a cause of lameness must not be overlooked. Such pain frequently is present without associated radiological change. A comparison of the responses to intra-articular analgesia and perineural analgesia (and the response to intra-articular medication) may be helpful. With subchondral pain, intra-articular analgesia may have a limited effect. Nuclear scintigraphy is a sensitive indicator of increased remodeling in the subchondral bone. Magnetic resonance imaging (MRI) has the potential to show subtle structural changes in the subchondral bone.

Until recently, soft tissue lesions within the hoof capsule have proved elusive to definitive diagnosis. Diagnostic ultrasonography, although possible, has marked limitations. Pool-phase scintigraphic images sometimes are helpful. Examination of the navicular bursa may yield useful information about the bursa, the deep digital flexor tendon, and the distal sesamoidean impar ligament. Advanced imaging techniques such as computed tomography and MRI have the best potential to demonstrate soft tissue pathological conditions, although determining the clinical significance of lesions is not necessarily easy.

False-positive results may be obtained if the horse is only mildly lame at investigation but has a history of a more obvious lameness. The detectable mild lameness may not necessarily reflect the original cause. Lameness that is induced when a horse is lunged in small circles on a concrete surface may not reflect the primary cause of lameness. Thus eliminating this lameness by nerve blocks may be misleading. Lameness induced by flexion also may not reflect the principal cause of lameness. Blocking the flexion response does not necessarily identify the primary cause of lameness.

A pony had moderate forelimb lameness that was markedly accentuated by lower limb flexion. The response to flexion was eliminated by either regional or intra-articular analgesia of the fetlock joint. However, the baseline lameness was unchanged and did not respond to any of the nerve blocks that were repeated on several occasions. Surgical removal of a large osseous fragment from the fetlock joint did not improve the lameness.

Multiple Sources of Pain in a Limb and More than One Lame Limb

Problems can arise in interpretation of nerve blocks in a horse that is lame in more than one limb, especially if there is more than one source of pain in a limb. Perineural nerve blocks usually last for up to 2 to 3 hours unless a long-acting local anesthetic agent, such as bupivacaine, is used. If a horse is lame in several limbs it is usually easiest to start with the lamest limb and block it first. Interpretation becomes difficult if there is a failure to desensitize all the lame limbs simultaneously. If the blocks in one limb are wearing off, then lameness in the least lame limb becomes less apparent. The horse's tolerance for nerve blocks may also compromise how much can be done. It may be necessary to start again on another occasion using bupivacaine. If simultaneous lameness of the ipsilateral forelimb and hindlimb is suspected, blocking should begin in the hindlimb. In this situation a substantial amount of the head nod probably originates from the hindlimb component. Because elimination of head nod is vital to improvement after blocking, forelimb diagnostic procedures cannot be fairly evaluated.

Very Low-Grade Lameness

Nerve blocks, especially in hindlimbs, often result in improvement rather than complete alleviation in lameness. Assessing improvement in subtle lameness is nearly impossible. If the horse has a history of more severe lameness previously, delaying further investigation often is worthwhile. The horse should be worked to accentuate the lameness and simplify interpretation of the response to local analgesic techniques.

Improvement of Lameness in Some Situations, but Unrelieved Lameness under All Situations: Which Is the Baseline Lameness?

Sometimes a horse has lameness that appears different in nature under different circumstances. Such findings may be related to more than one cause of lameness, and it is important to recognize this fact. For example, a dressage horse was admitted with left forelimb lameness that was only apparent to the rider when the horse was ridden on the right rein (to the right). Clinical examination revealed left forelimb lameness on the right rein on the lunge on a hard surface. This was alleviated by desensitization of the foot. However, desensitization did not alter the lameness that was apparent when the horse was ridden. The cause of the lameness could not be identified. It is vitally important to relate the results of the investigation to the history.

CHALLENGES TO LAMENESS DIAGNOSIS

Very Intermittent or Sporadic Lameness

Sometimes lameness is intermittent, and the horse may be perfectly normal between episodes. Lameness may be provoked only by maximal exercise in competition. It is very important to carefully assess the history and get the owner to pay great attention to any clinical features of the lameness when present. For example, mild, transient diffuse swelling in the midmetacarpal region medially may reflect axial impingement of a splint on the SL (Fig. 12-5) (see Chapter 73). Spontaneous resolution of hindlimb lameness after standing still is suggestive of aorto-iliaco-femoral thrombosis (see Chapter 52). Ask the owner to assess the reaction to manipulation of specific joints when the horse is lame. Pain on manipulation may suggest a joint problem such as hemarthrosis.

If it is not possible to examine the horse when it is lame, nuclear scintigraphic examination can be helpful but also has the potential to mislead (see page 141). For example, an Arab endurance horse had episodic right forelimb lameness that was present only immediately after rides longer than 30 miles. Comprehensive clinical evaluation revealed no evidence of lameness and no suggestions of the cause of previous lameness. Scintigraphic examination revealed increased uptake of the radiopharmaceutical in the third carpal bone of the lame limb (Fig. 12-6). Radiographic examination revealed marked sclerosis of the third carpal bone in the lame limb only, an unusual finding in an endurance horse and thought likely to be of clinical significance. Another Arab endurance horse had an acute-onset, severe right hindlimb lameness that resolved within

48 hours, but mild, extremely transient, and episodic lameness persisted over the next 3 weeks. Clinical evaluation revealed no suggestion of the cause and no current lameness. Nuclear scintigraphic examination revealed focal increased uptake of the radiopharmaceutical in the medial wing of the distal phalanx, and radiographic examination confirmed a recent fracture (Fig. 12-7).

If nuclear scintigraphic findings are negative, it is necessary to try to recreate the circumstances under which the horse exhibits lameness. For example, hemarthrosis can cause a very severe but extremely transient lameness. The horse may be completely normal between episodes. Diagnosis can be reached only by arthrocentesis at the time of acute episode, when there is usually some degree of joint capsule distention and pain on manipulation of the affected joint. Working the horse on a treadmill sometimes is helpful (see Chapter 101).

Lameness that Varies within and between Examinations

Sometimes lameness varies considerably in degree both within an examination period and between examinations. This variation makes interpretation of the response to nerve blocks potentially difficult, unless the veterinarian is aware of the fluctuation. It is important to watch a horse move for a sufficient length of time to appreciate any spontaneous changes in the degree of lameness. Horses with a subchondral bone cyst in either the distal scapula or the medial femoral condyle may behave in this way. Within a single examination period the horse may appear sound or lame. In such circumstances it is vital to compare the response to nerve blocks with the clinical signs exhibited. For example, if the characteristics of the lameness are suggestive of shoulder pain, but the lameness is apparently improved after desensitization of the foot, consider the possibility of spontaneous improvement in the lameness that is unrelated to the nerve block. This is, however, an unusual clinical situation, and generally it is best to rely on the results of the diagnostic blocks. A combination of nerve blocks, scintigraphic examination, and radiographic examination may enable a conclusive diagnosis to be reached.

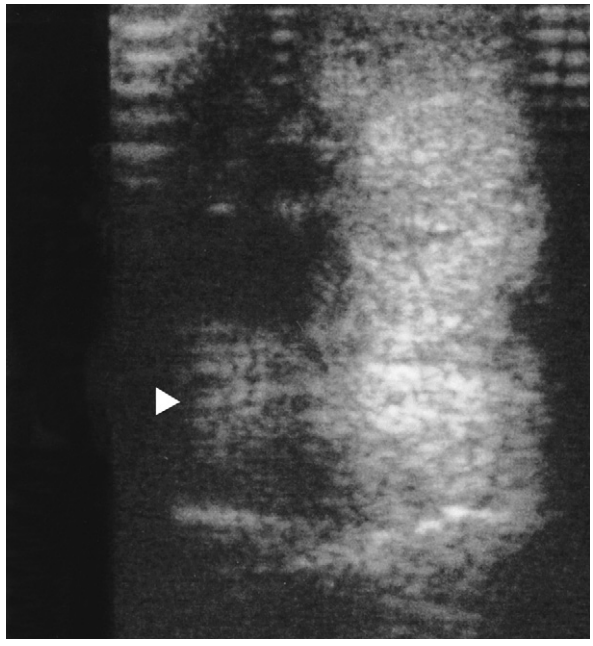

Fig. 12-5 Transverse ultrasonographic image of the palmar metacarpal soft tissues of the right forelimb of an endurance horse at 10 cm distal to the accessory carpal bone. Medial is to the left. The horse had low-grade lameness at the end of endurance rides that resolved completely within 24 hours. Note the echogenic tissue *(arrowhead)* next to the suspensory ligament (SL). This was a granulomatous reaction between an exostosis on the second metacarpal bone (McII) and the SL. The distal half of the McII was excised and the granulomatous tissue removed. The horse made a complete recovery.

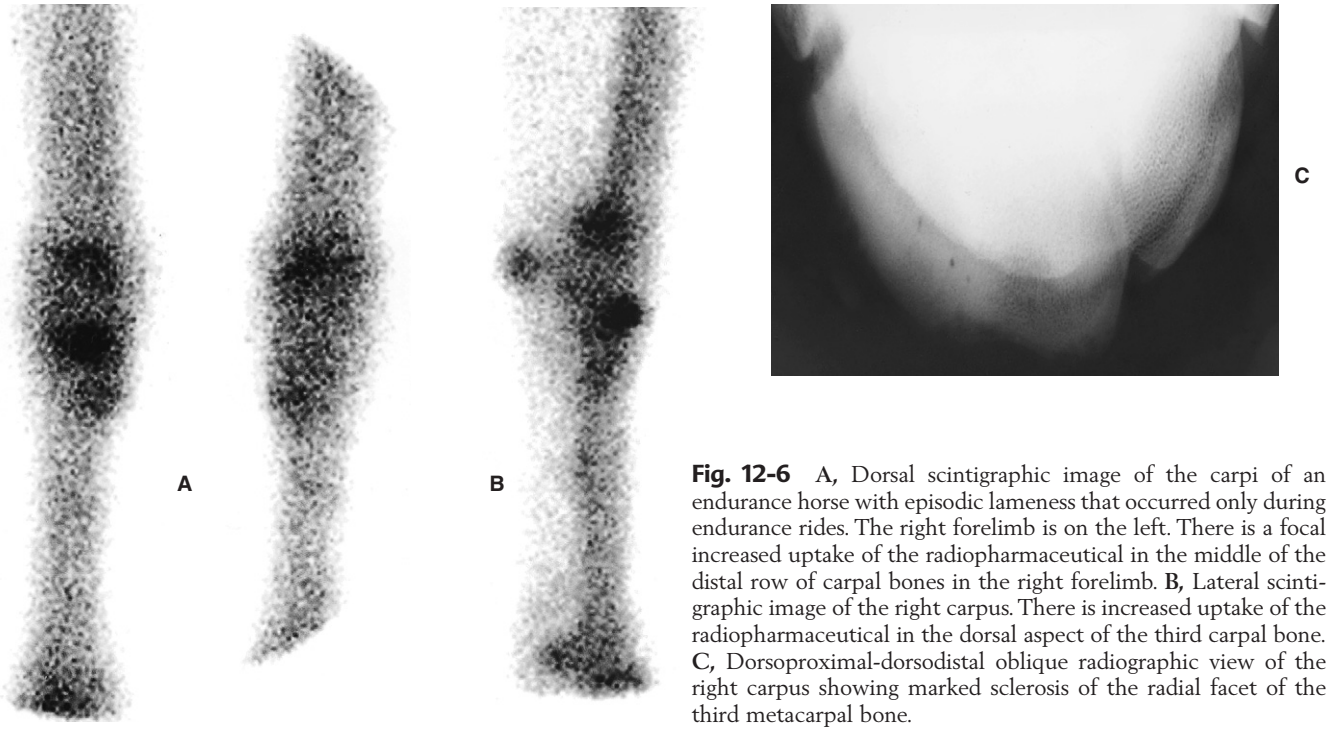

Fig. 12-6 **A,** Dorsal scintigraphic image of the carpi of an endurance horse with episodic lameness that occurred only during endurance rides. The right forelimb is on the left. There is a focal increased uptake of the radiopharmaceutical in the middle of the distal row of carpal bones in the right forelimb. **B,** Lateral scintigraphic image of the right carpus. There is increased uptake of the radiopharmaceutical in the dorsal aspect of the third carpal bone. **C,** Dorsoproximal-dorsodistal oblique radiographic view of the right carpus showing marked sclerosis of the radial facet of the third metacarpal bone.

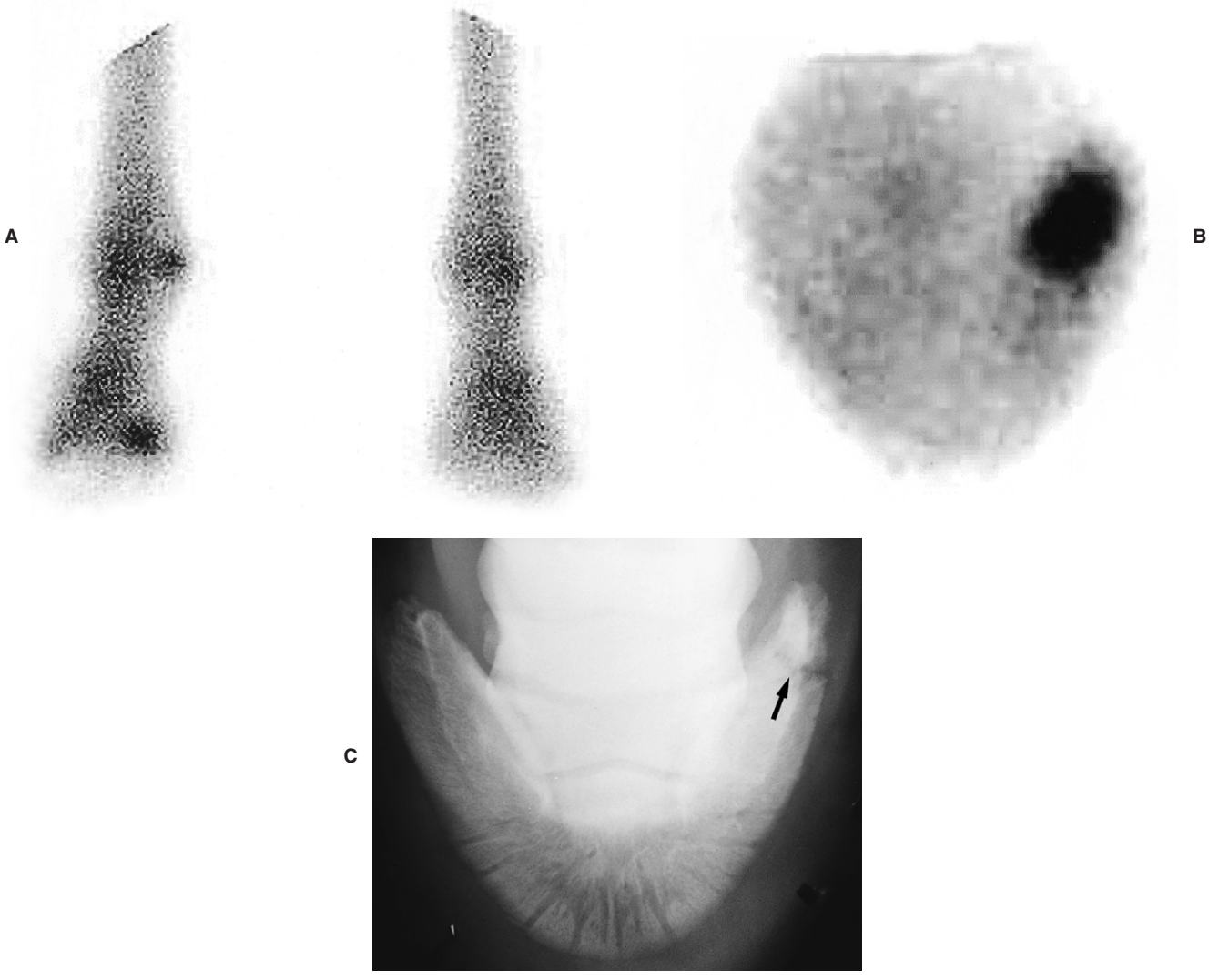

Fig. 12-7 **A,** Plantar scintigraphic image of the hind feet of an endurance horse with recent onset of left hindlimb lameness that was only apparent after an endurance ride. The horse appeared clinically normal at the time of the examination. There is moderate focal increased uptake of the radiopharmaceutical in the medial aspect of the left hind foot *(left)*. **B,** Solar scintigraphic image of the left hind foot. Medial is to the right. There is marked increased uptake of the radiopharmaceutical in the medial plantar process of the distal phalanx. **C,** Plantarodorsal radiographic view of the left hind foot. There is a fracture of the medial plantar process *(arrow)*. The horse was treated conservatively and made a complete recovery.

The Dangerous Horse and Nerve Blocks

Some horses do not tolerate needle placement and cannot be restrained safely. Nuclear scintigraphic examination sometimes indicates a diagnosis, but the results may be negative. Under such circumstances the horse can be sedated for each block. This approach obviously is time consuming and may be of low specificity, because time must be allowed for the sedative to wear off adequately before the response to the block can be assessed. During this time the local anesthetic solution has the potential to diffuse away from the site of injection and influence more remote pain. Sedation may result in a rather sloppy gait, which can hinder interpretation, especially with mild hindlimb lameness characterized only by a toe drag. Therefore patient selection is important. However, bearing in mind these limitations, it may be the only way to proceed. Xylazine is the shortest-acting α_2-agonist available and is the drug of choice, but with a difficult horse, combination with an opioid such as butorphanol may be necessary.

NEGATIVE RESPONSES

Negative Response to All Nerve Blocks: Where Next?

Occasionally a horse is evaluated for forelimb or hindlimb lameness that is not influenced by any local analgesic technique. Clinical signs may be suggestive of a source of pain (e.g., the foot). The reason for nerve blocks that result in apparent desensitization of a region but fail to eliminate or improve pain is not understood, but it does occur occasionally.[1] Alternatively, there may be no clinical clues for the source of pain. Nuclear scintigraphic examination may be helpful in either situation if the pain is bony in origin but is likely to be less helpful for soft tissue injuries.

Negative Responses to Nerve Blocks, No Clinical Clues, and Negative Scintigraphic Findings

Consideration may be given to systematic radiographic examination, bearing in mind that not all bony lesions are sufficiently active to yield positive scintigraphic findings. However, com-

prehensive radiographic examination is time consuming, expensive, and frequently unrewarding, and potentially results in unnecessary exposure to radiation. Thus it is usually discouraged. In the forelimb, unusual causes of lameness such as neurological disease (usually lower motor neuron diseases, such as equine protozoal myelitis), cervical nerve root pain (radiculitis), spondylosis of the cranial thoracic vertebrae, and pectoral, sternal, or rib pain may be considered. In the hindlimb, neurological disease and thoracolumbar or pelvic soft tissue injuries visible neither on pool nor bone phase scintigraphic images should be considered. Keep in mind that horses with forelimb or hindlimb lameness could potentially have a distant source of pain and comprehensive imaging (whole body bone scan, for instance) may be necessary.

NECK LESIONS AND FORELIMB LAMENESS

Forelimb lameness that is unassociated with primary limb pain has been recognized in association with bony lesions of the mid-cervical and caudal cervical vertebrae and the cranial thoracic vertebrae (see Chapter 55).[2] There are not necessarily any detectable clinical signs that can be related to the neck. In horses with confusing forelimb lameness, evaluation of the neck with radiography, scintigraphy, or both modalities is certainly indicated.

REFERRED PAIN

The concept of referred pain is well recognized in people but is more difficult in the horse. We must accept that referred pain originating from a lesion far removed from the lame limb may contribute to pain and thus cause lameness.

PREVIOUSLY UNRECOGNIZED CAUSES OF LAMENESS PROXIMAL TO THE CARPUS AND TARSUS

I think it is naive to consider that all potential causes of lameness proximal to the tarsus and carpus have been recognized. Injuries that primarily involve bone usually can be identified with nuclear scintigraphic examination. However, scintigraphy is rather insensitive in the identification of soft tissue injuries, although it may help identify some muscle injuries (Fig. 12-8). Muscles can be examined ultrasonographically, but we need to know where to look for damage. Acupuncture trigger point sensitivity may provide information. Use of muscle stimulators may help to identify superficial muscles that are damaged. We know that the horse can tear fibularis tertius, resulting in pathognomonic clinical signs. We do not know if minor injuries to this modified muscle could cause lameness. Tendonous and ligamentous pain without palpable abnormalities and therefore without a specific indication for ultrasonographic examination must always be considered. We must remain open-minded and search for other means of diagnosis.

MISINTERPRETED IMAGING FINDINGS THAT RESULT IN MISDIAGNOSIS

In horses in which the results of local analgesic techniques are equivocal or negative, it may be tempting to rely on the results of other diagnostic techniques, such as radiography, ultrasonography, and nuclear scintigraphy, without necessarily relating them to the initial clinical signs. Although these imaging modalities may help to confirm a clinical diagnosis, they also have the potential to mislead, especially if interpreted in isolation.

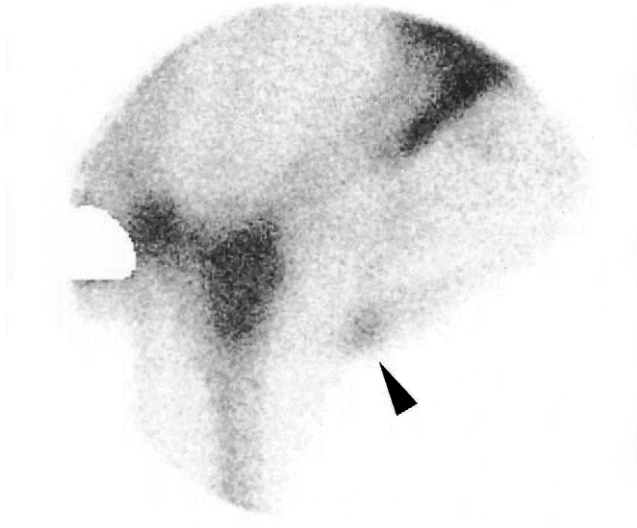

Fig. 12-8 Lateral scintigraphic image of the right elbow region of a 4-year-old Warmblood stallion with right forelimb lameness evident only at the walk, which was unaltered by any local analgesic technique. Uptake of the radiopharmaceutical was increased in the biceps brachii muscle *(arrow)*, which corresponded to an area of increased echogenicity compatible with fibrosis.

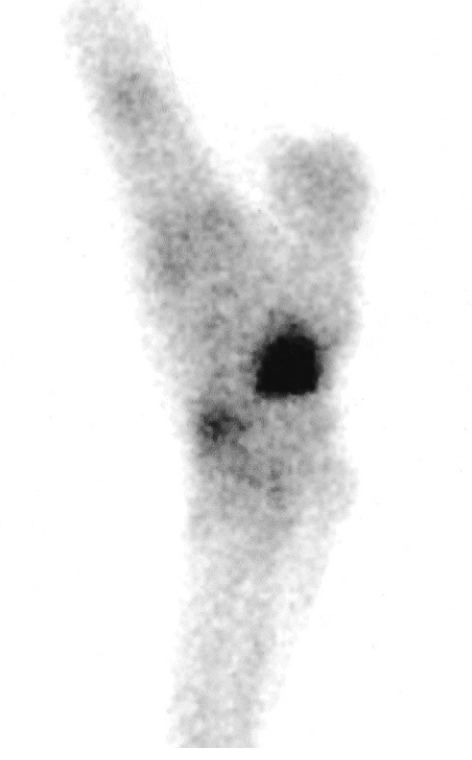

Fig. 12-9 Lateral scintigraphic image of the tarsus of a 7-year-old riding horse. Note the marked focal increased uptake of the radiopharmaceutical in the hock. Lameness was completely alleviated by desensitization of the fetlock region. There was no radiographic abnormality of the hock.

The horse illustrated in Fig. 12-9, with focal increased uptake of the radiopharmaceutical in the hock, was admitted with left hindlimb lameness that was alleviated by desensitization of the fetlock region. The horse illustrated in Fig. 12-10, which has increased uptake of the radiopharmaceutical in the distal tibia, was admitted with forelimb lameness. No hindlimb lameness

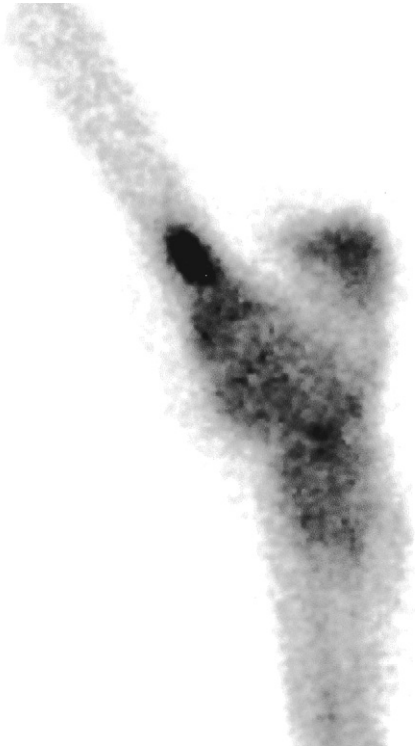

Fig. 12-10 Lateral radiographic view of the tarsus and tibia of a 12-year-old advanced event horse with bilateral forelimb lameness associated with distal interphalangeal joint synovitis. The horse had no history or evidence of hindlimb lameness and competed successfully thereafter. Note the intense increased uptake of the radiopharmaceutical in the tibia. The horse was reexamined approximately 8 months later with similar results. The horse had been competing successfully but had recurrent forelimb lameness.

was observed at any time, even after alleviation of the forelimb lameness. The horse in Fig. 12-11, with increased uptake of radiopharmaceutical in the region of insertion of the deep digital flexor tendon, had lameness associated with a fracture of the deltoid tuberosity of the humerus.

ODD LAMENESS APPARENT ONLY DURING RIDING

Some causes of lameness are apparent only when the horse is ridden. Some of these are easy to block, but a minority fail to respond to local analgesic techniques. Consideration must always be given to rider-induced lameness (see page 144 and Chapter 100), discomfort caused by tack, and gait abnormalities arising through thoracolumbar (see Chapter 54), sacral, and sacroiliac pain (see Chapters 52 and 53). The possibility of the rider's weight compressing muscles in the saddle and caudal neck region and nerve compression should be considered as potential causes of forelimb pain.

IDENTIFIABLE LESIONS: WHICH CONTRIBUTE TO THE CURRENT LAMENESS?

The presence of radiographic lesions is not necessarily synonymous with pain, or the pain may be low grade and not compromise the horse's gait sufficiently to be recognized by the rider. For example, a horse may have an acute lameness referable to the stifle. Several lesions may be identified radiographically (e.g., smooth flattening of the middle of the lateral trochlear ridge of the femur, modeling of the medial

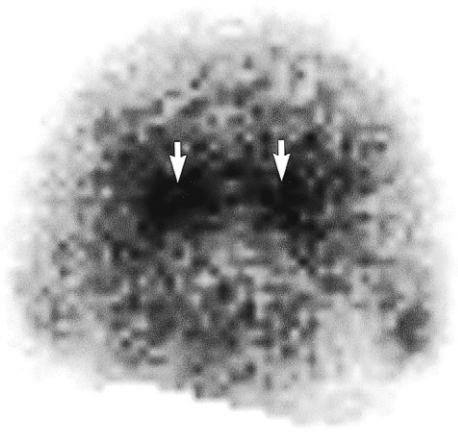

Fig. 12-11 Solar view of the right front foot of a 6-year-old Warmblood. There is increased uptake of the radiopharmaceutical in the region of insertion of the deep digital flexor tendon on the distal phalanx. The gait characteristics were typical of proximal limb lameness and lameness was unaltered by desensitization of the foot. The horse had a fracture of the deltoid tuberosity of the humerus.

articular margin of the tibial plateau, and a complete fracture of the proximal aspect of the fibula [Fig. 12-3]). It is likely that acute lameness is related to the fibular fracture. The horse in Fig. 12-3 had been coping despite radiographic evidence of both ostechondrosis of the femoropatellar joint and osteoarthritis of the medial femorotibial joint.

OTHER CAUSES OF LAMENESS

Lacerations and Occult Spiral Fractures

Acute-onset, moderate to severe lameness sometimes develops within 2 to 3 weeks of trauma to, or laceration over, a long bone (see Figs. 3-3 and 3-4). Sometimes lameness is first observed when the horse is still restricted to box rest and controlled exercise while the wound heals. There may or may not be any detectable focus of pain. Consideration must always be given to the possibility of an occult spiral fracture of, for instance, the radius, which was obviously sustained at the time of the initial injury. Occult spiral fractures are most common in the radius but can involve the third metacarpal or metatarsal bones and tibia. Radiographic examination should be performed to eliminate this possibility.

Rib Lesions

A fracture of one or more cranial ribs is a rather unusual cause of forelimb lameness that usually is a sequel to direct trauma, such as a collision with another horse or a gate post, or a fall. There are usually no localizing clinical signs, although secondary neurogenic muscle atrophy may develop within the next 10 to 14 days. The lameness may suggest an upper limb problem. Diagnosis is dependent on radiographic or scintigraphic identification of the fracture.

Sternal Injury

Sternal injury usually causes a change in behavior, such as a tendency to buck when first tacked up and mounted (i.e., extreme cold back behavior) rather than lameness. Frequently it is not possible to elicit pain by deep palpation.

Fracture of the Summits of the Dorsal Spinous Processes in the Withers Region

Acute fractures of the withers usually result in a very shortened forelimb stride and a tendency to move very closely in front ("the mini-skirt walk"). There is usually obvious palpa-

ble deformity of the withers region. Diagnosis is confirmed radiographically.

Temporomandibular Joint Pain

Pain associated with one or both temporomandibular joints may cause reluctance for the horse to take the bit properly, crookedness in the head and neck carriage, and secondary gait irregularities. The joints can be assessed by applying firm pressure over each joint, which may cause pain, and by opening the mouth and moving the upper and lower jaws relative to one another and assessing mobility. Thermography may be a sensitive indicator of local inflammation. If temporomandibular pain is suspected, further investigation can be performed using nuclear scintigraphic examination and diagnostic ultrasonography.[3] Radiography is relatively insensitive unless major bony changes are present.[4]

Neurological Problems and Lameness or Stiffness

Early compressive lesions of the cervical spinal cord can cause an apparent low-grade hindlimb lameness that is characterized by slight toe drag and asymmetrical movement of the hindquarters. Signs of weakness or ataxia may not be evident unless a comprehensive neurological examination is performed, especially if the horse is quite fit and fresh. Neurological signs may only be seen when the horse is tired and not compensating for its gait deficits.

Equine protozoal myelitis can cause rather bizarre forelimb or hindlimb gait abnormalities, either unilaterally or bilaterally. It should be considered in the differential diagnosis of odd gait abnormalities in horses that reside or have spent time in America.

Damage to the branch of the radial nerve, which results in an innervation of the extensor muscles of the carpus and digits, may cause a subtle gait abnormality that is characterized by a tendency to stumble and associated with slight knuckling of the carpus and distal limb joints.

Stiff horse syndrome[5-7] and equine motor neuron disease[8] are unusual neurological conditions in which the horse moves better in the trot and canter than at a walk and may stand abnormally.

Shivering behavior often is seen in one or both hindlimbs in association with hindlimb lameness. The two conditions generally are unrelated. Shivering behavior may be more apparent when the horse is stressed. The behavior can make it difficult to assess whether any manipulation of the limb causes pain, or whether the horse is uncomfortable with one hindlimb picked up.

Stringhalt, or exaggerated flexion of a limb, usually a hindlimb, results in a gait abnormality most obvious at the slower speeds of walk and trot. It is not usually directly associated with any other form of lameness. It may be sudden or insidious in onset.

Congenital abnormalities of the first ribs in association with abnormalities of the adjacent brachial plexus have been seen as a cause of persistent forelimb lameness (Fig. 12-12).

Acute-onset, severe, and persistent forelimb lameness has been seen in association with a mediastinal abscess that encroached on the nerve roots of the seventh cervical and first thoracic vertebrae, the stellate ganglion, and the first rib (Fig. 12-13). Measurement of fibrinogen may help to identify the presence of an infective or inflammatory process.

Lyme Disease

Lyme disease has frequently been incriminated as a cause of shifting lameness that involves several limbs, but confirmed cases are extremely rare.[9] Many horses that are in areas where there are many ticks have relatively high antibody titers to *Borrelia burgdorferi*,[9,10] but this is not consistent with clinical disease. Lyme disease may be suspected in adult horses in endemic areas when unusual synovitis develops in absence of any known injection history or presence of a wound (see Chapter 67).

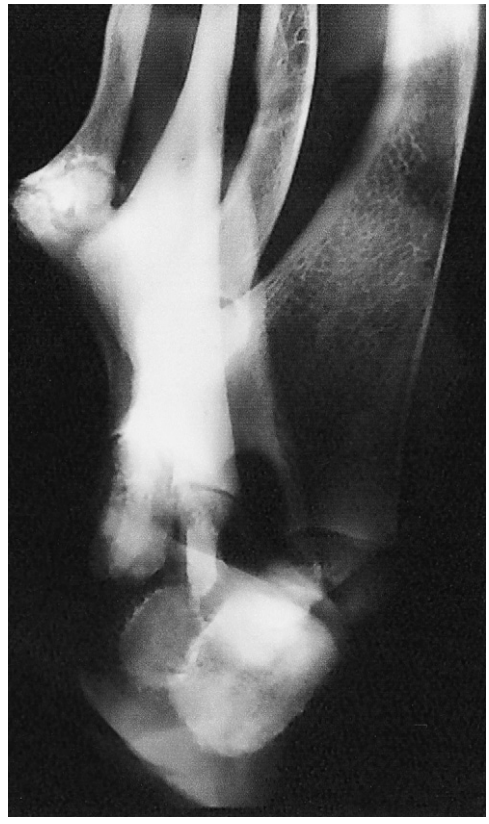

Fig. 12-12 Post-mortem specimen showing an anomalous first rib from a 3-year-old Thoroughbred with right forelimb lameness that was not altered by any local analgesic technique. There was an abnormal web of fibrous tissue that extended from this anomalous rib to incorporate part of the brachial plexus on the right side.

Fig. 12-13 A 7-year-old pony with severe right forelimb lameness that progressively deteriorated. The pony had an increased fibrinogen level and intermittent pyrexia. There was a mediastinal abscess that encroached on the roots of the seventh cervical and first thoracic nerves, the stellate ganglion, and the first rib.

Immune-Mediated Polysynovitis

Immune-mediated polysynovitis is relatively uncommon but may result in generalized stiffness associated with transient synovial distention of several joint capsules. It is frequently not

possible to identify the underlying cause, but this condition usually responds to corticosteroid medication (see chapter 67).

Tack-Induced Pain

An ill-fitting saddle can induce back pain and restricted action or poor performance. The bit can induce pain through poor fit (too narrow or too wide), being too low in the horse's mouth, banging on the canine teeth, pinching the corners of the horse's mouth, or being too severe. Any oral pain related to the bit, sharp teeth, or lacerations of the tongue, cheeks, or corners of the horse's mouth may cause reluctance to accept the bit properly and gait irregularities.

Rider-Induced Problems

The rider has a potentially huge influence on the gait of the horse. If a horse is not going forward properly, either because the rider is restricting it or because the rider is not asking it to go forward properly, the forelimb and hindlimb gaits may appear irregular, mimicking pain-induced lameness. An overweight rider who is too heavy for the horse may induce hindlimb lameness. A rider who constantly sits crookedly may induce back pain and hindlimb lameness. (Rider-related problems are discussed further in Chapter 100.)

Physical Limitations of the Horse, Temperament, and Confidence

With appropriate handling and training, most horses are cooperative. However, a previously compliant horse can very rapidly change if regularly handled and ridden by someone who lacks confidence, technique, or strength. Such a horse can quickly develop evasions, such as not going forward properly, rearing, bucking, or taking off. These problems may be pain related, but not necessarily. A horse that has never been trained properly may be very difficult and even potentially dangerous to the rider. Horses homebred by amateur enthusiasts are high-risk candidates. Some horses are innately lazy and unwilling to go forward. Others are very exuberant and excessively forward going and "fizzy." The veterinarian may be asked to investigate any of these behavioral features as a potentially pain-related problem.

It is important to establish how the horse was previously. It may be useful to see a video. Determine if there has been a change in rider or management. Establish what the horse is being fed and how much work it is getting: there is a tendency for people to overfeed and underwork horses. A comprehensive clinical evaluation may need to be repeated on several occasions before you can determine if it is a pain-related problem. Nuclear scintigraphic examination of selected areas may be useful to eliminate the presence of any underlying problems and to convince the owner that there is not a physical problem. A change of rider or work pattern may be necessary. The use of high doses of non-steroidal, anti-inflammatory drugs (e.g., 2 to 3 g twice daily for at least 7 to 10 days) can be helpful to determine if the problem is pain related, but be aware that there may be a placebo effect. Not all pain responds to phenylbutazone, so a negative response does not preclude a pain-induced problem (see Chapter 100.)

Reproductive Problems

Some mares become more difficult to handle and ride when in season and the judicious use of a synthetic progestagen such as altrenogest (Regumate, Intervet, Millsboro, DE) to stop the mare's cycling can sometimes be helpful. While some stallions can be used successfully for both competition and breeding, especially if the management is good and a very clear distinction is made with handling routines, others cannot focus adequately and thus perform athletically below expectations. Performance may be enhanced by castration. However, although malorientation of testicles is frequently blamed for lameness or poor performance, in the Editors' experience this is highly unusual.

A schirrhous cord in a gelding or mastitis in a mare can cause loss of hindlimb action and hindlimb stiffness. Chronic episodic, transient hindlimb lameness associated with strenuous work (jumping) has been seen in a stallion with large internal inguinal rings.[11] The lameness resolved after herniorrhaphy.

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CHAPTER • 13

Assessment of Acute-Onset, Severe Lameness

Sue J. Dyson

FIELD DIAGNOSIS OF THE INJURED HORSE

The assessment of an acutely lame horse presents a challenge in diagnosis and in dealing with the people associated with the animal, particularly if lameness occurs at a competition. The horse may have fallen and been lame immediately or may have pulled up lame, and the veterinary surgeon may be called to examine the horse on course in full view of the public.

Ideally the horse should be transported to an examination area for comprehensive evaluation, but the veterinary surgeon must establish whether the injured limb requires support, before moving the horse. Although the horse may be very lame, establishing a definitive diagnosis for the cause of the lameness at this stage may be difficult.

This may surprise riders, trainers, and owners, and maintaining their confidence in what is an emotionally charged situation can be quite difficult. If a fracture is suspected, pressure to destroy the horse humanely without delay may be felt. Although some fractures are catastrophic and horses merit immediate destruction on humane grounds (e.g., a spiral fracture of the humerus), other serious fractures can be repaired. Therefore as much information as possible about the site of the fracture and its configuration should be obtained before a decision is made. The limb should be supported appropriately before the horse is moved for radiographic examination. If a horse must be destroyed on humane grounds at a competition, this should be done off the course.

Although a diagnosis may be obvious in some horses immediately after the onset of lameness, the veterinarian must recognize that severe lameness may occur without an evident cause. Serial re-examinations over the following hours or days may be required before a diagnosis can be reached. Sometimes the lameness resolves spontaneously within 12 to 18 hours and its cause is never established.

The clinician must be aware of the most common causes of acute-onset, severe lameness, which include the following:

- Sub-solar abscess
- Fracture
- Laminitis
- Intra-synovial sepsis
- Periarticular cellulitis

When lameness occurs during training or competition, a spectrum of other injuries must be considered. However, a history of acute-onset, severe lameness during exercise must not mislead the clinician into thinking that lameness must be caused by internal or external trauma associated with exercise. Lameness may still be caused by pain from a sub-solar abscess.

This chapter describes a systematic approach to management of a horse with sudden-onset, severe lameness and focuses particularly on injuries that occur during work.

ASSESSMENT

Medical History

While performing an initial visual appraisal of the horse, establishing a history is useful. The examiner must determine the following:

- Any previous lameness, tendon or ligament injury, or history of tying up
- Date of last shoeing
- Circumstances of lameness: whether the horse was performing normally, fell, hit a fixed fence, or collided with a fixed object such as a guide rail or a tree. The horse may have reared in the starting stalls or reared and fallen over backward. Another horse may have kicked it.

The clinician should also be aware of common injuries in the discipline in which the horse is competing.

The horse may be distressed because of the severity of pain and excited because of the atmosphere of a competition and thus difficult to restrain and examine adequately. Sedation with romifidine or detomidine, with or without butorphanol, may be necessary to facilitate examination of the horse.

The horse's posture should be observed while it stands still and walks a few steps. If the horse bears weight only on the toe, it may be inapparent that a horse has lost some support of the fetlock because of rupture of the superficial digital flexor tendon (SDFT) in the metacarpal region or at the musculotendonous junction in the antebrachium, unless it walks a few steps.

Limb Examination

The veterinarian should establish whether the horse is able and willing to bear weight on the limb, bearing in mind that after a fall, a neurological component may contribute to the lameness, in addition to the pain. The horse's demeanor should be assessed; the degree of pain and distress usually but not invariably reflects the severity of the injury. The horse may be greatly distressed, shifting weight constantly between limbs, and may be reluctant to move. Reluctance to move may be due to a bilateral problem (e.g., bilateral severe superficial digital flexor [SDF] tendonitis) or a more generalized problem such as equine rhabdomyolysis (tying up).

The horse should be carefully appraised visually to identify areas of swelling or a laceration. If the horse's limbs are covered in mud or grease (commonly applied to the limbs during the speed and endurance phase of a Three-Day Event), this should be washed off before proceeding with the evaluation. Boots, bandages, and the saddle and martingale should also be removed. Temporary studs in the shoes should be removed, because they may be more difficult to remove later if the injury is severe.

The horse may be obviously lame on a hindlimb or forelimb, but this may mask a similar, less severe injury in a contralateral limb or a different injury; therefore all limbs should be assessed carefully. For example, a racehorse may develop a lateral condylar fracture of the third metacarpal bone in one limb and SDF tendonitis in the contralateral limb. Although the former injury results in a more severe lameness, the latter may be more important to the horse's long-term prognosis.

Occasionally, forelimb and hindlimb lameness are concurrent. Each limb should be palpated systemically with the horse bearing weight and not bearing weight. The examiner should pay careful attention to heat, swelling, abnormal muscle texture, pain on firm pressure, pain induced by manipulation of a joint, restriction of flexibility of a joint, an abnormal range

of motion of the joint, audible or palpable crepitus, and the intensity of the digital pulse amplitudes.

The position of the shoe should be assessed carefully. A shoe that has moved slightly may result in nail bind. Hoof testers should be systemically applied across the wall and sole, gently at first and then firmly. Percussion should also be applied to the sole of the foot with the limb picked up and to the wall with the limb bearing weight. The clinician should not forget that if the sole is very hard, eliciting pain with hoof testers may not be possible, despite the presence of a sub-solar abscess.

The limbs should be carefully assessed for lacerations. Serious damage may occur to underlying structures if the laceration was sustained while the horse was moving at speed, and the position of the laceration and the site of damage to underlying structures may not coincide.

Shoulder and Chest

Injuries to the shoulder region usually result from a fall or collision, which may result in severe bruising only or a fracture. A fracture of the supraglenoid tubercle of the scapula results in severe lameness (Fig. 13-1). Slight soft tissue swelling may develop, usually without audible or palpable crepitus, and pain on palpation may be difficult to differentiate from that caused by severe bruising alone. Articular fractures of the scapula may be associated with audible crepitus on manipulation of the limb. Fractures of the body of the scapula or the humerus are usually associated with severe lameness, soft tissue swelling, and pain in that area.

After collision with a fixed object, or occasionally a fall, the scapulohumeral joint may become luxated or subluxated, with or without a fracture of the glenoid cavity of the scapula. The horse bears weight on the limb reluctantly, soft tissue swelling develops rapidly, and the distal aspect of the scapular spine may become more difficult to palpate. The limb may appear straighter than usual. A collision also may result in collateral instability of the shoulder, so-called shoulder slip, usually caused by trauma to nerves of the brachial plexus. The horse may have pain-related lameness because of bruising, together with mechanical lameness caused by neurological dysfunction (Fig. 13-2).

Although major fractures of the scapula and humerus are usually readily evident by clinical signs, most other shoulder injuries require radiographic and sometimes ultrasonographic examination to reach a diagnosis.

Pectoral muscle tears may result in similar clinical signs, with severe lameness and distress. Repeated clinical examinations may reveal the site of muscle rupture, with increasing evidence of hemorrhage, inflammatory effusion, and edema.

Rib fractures can also result from direct trauma or from falls and occur most commonly in steeplechasers and polo ponies. Fractures in the region of the scapula and triceps result in acute, severe forelimb lameness. More caudal fractures may result in extreme stiffness and may cause severe respiratory embarrassment.

Lameness associated with the upper forelimb may also be caused by strain of the biceps brachii or brachiocephalicus muscles or hematoma formation. Careful, deep palpation of these muscles is required to identify focal pain and possibly swelling or abnormal muscle texture.

Elbow and Carpus

Acute-onset lameness associated with pain arising from the elbow region is rare, except as the result of a fall or kick. Fracture of the olecranon process of the ulna is the most common injury (Fig. 13-3). If the fracture is non-displaced, the horse may stand normally, but with severe pain or if the fracture is complete with loss of triceps function, the horse stands with a dropped elbow.

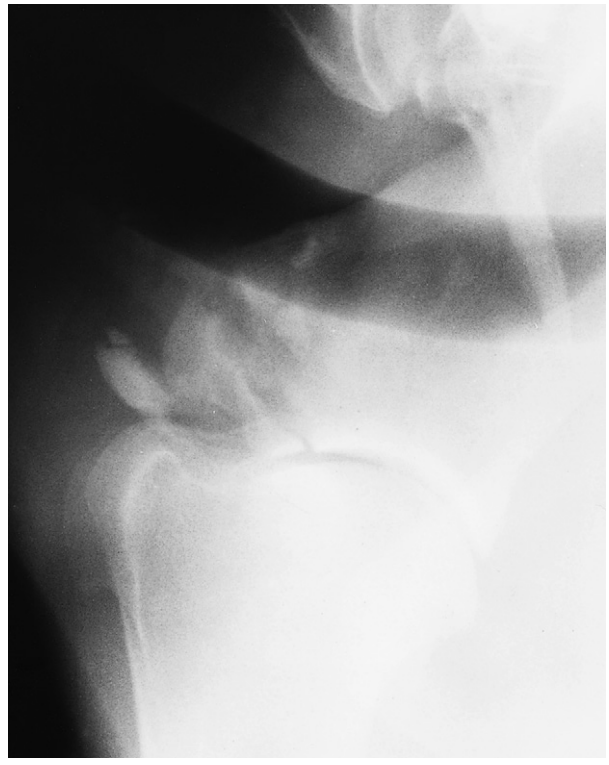

Fig. 13-1 Mediolateral radiographic view of the left shoulder of an advanced event horse that fell during competition 3 days previously and developed severe left forelimb lameness from a displaced comminuted fracture of the supraglenoid tubercle of the scapula.

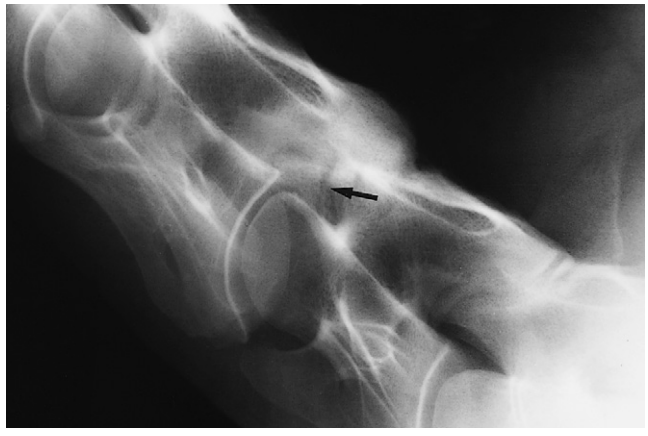

Fig. 13-2 Lateral radiographic view of the mid-cervical region of a 6-year-old event horse that fell on a cross-country course. The horse was not bearing weight on the left forelimb and showed moderate hindlimb ataxia. The synovial facet joints were fractured between the sixth and seventh cervical vertebrae (*arrow*). A cause of the forelimb lameness was not identified and the lameness resolved within 24 hours. The horse had a complete functional recovery but had some residual neck stiffness.

Acute-onset lameness associated with the carpus occurs most commonly, in racehorses, both flat racehorses and steeplechasers, and usually is associated with a chip or slab fracture or, less commonly with hemarthrosis. Synovial effusion within the antebrachiocarpal or middle carpal joint usually develops rapidly. The horse may resent maximal flexion of the carpus,

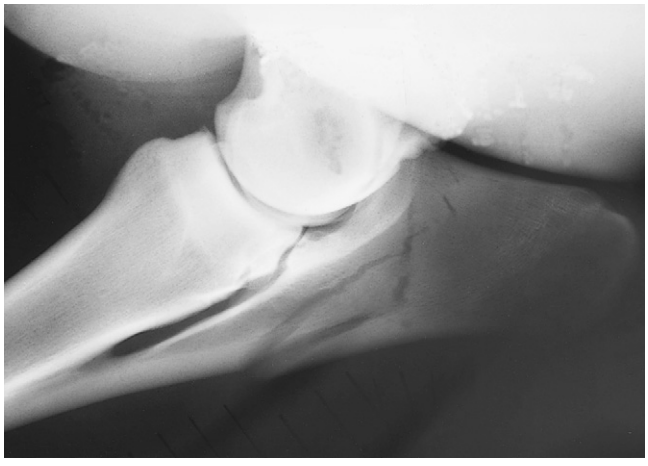

Fig. 13-3 Mediolateral radiographic view of left elbow of an event horse that was very lame after a fall and stood with the elbow dropped. The olecranon of the ulna sustained a displaced, comminuted fracture.

and direct palpation of the carpal bones may elicit pain. Fracture of the accessory carpal bone usually results from a fall and occurs most commonly in steeplechasers; such fractures may be associated with effusion within the carpal sheath. Acute tears of the accessory ligament of the SDFT sometimes occur in polo ponies, with associated distention of the carpal sheath.

Forelimb Soft Tissue Injuries

Injuries of the forelimb suspensory ligament (SL) (proximal, mid-body, and branch lesions), and of the forelimb SDFT occur most commonly in racehorses and event horses, whereas desmitis of the accessory ligament of the deep digital flexor tendon (ALDDFT) occurs more commonly in show jumpers, older steeplechasers, and polo ponies.

Severe, apparently acute onset lesions of the SDFT occasionally occur in show jumpers, especially those of international standard. Evaluation of the posture of the limb and the presence of heat, pain, and swelling are important for accurate diagnosis. Clinical signs associated with these injures can vary markedly. Substantial lesions of the SDFT can develop without detectable lameness, whereas a large tear can result in acute, severe, non–weight-bearing lameness. Bilateral tears may result in extreme distress, a reluctance to move, and a laminitic-like stance. In event horses, lameness can develop after the speed and endurance phase of a Three-Day Event, associated with SDF tendonitis, but no clinical signs may suggest the injury. Swelling or localized heat and pain may take several days to develop despite improvement or resolution of the lameness. Therefore careful re-appraisal of the horse over the next few days is strongly recommended if an event horse develops an acute-onset, forelimb lameness for which no diagnosis can be identified.

Rupture of the SDFT results in hyperextension of the fetlock with normal foot placement. Elevation of the toe with normal angulation of the fetlock indicates disruption of the deep digital flexor tendon (DDFT). Hyperextension of the fetlock and elevation of the toe reflect laceration or rupture of the SDFT and the DDFT. Sinking of the fetlock to the ground indicates disruption of the suspensory apparatus, with or without the flexor tendons.

Severe lameness and distress, with hyperextension of the fetlock, without obvious swelling in the metacarpal region suggests rupture of the SDFT at the musculotendonous junction. This injury occurs most commonly in steeplechasers. Rupture of the SDF tendon usually is a sequel to a previous injury; therefore the tendon is generally chronically enlarged. Detection of the rupture is easiest at the peracute stage, before exudate and hemorrhage fill the deficit. The site of rupture is usually in the mid-metacarpal region.

Desmitis of the ALDDFT usually causes acute-onset, moderate to severe lameness with rapid development of soft tissue swelling in the region of the ligament. However, recurrent injuries can develop with no detectable alteration of a chronically enlarged ligament. The degree of lameness associated with a SL injury varies from mild to moderate but may be worse if a concurrent fracture of the second or fourth metacarpal bone or of the apex of a proximal sesamoid bone exists, and these structures should be evaluated carefully. Some severely lame horses with proximal suspensory desmitis have no palpable abnormalities.

Each of the tendons and ligaments in the metacarpal region should be palpated carefully, from proximally to distally with the limb bearing weight and picked up. The size, shape, and consistency of the tendons and ligaments and any pain on palpation should be carefully assessed. With severe SDF tendonitis the horse may be distressed, and peritendonous edema rapidly develops, which makes accurate palpation of the tendon difficult. Bilateral SDF tendonitis sometimes occurs, with the only detectable palpable abnormality being slight enlargement of each tendon and rounding of its margins. The clinician must be aware of this, because these lesions may be missed if one assumes that because the limbs feel symmetrical, the tendons are normal.

Fractures of the Distal Aspect of the Limbs

A fracture of the lateral, or more rarely the medial, condyle of the third metacarpal or metatarsal bone results in acute-onset, severe lameness. If the fracture is incomplete and non-displaced, no palpable abnormality may be detectable, although some effusion in the metacarpophalangeal joint usually develops within 12 to 24 hours. The horse may resent fetlock flexion; however, some horses become so distressed that in the acute phase, determining whether pressure or joint manipulation causes pain is impossible. The same can apply for a fracture of the proximal phalanx.

Subluxation of the metacarpophalangeal joint occasionally occurs, with disruption of a collateral ligament, with or without an associated fracture. The horse may be very lame, but during normal load bearing the joint may appear to be aligned normally. Instability of the joint may only be detectable with the joint stressed with the limb not bearing weight.

Feet

Trauma to the palmar aspect of the pastern may result in an innocuous skin wound but severe damage to the underlying soft tissue structures. The branches of the SDFT, the DDFT, and the digital flexor tendon sheath are particularly vulnerable. Posture of the limb should be carefully assessed to determine which structure or structures may be involved.

An over-reach on the bulb of the heel can result in severe, deep-seated bruising and lameness, especially on hard ground. Injuries of the foot are common, especially in event horses. The differential diagnosis should include sub-solar hemorrhage (especially corns), nail bind, a sub-solar abscess, and a fracture of the distal phalanx.

If the horse shows any reaction to percussion of the foot or pressure applied with hoof testers, the shoe should be removed for further exploration of the foot. If the horse is very lame, removal of each nail individually using nail pullers may be preferable to levering off the shoe.

Hindlimb Injuries

When presented with a horse with acute hindlimb lameness that developed during exercise, consideration should always

be given to tying up, even if the hindlimb musculature feels soft and local pain cannot be elicited and the horse is not unduly distressed. The clinical manifestations of tying up vary considerably from acute, severe bilateral or unilateral hindlimb lameness with obvious firmness of the muscles of the hind quarter, with or without swelling, to a moderate unilateral hindlimb lameness that developed after the horse was not moving as freely as normal, with no detectable palpable abnormality. This lameness may persist for several hours but usually resolves within 12 to 18 hours. Occasionally, tying up can affect forelimbs, alone or together with the hindlimbs.

Measurement of substantially raised serum creatine kinase concentration 3 to 24 hours after the onset of lameness may be the only way to reach a definitive diagnosis. Alternatively, a horse may not be moving as freely as normal during the competition and subsequently may be withdrawn; further clinical signs may not develop.

Major hindlimb muscle rupture of quadriceps, semimembranosus, gastrocnemius, adductor, or belly muscles results in severe lameness and distress, but diagnosis may be difficult. Careful palpation may reveal a site of rupture. Detection of acute muscle strains may be possible when superficial muscles are involved. Pain on palpation and sometimes swelling may be present; however, this is not always the case, and evaluation of deep muscles is limited.

Stifle trauma is common in horses that jump fixed fences at speed, even if the rider cannot recollect the horse hitting a fence. Lameness caused by a fracture may be sudden and severe in onset, and the horse may pull up, but in some horses it does not become apparent until the horse has finished.

Because the cranial aspect of the stifle is relatively poorly covered with soft tissues, the bones are particularly susceptible to bruising or fracture (Fig. 13-4). The degree of pain on palpation does not necessarily reflect accurately the severity of the injury. Development of femoropatellar or femorotibial effusion or marked periarticular soft tissue swelling suggests a fracture. Sometimes a displaced fragment of bone can be palpated. The superficial location of the femoropatellar joint capsule also makes it vulnerable to puncture and the introduction of infection, and effusion and severe lameness may develop rapidly.

Fig. 13-4 Cranioproximal-craniodistal oblique radiographic view of right stifle of an event horse that hit the penultimate fence at the World Equestrian games. The horse completed the course, pulled up slightly lame, and was very lame within a few hours. The *arrows* show an articular fracture of the medial pole of the patella (*arrows*).

Most horses with stifle injuries require radiographic examination to establish or confirm the presence of a fracture. Although lameness associated with bruising may initially be severe, the horse generally rapidly improves within 24 to 48 hours, whereas with most fractures the lameness usually persists unchanged. The horse may be reluctant to extend the stifle and tends to stand with the limb semi-flexed and the weight on the toe, a posture also typical of severe foot pain. Occasionally severe ligamentous injury occurs in the stifle, which cannot be detected radiographically.

Rupture of fibularis tertius usually results from a traumatic episode, with the limb getting trapped in an extended position, and results in a severe lameness with the horse unwilling to load the limb fully. Passive extension of the hock is pathognomonic.

Acute injuries to the hock are uncommon, unless the horse has a severe fall resulting in damage to the hock joint capsules and the collateral ligaments or a lateral malleolar fracture of the distal tibia. The horse usually is severely lame and rapidly develops distention of the tarsocrural joint capsule and periarticular soft tissue swelling. Radiographic examination is indicated to determine the extent of the damage. Less commonly, slab fractures of the central or third tarsal bone cause acute, severe lameness. Kick injuries may result in a fracture.

Displacement of the SDFT from the tuber calcanei may occur suddenly, resulting in marked distress, especially if the tendon continues to move on and off the tuber calcanei. The tendon may slip laterally, or less commonly medially, and occasionally splits. Peritendonous soft tissue swelling develops rapidly. The horse is reluctant to bear weight on the limb and characteristically shows extreme distress, caused by pain or instability. Careful palpation usually confirms the diagnosis, although acute soft tissue swelling may make this difficult in the initial period after injury.

Periarticular cellulitis of the tarsal region results in an extreme lameness associated with the rapid development of extensive soft tissue swelling, which is exquisitely sensitive to touch. The horse is often lamer than with a fracture, and swelling is more extensive than with synovial infection.

Injuries to the soft tissue structures of the metatarsal and pastern regions are less common than those of the metacarpal and forelimb pastern regions. Suspensory branch injuries are the most common injuries resulting from direct blunt trauma. Acute tears of the DDFT may occur within the digital flexor tendon sheath, with rapid development of effusion. Fractures of the third metatarsal bone and phalanges are also less common than in the forelimbs, except in barrel racing or cutting horses or polo ponies. Plantar process fractures of the proximal phalanx are rare but do occur in racehorses and result in moderate to severe lameness, with pain on manipulation of the fetlock, effusion, and in some horses periarticular soft tissue swelling especially on the plantar aspect. Stability of the fetlock should be assessed carefully.

Stress Fractures

In young Thoroughbred racehorses the possibility of a fatigue or stress fracture must always be considered. The most common sites are the humerus, radius, ilial wing, tibia, third metacarpal bone, and tarsus. With the exception of ilial wing fractures, in which asymmetry of the tubera sacrale and pain on palpation in this area may be obvious, localizing clinical signs may otherwise be absent. A definitive diagnosis can rarely be made by clinical examination alone.

Hemarthrosis

Hemarthrosis may occur in any joint and results in acute onset, non–weight-bearing lameness associated with distention of the joint capsule. Lameness often improves rapidly

within the following 48 hours. Diagnosis is based on ultrasonography and synoviocentesis. Draining blood from the joint produces rapid relief of clinical signs.

TRANSPORTATION

In any horse with severe lameness in which making a tentative diagnosis based on a preliminary but thorough clinical examination is not possible, a decision has to be made about whether the horse is fit to travel, whether any risks are involved in travel, and whether the horse should be stabled as close as possible and be reassessed later or the following day (if this is practical). Whether the horse is insured should be established, together with the terms of the insurance policy.

The majority of horses can be taken safely to the nearest adequate diagnostic facility, which ideally should have a loading ramp, a veterinarian experienced in orthopedics, facilities for hospitalization, and high-standard radiographic, ultrasonographic, and possibly nuclear scintigraphic, equipment.

Facilities for orthopedic surgery are not necessarily essential, although desirable, because the first step must be to reach an accurate diagnosis. Having reached a diagnosis, the limb may then be appropriately supported to minimize risks of exacerbating the injury, if the horse is to be treated conservatively, or during induction of general anesthesia or for transfer to a suitable surgical facility. If a hindlimb fracture is suspected, the horse should be tied up (cross tied). For transport for further diagnostic investigation or surgical treatment the injured limb should usually be supported using a Robert Jones bandage,[1] with or without splints, or an appropriate commercial splint, bearing in mind the proposed site of injury[2] (see Chapter 87). For forelimb injuries the horse should ideally travel facing backward, although some low-loading ambulances are not designed for loading from the front.[3] When possible a low-loading trailer should be used, but if this is not available, the ramp of the vehicle should be placed on a slope to minimize the gradient for loading and unloading. If a diagnosis has been made for a horse with an injury that requires rapid surgical treatment, the limb should be supported in the most appropriate way and the horse referred to the nearest surgical facility, or to the best surgical facility in close proximity to the horse's place of origin, or to the person with the most expertise and experience dealing with that kind of injury. Provided that the limb is adequately immobilized and adequate pain relief is possible, the horse should be fit to travel several hours safely and humanely.

GUIDELINES FOR HUMANE DESTRUCTION OF AN INJURED HORSE

If the horse is insured for all risks of mortality, owners, trainers, and other interested people may request humane destruction. The American Association of Equine Practitioners (AAEP) and the British Equine Veterinary Association (BEVA) have issued guidelines indicating the requirements that should be fulfilled to satisfy a claim under a mortality insurance policy. Nonetheless, the decision to advise an owner to destroy a horse on humane grounds must be the responsibility of the attending veterinary surgeon, based on the assessment of clinical signs at the time of the examination or examinations, regardless of whether the horse is insured. The veterinary surgeon's primary responsibility is always to ensure the welfare of the horse. On occasion the attending veterinary surgeon will advise euthanasia, but such a decision may not necessarily lead to a successful insurance claim. It is important that all parties are aware of these potential conflicts of interests before a horse is destroyed. The owner's responsibility is to ensure compliance with any policy contract with an insurer.

The AAEP has issued the following guidelines for recommending euthanasia:

> The AAEP recommends that the following criteria should be considered in evaluating the immediate necessity for intentional destruction of a horse. It should be pointed out that each case should be addressed on its individual merits and that the following are guidelines only. Not all criteria must be met in each case.
>
> 1. Is the condition chronic, incurable and resulting in unnecessary pain and suffering?
> 2. Does the immediate condition present a hopeless prognosis for life?
> 3. Is the horse a hazard to itself or its handlers?
> 4. Will the horse require continuous medication for the relief of pain for the remainder of its life?
>
> Justification for euthanization of a horse for humane reasons should be based on medical grounds, not economic considerations; and further the same criteria should be applied to all horses regardless of age, sex or potential value.

The BEVA guidelines for compliance for a mortality insurance policy are as follows:

> That the insured horses sustains an injury, or manifests an illness or disease, that is so severe as to warrant immediate destruction to relieve incurable and excessive pain, and that no other options of treatment are available to that horse, at that time.
>
> If immediate destruction cannot be justified, then the attending veterinary surgeon should provide immediate first aid treatment before:
>
> 1. Requesting that the insurance company be contacted, or, failing that
> 2. Arranging for a second opinion from another veterinary surgeon.

It is essential that the attending veterinary surgeon keep a written record of the injuries sustained by the horse, its identification, and the date, time, and place. The owner or agent should whenever possible sign a form consenting to euthanasia. Insurance companies frequently require some form of examination after death and may request an independent postmortem examination, and this must be borne in mind when arranging for disposal of the carcass.

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CHAPTER • 14

The Swollen Limb

Sue J. Dyson

The development of diffuse or more localized swelling in one or more limbs can present a diagnostic challenge, requiring a systematic approach to identify the cause. Although the metacarpal, metatarsal, and pastern regions are most commonly affected, an entire limb may be swollen, or swelling may initially be restricted to the antebrachium or crus or the carpus or tarsus, with swelling subsequently spreading distally. This chapter discusses an approach to diagnosis and management but does not provide exhaustive differential diagnoses and treatments. Some conditions are discussed in more detail in other chapters.

DIAGNOSIS

Many stabled horses develop some degree of enlargement of the distal limbs, especially the hindlimbs, that dissipates with work. Termed *filled legs* or *cold edema* (stocked-up, stoved-up), this swelling may be controlled by applying stable bandages and is of no consequence.

History

Accurate diagnosis of the cause of limb swelling requires knowledge of the history.

- Was the problem sudden or gradual in onset?
- What is the duration of the swelling?

It may be pertinent to establish when the owner last thought that the horse was normal, especially in horses that are not inspected reguarly while kept at pasture. The actual duration of swelling may be longer than the owner recognized. The veterinarian also should bear in mind that some owners are remarkably unobservant, despite maintaining that they veterinarian and groom the horse thoroughly daily.

- Were any swellings pre-existing?
- What was the initial distribution of the swelling and has this changed?
- Has similar swelling appeared previously (lymphangitis may be a recurrent problem)?
- Does any history of trauma exist?
- Has the limb been bandaged, or have boots or bandages been used for exercise? Overly tight bandages can rapidly result in severe cellulitis, skin excoriation, and hair loss. Sand or grit inside a boot can provoke severe skin inflammation and diffuse soft tissue swelling.
- Has the horse had tendonitis or desmitis? Recurrent injury may be much more severe and result in diffuse swelling, prohibiting accurate palpation of the injured structure.
- Has the horse had any other recent clinical problems (strangles may predispose to purpura hemorrhagica)?
- Does the horse show any other clinical signs, and could the limb swellings reflect systemic disease?
- Is the horse lame, and when did lameness develop relative to the recognition of swelling? If swelling preceded the lameness, more than one problem may exist.
- Has the horse received any treatment, and what was the response? Has anything topical been applied to the limb that may be irritant?

Swelling in a single limb usually reflects a local problem, whereas swelling in several limbs may be due to systemic disease or a primary skin problem. The differential diagnosis should include the following: sub-solar abscess (see Chapter 28); mud fever or scratches; scabby skin lesions on the palmar aspect of the fetlock; other bacterial pyodermas; hemorrhage or thrombosis (see Chapter 38); desmitis or tendonitis (see Chapters 70, 72, and 73); cellulitis associated with superficial digital flexor tendonitis (see Chapter 70); skin necrosis and cellulitis after topical application of proprietary products; infected tendon or tendon sheath; cold edema; cellulitis caused by trauma or infection; fracture; or hypertrophic osteopathy (see Chapter 38); muscle rupture (see Chapter 13); muscle trauma resulting in compartment syndrome (see Chapter 84); lymphangitis; photosensitization; equine viral arteritis; heart failure; and hypoproteinemia.

Clinical Examination

A systematic clinical examination should be performed by careful observation and palpation. The veterinarian should assess the horse's posture, demeanor, and attitude. Depression may reflect pain or infection.

- Is the horse febrile? The clinician should determine whether one or more limbs are involved.
- What is the distribution of the swelling? Is it localized or more diffuse? Swelling associated with a sub-solar abscess is usually diffuse and involves the pastern and metacarpal or metatarsal region, extending a variable degree proximally, and is symmetrically distributed around the limb. In contrast, swelling associated with direct trauma may be restricted to the metacarpal region in the acute phase and only later spread distally.
- Does the swelling relate to a joint or to a long bone and the surrounding soft tissues?
- Where is swelling maximal? Is swelling predominantly on one side of the limb, which may reflect trauma, because of the horse becoming cast?
- Does the metacarpal (metatarsal) region have a straight palmar (plantar) contour? Diffuse swelling in the metacarpal region is unlikely to reflect superficial digital flexor tendonitis if the palmar aspect of the limb is straight.
- Is the swelling cool, warm, or hot? Hot swelling is most likely to be associated with infection.
- Is the swelling tense, often reflecting infection, or soft?
- Is evidence of pitting edema present?
- Does light digital pressure or firm pressure elicit pain? Pain caused by only light pressure is often associated with cellulitis caused by infection or less commonly by intra-tendonous infection.
- How does the location of pain relate to the site of maximal swelling?
- How easily can underlying anatomical structures be palpated, and does palpation cause pain? The

veterinarian should note that scabby skin lesions overlying soft tissue swelling can in themselves be remarkably painful.
- Does a draining tract suggest an abscess, cellulitis, or infectious osteitis or osteomyelitis?
- Does joint manipulation cause pain?
- Do any skin lesions exist through which infection may have entered? This may not be readily apparent in a horse with a long hair coat, and clipping may be necessary. In a horse kept at pasture the limb may be caked in mud, and thorough cleaning may be necessary before an accurate clinical examination can be made.
- Do any other skin lesions reflect a primary dermatological problem? The clinician should try to categorize any lesions identified.

The digital pulse amplitudes should be assessed: any increase highly suggests a primary foot problem. The response to pressure and percussion applied with hoof testers should be evaluated. Pulse rate, the quality of the peripheral pulses, capillary refill time, and careful auscultation of the heart and lungs should reveal whether a primary cardiac problem exists.

The mucous membranes should be examined for evidence of petechial hemorrhages, which can be seen with purpura hemorrhagica. The veterinarian should relate the swelling to the color of the limbs; swelling confined to white limbs may result from photosensitization.

The clinician should establish the degree of lameness and bear in mind that mild stiffness may result from extensive limb swelling and mechanical restriction. Extremely severe lameness often reflects infection, either periarticular (e.g., peritarsal cellulitis; see Chapter 45), intra-articular, intra-thecal, intra-tendonous, or sub-solar. A horse with a fracture may be less lame.

The results of this clinical examination should suggest the likely causes of the swelling, but definitive diagnosis might not be possible without further investigation. This may include radiography, ultrasonography, routine hematological testing, measurement of total protein and fibrinogen, and liver enzyme levels if indicated. Paired serum samples may be required to confirm equine viral arteritis. Treating the horse symptomatically to reduce the soft tissue swelling may be helpful to facilitate more accurate palpation. This may include the use of non-steroidal anti-inflammatory drugs (NSAIDs), hydrotherapy, poulticing or leg sweats, bandaging, and walking, with or without antimicrobial medication. Without evidence of a primary infectious process the response to corticosteroids may be helpful diagnostically, because limb filling may be an immune-mediated response. The clinician should be prepared to make repeated examinations if a primary diagnosis is not readily apparent.

Early periosteal new bone associated with hypertrophic osteopathy is readily overexposed, and greatly reduced exposure factors are required for its radiographic detection. On the first day of examination the results of radiographic and ultrasonographic examinations may be misleading, and repeated examinations may be necessary. Following trauma, laceration, or both injuries, to the antebrachium, crus, and metacarpal or metatarsal regions, delayed-onset lameness caused by an occult spiral fracture of the radius, tibia, or third metacarpal or metatarsal bones is possible. Many oblique radiographic views or follow-up examination be may be necessary to identify the fracture. If extensive cellulitis occurs around a joint or tendon sheath, but intra-articular or intra-thecal infection are suspected, the examiner should be cautious about performing synoviocentesis through infected tissues, because iatrogenic intra-articular or intra-thecal infection may ensue. If skin lesions are identified as a possible primary cause of limb swelling, but these fail to respond to topical or systemic treatment, obtaining skin biopsies for culture and histological examination or seeking specialist advice from a dermatologist may be necessary.

MANAGEMENT

Mud Fever

Mud fever (scratches or pastern dermatitis) is associated with bacterial or fungal skin infection and usually is restricted to the palmar or plantar aspect of the pastern but sometimes extends farther proximally if severe. Mud fever is associated with many excoriated skin lesions, which may develop severe crusting. Deep fissures may develop in the skin, especially if the condition goes unrecognized or in horses with many skin folds in the pastern region. Extensive edematous swelling often extends up the metacarpal and metatarsal regions. If the condition is mild, no associated lameness may occur, but severe lesions are associated with marked stiffness. The condition can occur in horses kept out in wet, muddy conditions or in horses that are stabled but work in a muddy environment. Certain soil types seem to be associated with a higher occurrence. Some horses seem prone to recurrent episodes, although this may in part reflect management practices. The condition is difficult to manage if the horse is left in wet, muddy pasture and it must be stabled. The affected areas should be clipped and thoroughly cleaned with chlorhexidine solution. The scabs should be softened to facilitate removal. If the condition is mild, no further treatment may be required, but if the condition is more severe, daily topical application of lanolin-based emollient cream with trimethoprim and sulfadiazine and dexamethasone is indicated, sometimes combined with systemic antimicrobial treatment. Alternatively, a proprietary topical preparation can be used. The limbs should be carefully cleaned and dried after exercise.

Scabs on the Palmar Aspect of the Fetlock

Some horses seem prone to develop many small skin scabs on the palmar or plantar aspects of the fetlock. The scabs appear to be related to work on specific surfaces, which presumably cause skin irritation and subsequent bacterial infection. These skin lesions are often associated with diffuse swelling and can be exquisitely painful. The lesions rarely resolve spontaneously but usually resolve with penicillin therapy.

Cellulitis Caused by Trauma

Direct trauma to a limb may result in extensive edematous soft tissue swelling unassociated with infection. If skin abrasion is concurrent, the site of the wound relative to synovial structures susceptible to infection must be evaluated carefully. Lameness may vary in degree, but if severe, the possibility of fracture must be considered. If little soft tissue covers the underlying bones, radiographic examination is prudent to eliminate the possibility of a fracture. With primary cellulitis, treatment with NSAIDs and rest and controlled exercise is usually all that is required.

Cellulitis Caused by Infection

Cellulitis associated with infection from a penetrating wound usually results in fairly extensive soft tissue swelling, which tends to be warmer and more painful than noninfectious cellulitis (see Fig. 5-8). Associated lameness may also be severe, depending in part on the location of infection. If untreated, abscessation may develop in muscular areas and may require surgical drainage. Cellulitis may also be concurrent with infectious osteitis or osteomyelitis (see Chapter 38). Horse with acute infectious cellulitis usually respond well to systemic broad-spectrum antimicrobial treatment, unless clostridial organisms are involved (see Chapter 84).

Lymphangitis

So-called lymphangitis occurs more commonly in hindlimbs than forelimbs and is often unilateral but may be bilateral. Diffuse soft tissue swelling occurs throughout the limb, often

extending distad from immediately below the stifle. The superficial lymphatic vessels may appear more prominent than usual. Serum may ooze through taut skin. The degree of swelling usually results in mechanical stiffness that improves with progressive walking. Careful inspection may reveal some small skin lacerations, often in the more distal part of the limb. Once a horse has had a severe attack of lymphangitis, it seems prone to recurrence, often after seemingly innocuous skin abrasions. Although the condition appears to be triggered by infection, antimicrobial treatment alone is inadequate and usually must be combined with long-term corticosteroid treatment (dexamethasone 0.05 to 0.2 mg/kg once daily intravenously or intramuscularly, using the lowest dose necessary to control edema, and replacing with prednisolone 0.5 to 1 mg/kg intramuscularly or by mouth twice daily, when the dexamethasone dose is <0.04 mg/kg), together with aggressive hydrotherapy and walking exercise, with or without bandaging and topical application of leg sweats. Bandaging the metatarsal region in the face of more proximal limb swelling tends to result in persistent proximal limb swelling that cannot move distally. Although prompt aggressive treatment may resolve clinical signs, persistence of marked filling for more than a week may result in chronic enlargement of the limb. Ulcerative lymphangitis occurs much less frequently and is caused by bacterial or fungal infection.

Purpura Hemorrhagica

Purpura hemorrhagica usually occurs as a sequel to previous streptococcal respiratory infection but occasionally follows other antigenic stimuli. Purpura hemorrhagica results in extensive submucosal petechial hemorrhages, evident clinically in the mucous membranes but actually more widespread, including muscle and viscera. Facial swellings and limb edema may be extensive. The horse is usually depressed, inappetant, and pyrexic. Purpura hemorrhagica is an acute, probably immune-mediated, necrotizing vasculitis. Aggressive treatment with penicillin and corticosteroids (see the previous discussion) is required.

SECTION • 2
Diagnostic Imaging

CHAPTER • 15

Radiography and Radiology

Sue J. Dyson

Radiography is an important part of the diagnostic armamentarium in the evaluation of lameness. Its most important role is to give information about bones and joints. However, it also can provide information about soft tissues, most particularly tendon, ligament, and joint capsule insertions. If radiography is to be used properly, then the area under investigation must be evaluated comprehensively and appropriately. A sufficient number of views, all which have been appropriately centered and exposed, should be obtained.

Obtaining high-quality radiographs requires attention to detail. The horse must be correctly positioned and adequately restrained or sedated. For most weight-bearing examinations the horse should stand with the cannon bone of the limb to be examined in a vertical position. The horse should be standing on all four limbs, not resting a limb. The area under investigation should be cleaned to remove any surface dirt. For examinations of the foot, the shoe should be removed (if possible) to facilitate proper paring of the sole and frog, to ensure it is adequately clean, and to avoid superimposition of a radiodense shoe over the distal phalanx and navicular bone. The tail should be tied to facilitate correct positioning of the cassette when examining the stifle and hock regions.

RADIOGRAPHIC DETAIL

The aim is to obtain as highly detailed radiographic images as possible. The detail that can be obtained is influenced by a number of factors, including the following:

- Film screen combination. High-definition screens for use with single-emulsion and relatively slow film provide the best definition in the distal limbs. When higher exposure factors are required to gain adequate penetration of the more proximal parts of the limb, rare earth screens are required to minimize exposure times and thus reduce the risk of movement blur.
- Proper contact between the film and the screen. Old screens become warped and result in loss of image quality.
- Cleanliness of the screens. Dust and hair accumulate easily within cassettes, resulting in radiopaque artifacts and lines on the films. Screens must be cleaned regularly. Careful darkroom technique is essential to reduce the risk of dust build-up.
- Power of the x-ray machine. The use of high-definition screens is possible only with x-ray machines capable of an output of 100 kV and 100 mAs. Otherwise, exposure times are too long, resulting in movement blur.
- Choice of appropriate exposure factors. This step is less critical with digital radiography when post-processing can be used.
- Use of the correct focus film distance (FFD). X-rays obey the inverse square law, so that an alteration in the FFD potentially has a big effect. Single-emulsion film is particularly sensitive to a slight change in the FFD.
- Exposure time. The exposure time should be as short as possible to reduce movement blur.
- Use of grids. Grids are required in areas with a large amount of soft tissue, which results in scattered radiation. The use of a grid requires a higher exposure factor. If a focused grid is used, the x-ray beam must be perpendicular to the grid and centered on it, and the correct FFD should be used. Parallel grids have slightly more latitude when a FFD of more than 120 cm is used. The higher the grid ratio and lines per centimeter, the more effective the grid is in reducing scattered radiation, but the higher the grid factor. The grid factor denotes how much an exposure must be increased from non-grid values for comparable opacity. For example, if the grid factor is 2, then the milliamperes seconds (mAs) should be doubled.
- Lead. Lead is placed behind the cassette when large exposures are used to reduce the amount of backscatter.
- Collimation of the x-ray beam.
- Use of stationary cassette holders when possible and practical to reduce movement of the cassette.
- Size and shape of the horse. These factors become particularly important when examining the thoracolumbar region. The amount of muscle mass and fat influence the exposure factors; with very large horses the risk of the radiographic images becoming flat as the exposure is increased. The shape of the horse's barrel also influences how closely the cassette can be placed to the back and thus influences magnification. The use of an aluminum wedge filter allows the intensity of the beam to be reduced in specific areas and is of particular value when examining areas with a marked change in soft tissue thickness from one side of the film to the other (e.g., the dorsal spinous processes in the thoracolumbar region, the shoulder, and the stifle).
- Amount of soft tissue swelling. If marked periarticular soft tissue swelling is present, higher exposures are required to achieve adequate penetration. The use of a grid in these circumstances helps to reduce scattered radiation.
- Cooperation of the patient.

Image Resolution

Image resolution is the objective measurement of how much detail can be provided by a film screen combination and is measured in line pairs per millimeter. Resolution indicates the size of the smallest object that the system will record (i.e., the smallest distance that must exist between two objects before they can be distinguished as two separate entities). *Image definition* cannot be quantified but is the subjective impression of the amount of detail that can be seen on a radiograph.

Image Contrast

Contrast is the degree of definition on a radiograph between adjacent structures of differing radiopacities. *Opacity* or *radiopacity* is the degree of whiteness of the object being radiographed. The denser the physical structure, the greater the degree to which the tissue absorbs x-rays, and the more opaque it appears on a radiograph.

Exposure Factors

Exposure factors affect the opacity and contrast of the radiographic image. The kilovoltage governs the quality and intensity of x-rays and affects both contrast and opacity. The quantity of x-rays reaching the x-ray film is the product of milliamperage and time, and is also influenced by the FFD, according to the inverse square law. The following equation is used to calculate the exposure of a change in distance:

$$\text{Old mAs} \times \frac{(\text{New FFD})^2}{(\text{Old FFD})^2} = \text{New mAs}$$

The milliamperes seconds and FFD affect the opacity of the image, not the contrast.

Exposure Latitude

Exposure latitude is the degree of over-exposure or under-exposure that can be tolerated in a correctly developed film and still produce an image of acceptable radiographic quality. A low kilovoltage yields high contrast with low latitude, whereas a high kilovoltage results in low contrast but has wide latitude. For good bone detail the kilovoltage should be less than 70 kV. Attenuation of the x-ray beam depends heavily on the atomic number of the tissues, and it is desirable that photoelectric absorption predominates. Increasing the kilovoltage also results in more forward-scatter.

To obtain a radiograph with the same opacity as the original, but with decreased contrast, the milliamperes seconds are halved and the kilovoltage is increased by 15% (approximately 10 kV). To increase the contrast but maintain the opacity, the milliamperes seconds are doubled and the kilovoltage is reduced by approximately 15%.

Image Sharpness and Resolution

Lack of image sharpness can be caused by a number of factors, including movement of the patient. Short exposure times help to reduce the risk of movement blur. Reducing the FFD can increase the amount of x-rays reaching the patient and therefore the exposure time could be reduced. However, reduction of FFD results in an increase in geometric indistinctness or *penumbra*. Image blur also may be the result of poor screen film contact. Contact can be tested by placing a wire mesh on top of the cassette and making an exposure using a large FFD. Any loss of image sharpness is due to poor film screen contact.

Image resolution also can be influenced by the focal spot size of the x-ray machine. High-output x-ray machines usually have different size focal spots. A small focal spot (e.g., 0.6 mm) usually results in better image resolution, but an increased exposure time is required to achieve the same milliamperes. When movement is likely to be a problem (e.g., proximal limb examinations), a larger focal spot (1.5 to 2.0 mm) is preferable to reduce exposure times.

Film and Screen Factors

Relative speed ratings of screens and distance factors must be considered when selecting exposure factors. Speed classification of film screen combinations allows comparison of systems between manufacturers. Some manufacturers use 100, 200, 400, and so on, and others use 2, 4, 8, and so forth, but the interrelationship is the same. Speed 8 screens require half the exposure (milliamperes seconds) needed for speed 4 screens; speed 200 requires twice the exposure (milliamperes seconds) of speed 400. Although the same exposures are required to provide the same image opacity using similar film screen speeds, the detail and resolution may vary. Generally when only one screen from a pair is used (when using single-emulsion film), the speed of the system will halve. Thus if one screen from a pair rated 400 is used, the speed will be 200.

RADIATION SAFETY

Radiation safety (i.e., ensuring that personnel around the horse do not receive doses of radiation) is essential. Different codes of practice apply in various countries, but the basic principles can be summarized as follows:

- The number of people present during radiography should be kept to the absolute minimum required to restrain the horse, position the limb and the x-ray cassette, and obtain the radiograph.
- Appropriate restraint or sedation of the horse is essential to ensure it remains still during exposures. This practice will limit the number of repeat exposures needed.
- Collimation of the x-ray beam. Use of a light beam diaphragm permits maximum collimation. No part of any attending person, even if covered with protective clothing, should be placed in the primary x-ray beam. Protective lead clothing protects from scattered radiation only, not the primary beam. The primary beam continues beyond the patient and cassette, and personnel standing on the opposite side also are at risk.
- All personnel who must remain present for the exposure must wear protective lead gowns. If near the primary beam, they should also wear protective lead gloves, hand and arm protection, and a thyroid protector.
- All protective clothing should be checked annually and replaced when necessary.
- All personnel working with and around the x-ray machine must be monitored using a film badge system.

RESPONSE OF BONE TO STIMULI: WOLFF'S LAW

Correct interpretation of radiographic images requires knowledge of the ways in which bone responds to various stimuli. Bone models according to Wolff's law: it models according to the stresses placed on it so that it can be functionally competent with the minimum amount of bone tissue. The use of the terms *modeling* and *remodeling* creates considerable confusion because usage differs in histology and radiology. *Histologically, bone remodeling* refers to resorption and formation of bone that is coupled and occurs in basic multicellular units. This regulates the microstructure of bone without altering its shape and is a continuous process, replacing damaged bone with new bone. Thus remodeling cannot be appreciated radiographically. *Radiographically,* the term has been used to describe reshaping of bone to match form and function (e.g., after fracture repair). Strictly speaking, the term *modeling* should be used to describe the change in the shape of a bone as it adapts to the stresses applied to it.

Bone is a dynamic tissue, constantly reacting to the stimuli that it receives both internally and externally. However, it takes time to respond, and a 40% change in bone density must occur before changes are evident radiographically. Therefore radiographs, although anatomically accurate, are relatively insensitive in the early stages of a disease process. This is known as the radiographic *latent period*. It is critical to appreciate these limitations when interpreting radiographs. Bone can be undergoing abnormal modeling without identifiable

structural change. Once radiographic abnormalities have developed, some will persist over the long term without necessarily being associated with ongoing pain. Thus in effect these changes remain as scars reflecting previous injury. Aging of such lesions is impossible, and assessing clinical significance must be evaluated in the light of the clinical signs.

Bone can react to stimuli in only a limited number of ways. Bone can produce new bone, such as periosteal new bone, endosteal new bone, cortical thickening, increased thickness of trabeculae, callus formation, osteophyte and entheseophyte formation, and the pallisading periosteal new bone typical of hypertrophic osteopathy. New bone often results in what is described radiographically as *sclerosis*: increased opacity of the bone, caused by either new bone being laid down within the bone or superimposition of new bone on the surface of the bone. More than one radiographic view usually is required to determine why a structure appears sclerotic. Strictly speaking, however, sclerosis is a localized increase of opacity of the bone caused by increased bone mass within the bone.

O*steolysis* is resorption of bone resulting in a radiolucency. Again, a lag period, usually of at least 10 days, occurs between the onset of lysis and its radiographic detection. Osteolysis occurs for a variety of reasons, including pressure, infection, as part of early fracture repair, and as part of the disease process in osteoarthritis, osteochondrosis, osseous cyst-like lesions, and subchondral bone cysts. Bone destruction and resorption usually are seen more easily in cortical bone, rather than cancellous bone, because of the greater contrast.

Generalized demineralization, or *osteopenia*, of bone throughout the body rarely occurs in the horse. Localized demineralization in a single limb usually is the result of disuse and is characterized by thinning of the cortices and more obvious trabecular pattern. The proximal sesamoid bones are particularly sensitive indicators of disuse osteopenia in the horse.

Focal demineralization and loss of bone may be due to pressure, for example, as seen in chronic proliferative synovitis in the fetlock, resulting in erosion of the dorsoproximal aspect of the sagittal ridge of the third metacarpal bone (McIII). It may be the result of infection, invasion by fibrous tissue, or a neoplasm.

Cortical thickness changes (models) according to Wolff's law as an immature athlete develops into a mature, trained athlete. The dorsal cortex of the third metacarpal and third metatarsal bone becomes thicker. If a horse has marked conformational abnormalities, such as "offset or bench knees," the bones model accordingly with the lateral cortex of the distal limb bones becoming thicker.

Periosteal New Bone

Blunt trauma to bone can lead to subperiosteal hemorrhage, resulting in lifting of the periosteum away from the bone. This process may stimulate the production of periosteal new bone (Fig. 15-1). Some bones, such as the second and fourth metacarpal and metatarsal bones, seem particularly prone to such reactions. The variation in susceptibility to such reactions between horses appears to be individual. Usually a lag period of at least 14 days occurs between trauma and the radiographic detection of periosteal new bone. Such bone usually is much less dense than the parent bone; therefore soft exposures (or low kilovoltage) are essential for detection of this bone, which initially has a rather irregular outline. As the bone gradually consolidates and then models, it becomes more opaque and more smoothly outlined. Curiously, although a well-established splint would be expected to become inactive, many appear slightly hotter than the parent bone if examined scintigraphically.

Periosteal new bone can also develop as a result of fracture, infection, inflammation, and neoplasia. Inflammation of the interosseous ligamentous attachment between the second and third metacarpal or fourth and third metacarpal bones caused by movement and loading can result in periosteal new bone formation and a splint. It is curious that some of these formations develop rapidly without associated pain, whereas others can cause persistent pain and lameness for many weeks, despite a similar radiographic appearance. The bony protuberances that develop on the proximolateral aspect of the metatarsal regions, often bilaterally, are even more enigmatic. They are rarely associated with clinical signs, although they often appear hot scintigraphically despite having been present for several years.

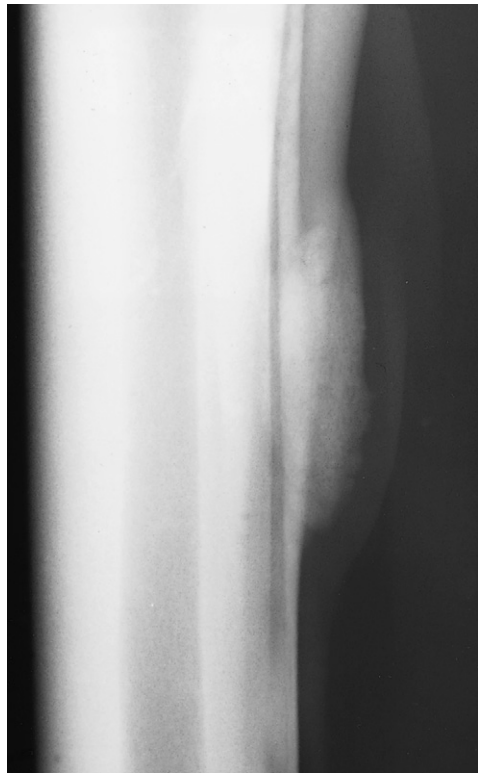

Fig. 15-1 Dorsomedial-palmarolateral oblique view of the metacarpal region. Soft tissue swelling overlies periosteal new bone on the mid-diaphyseal region of the second metacarpal bone.

Endosteal New Bone

Endosteal new bone may develop as a result of trauma (e.g., a cortical or subcortical fracture) or inflammation, infection, or less commonly, a tumor (Fig. 15-2). Stress fractures of the dorsal cortex of the third metacarpal bone are accompanied by the development of endosteal new bone, which may be more readily detected than the fracture itself.

Sclerosis

Sclerosis is the localized formation of new bone within bone and results in increased bone mass. It is most easily identified in trabecular bone (Fig. 15-3) and occurs in response to several stimuli, including the following:

- Stress (e.g., subchondral sclerosis in osteoarthritis and sclerosis of the medulla of the navicular bone in navicular disease)
- Protection of a weakened area (e.g., sclerosis surrounding an osseous cyst-like lesion or a subchondral bone cyst)
- Walling off infection (e.g., adjacent to a sequestrum or in the medullary cavity adjacent to an area of osteomyelitis)

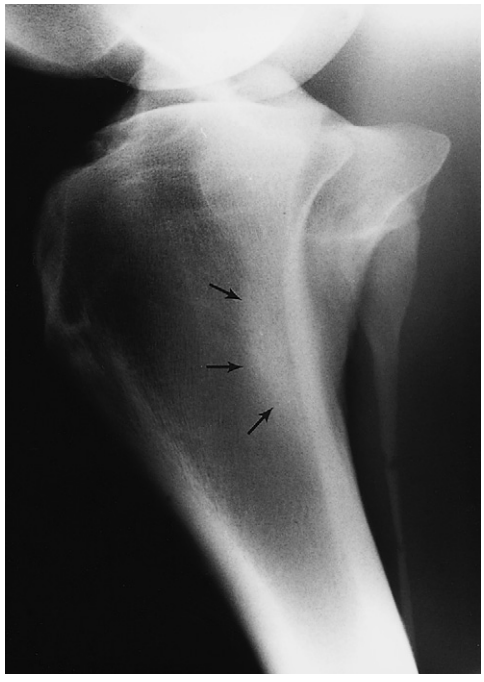

Fig. 15-2 Lateromedial view of the proximal tibia. There is increased opacity of the subcortical bone of the proximocaudal aspect of the tibia. Endosteal new bone *(arrows)* is associated with a fatigue fracture.

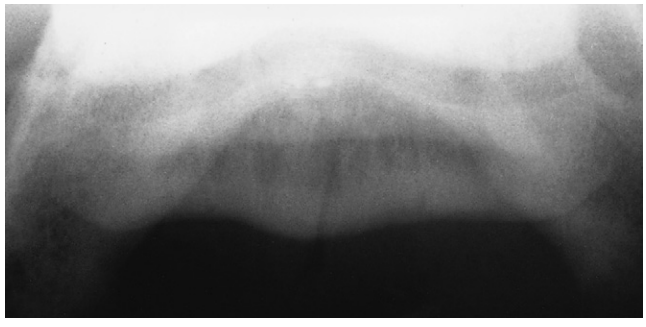

Fig. 15-3 Palmaroproximal-palmarodistal oblique view of a navicular bone. There is increased thickness of the trabeculae of the medulla of the navicular bone, resulting in sclerosis. Several large, oval-shaped lucent zones are present within the medulla. The flexor cortex is thickened and corticomedullary demarcation is poor.

Enostosis-like lesions are the development of bone within the medullary cavity or on the endosteum, resulting in a relatively sclerotic area of variable size. They frequently occur adjacent to the nutrient foramen, and the origin is unclear. They may develop as a focal or multifocal lesion. They vary in size and generally are seen in the diaphyseal regions of long bones in the horse. These lesions have been seen most frequently in the humerus, radius, and tibia and the third metacarpal and third metatarsal bones. When these lesions develop, they are very hot scintigraphically and may be associated with pain and lameness. However, they also are seen as incidental findings.

Small focal opacities in the proximal metaphyseal region of the tibia have been seen. The origin and significance are not known. Care must be taken in the fetlock region not to misinterpret the radiopacity caused by the ergot as an opaque lesion within the proximal phalanx.

Osteophyte Formation

An *osteophyte* is a spur of bone on a joint margin that develops as a result of a variety of stimuli, including joint instability, or in association with intra-articular disease, particularly osteoarthritis. Not all periarticular modeling changes at the junction of the articular cartilage and periarticular bone are associated with ongoing joint disease, but radiological differentiation between a subclinical osteophyte and a clinically significant one is difficult. Small spurs frequently are seen on the dorsoproximal aspect of the third metatarsal bone close to the tarsometatarsal joint. Some are quiescent, unassociated with articular pathological findings, whereas others are progressive. Small spurs on the dorsoproximal aspect of the middle phalanx are frequent incidental findings in Warmblood breeds. Mature horses with offset or bench-knee conformation frequently have spurs on the lateral aspect of the antebrachiocarpal joint without associated clinical signs.

The time of development of an osteophyte after stimulus varies depending on the inciting cause and individual variation. Two weeks to several months may pass before an osteophyte may be identified radiographically. A smoothly marginated osteophyte of uniform opacity is more likely to be long-standing, whereas a poorly marginated osteophyte with a lucent tip is likely to be active.

Some joints seem to have a greater propensity than others for the development of periarticular osteophyte formation. The reason for this tendency is unknown and may in part reflect the ease with which osteophytes can be detected radiographically. Even within what is currently considered a single disease process, osteoarthritis of the distal hock joints (bone spavin), some horses develop predominantly periarticular osteophytes (Fig. 15-4), whereas others have narrowing of the joint space and subchondral sclerosis. A third group develops extensive radiolucent areas (Fig. 15-5).

Entheseophyte Formation

Entheseophyte formation is new bone at the site of attachment of a tendon, ligament, or joint capsule to bone. Entheseous new bone reflects the bone's response to stress applied through these structures, such as ligamentous tearing or capsular traction (Fig. 15-6). Like osteophytes, entheseophyte formations take several weeks to months to develop and may or may not be associated with clinical signs. Knowledge of ligament, tendon, and capsular insertions is essential to determine which soft tissue structure may have been damaged. In some locations, such as the hock, differentiation between entheseophyte and osteophyte formation is not easy. The attachments of cranialis tibialis and fibularis tertius are close to the joint margins of the tarsometatarsal joint, and differentiation between entheseous new bone at these attachments and periarticular osteophyte formation may be difficult. Entheseous new bone may take the form of spur formation, such as at the site of attachment of the common digital extensor tendon on the distal phalanx or the site of attachment of the middle distal sesamoidean ligament on the palmar aspect of the middle phalanx. At other sites, such as the origin of the suspensory ligament (SL) on the plantar aspect of the third metatarsal bone, new bone formation may be more diffuse. Diffuse new bone formation on the caudal aspect of the occiput reflects tearing of the attachment of the nuchal ligament. New bone on the dorsal aspect of the radial, intermediate, ulnar, or third carpal bones may reflect entheseous new bone at the site of attachment of intercarpal ligaments or joint capsule. Such new bone does not necessarily reflect osteoarthritis, but it may reflect slight joint instability, which itself may predispose to the development of osteoarthritis. Similarly, joint trauma resulting in sprain of periarticular ligaments and subsequent entheseous new bone may ultimately also result in osteoarthritis.

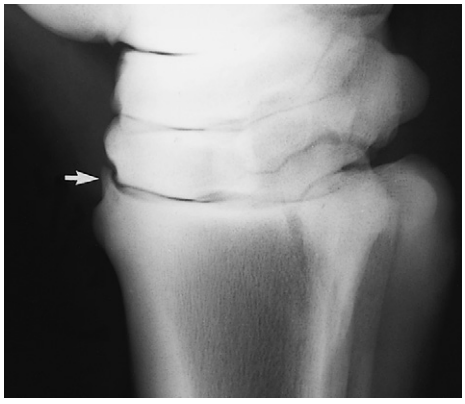

Fig. 15-4 Lateromedial radiographic view of a hock. There is a large periarticular osteophyte on the dorsoproximal aspect of the third metatarsal bone, close to and traversing the tarsometatarsal joint *(arrow)*.

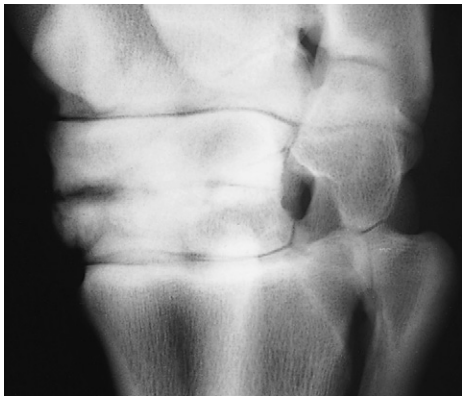

Fig. 15-5 Dorsolateral-plantaromedial oblique radiographic view of a hock. There is narrowing of the centrodistal joint space. Lytic regions are seen in the subchondral bone of the central and third tarsal bones dorsomedially. There is loss of trabecular architecture due to medullary sclerosis in the central and third tarsal bones.

Fracture of an entheseophyte and mineralization within the tendon or ligament attachment also may occur. A relatively common site is the radial tuberosity at the attachment of biceps brachii. Small linear opacities may be seen dorsal to the summits of the spinous processes in the thoracic region, which is associated with tearing of the attachment of the supraspinous ligament.

Spondylosis

Ossifying spondylosis occurs in the caudal half of the thoracic region in the horse. Spondyles (osteophytes) arise from the ventral aspect of the vertebral bodies near the intercentral articulations. The osteophytes extend across the intercentral articulation toward similar osteophytes on adjacent vertebrae. Usually a lucent line persists between the two spondyles, although sometimes complete bridging does occur. Spondylosis may be progressive in cranial and caudal directions, although in some horses it remains static.

New Bone of Unknown Origin

New bone sometimes develops on the dorsal aspect of the diaphysis and distal metaphyseal region of the middle phalanx. The cause of this is unknown. It may be asymptomatic.

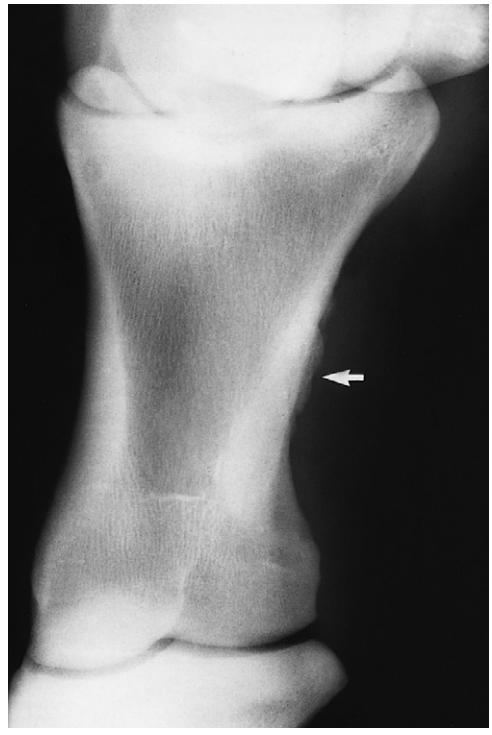

Fig. 15-6 Dorsolateral-palmaromedial oblique radiographic view of a pastern. There is entheseous new bone *(arrow)* on the mid-diaphyseal region of the proximal phalanx at the site of attachment of the middle (oblique) distal sesamoidean ligament.

Hypertrophic Osteopathy

Hypertrophic osteopathy (Marie's disease) is typified by palisading periosteal new bone, which appears to be perpendicular to the cortices and irregular in outline in the acute stage. It affects principally the diaphyseal and metaphyseal regions of long bones and spares the joints. In the early stages, very soft exposures must be used to avoid overexposing this relatively radiolucent bone. Later the margins of the new bone become more opaque and smoother and the distinction between the original cortex and the new bone becomes less obvious. These bony lesions develop secondary to a tumor, abscess, or other lesion in the thorax or abdomen, or in association with diffuse granulomatous disease. The bony lesions may regress and model if the primary lesion can be identified and successfully treated.

Osteitis

Osteitis is inflammation of bone. It may be non-infectious or infectious. Non-infectious osteitis usually is the result of trauma or inflammation of the adjacent soft tissues. It is characterized by new bone formation and, less commonly, bone resorption.

Infectious Osteitis and Osteomyelitis

Infectious osteitis is inflammation of bone as a result of infection. In bones with a myeloid cavity the term *osteomyelitis* is used if the myeloid cavity is affected. Infection results in soft tissue swelling, new bone formation, and bone resorption. In the distal phalanx and the distal sesamoid (navicular) bone, bone lysis predominates with little new bone formation. In other bones a combination of loss of bone and new bone formation usually occurs. A piece of dead radiopaque bone, or a *sequestrum*, may develop, surrounded by an area of lucent granulation tissue, the *involucrum* (Fig. 15-7). An area of more radiopaque bone may be laid down surrounding the infected area to wall off infection. A radiolucent tract, a *sinus,* may

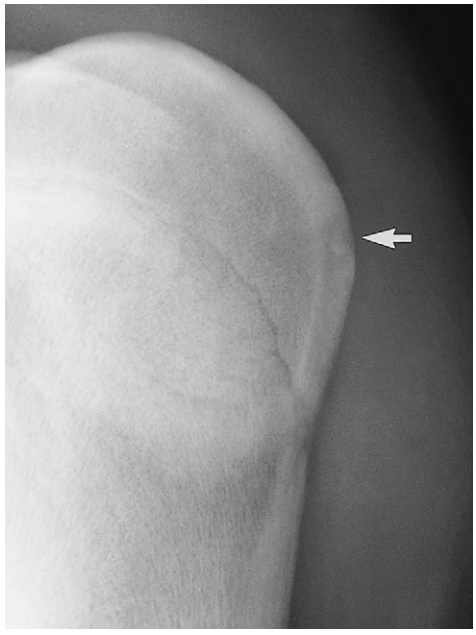

Fig. 15-7 Dorsolateral-plantaromedial oblique view of the tuber calcanei. There is a small radiolucent region on the plantarolateral aspect with a radiopaque center *(arrow)*. These are an involucrum and sequestrum caused by infectious osteitis.

develop between the infected tissues and the skin. In the early stages, diagnostic ultrasonography may be more sensitive than radiography for detecting infection.

Osseous Cyst-Like Lesions and Subchondral Bone Cysts

Osseous cyst-like lesions are solitary, circular, or semicircular lucent areas in a bone; they usually develop in the subchondral bone. Sometimes a neck can be identified connecting the cyst to the joint cavity. Cyst-like lesions may be unicameral (single chambered) or, less commonly, multicameral. A sclerotic rim frequently surrounds osseous cyst-like lesions. Those that develop before skeletal maturity often appear to migrate away from the joint surface as normal endochondral ossification occurs. These single osseous cyst-like lesions, which are not associated with any pathological changes in the articular cartilage, should be differentiated from the more poorly defined lucent zones that develop in the subchondral bone in osteochondrosis.

The cause of osseous cyst-like lesions is probably multifactorial. Some are true subchondral bone cysts and have a fibrous cystic lining. There is increasing evidence that some of these lesions develop as a response to trauma to the articular cartilage and subchondral bone. They tend to occur most commonly in the medial femoral condyle of the femur and in the center of the glenoid cavity of the scapula. Other osseous cyst-like lesions may have a different histological appearance, despite similar radiographic appearance. Osseous cyst-like lesions in the proximal medial aspect of the radius tend to be associated with periosteal new bone in the region of insertion of the medial collateral ligament. Other common locations include the distal aspect of the third metatarpal bone and the proximal, distal, and middle phalanges.

Care should be taken when evaluating some bones, especially the phalanges, not to confuse the myeloid cavity with an osseous cyst-like lesion.

The development of some osseous cyst-like lesions can be followed radiographically. Some start as a small, elliptical depression in the articular surface that progressively enlarges into the subchondral bone. A sclerotic margin develops.

Osseous cyst-like lesions may be associated with lameness. Lesions deep in the bone, such as those seen in the first, second, and ulnar carpal bones, are rarely associated with lameness. Cyst-like lesions that are close to an articular surface are more likely to be associated with lameness. However, the lameness may be remarkable in its variable nature and severity. Lameness may resolve spontaneously, or after surgical intervention, despite persistence of a radiographic lesion.

The incidence of the development of osseous cyst-like lesions is higher in young horses than older horses. Some horses develop a subchondral bone cyst early in life that remains clinically quiescent, only to cause clinical signs later in life. However, subchondral bone cysts and osseous cyst-like lesions can develop in skeletally mature horses.

It can be difficult to determine the likely clinical significance of a subchondral bone cyst or an osseous cyst-like lesion based purely on radiographic appearance. Scintigraphic evaluation may be helpful, since many are hot, but it can also be confusing because a long-established cyst-like lesion that has been clinically quiescent can suddenly become a source of pain without evidence of active bone modeling. It is always important to evaluate radiographically the joint in its entirety, since evidence of secondary osteoarthritis warrants a more guarded prognosis.

Osteochondrosis

Osteochondrosis is considered a disorder of endochondral ossification, although there is increasing evidence that primary subchondral bone lesions also may occur. Osteochondrosis can cause osseous cyst-like lesions and osteochondritis dissecans. Osteochondritis dissecans may be generalized although it is only evident clinically and radiographically in certain joints. The femoropatellar, tarsocrural, fetlock, and scapulohumeral joints are most commonly affected. Radiographic abnormalities vary depending on the joint involved and include the following:

- Discrete osteochondral fragments
- Flattening or depression in the articular surface
- Subchondral lucent zones
- Sclerosis in the subchondral bone, surrounding lucent zones, or parallel with the joint surface
- Secondary osteoarthritis

Radiographic changes are not always clinically significant but must be interpreted in the light of clinical signs. Flattening of contour of the lateral trochlear ridge of the femur, with sclerosis of the subchondral bone, may be seen in the absence of lameness in mature horses. However, identical changes may be present in horses with poor hindlimb action referable to pain associated with the femoropatellar joints (Fig. 15-8). Flattening of contour or elliptical depressions in the trochleas of the talus with normal subchondral bone opacity are rarely associated with clinical signs. The more change in the subchondral bone, the more likely that clinical signs will be present. Small osteochondral fragments may become lodged in the synovial membrane and become progressively larger. Trauma may result in such lesions becoming dislodged and mobile and result in clinical signs.

FRACTURE

A *fracture* is a discontinuity of bone that may be seen radiographically as one or more lucent lines. Mach lines or bands should not be confused with a fracture. A *Mach line*, or band, is a radiolucent line created by edge enhancement when one bone edge is superimposed over another bone. Superimposition of the second and fourth metacarpal bones on the third metacarpal bone commonly results in Mach lines. In a caudocranial view of the proximal tibia, a Mach line is created by superimposition of the tibial crest on the tibia.

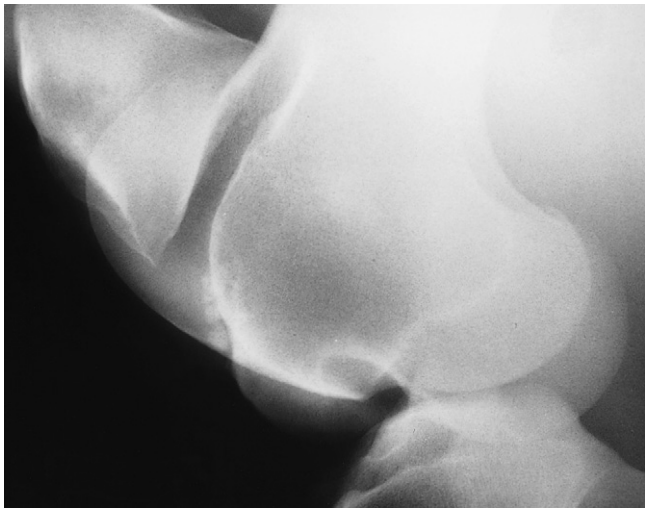

Fig. 15-8 Caudolateral-craniomedial oblique view of a stifle of a 5-year-old Thoroughbred with mild lameness associated with an effusion of the femoropatellar joint. A concave depression is seen in the middle of the lateral trochlea of the femur, with subchondral sclerosis and ill-defined lucent areas. In addition, several radiopaque bodies are visible in the concave depression. This is osteochondrosis.

Large exostoses involving the second or fourth metacarpal bone or fourth metacarpal or fourth metatarsal bone often result in confusing lucent lines, apparently within the bone, which must not be interpreted as fractures. Likewise, some bony exostoses in these locations incorporate some fibrous tissue, resulting in a persistent lucent line that should not confused with a fracture. A nutrient foramen that traverses a bone should also not be interpreted as fracture. Most nutrient foramina are broader than a recent fracture would be expected to be. A notable exception is the vertically orientated nutrient foramen in the third metacarpal or second metatarsal bone and the fourth metacarpal or fourth metatarsal bone. It is important not to misinterpret lucent lines in the foot created by the frog clefts as fracture lines. Despite careful packing of the frog clefts and sulcus (with, for example, Play-Doh; Hasbro, Inc., Pawtucket, RI), complete packing is impossible if these are very deep and will result in narrow lucent lines. Care should be taken to see if such a lucent line (e.g., apparently in the navicular bone) extends beyond the bone margins. If the angle of projection is altered slightly, does the position of the line change relative to the medial and lateral borders of the bone? Can the same lucent line be seen in a dorsopalmar projection? Care must also be taken not to confuse the lucent bands between separate centers of ossification with fracture lines. The cartilages of the foot and the fibula frequently develop as separate centers of ossification and bony union may never develop between the different ossification centers, which results in lucent gaps within each bone that persist over the long term. A long, thick hair coat that is wet sometimes can result in the appearance of a lucent line or lines superimposed over bone, which must not be interpreted as a fracture.

Some stress or fatigue fractures are never detectable radiographically. Some can be seen as one or more lucent lines, or if chronic, there may be endosteal new bone or medullary sclerosis. If a chronic stress fracture traverses a cortex, there may be periosteal callus, although the fracture line itself may not be detectable.

Ideally, the postulated radiolucent fracture line should be detectable on more than one radiographic projection to confirm the presence of a fracture. However, this is not always possible, especially if the fracture is incomplete and non-displaced. This is particularly true for fractures of the distal phalanx and incomplete dorsal cortical fractures of the proximal phalanx. For best results, the x-ray beam must be perpendicular to the plane of the fracture line. Many different views that differ by only 5° in angle of projection may be required to detect a fissure fracture. With a complete fracture, many different views may be required to determine the precise configuration of the fracture.

Some fractures are not detectable in the standard radiographic projections of the area under examination. A fracture of the lateral palmar process of the distal phalanx may not be detectable in lateromedial, dorsopalmar, and dorsoproximal-palmarodistal oblique views. Dorsolateral-palmaromedial oblique or sometimes a palmaroproximal-palmarodistal oblique or palmaroproximal-palmarodistal oblique views, or a dorsal 60° proximal 45° lateral-palmarodistal medial oblique view, are required. Sagittal and some slab fractures of the third carpal bone are only visible in dorsoproximal-dorsodistal oblique views of the flexed carpus (the skyline projection). Similarly, fractures of the medial pole of the patella are only detectable in cranioproximal-craniodistal oblique views of the flexed stifle.

The normal healing process of a fracture involves osteoclasis along the fracture line within 5 to 10 days, resulting in initial broadening of the fracture line. Therefore if a fracture is suspected but cannot be seen in the acute stage, radiography should be repeated after 5 to 10 days. Care should be taken not to underexpose the radiograph, since a faint lucent line may be missed. Paradoxically, early callus formation, which develops within 14 to 21 days, will be missed if the radiograph is overexposed or not viewed over high-intensity illumination.

A fracture should be evaluated to determine whether it is complete or incomplete, simple or comminuted, whether fracture fragments are displaced, presence of articular involvement, and whether any other concurrent pathological condition is present that may influence the prognosis. An apical sesamoid fracture may be accompanied by severe desmitis of the branch of the SL, which prognostically is the more severe injury. Biaxial transverse fractures of the proximal sesamoid bones result in disruption of the suspensory apparatus and severe risk of compromising the blood supply to the distal part of the limb. An avulsion fracture of the lateral collateral ligament of the metacarpophalangeal joint from the proximal phalanx will result in loss of joint stability.

It is important to be aware that with a single, complete fracture, two lucent lines representing the fracture traversing, for example, the dorsal and palmar cortices, may be seen. These lines should not be confused with two fractures. However, care must always be taken to make sure that there is not more than a single fracture line. For example, a common fracture in steeplechasers that fall is a vertical fracture through the body of the accessory carpal bone. These horses have a good prognosis with conservative management. Rarely, one or more chip fractures involving the articular margins of the bone occur; such injuries warrant a much more guarded prognosis. An event horse that hits a fixed fence during cross-country jumping may fracture the medial trochlear ridge of the femur. Fracture fragments may readily be seen in a lateromedial projection of the stifle. Such fractures also can be accompanied by a fracture of the medial pole of the patella, which can be seen only in a skyline projection. Therefore it is essential to obtain a complete radiographic series and interpret each radiograph in its entirety.

Intra-articular fractures occur when a break in the articular surface occurs. Unless some degree of displacement is present, damage to the articular cartilage may not be seen but is assumed to exist. A small degree of displacement is indicated

by the presence of a slight step in the two sides of the articular portion of the fracture line. Comminution at the joint surface sometimes occurs. This may not be evident in standard radiographic projections. For example, lateral condylar fractures of the third metacarpal bone may be accompanied by a Y fragment at the articular margin that is detectable only in dorsodistal-palmaroproximal oblique projections.

Fractures on an articular margin are called *chip fractures*. In some locations, such as the antebrachiocarpal and middle carpal joints, it is important to assess the radiographs carefully for evidence of pre-existing osteoarthritis, which predisposed to the fracture and may influence the prognosis. The veterinarian should bear in mind that more than one chip may exist.

Chip fractures of the articular margins should be differentiated where possible from separate centers of ossification, avulsion fractures that occurred in the neonatal period, and ectopic mineralization. The position of the mineralized body relative to the articular margin, the size and shape of the body, and the contour of the articular margin should all be assessed carefully. A recent chip fracture may have a sharp edge and a detectable fracture bed from which it originated may be seen. Separate centers of ossification usually are well rounded and uniformly opaque with no discernible fracture bed. It is important to recognize that such pieces may be clinically silent, but some are associated with lameness. The significance must be interpreted in the light of clinical signs and the response to local analgesic techniques. Ectopic mineralization may be present within the joint capsule.

Osteochondral fragments, the result of osteochondritis dissecans, should not be confused with a fracture. Osseous fragments may be seen, for instance, distal to the trochleas of the talus at the entrance to the talocalcaneal-centroquatral (proximal intertarsal) joint. These fragments may have originated from the distal intermediate ridge of the tibia and usually are of no clinical significance. Well-rounded, smoothly marginated osseous opacities may be seen on the proximoplantar aspect of the proximal phalanx. These may be the result of avulsion fractures of the short, deep cruciate sesamoidean ligaments that were sustained early in life; they are not always clinically significant but may compromise performance at maximum levels. The variably present first and fifth carpal bones should not be confused as fractures.

In some joints, it may not be possible to determine radiographically the origin of a fracture fragment. For example, fragments in the femorotibial joint frequently are not associated with any detectable disruption in bone outlines and only arthroscopic evaluation of the joint allows determination of the origin. It also may not be possible to determine whether an accompanying soft tissue pathological condition exists. A fracture of the medial intercondylar eminence of the tibia may occur alone. The prognosis is good with surgical removal. Sometimes, however, concurrent cranial cruciate ligament damage exists at its insertion cranial to the eminence, resulting in a much more guarded prognosis.

A *slab fracture* is a fracture that extends from one joint surface to another (e.g., from the proximal to distal aspect of the third carpal or tarsal bones). These fractures can be extremely difficult to detect in the acute stage if not displaced.

In some locations the origin of a fracture fragment is facilitated by ultrasonographic examination. For example, a fragment on the palmar aspect of the fetlock may be an avulsion fracture of the insertion of the palmar annular ligament on the proximal sesamoid bone, and this is readily confirmed ultrasonographically.

Fracture healing depends on many factors, including the age of the horse, its nutritional and metabolic status, the site and stability of the fracture, the presence or absence of periosteum or infection, and the blood supply to the bone. After initial mineral resorption along the fracture line and formation of fibrous callus, calcified periosteal and endosteal callus develop. The amount and quality of callus that develops depends on the degree of stability at the fracture site and the presence or absence of infection. If a fracture is stable, either because it is incomplete or because it has been stabilized by internal fixation, healing is predominantly by primary union and endosteal reaction with minimal periosteal callus. Instability results in the development of periosteal callus.

The fracture may become stable long before the fracture line disappears radiographically. Healing may be complete within 6 to 12 weeks, with progressive narrowing and ultimate disappearance of the fracture line. However, healing of some fractures takes considerably longer. A horse may be sound and able to withstand full work despite the persistence of a radiolucent fracture line. In some locations (e.g., lateral condylar fractures of the third metacarpal bone) a persistent lucent line may be associated with recurrent and persistent pain.

Some bones tend to heal by fibrous union, resulting in a persistent lucent line. These include the accessory carpal bone, the proximal and distal sesamoid bones, and the distal phalanx. Unless a fracture of the navicular bone is stabilized by internal fixation, lucent areas tend to develop in the adjacent bone along the fracture line. These indicate a chronic fracture of the navicular bone of at least 6 to 8 weeks' duration.

If a fracture line persists beyond 6 months, it can be considered to be a delayed union. Sclerosis of the bone adjacent to the fracture line may be present, and the ends of the bone may be slightly flared. Delayed union is not uncommon, but non-union is unusual in the horse except in those bones that tend to heal by fibrous union.

Aging of a fracture is not easy to determine radiographically with any accuracy. The presence of periosteal callus (Fig. 15-9) indicates a fracture of at least 14 days' duration and often substantially longer. An acute fracture has very clearly defined margins, which become less distinct as resorption occurs along the fracture line during early healing.

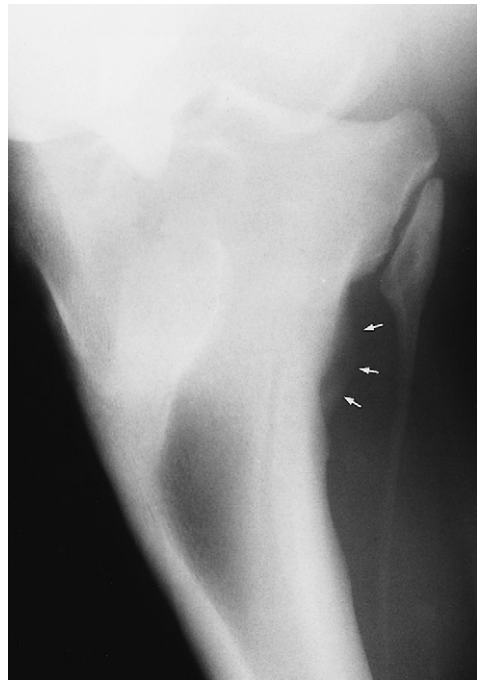

Fig. 15-9 Craniolateral-caudomedial oblique view of the proximal tibia of a 3-year-old Thoroughbred filly. Periosteal callus formation *(arrows)* on the caudoproximal aspect of the tibia is associated with a stress fracture. The oblique lucent line in the proximal tibia is a nutrient foramen.

DEGENERATIVE JOINT DISEASE: OSTEOARTHROSIS OR OSTEOARTHRITIS

Arthritis means inflammation of a joint. *Osteoarthritis* or *osteoarthrosis* indicates that bone has become involved and that a soft tissue component may (-itis) or may not (-osis) be present. The term *secondary joint disease* sometimes is used to denote that the degenerative changes are secondary to a known condition, such as infection or osteochondrosis. Any condition that damages cartilage, causes joint instability, or places abnormal forces on the joint may result in osteoarthritis. It may develop as a result of abnormal conformation or be the result of wear and tear on an athletic horse. However, advanced osteoarthritis sometimes is seen in relatively young horses with no identifiable predisposing cause.

Radiographic abnormalities associated with osteoarthritis include the following:

- Periarticular osteophyte formation
- Narrowing of the joint space
- Subchondral lucent zones, either well or poorly defined
- Subchondral bone sclerosis; loss of trabecular pattern
- Thickening of the subchondral bone plate
- Joint capsule distention

However, it is important to recognize that in some joints advanced osteoarthritis may be present without any detectable radiographic change. Periarticular osteophytes are not necessarily synonymous with clinically significant osteoarthritis. It is relatively unusual to see subchondral lucent zones in high-motion joints. These occur more commonly in the low-motion joints, such as the distal hock and proximal interphalangeal joints.

A relatively poor correlation exists between the degree of radiological change associated with osteoarthritis and the degree of pain and thus lameness. Advanced radiological change may be present when lameness is first recognized, which clearly must have pre-dated the onset of recognizable clinical signs. In contrast, obvious lameness associated with joint pain may be present without detectable radiological change. Widespread wear lines may be present on the articular cartilage without associated radiological change.

Therefore dating the likely onset of clinical signs based on the radiological appearance of a joint can be difficult. It also can be difficult to predict the likely progression of minor radiological changes, such as small periarticular osteophytes, which may be present without clinical signs at, for example, a prepurchase examination.

LUXATION AND SUBLUXATION

Luxation is the complete loss of contact between articular surfaces. *Subluxation* is partial loss of contact between joint surfaces and may be intermittent. Both are usually the result of trauma, although congenital luxation of the patella occurs occasionally. Subluxation of the proximal interphalangeal joint may occur without any obvious cause, especially in hindlimbs. Subluxation is easy to identify radiographically, but the radiographs must be evaluated carefully to identify any concurrent fracture that will adversely influence prognosis.

Subluxation may not be obvious radiographically and stress radiographs obtained with the limb not bearing weight, with pressure applied in a mediolateral or dorsopalmar direction, may be necessary to determine if the bones can be moved relative to each other.

DYSTROPHIC AND METASTATIC MINERALIZATION

Soft tissue mineralization is classified as metastatic or dystrophic. *Metastatic mineralization* is the deposition of mineral in normal tissues and is associated with hypercalcemia, hyperphosphatemia, or hypercalciuria and is unusual in the horse. *Dystrophic mineralization* is the deposition of mineral in injured, degenerating, or necrotic tissue and occurs quite commonly in the horse in, for example, a damaged SL. It can occur secondary to any injury to soft tissue subsequent to infarction, hemorrhage, or inflammation. Dystrophic mineralization may ultimately become ossified.

RADIOGRAPHIC EXAMINATION

For each joint or region to be examined there is a standard radiographic technique that includes a basic minimum number of views for routine evaluation of the region. This usually includes a minimum of four views for joints distal to the elbow and stifle. However, radiographic technique must be flexible. For example, if a fracture is suspected, various views differing only slightly in angle of projection may be required. Some fractures are visible only on special projections. For example, a fracture of the medial pole of the patella may be detected only in a cranioproximal-craniodistal oblique view. Exposure factors need to be altered depending on the size of the horse and the area being evaluated. Exposure factors ideal for assessing trabecular structure within medullary bone result in overexposure of immature periosteal new bone. Underexposure may result in a fracture line or alterations in corticomedullary demarcation and trabecular structure being missed.

It is also important to realize how position-sensitive some radiographic abnormalities are and how easily artifacts can be created. For example, the accurate evaluation of corticomedullary demarcation and trabecular structure within the navicular bone in a palmaroproximal-palmarodistal oblique view depends highly on the position of the limb and the angle of the x-ray beam. The optimum angle of the x-ray beam depends on the shape of the foot. Inappropriate positioning of the foot or using an x-ray beam that is not tangential to the flexor surface of the navicular bone will result in artifacts.

The x-ray beam should be coned down on the area of interest as much as possible and centered on the area of interest. Lesions will be missed if an attempt is made to evaluate too much in a single view (e.g., the fetlock, pastern, and foot). It frequently is helpful to obtain comparative views of the contralateral limb. These should be obtained with identical exposure factors and similar positioning of the limb and angulation of the x-ray beam for accurate evaluation. Comparison of radiographs with a known normal example of similar age can also be helpful. It is thus useful to compile an image library of normal examples.

INTERPRETING RADIOGRAPHS

Radiographs should be evaluated in a systematic way. First, the quality of the radiographs should be assessed. Is the horse positioned appropriately? Are the radiographs of adequate quality? Are there any artifacts? The films should be viewed on a proper viewing box, both close to and from far away. The veterinarian should view the films under both normal and high-intensity illuminations and interpret the entire film, and should follow all the bone margins and then evaluate the internal architecture. The veterinarian should avoid lesion or disease spotting but aim to describe the radiological abnormalities and then deduce potential causes of the abnormality.

If a lesion is suspected but further information is required, the clinician should consider coning down, altering the exposure factors, slightly changing the angle of projection, or using special views. The radiographs should be compared with normal bone specimens, with an awareness that not all radiographic abnormalities are necessarily of clinical significance.

Knowledge of normal anatomy and normal variations is essential, together with knowledge of the sites of ligament, tendon, and joint capsule insertions.[1] Recognition of breed differences also is important. Radiographic abnormalities must be interpreted in the light of clinical signs. It is also important to recognize that there are discipline differences in the potential significance of some lesions. For example, mild osteoarthritis in the antebrachiocarpal or middle carpal joint may compromise the performance and career of a flat racehorse but be of little significance for a show horse or low-level show jumper. It must also be recognized that it is often not possible to predict the future development of lesions. A horse with a small osteophyte on the dorsoproximal aspect of the third metatarsal bone may look identical in a later year, but a different horse may have developed extensive periarticular osteophytes involving both the centrodistal and tarsometatarsal joints.

Determining the Age of a Lesion

It is often not possible to accurately determine the age of a lesion that is identified radiographically. Periosteal new bone usually takes at least 14 days to be visible radiographically following trauma. Once new bone is well consolidated and smoothly marginated, it is impossible to determine how long it might have been present. A non-displaced fracture may take up to 10 days before it is evident radiographically; loss of clarity of the fracture margins may indicate that it has been present longer. Radiological changes compatible with osteoarthritis can precede the onset of clinical signs and may be relatively advanced when lameness is first recognized. It is not possible to determine when the changes first developed.

RADIOGRAPHIC TECHNIQUE

Standard radiographic projections are outlined in Table 15-1. For detailed descriptions of radiographic technique for all regions of the musculoskeletal system, readers are referred to *Clinical Radiology of the Horse*.[2] Throughout this text, radiographic views are described using the technique advocated by the American College of Veterinary Radiologists.[3]

All radiographs should be permanently labeled photographically on the film, either by use of special tape attached to the cassette when the film is exposed or by a labeling light box system in the darkroom. Labels should include at least the identity of the horse, the date, the limb examined, and medial or lateral when appropriate. The following facts also are preferable on the label: the name of the owner or agent of the horse, the name of the veterinary practice, and the identity of the view (e.g., PaL-DMO). For ease of identification, it can be helpful if the horse's label is always positioned on the lateral aspect of the cassette; if a medial marker was omitted, it is then still possible to differentiate between the medial and lateral sides.

Radiographs are part of the horse's permanent medical and legal record and should always be retained by the veterinary practice for future reference unless the owner of the horse gives permission for the radiographs to be transferred to another veterinary practice. Copy radiographs are easily produced if a client wishes to have his or her own copy.

Digital radiography generally uses imaging plates with a larger exposure latitude compared with traditional film screen combinations. Therefore it is potentially easier to obtain good-quality images with a single exposure. With a computerized system the images can be manipulated to enhance contrast and magnify areas of interest; therefore lesions that might previously have been missed may be more readily identified. With an appropriate archiving system, all images can be stored digitally rather than printed. Digital radiography is not a substitute for good radiographic technique but offers the advantage of providing images that may be of superior quality and can be stored and transmitted electronically.

Table • **15-1**

Standard Radiographic Projections and Suggested Extra Views

REGION	NOTES ON RADIOGRAPHY	NOTES ON RADIOLOGY
Front Feet		
LM	• Center on position of navicular bone • Angle the x-ray beam at a tangent to the bulbs of the heels • Position the foot on a block so that the cassette can be placed lower than the solar surface of the foot • Place a radiodense marker on the dorsal hoof wall with the proximal aspect at the coronary band to determine any possible deviation of the distal phalanx • Soft exposures needed to see remodeling of distal phalanx	• Variable shape of extensor process • Small osseous opacities on the proximodorsal aspect of the distal phalanx may be insignificant • Smoothly outlined depression in sagittal ridge of navicular bone is normal
DPa	• Place foot on a block • Horizontal x-ray beam centered midway between the coronary band and the ground and aligned perpendicular to a tangent to the bulbs of the heels	• Useful for assessing mediolateral balance and joint space width • Mineralization of the cartilages of the foot (sidebone) rarely significant • Mineralization of cartilages may be from separate centeres, resulting in permanent lucent lines that are not fractures

L, Lateral; *M*, medial; *D*, dorsal; *Pa*, palmar.

Table • **15-1**

Standard Radiographic Projections and Suggested Extra Views—cont'd

REGION	NOTES ON RADIOGRAPHY	NOTES ON RADIOLOGY
Front Feet—cont'd		
DPr-PaDiO	• Remove shoe and clean thoroughly • Pack the frog clefts • Different exposures needed for distal phalanx and navicular bone • Avoid excessive flexion of the fetlock • Dorsal hoof wall should be just in front of the vertical	• Variable shape and size of crena of distal phalanx • Variable smoothness of solar margins of distal phalanx • Entheseophyte formation on proximolateral aspect of navicular bone a common finding in normal horses • Seven or less small radiolucent zones along the distal border of navicular bone often normal
Pa 45° Pr-PaDiO	• Remove shoe and clean foot • Angle the x-ray beam according to heel height: if the heels are low, reduce the angle to 35°-40° • Position the foot to be examined behind the contralateral limb • Center the beam between the bulbs of the heel	• Artifacts (e.g., reduced corticomedullary demarcation) easily created by poor technique
Extra views		
Flexed oblique views of interphalangeal joints (D 60° L-PaMO) or of palmar processes of distal phalanx (D 45° L-PaDiO)	• Remove shoe and clean foot • Pack frog clefts	• Useful for detection of modeling changes of articular margins of DIP joint • Essential for diagnosis of some distal phalanx fractures
Hind Feet		
As for front feet, but easier to obtain PlPr-DDiO views of distal phalanx and navicular bone		
Pasterns		
LM		• Entheseous new bone on the palmar aspect of the proximal phalanx is often seen without associated clinical signs • A small spur on the dorsoproximal aspect of the middle phalanx is commonly seen in Warmblood breeds
DPa	• Angle the x-ray beam downward, perpendicular to the pastern	• Best view for evaluation of joint space narrowing
Flexed DL-PaMO and DM-PaLO	• Flexed oblique views open up the proximal interphalangeal joint, allowing much better appreciation of the joint margins than in weight-bearing views	
Fetlocks		
LM or flexed LM	• Flexed LM gives more information about the distal aspect of the sagittal ridge of the third metacarpal bone, supracondylar region, and articular margins of the proximal sesamoid bones • For LM of hindlimb fetlock, angle the x-ray beam from 10° plantar to a tangent to the bulbs of the heel	• Well-rounded osseous fragments may be seen distal to the proximal sesamoid bones, which may be previous avulsions from either the palmar/plantar aspect of the proximal phalanx or from the sesamoid bones

D, Dorsal; *Pr,* proximal; *Pa,* palmar; *Di,* distal; O, oblique; *L,* lateral; *M,* medial; *Pl,* Plantar.

Continued

Table • **15-1**

Standard Radiographic Projections and Suggested Extra Views—cont'd

REGION	NOTES ON RADIOGRAPHY	NOTES ON RADIOLOGY
Fetlocks—cont'd		
DPa or DPl	• Angle the x-ray beam proximodistally at least 10° to avoid superimposition of the proximal sesamoid bones over the joint	• The ergot is seen as a radiopaque area superimposed over the proximal phalanx
D 45° L-PaMO D 45° M-PaLO	• Make sure the horse is standing with the cannon bone vertical, or in front of the vertical, so that the sesamoid bones are not superimposed over the proximal phalanx	• Prominent lucent zones within the proximal sesamoid bones or entheseous new bone may reflect suspensory branch desmitis
Extra views		
L 45° Pr-MDiO	• Highlights the abaxial surface of the medial proximal sesamoid bone	
M 45° Pr-LDiO	• Highlights the abaxial surface of the lateral proximal sesamoid bone	
D 30° Pr 70° L-PaDiMO	• Useful for evaluation of the proximal articular margins and lateral palmar process of the proximal phalanx and projects the lateral proximal sesamoid bone distal to the medial proximal sesamoid bone	
D 30° Pr 70° M-PaDiLO	• Useful for evaluation of the proximal articular margins of the proximal phalanx and medial palmar process of the proximal phalanx and projects the medial proximal sesamoid bone distal to the lateral proximal sesamoid bone	
D 45° Pr 45° L-PaDiMO	• Useful for evaluation of the subchondral bone in the lateral condyle of the third metacarpal bone	
D 45° Pr 45° M-PaDiLO	• Useful for evaluation of the subchondral bone of the medial condyle of the third metacarpal bone	
DPr-DDi (flexed)	• Useful for evaluation of lesions of the subchondral bone of the condyles and sagittal ridge of the third metacarpal bone	
Tangential dorsopalmar views	• Place foot flat on block and angle x-ray beam distoproximally at approximately 125° to the metacarpal region to highlight the palmar aspect of the condyles of the third metacarpal bone • Flexed dorsopalmar view moves proximal sesamoid bone further proximal and useful for detection of axial lesions of the sesamoid bones and some third metacarpal bone condylar fractures	
Metacarpal or Metatarsal Regions		
LM DL-PaMO DM-PaLO DPa	• Select views depending on region of interest • Several similar views with slightly different angles of projection may be useful • Soft exposures needed for evaluation of periosteal new bone	• Variable degree of ossification of interosseous ligament • Nutrient vessels in second, third, and fourth metacarpal bones • Exotoses on second and fourth metacarpal bones common
Carpus		
LM or flexed LM	• Flexed LM opens up the intercarpal joints; the radial carpal bone drops down	• Entheseous new bone on the dorsal aspect of the carpal bones common in horses that have raced
DL-PaMO PaL-DMO		• Lucent zones in the ulnar and second carpal bones are quite common • Presence of first and fifth carpal bones variable

D, Dorsal; *Pr,* proximal; *Pa,* palmar; *Di,* distal; O, oblique; *L,* lateral; *M,* medial; *Pl,* Plantar.

Table • **15-1**

Standard Radiographic Projections and Suggested Extra Views—cont'd

REGION	NOTES ON RADIOGRAPHY	NOTES ON RADIOLOGY
Carpus—cont'd		
Extra views		
Flexed D 85° Pr-DDiO	• Highlights the distal radius	
Flexed D 55° Pr-DDiO	• Highlights the proximal row of carpal bones	
Flexed D 35° Pr-DDiO	• Highlights the distal row of carpal bones	
Radius		
LM CrL-CdMO CrM-CdLO CrCd	• Views dictated by region under investigation	
Elbow		
ML	• Protract the limb to be examined so that the olecranon is cranial to the contralateral pectoral muscles	
CrCd	• Rotate the cassette so that it can be held as high up under the thorax as possible	
Extra views CrM-CdLO		
Shoulder		
ML	• Protract the limb to avoid superimposition of the scapulohumeral joints • Use a grid	
Cr 45° M-CdLO		
Extra views Skyline views of the humeral tubercles		
Hock		
LM	• Center at the level of the centrodistal joint and angle the x-ray beam 10° proximodistally to cut through the centrodistal joint space • Position the limb with the metatarsal region vertical	• The distal aspect of the medial trochlea of the talus has a protuberance that varies greatly in size, shape, and opacity • Small spurs on the dorsoproximal aspect of the third metatarsal bone are common
D 45° L-PlMO D 45° M-PlLO		• Best view for evaluating the distal intermediate ridge of the tibia; small osseous fragments may not be of significance
DPl	• Because of the undulations of the centrodistal joint, a horizontal x-ray beam may not cut through all the joint space and one side may look narrowed; repeat the view angling the x-ray beam 5° proximodistally to determine if narrowing is real	• The best view to assess joint space narrowing
Stifle		
LM or flexed LM	• Flexed LM view gives better ecaluation of the patella and cranioproximal aspect of the tibia	• Slight flattening of the lateral trochlear ridge of the femur in a mature horse may not be of clinical significance
Cd 15° Pr-CrDiO	• Position the limb to be examined slightly caudal to the contralateral limb	• The radiolucent lines between separate centers of ossification of the fibula should not be confused as fractures

D, Dorsal; *Pr,* proximal; *Pa,* palmar; *Di,* distal; *O,* oblique; *L,* lateral; *M,* medial; *Pl,* Plantar.

Continued

Table • **15-1**

Standard Radiographic Projections and Suggested Extra Views—cont'd

REGION	NOTES ON RADIOGRAPHY	NOTES ON RADIOLOGY
Stifle—cont'd		
Extra views		
Flexed CrPr-CrDiO CdL-CrMO	• Essential for identification of some patellar fractures • May provide more information about the trochleas of the femur	
Pelvis		
VD in standing horse	• Provides acceptable images of the ischium, caudal ilium, acetabulum, and femoral head and neck for detection of gross fractures	
VD with horse under general anesthesia		
Cervical Vertebrae		
Lateral views only in standing horse	• Keep the head and neck as straight as possible • Use a grid	
Thoracolumbar Vertebrae		
Lateral views	• Horse should stand squarely bearing weight evenly on all four limbs • Evaluate the dorsal spinous processes, facet joints, and vertebral bodies separately	

Cr, Cranial; *Pr,* proximal; *Di,* distal; *O,* oblique; *Cd,* Caudal; *L,* lateral; *M,* medial; *V,* ventral; *D,* dorsal.

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CHAPTER • 16

Ultrasonographic Evaluation of the Equine Limb: Technique

Norman W. Rantanen, Joan S. Jorgensen, and Ronald L. Genovese

Diagnostic ultrasonography was introduced in the early 1980s as a practical imaging modality to evaluate soft tissue injuries of the equine limb.[1] It continues to be used extensively for evaluation of tendonous and ligamentous structures to identify, confirm, and monitor soft tissue injury (Box 16-1).[2-8] Ultrasonography is now the imaging modality of choice for soft tissue evaluation. However, it is user specific, and diagnostic information relies heavily on adequate equipment, limb preparation, and the scanning skills of the ultrasonographer. This chapter describes terminology and techniques and provides advice about interpretation based on our collective experiences. We describe our systematic approach that includes both qualitative and quantitative ultrasonographic analysis of distal limb injuries. The resulting data can be used to categorize the severity of the injury and compare with subsequent examinations. The state of the art

Table • **15-1**

Standard Radiographic Projections and Suggested Extra Views—cont'd

REGION	NOTES ON RADIOGRAPHY	NOTES ON RADIOLOGY
Stifle—cont'd		
Extra views		
Flexed CrPr-CrDiO CdL-CrMO	• Essential for identification of some patellar fractures • May provide more information about the trochleas of the femur	
Pelvis		
VD in standing horse	• Provides acceptable images of the ischium, caudal ilium, acetabulum, and femoral head and neck for detection of gross fractures	
VD with horse under general anesthesia		
Cervical Vertebrae		
Lateral views only in standing horse	• Keep the head and neck as straight as possible • Use a grid	
Thoracolumbar Vertebrae		
Lateral views	• Horse should stand squarely bearing weight evenly on all four limbs • Evaluate the dorsal spinous processes, facet joints, and vertebral bodies separately	

Cr, Cranial; *Pr,* proximal; *Di,* distal; *O,* oblique; *Cd,* Caudal; *L,* lateral; *M,* medial; *V,* ventral; *D,* dorsal.

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Box • 16-1

Indications for Ultrasonographic Evaluation of Limbs

- Diagnosis of soft tissue injuries, including muscular, vascular, tendon, tendon sheath, ligament, joint capsule, or bursal defects
- Assessment of fluid accumulation
- Evaluation of bony surfaces
- Monitoring of the healing progress
- Monitoring of the effect of training on soft tissue structures, especially tendons and ligaments

is still developing, and changes will evolve to improve the clinician's ability to identify and substantiate clinical findings leading to improved management of soft tissue injury in the horse.

EQUIPMENT

Ultrasound machines used for musculoskeletal imaging require a range of transducer frequencies to examine superficial and deep structures. Soft tissues within 5 to 7 cm of the skin surface should be examined using a transducer with a frequency of at least 7.5 MHz or higher. Tissues within 7 to 15 cm of the skin surface are best evaluated with a 5.0-MHz transducer, whereas deeper tissues require a transducer in the 2.5- to 3.5-MHz range.

Sound is attenuated at 1 dB/cm depth per megahertz. Higher frequencies are attenuated at higher rates, thus limiting penetration. Conversely, lower frequencies, attenuated at lower rates, have greater penetration. The highest frequency possible should be used to obtain maximum resolution.

Claiming that one instrument is the only one to perform musculoskeletal examination is naive at best and totally inaccurate. All state-of-the-art machines have similar focusing capabilities and resolving power. Image appearances made by different machines at the same frequencies have subtle differences; however, resolution is about the same. Images may appear more crisp from transducers with a greater number of elements. Operator proficiency (or lack of it) largely determines image quality.

Large muscle masses or bone surfaces, such as the pelvis, are more effectively scanned with convex, annular array, or sector transducers. The small skin surface imprints and divergent beam are advantageous when imaging tissues at greater depths. However, annular array and sector transducers have focal zones that usually favor the central portion of the beam and have beam divergence, which causes far-field, lateral beam width artifact. Convex array transducers have electronic focusing, and the examiner can control focal zone placement. It is common for sector and annular array transducers to penetrate up to 30 cm, but most convex array transducers are limited to 24 cm.

Linear array transducers are the most popular for tendon and ligament scanning because they facilitate anatomical recognition of structures and evaluation of longitudinal tendon and ligament fiber alignment parallel to the skin surface. Convex array and sector transducers, if used properly, work equally well. However, they are more difficult to use because it is easier to change the beam direction because of the smaller contact area. It is necessary to keep the beam at 90° to the tendon or ligament fibers. Anatomical features may be more difficult to recognize. Standoff pads are essential with any transducer to image the most superficial structures.

Sector and convex array transducers are preferable for use on contoured skin surfaces. For instance, scanning between the heel bulbs is difficult with a flat-face, linear array transducer but simple with convex or sector transducers. It is easier to steer a divergent beam and look around from a smaller contact area compared with the flat-face, linear array transducer's broad contact area and rectangular sound beam.

Dual-frequency scanheads are available from most manufacturers; these scanheads allow operators to change frequencies relative to tissue depths without changing transducers. Newer broadband scanhead technology ensures quality, and there are no disadvantages in using these scanheads for musculoskeletal scanning provided basic ultrasound principles are practiced.

PATIENT PREPARATION

The horse should be adequately restrained, because an anxious, fidgety patient may cause a hurried or inadequate examination, resulting in poor-quality images and interpretive errors. One author (R.L.G.) prefers to sedate most horses that are not entered in drug-tested competition to ensure that the horse stands quietly during the examination. The horse should be held by a person who has responsibility for the safety of the ultrasonographer and the equipment. A calm, well-positioned patient allows a thorough, tension-free evaluation.

Hair, air, scurf, scabs, and dirt induce artifacts and reduce image quality. The hair should be clipped with a No. 40 or No. 50 blade, then cleaned with an antiseptic scrub, followed by a generous rinse with water, taking care to follow the growth pattern of the hair. In some horses an additional close shave with a disposable razor may improve image quality. Acoustic coupling gel is applied to improve transducer contact. Excessive application of gel causes a lateral image artifact that may impair assessment of the structures being examined, especially if peritendonous or peri-ligamentous neural, vascular, and connective tissue structures are being evaluated. Removal of the excess gel from the transducer head or the limb corrects the artifact.

If the hair coat is fine and clipping or shaving is not possible or desired, then the limb can be washed. The examiner should spend some time wetting and soaking the hair to improve transducer contact. Topical application of alcohol while stroking the hair along its growth path may improve transducer contact,[5] but *alcohol can be harmful to some transducers*. The operator should contact the manufacturer of the equipment to ensure that it is safe to use the transducer with alcohol.

MEDICAL RECORDS

Ultrasonographic images should be recorded using thermal prints, video recording, or digital recording. Thermal images are used most commonly.

Thermal Print Storage Envelope

Practical hints and suggestions for medical record documentation that can simplify case records, enhance clinical information for serial studies, and improve documentation are provided in the following text. We recommend a simple form stamped on the outside of a storage envelope. The information on the envelopes can be quickly filled in using symbols or checkmarks and should include the following data: horse identification, date, owner, trainer, limb or limbs examined, and structure or structures evaluated (superficial digital flexor tendon [SDFT], deep digital flexor tendon [DDFT], accessory ligament of the DDFT [ALDDFT], main body of the suspensory ligament [SL], SL branch, pastern, hock, antebrachium,

crus, and other). It is also useful to record exercise level, the reason the examination was performed, and a brief summary of clinical findings. This checklist indicates which structures have been targeted and assists in interpretation of an actual lesion versus an off-incidence angle artifact. This is especially helpful if the images are reassessed at a later time.

We have developed an alphanumerical system for ranking exercise levels (Box 16-2). The current exercise level may have an important impact on diagnosis, treatment, future exercise control, and prognosis. For instance, if a horse at pasture injured a SDFT, advising pasture rest would be contraindicated. A chart for ranking exercise levels can be duplicated easily, laminated, and taped to equipment for easy reference.

Clinical findings may be recorded concisely as follows:

Leg	Which limb.
S	Subcutaneous swelling on a scale of 0 to 5. This does not refer to tendonous or ligamentous enlargement.
L	Lameness on a scale of 0 to 5.
T	Thickening of a tendon or ligament on a scale of 0 to 5. This is independent of subcutaneous swelling.
Sen	Response to digital palpation of a tendon or ligament on a scale of 0 to 5.
H	Heat or skin temperature on a scale of 0 to 5.
TS	Distention of the digital flexor tendon sheath (tenosynovitis) on a scale of 0 to 5.
AS	Ankle sinking or hyperextension of the metacarpophalangeal joint, either at rest or during movement, on a scale of 0 to 5.
Qualitative diagnosis	Comments on preliminary qualitative ultrasonographic interpretation.
New	Whether this is the first time this horse has been examined ultrasonographically for this complaint.
Re-Ck	A recheck of a previous injury.
Normal leg	This indicates which is the clinically normal limb, which may not be normal ultrasonographically. It is strongly recommended that both limbs be examined routinely.
Both abn	Both limbs are clinically abnormal.

Image Labeling

All ultrasonographic images should be labeled with the date of examination, the horse's name, the owner's or trainer's name, the limb being examined, and the location of the image. In addition, it is helpful to include the age, breed, use of the horse, and the current exercise level. Most machines automatically record the frequency of the transducer and the focal zone.

Box • 16-2

Exercise Level Grading Scale

Exercise Level	*Type of Exercise Allowed*
0	Complete stall rest.
1A	Hand walk for 15 to 30 minutes once a day.
1B:	Hand walk ≥30 minutes a day or walk on a mechanical walker. We believe horses usually are more active walking on a mechanical walker than in hand.
2A	Thoroughbred (TB) and Standardbred (STB) racehorse, event horse (EV), and sport horse (SH): exercise level- 1A/B plus trotting *in hand* for 5 to 10 minutes a day.
2B	TB racehorse, SH, and EV: trot under saddle 10 to 15 minutes once a day or swim. STB racehorses: walk *only* in the bike or swim. This level also includes 10 to 15 minutes of trotting on a treadmill.
3A	Small paddock turnout for all horses. Small paddock implies small enough not to be able to work up to a sustained canter or gallop.
3B	Large paddock turnout for all horses.
4A	TB and SH: 15 to 20 minutes a day of walk, trot, and canter under saddle, 3 days per week, plus any of the above levels. This level does not apply to most STBs.
4B	TB: 20 to 30 minutes a day of walk, trot and canter under saddle, 3 to 4 days a week. TB racehorse: "ponying" (being led from another horse) on the racetrack. This level does not apply to most STBs.
5	TB racehorse and EV: all of the above plus normal galloping. STB racehorse: all of the above plus jogging. SH: all of the above plus normal arena flat work with limited low fence jumping where applicable. Dressage horses: normal work minus lateral movements and special gaits.
6	TB racehorse: all of the above plus faster gallops. EV: all of the above plus jumping. STB racehorse: all of the above plus training miles ≥2:10. SH: all of the above plus unlimited low fence jumping where applicable. Dressage: all of the above plus lateral movements and special gaits. Contest horses (reiners, cutters, and so on): all of the above plus practicing specific turns and movements.
7	In essence, this is the maximal work level for any type of athletic horse. TB and EV: racing, fast works, and competing. STB racehorse: training miles <2:10 and racing. SH: showing, jumping, hunting, and so on. Dressage and contest horses: competing.

EXERCISE LEVELS

One of the basic concepts in the management of tendon and ligament injuries is to relate increasing levels of exercise to increases in tendon and ligament loading. For instance, walking a Thoroughbred (TB) racehorse in hand results in far less SDFT loading (stretching) than racing at 35 mph. Understanding the current exercise level is important in interpretation of ultrasonographic information and advising management programs for controlled exercise during rehabilitation from an injury (see Box 16-2).

For example, if a TB racehorse with a SDFT injury of 4 months' duration was re-examined and the SDFT showed little improvement, despite box or stall rest, the veterinarian would conclude that the repair was of poor quality with a poor prognosis for a return to racing. However, if the horse had been turned out for 6 hours a day with other horses that ran around, the delayed healing would be interpreted as being caused by too much tendon loading and the lack of improvement was due to low-level ongoing injury. The client would be advised to reduce the exercise level and would be given a little more positive outlook for future racing soundness.

In the rehabilitation of a tendon or ligament injury, the odd exercise level numbers result in the most salient changes in tendon or ligament loading. Exercise level 1 is limited to walking, resulting in little structure loading, whereas exercise level 3 is a major step up in exercise and structure loading because it allows free movement of the horse. Exercise level 5 signals the return to active training. Finally, exercise level 7 is the ultimate goal of rehabilitation and is the return to maximal athletic use. During rehabilitation the ultrasonographer assesses the current morphological status of a structure and advises changes in exercise levels consistent with those findings. Readily available and definable exercise levels make exercise decisions more efficient and consistent.

SCANNING TECHNIQUE

The basic objective of an ultrasonographic evaluation is to characterize the morphological characteristics of the soft tissue structures and bony surfaces of each designated anatomical area. Although physical examination findings may direct the clinician's attention to a specific structure, a thorough examination of all soft tissue and bone surface structures is imperative. In many instances, lesions in more than one anatomical region may be identified and often result in a completely different recuperation regimen. To avoid off-incidence artifacts, the ultrasound beam must be perpendicular to the target structure. Although more than one structure may be perpendicular to the ultrasound beam simultaneously in either transverse or longitudinal images, often it is necessary to redirect the ultrasound beam to adequately examine all structures at each level. Initially the limb should be systematically examined from proximal to distal to assess the morphological features of the vasculature, peri-ligamentous and peri-tendonous tissue, tendons, and ligaments. Once specific abnormal or normal structures are identified, each tendon or ligament is analyzed systematically by ultrasonographically targeting the transducer to the structure at each zone and simultaneously documenting the scan on tape or print record.

ARTIFACTS

Artifacts cause major problems with any method of imaging, but especially with ultrasonography, because the operator steers the ultrasound beam and sets the instrument parameters. Three primary artifact sources are common with diagnostic ultrasonography: operator error, ultrasound-tissue interaction, and inherent instrument design artifacts. Nothing can be done about the limitations of the imaging system except to recognize the artifacts in special scanning situations. Sound-tissue interaction creates a myriad of artifacts, some of which are useful and help with diagnosis. Others are annoying and cannot be overcome. Readers are referred to several publications that address the majority of artifacts peculiar to ultrasonography.[9-14] Operator error and ultrasound-tissue interactions are discussed further.

Operator Errors

Inadequate Skin Preparation

Excessively long or dirty hair and unclean skin attenuate the ultrasound beam and produce image artifacts. Improper scanhead coupling to the skin caused by a lack of gel or the presence of scabs or scurf is common (Fig. 16-1). Artifacts created by improper skin preparation result from poor contact. Images may be dark even though gain settings may be set at the highest limits. The tendons do not have a fine texture and are more grainy or mottled than normal. Tendon margins may not be seen, and reverberation artifacts caused by trapped air within the hair may appear within images, especially if standoff pads are used. Hypoechoic streaks may be present in the images.

Ultrasound Beam Angle

Reflection of the ultrasound beam is dependent on the sound-interface and tissue-interface geometry. Ideally the ultrasound beam should strike tissue interfaces at 90° to produce the best echo reflection back to the transducer crystals, which also act as receivers. If the beam strikes a tissue interface at a smaller angle, a portion of the ultrasound is reflected away from the primary beam direction, and the interface is not seen as well or at all. This is especially important in the evaluation of tendons and ligaments in the metacarpal and metatarsal regions because the fibers usually are parallel to the skin. If the ultrasound sound beam is not perpendicular to a tendon in a transverse image, information is lost and hypoechogenic areas, which mimic lesions, are created (Fig. 16-2). The problem is not seen in longitudinal images of the metacarpal or metatarsal region obtained using a linear array transducer because its surface is parallel to the fibers. However, with convex array and sector transducers that have divergent ultrasound beams, there are only small areas within longitudinal tendon fiber images in which valid information is found (Fig. 16-3). In the divergent area of the beam, sound is reflected away from the beam path and is lost to the image. It is important not to confuse these

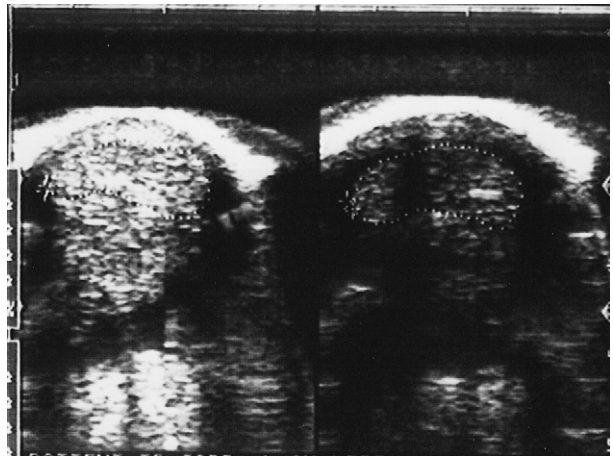


Fig. 16-1 Transverse ultrasonographic images of the palmar metacarpal region. Improper skin preparation causes artifacts within the image on the right.

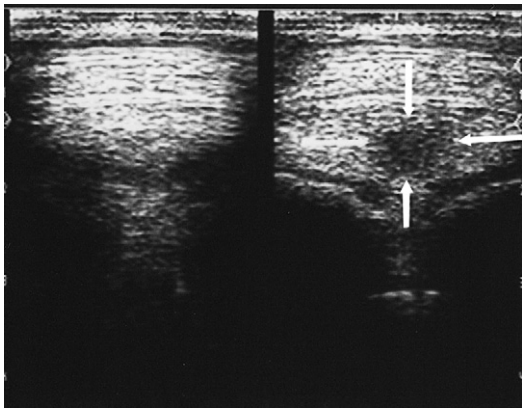


Fig. 16-2 Transverse ultrasonographic images of the distal palmar aspects of the metacarpal region. The left deep digital flexor tendon image has a normal tendon fiber pattern. The right image has an apparent central core artifact *(arrows)* that was produced by slightly changing the angle of the ultrasound beam.

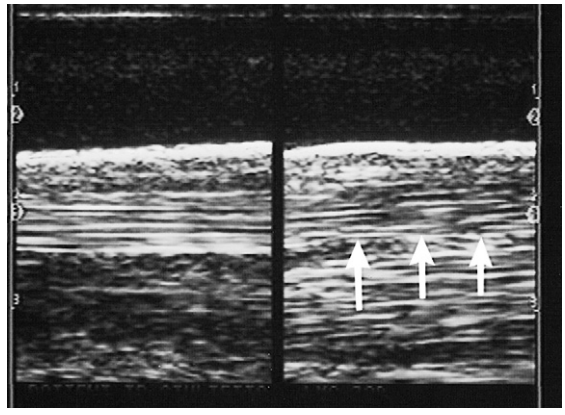


Fig. 16-4 Longitudinal ultrasonographic images of the palmar metacarpal region. The left image was obtained with a linear array transducer parallel to the tendon fibers. The right image was obtained with the transducer slightly oblique to the tendon fibers, causing them to appear as short segments instead of continuous fiber strands *(arrows)*.

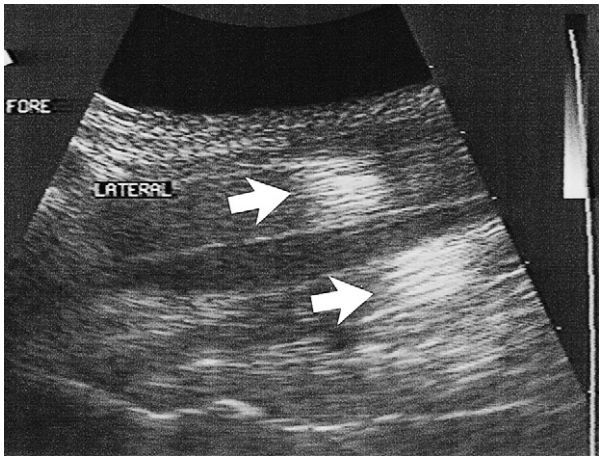


Fig. 16-3 Longitudinal image of the proximal metacarpal region. The *arrows* point to tendon and ligament sections where the ultrasound beam is at 90°, which allows the fibers to be seen. The remaining parts of the image provide no diagnostic information.

areas with pathological conditions of the tendon. The ultrasound beam must be positioned parallel to the tendon fibers. Normal tendon or ligament fibers should be seen as continuous linear echogenic structures across the image (Fig. 16-4). If the transducer is turned slightly, the fibers appear as short linear segments as the beam cuts obliquely across the longitudinal axis. Because fiber alignment is an important criterion to assess in diagnosis and rehabilitation of tendon injury, care must be taken to not create this artifact.

Improper Gain Settings

Ultrasound machines have gain settings referred to as *overall*, *near,* and *far gain*. As ultrasound penetrates normal soft tissues, it is attenuated at the rate of 1 dB/cm of tissue thickness per MHz. Obesity and dehydration cause greater attenuation and can limit penetration. Because energy is lost from the ultrasound beam as it passes into the tissues, the instrument gain must be increased to allow echo detection from the deeper tissue depths. Overall gain changes the brightness and darkness over the entire image. The near-gain adjustment affects the echoes closest to the transducer. Increasing the near gain brightens the echoes and decreasing it darkens them. The far gain affects the deeper image. Some machines display a time gain compensation curve at the side of the screen. Adjustment of the gain settings should produce an image in which the gray scale is similar over the entire image. Bright whites in the near field should prompt decreasing the near gain; conversely, if echoes are difficult to see, increase in the gain is necessary (Fig. 16-5). Improper near-gain settings are the most common error in settings. Visibility of far-field echoes is enhanced by far-gain increases.

Improper Focal Zone Use

Before the advent of moveable and multiple focal zones, transducers had fixed focal points and well-defined focal zones. The ultrasound beam was narrowest in the focal zone in which the lateral resolution produced the best image quality. If the tissues of interest were outside the focal zone, image quality was not ideal. Use of transducers with appropriate focal zones became necessary. If a sector transducer is used to image the SDFT, a standoff pad is necessary to place the tendon in the focal zone. Variable focal zones have eliminated this problem and allow focal zone placement throughout the image depth. Modern linear array and convex array transducers have multiple and moveable focal zones. However, they need to be set in the proper locations to investigate the areas of suspected abnormality. If the focal zones are not placed properly, significant tendon and ligament fiber damage can be overlooked (Fig. 16-6).

Incorrect Frequency Transducer

Tissue depth dictates the optimal transducer frequency. Tissues within 5 to 7 cm of the skin surface should be examined with a 7.5-MHz or higher-frequency transducer. A 5.0-MHz transducer is required to examine tissues from 7 to 12 and up to 15 cm (depending on the instrument), and a 3.0-MHz or lower-frequency transducer is necessary for tissues 15 to 30 cm deep.

Imaging the flexor tendons or SL with a transducer of 5.0 MHz or less results in major compromise of image quality because of lateral beam width artifact. Two types of resolution are peculiar to ultrasound. Resolution along the beam axis, or axial resolution, is frequency dependent; the higher the frequency, the better the axial resolution. Resolution in the transverse plane, or lateral resolution, is dependent on the width of the ultrasound beam; the better the focusing or sound beam narrowing, the better the lateral resolution. Smaller crystals produce narrower sound beams, hence increased lateral resolution. A transducer with a minimum frequency of 7.5 MHz should be used for superficial tendons and ligaments for best axial and lateral resolution.

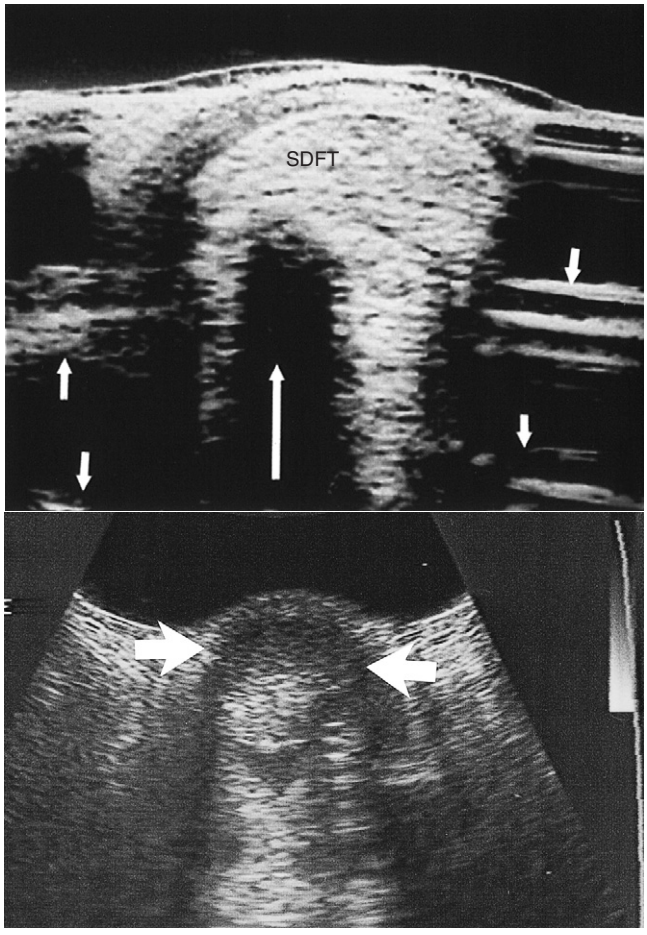


Fig. 16-5 **A,** Transverse sector scan image of the palmar metacarpal region with the near gain set too high. The superficial digital flexor tendon (SDFT) echoes are too bright and cannot be differentiated. Acoustic shadowing *(large arrow)* is caused by flexor tendon and reverberation artifacts caused by the standoff pad *(small arrow)*. **B,** The near gain set too low, so the SDFT cannot be seen *(arrows)*.

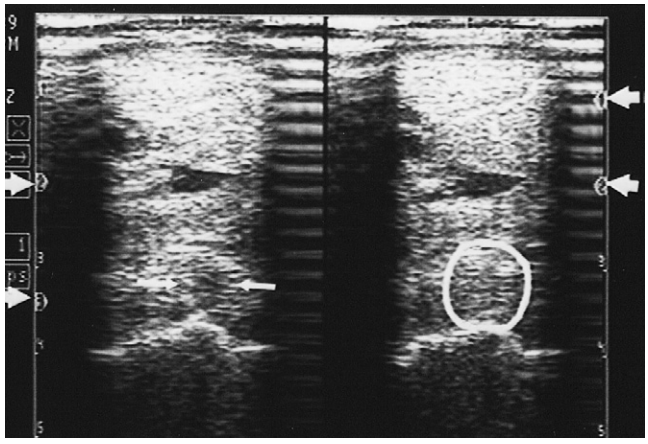


Fig. 16-6 Transverse images of the proximal metacarpal region. A focal lesion in the suspensory ligament *(arrows)* is visible on the left image. The lesion disappears *(circle)* in the right image because the focal zones are improperly placed. *Arrows* in the left and right margins point out the focal zone levels.

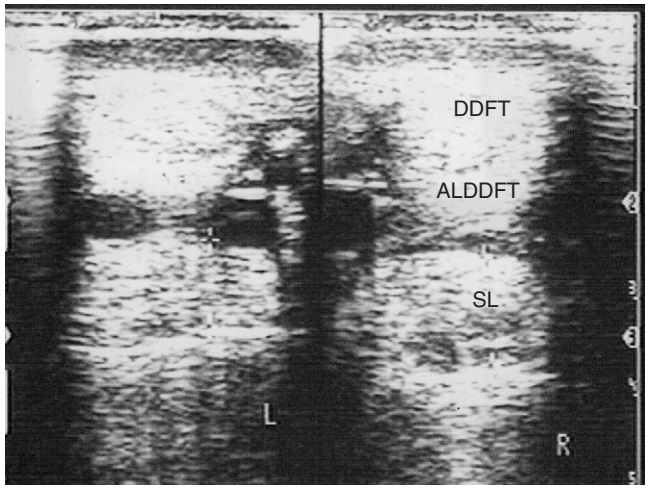


Fig. 16-7 Transverse images of the metacarpal region obtained without a standoff pad showing the deep digital flexor tendon (DDFT), accessory ligament of the accessory ligament of the DDFT, and the suspensory ligament. The brightness is set too high, causing tendon fiber detail loss.

Recording Images

A thermal printer is the most popular method for recording ultrasonographic images, but excellent images can be misrepresented. Brightness may be set too high, causing echoes to have no differentiating characteristics (Fig. 16-7), or set too low, causing the image to be too dark (Fig. 16-8). The same problem occurs with contrast settings; too high causes excessive contrast, and too low causes the image to be washed out and too dark.

A series of dual inline package switches in the back of the printer can change the image and produce a myriad of artifacts. It is important to consult the instruction manual or company representative to ensure that the switches are set properly. Proper settings also are necessary for the paper type. Interpreting improperly recorded (sub-optimal) images causes substantial diagnostic error. Images must be assessed for photographic quality to preclude overlooking lesions or over-interpretation. Sub-optimal images should be repeated to preclude inaccurate interpretation and misdiagnosis. Images should always be frozen before capture.

Recording digital images on floppy disks is an effective way to capture all of the information. Depending on the system, images may be recorded in bitmap (bmp) or Joint Photographic Experts Group (JPEG) formats. These may be archived on a hard drive and may be sent by e-mail if desired. External image capture devices function well, and some instruments have internal floppy drives. Hard copy from the disk can be obtained by using a thermal or other type of printer at a later time if necessary. It must be remembered that the image is an accurate copy of the image created by the operator and contains any artifacts that are due to operator error.

Various video formats, including video home system (VHS), 8 mm, S-Video, cineloop, and videodisk, are used to record motion. Recording static tissue (e.g., tendon) studies while moving the scanhead during recording can produce errors if the transducer is moved too rapidly for the frame rate to keep pace. Tendon examinations should be performed at relatively slow frame rates, as low as 7 per second, if multiple focal zones are activated. Moving the scanhead too fast causes ghosting of the image that blurs the anatomy. Activating multiple focal zones and the resultant slow frame rate can cause a similar problem with minimal scanhead movement.

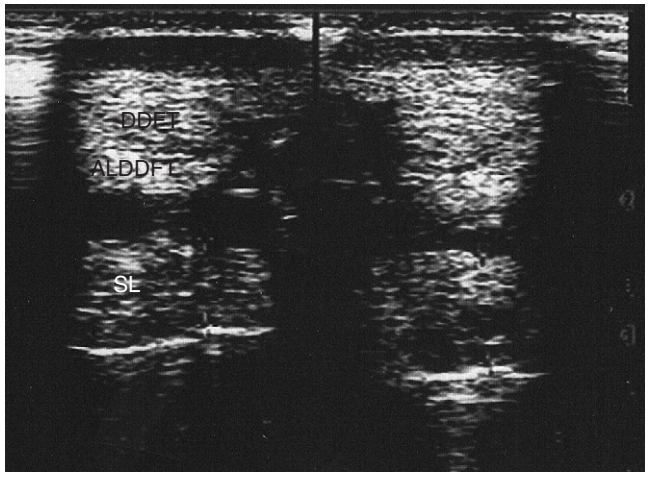


Fig. 16-8 Transverse images of the metacarpal region obtained without a standoff pad showing the deep digital flexor tendon *(DDFT)*, accessory ligament of the accessory ligament of the DDFT *(ALDDFT)*, and the suspensory ligament *(SL)*. The brightness is set too low, causing the image to be too dark.

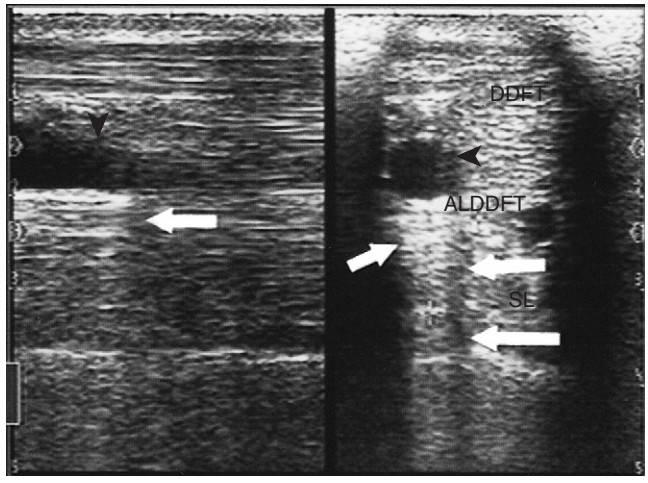


Fig. 16-9 Longitudinal image *(left)* and transverse image *(right)* of the metacarpal region obtained without a standoff pad showing the deep digital flexor tendon *(DDFT)*, accessory ligament of the accessory ligament of the DDFT *(ALDDFT)*, and the suspensory ligament *(SL)*. The tissue brightness *(short arrows)* increases deep to the blood vessels. An anechoic line *(arrow)* extends deep to the blood vessels *(arrowheads)*.

Standoff Pads

Standoff pads are necessary when scanning structures near the skin, such as the SDFT and SL branches. Artifacts are produced each time they are used and can compromise image quality. It is important to recognize them. Transducers with built-in fluid standoff devices are no different. The artifacts are caused by reverberation within the standoff material and occur at a depth equaling the pad thickness (see Fig. 16-5), which may obscure detail. For instance, examination of SL branches with a standoff pad can place artifacts in or near the axial border of the SL. This technique can obscure small axial border tears that are fairly common in sports horses. If the standoff pad thickness produces interfering artifacts, the SL branch should be examined with and without a standoff pad.

Ultrasound Tissue Interaction Artifacts (Patient-Produced)

Ultrasonographic images are composed of returning echoes from many tissue interfaces, regardless of whether they are real or artifact. Some artifacts are useful in diagnosis and provide clues to tissue composition. Others are annoying and do not contribute to the diagnosis. Echoes are generated at tissue interfaces because of differences in acoustic impedance. Acoustic impedance is the product of tissue density (grams per cubic centimeter) and sound propagation velocity (meters per second). Substantial changes in either parameter produce acoustic barriers proportional to the magnitude difference, which causes sound reflection at the interface. Higher (brighter) amplitude echoes are created by greater acoustic impedance differences at tissue interfaces.

Skin Surface Contact

Debris and air trapped in the hair create large acoustic impedance differences and do not allow adequate sound penetration (see Fig. 16-1).

Acoustic Enhancement

Whenever ultrasound passes through non-attenuating tissues, such as fluid with few or no interfaces, a slight increase in ultrasound intensity occurs in addition to less attenuation. The adjacent tissues attenuate the ultrasound normally. This causes brighter (enhanced) echoes deep to the less attenuating tissues. A good example is the enhancement seen deep to the metacarpal and metatarsal blood vessels. The SL has brighter echoes deep to the blood vessels, whereas between the blood vessels it is less echogenic (darker); it is tempting to interpret such results as abnormal (Fig. 16-9). This is an inherent, tissue-produced artifact that can lead to interpretation errors but may help identify less attenuating areas within tissues such as muscle.

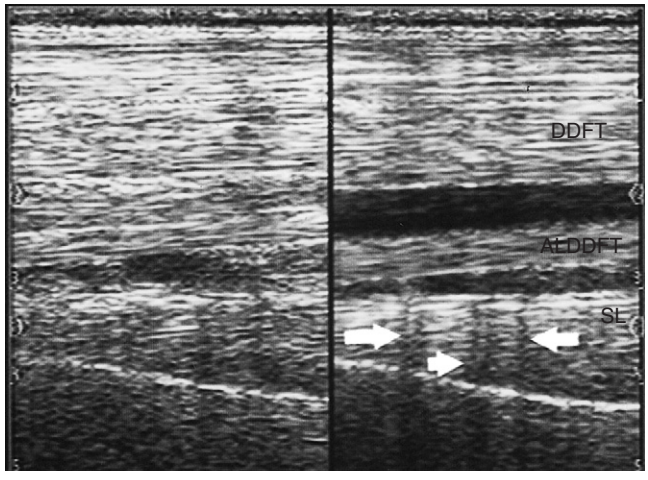


Fig. 16-10 Longitudinal images of the proximal palmar metacarpal region. Proximal is to the left. Refractive scattering *(arrows)* caused by the veins between the origin of the suspensory ligament *(SL)* and the accessory ligament of the deep digital flexor tendon *(ALDDFT)*. *DDFT*, Deep digital flexor tendon.

Refractive Scattering

If the ultrasound beam is not perpendicular to a tissue interface, hypoechoic artifacts are caused by refraction. These artifacts can be a major problem in assessing the origin of the SL because of the anastomotic veins between it and the ALDDFT (Fig. 16-10). The curved blood vessel walls create hypoechoic lines that extend deep to the vessel walls in transverse images (see Fig. 16-9). This is called *refractive scattering* and should not be misinterpreted as a lesion.

Acoustic Shadowing

A high acoustic impedance difference blocks the ultrasound beam and causes an anechogenic shadow deep to the reflecting interface. Bone and other mineralized tissue are much denser than soft tissues, and the sound propagation velocity is much faster. This creates an impenetrable acoustic barrier and, characteristically, the reflector surface has bright echoes and the

deeper tissues cannot be seen. These artifacts are useful because they identify soft tissue mineralization (see Fig. 16-5). Bone surfaces are easily recognized because of shadowing. Dense scar tissue may create incomplete shadowing.

Reverberation

Reflection of ultrasound back and forth to the transducer from high acoustic impedance interfaces produces reverberation artifacts. The classic example is an air-filled lung surface that produces characteristic concentric reverberation echoes. Reverberation artifacts are not a major problem in tendon and ligament scanning; however, they can be important in certain circumstances. Gas production by anaerobic bacteria and air accidentally injected during diagnostic analgesia are two examples in which reverberation can be found in soft tissues. Standoff pads also create reverberation artifacts (see Fig. 16-5).

Mirror Image Artifacts

Mirror image artifacts are not common problems in musculoskeletal ultrasonography and are more common in thoracic and abdominal scanning. They usually are found deep to interfaces that are highly reflective, such as air-filled lung.

TERMINOLOGY AND QUANTITATIVE MEASUREMENTS

The basis of diagnosis is to determine the morphological variation from normal, which is not always easy. The goal is to determine the size, shape, echogenicity, fiber pattern, and surrounding inflammatory reaction of any structure. These findings should be considered carefully in conjunction with clinical impressions and the current athletic use of the horse. Box 16-3 lists parameters used for characterizing tendon and ligament lesions.[15]

Echogenicity

Echogenicity refers to the whiteness or brightness of a structure. Each tendon and ligament has a characteristic echogenic pattern at specific anatomical sites. Lesions vary in echogenicity depending on morphological consistency at the time of the examination. A scoring system can be used to improve objectivity when assessing the severity of an injury or the response to therapy (Box 16-4). Such a system may improve case management and illustrate to the client the changes in echogenicity that correlate with repair of an injury.

The following terms are used to describe ultrasound images:

Isoechoic	The echogenicity of the structure is normal and is scored as 0.
Hypoechoic	The lesion is less than isoechogenic. There are two categories of reduced echogenicity defined under the term hypoechoic. The first is gray or slightly off-white. This is a type 1 hypoechogenic lesion (Fig. 16-11). If the lesion is of mixed black and white tones, it is scored as a type 2 hypoechogenic lesion (Fig. 16-12).
Anechoic	The lesion is mostly black, a type 3 lesion.
Hyperechoic	Denoix[8] grades hyperechogenic lesions as hyperechoic 1, which indicates that the lesion is brighter than isoechoic and represents dense scar tissue without an acoustic shadow. Hyperechoic 2 lesions (see Fig. 16-5) represent soft tissue mineral deposition, which is characterized by casting an acoustic shadow. These echo patterns are uncommon and generally seen in long-standing and repetitively injured tendons and ligaments.

Box • 16-3

Parameters Used for Characterizing Tendon and Ligament Lesions

- Region or location of the lesion
- Length of the lesion
- Alteration in echogenicity
- Pattern of altered echogenicity (i.e., homogeneous, heterogeneous, focal, and diffuse)
- Alteration of fiber pattern in longitudinal images
- Percent of cross-sectional area of tendon injury
- Changes in the character of the lesion over time

Box • 16-4

Type Scores

Score	*Description*
0	Isoechoic
1	Slightly hypoechoic; mostly echoic
2	Mixed echogenicity (50% echoic and 50% anechoic)
3	Mostly anechoic or anechoic

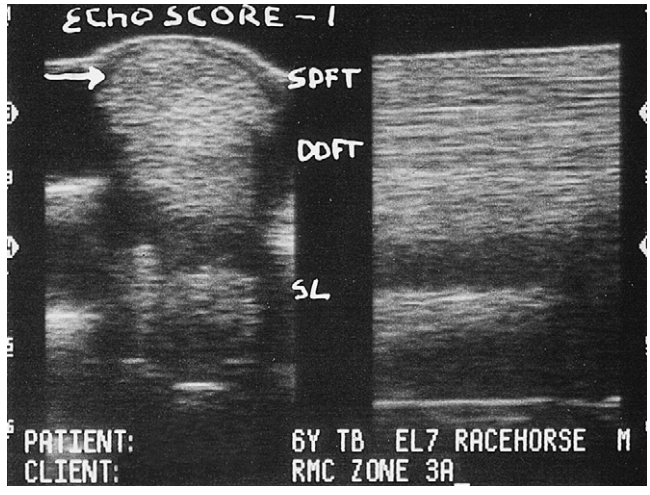


Fig. 16-11 Transverse *(left)* and longitudinal *(right)* images of zone 3A of the metacarpal region obtained using a standoff pad. A type 1 hypoechogenic lesion is present in the superficial digital flexor tendon (SDFT) *(arrow)*. (See text for zone descriptions.) *DDFT,* Deep digital flexor tendon; *SL,* suspensory ligament.

Fiber Alignment Pattern Assessment

Assessment of fiber bundle alignment in longitudinal images determines the nature of a tendon's or ligament's fiber arrangement. Normally the fibers are aligned in parallel except in some anatomical areas, such as the origin of the hindlimb SL. When injury occurs, the expected parallel fiber pattern is disrupted. During healing of an injury, random orientation and cross-linking of new collagen fibers may create scar tissue that results in a substantial improvement in echogenicity but constitutes a poor scaffold for parallel alignment of fibers. Parallel alignment is more physiologically advantageous than random orientation

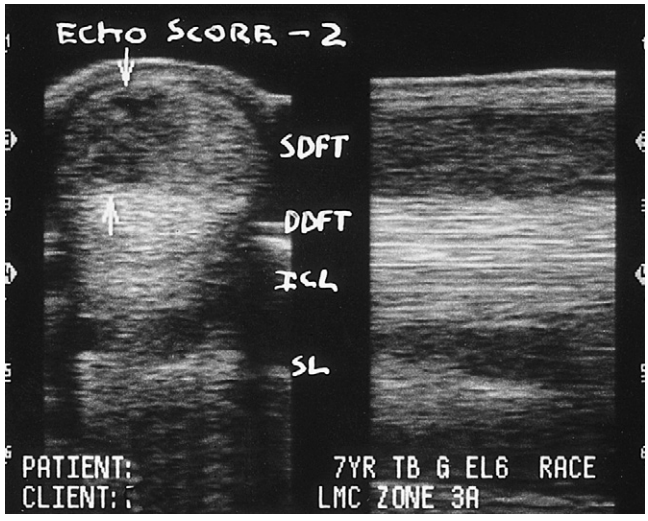


Fig. 16-12 Transverse *(left)* and longitudinal *(right)* images of zone 3A of the metacarpal region. A type 2 hypoechoic lesion is present in the superficial digital flexor tendon *(SDFT; arrows)*. *DDFT*, Deep digital flexor tendon; *ICL*, inferior check ligament (accessory ligament of the deep digital flexor tendon); *SL*, suspensory ligament.

of a scar, because randomly oriented scar tissue is susceptible to re-injury, especially during increased exercise. In a new injury or re-injury, longitudinal images are used to confirm the presence of a lesion identified in transverse images. When monitoring repair, fiber alignment can be assessed.

The longitudinal image must be obtained through the area of fiber bundle compromise to accurately assess the lesion. This is easy if a large proportion of the cross-sectional area is involved. If a lesion is small or located on the margins of a tendon or ligament, it is more difficult. It is important to remember that fiber alignment, *not* echogenicity, is being assessed. In some instances, such as new injuries or re-injuries in the SDFT, the echogenic and fiber alignment scores may be the same. This is rarely true for SL injuries.

An arbitrary scoring system for fiber alignment has been reported (Box 16-5).[6,8] In this system, a score is assigned to semi-quantitate fiber alignment of the target path. The veterinarian should bear in mind that the target path is wide with large lesions, but in small lesions it is quite narrow.

During repair the echogenicity score usually improves at a more rapid rate than the scar remodeling and fiber alignment score. The difference in these scores may be important for tailoring an appropriate controlled exercise program. A delay in fiber alignment improvement suggests that scar remodeling must continue and prompts conservative progression in exercise management.

It is more difficult to identify the target path of fiber bundles during healing once echogenicity has improved. Printed images of the initial examination can be reviewed to identify the exact location of the lesion. If the original scans are not available, one author (R.L.G.) prefers to scan each zone from medial to lateral (or vice versa) and document the longitudinal image with the most abnormal fiber alignment. Fiber alignment score (FAS) (see Box 16-5) has been useful in predicting the prognosis for return to racing.[16] A horse with mean FAS of less than or equal to 0.5 at 4 months after treatment of SDFT was more likely to return to racing than a horse with a mean FAS greater than 0.5. An improvement in total FAS of greater than 75% was associated with a greater chance of returning to racing.[17]

Box • 16-5

Fiber Alignment Scoring

Score	*Definition*
0	Target path ≥75% parallel
1	Target path 50% to 75% parallel
2	Target path 25% to 50% parallel
3	Target path ≤25% parallel

Cross-Sectional Area

Cross-sectional area (CSA) measurements of transverse images of a ligament or tendon are useful, especially in assessing a subtle injury or determining the significance of a localized, focal hypoechogenic fiber bundle path. When assessing the proximal SL, it is useful to determine whether a focal hypoechogenic fiber bundle is likely to be a lesion or the remains of muscle fibers. An injured tendon or ligament usually is enlarged, and although this finding may not be obvious subjectively, measurements of CSA confirm structural thickening.

CSA is also a useful parameter to evaluate the quality of repair, because decreasing size during rehabilitation indicates resolution of the inflammatory component of healing or may indicate favorable scar remodeling. Randomly oriented scars usually form in a rounded fashion and occupy more space. As the scar remodels into a more parallel alignment, less space is necessary, resulting in a reduction in CSA.

The transducer must be perpendicular to the structure to obtain accurate CSA data; otherwise the CSA may be falsely enlarged. The most common technique is to trace the structure on the monitor and the computer software automatically calculates the size. This technique also can be used to calculate lesion size and the proportion of the CSA involved (Fig. 16-13). Initially this method of quantifying CSA may be time consuming, especially when assessing several structures. Eventually the veterinarian can become extremely proficient at producing accurate measurements.

One author (R.L.G.) uses a computer-assisted method that is not commercially available. Transverse images are traced with a microfine-point felt pen after the ultrasonographic study. Using a digitizing pad and software program, surface area data are produced from the tracings of structures and lesions. This method is equally accurate but saves time because scans can be traced more quickly and at a more convenient time. The disadvantage is that the final quantitative information is not immediately available, but it does allow the clinician time to review the images before giving a conclusion to the owner. An alternative method is to save images on a computer disk using commercially available software (Metron-V, EponaTech, Creston, Calif.)

Another simple and inexpensive method to determine structure and lesion CSA is creation of thermal images at all zones, and then at a convenient time, use of handheld calipers to make structures and lesions into two-dimensional squares or rectangles. This is done by measuring the palmar/plantar to dorsal length and the medial to lateral length. These measurements are multiplied to determine the CSA. CSA obtained with handheld calipers is likely larger than the actual size. However, absolute values are less important than the relative difference between normal and suspected abnormal values. Thus if measurements are performed in a consistent manner, handheld calipers provide valid relative CSA data.

Relative sizes are best obtained by comparing the injured limb with the normal contralateral limb, but this is not possible if the injury is bilateral. The veterinarian must rely on the

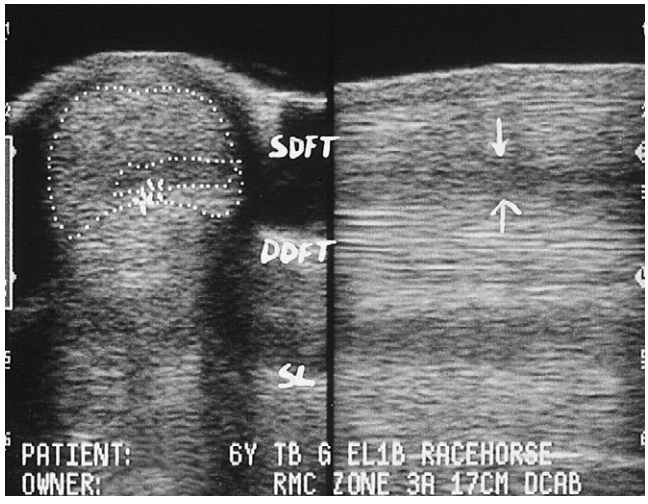


Fig. 16-13 Transverse *(left)* and longitudinal *(right)* images of zone 3A of the metacarpal region. The cross-sectional area (CSA) of the superficial digital flexor tendon *(SDFT)* is 3.05 cm^2. A dorsolateral type 1 hypoechoic lesion is present; the lesion is 43 mm^2 and represents 14.1% of the CSA of the tendon. The fiber alignment score of the targeted fiber path is 3 *(between arrows)*. *DDFT*, Deep digital flexor tendon; *SL*, suspensory ligament.

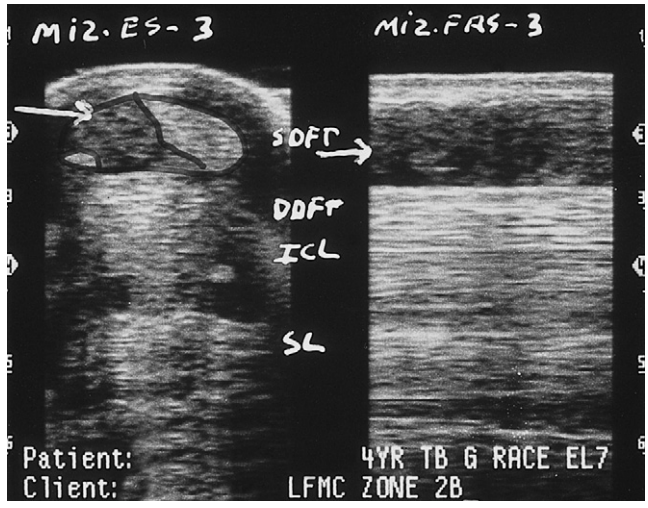


Fig. 16-14 Transverse *(left)* and longitudinal *(right)* images of zone 2B, obtained using a standoff pad. There is a large lateral border type 3 lesion of the superficial digital flexor tendon (SDFT; *horizontal arrow)* confirmed on longitudinal scan *(horizontal arrow)*. *DDFT*, Deep digital flexor tendon; *MIZ-ES*, maximal injury zone echo score; *MIZ-FAS*, maximal injury zone fiber alignment score; *SL* suspensory ligament.

expected values for the age, size, and breed of the horse and level or zone of the image based on personal experience and previous images if availabe. Normal values for various structures are difficult to determine and few studies have been published. Normal values for tendon and ligament sizes for TB and SB horses have been reported.[18] It is essential to measure both limbs at the same depth setting on the ultrasound machine.

In summary, quantifying the ultrasonographic image using echogenicity and fiber alignment scores, along with CSA of the lesion, provide powerful resources to aid in interpreting lesions (Fig. 16-14). When assessing an injury from proximal to distal, these scores can be summed to estimate the volume of the injury. These values can be used to categorize the injury and to monitor repair objectively.

ZONE DESIGNATIONS

The anatomical location of an ultrasonographic image can be designated by measuring the distance from a fixed landmark, such as the accessory carpal bone, or dividing the limb into zones.

Distal Forelimb

The palmar metacarpal and metacarpophalangeal regions may be separated into seven zones, or eight zones if the medial aspect of the carpus is included (Fig. 16-15). The pastern also may be divided into zones (see page 180). Many of these zones have unique anatomical features. The measurements outlined in the following text refer to a 16.1-hand TB horse.

Zone Definitions

Zone 0 Zone 0 (~5 cm in length) is located along the medial aspect of the carpus adjacent to the accessory carpal bone (Fig. 16-16). Off-incidence angle artifacts may be present on the dorsal border of the DDFT, and rarely, hypoechogenic muscle bundles are seen (Fig. 16-17). At this level the SDFT is uniform in echogenicity, but further proximally are hypoechogenic muscle bundles that should not be confused as lesions. The carpal sheath, common digital artery, and palmar carpal fascia are visible.

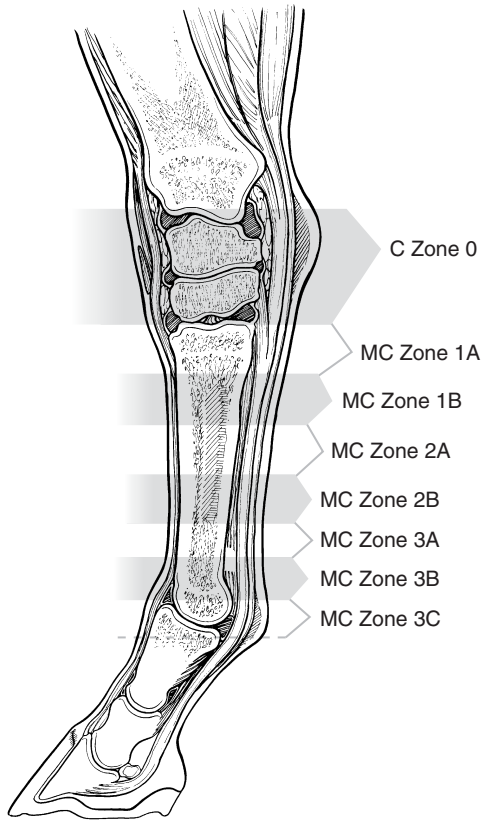


Fig. 16-15 Ultrasonographic zone designations for the carpal *(C)* and metacarpal *(MC)* regions.

Zone 1A Zone 1A extends from approximately 0.5 to 4 cm distal to the base of the accessory carpal bone. The SDFT is slightly medially placed, oval shaped, and readily visible on the midline. The carpal retinaculum covers its palmar border proximally. The common digital artery lies close to the medial

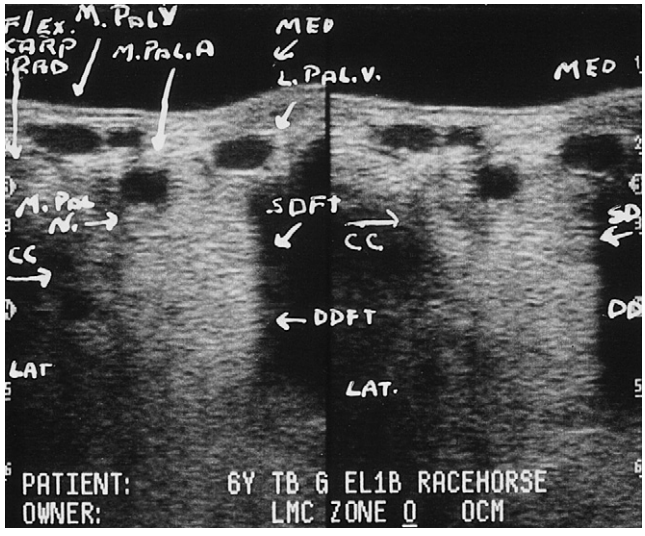


Fig. 16-16 Transverse images of a forelimb zone 0 obtained along the medial aspect of the accessory carpal bone with a standoff pad. The left image is focused on the superficial digital flexor tendon *(SDFT)* and the right image is focused on the deep digital flexor tendon *(DDFT)*. CC, Carpal canal; *Flex Carp Rad*, flexor carpi radialis; *Lat*, lateral; *L Pal V*, lateral palmar vein; *M Pal A*, medial palmar artery; *M Pal N*, medial palmar nerve; *M Pal V*, medial palmar vein; *Med*, medial.

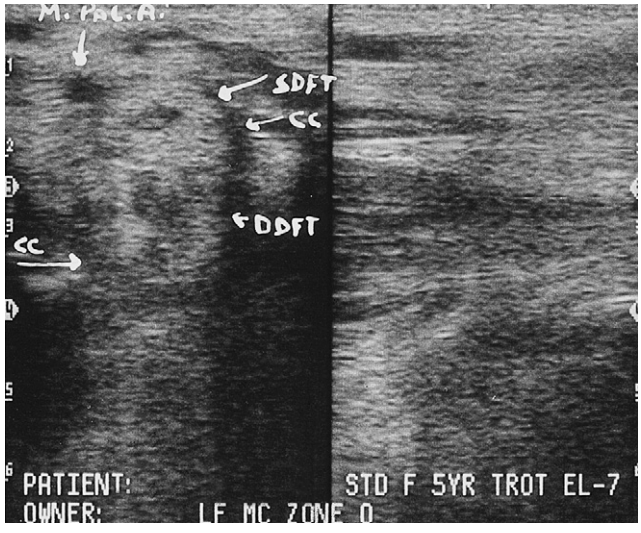


Fig. 16-17 Transverse *(left)* and longitudinal *(right)* images of the forelimb zone 0 obtained from the medial aspect of the carpus, immediately proximal to the accessory carpal bone, without a standoff pad. Note the centrally located muscle bundles of the superficial digital flexor tendon *(SDFT)* and the deep digital flexor tendon *(DDFT)*. This is normal and should not be confused with a lesion. CC, Carpal canal; *M Pal A*, medial palmar artery.

border of the SDFT. The DDFT is triangular, and the lateral border is more rounded (Fig. 16-18). The ALDDFT and SL are rectangular in shape. The medial and lateral borders of the ALDDFT and SL may not be visible from the palmar midline; therefore the transducer should be moved medially and laterally. However, to measure CSA of these structures, the veterinarian should use what can be seen from the palmar aspect. In most horses, anechoic fluid in the carpal sheath is seen between the dorsal border of the DDFT and the palmar

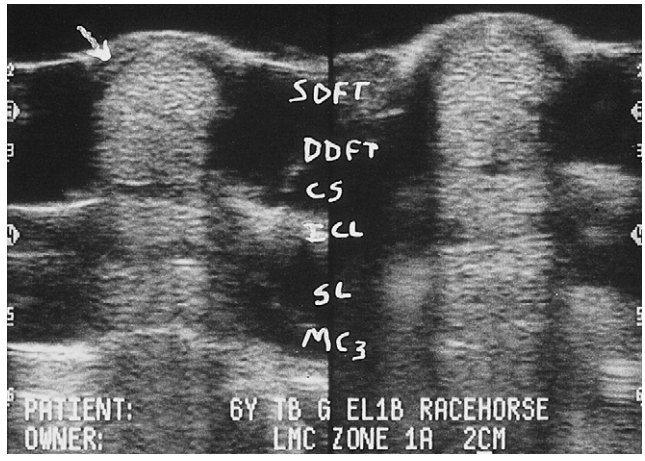


Fig. 16-18 Transverse images of the forelimb zone 1A. Carpal retinaculum *(arrow)*. *CS*, Carpal sheath; *DDFT*, deep digital flexor tendon; *ICL*, accessory ligament of the DDFT; MC_3, palmar border of the third metacarpal bone; *SL*, suspensory ligament; *SDFT*, Superficial digital flexor tendon.

border of its accessory ligament. The medial aspect of the rectangular carpal sheath is wider than the lateral border.

The origin or enthesis of the SL is approximately 3 to 4 cm long. The SL rapidly increases in palmar to dorsal thickness as it descends distally. In contrast, the ALDDFT rapidly decreases in palmar to dorsal thickness. Comparative CSA measurements in each forelimb should be done at precisely the same distance distal to the accessory carpal bone in zone 1A.

In the proximal metacarpal region a ligamentous band often extends proximally from the origin of the SL to insert on the palmar carpal fascia of the distal row of carpal bones (Fig. 16-19). It is located between the dorsal border of the ALDDFT and the palmar recess of the carpometacarpal joint, which lies between the fibrous band and the third metacarpal bone (McIII). Anechoic fluid in the carpometacarpal joint should not be confused with a lesion of the SL. The proximal aspect of zone 1A is proximal to the enthesis of the SL on the McIII.

At 2.5 cm distal to the accessory carpal bone the origin of the SL is firmly attached to the McIII (Fig. 16-20). Entheseal fiber tearing usually occurs 1 to 3 cm distal to the accessory carpal bone. The oblique orientation of the McIII at the origin of the SL attachment can be seen on a longitudinal view (see Fig. 16-20). Fig. 16-21 is at the distal extent of zone 1A and the distal extent of the enthesis of the SL. Note the palmar to dorsal width at this point in zone 1A and the firm bony attachment of the entire dorsal surface of the SL. In this example, there is complete dorsal attachment of the SL. However, in many normal horses the origin of the SL has a bilobed appearance (medial and lateral attachments), with a rounded or triangular-shaped hypoechoic area dorsally in the midline. Clinicians must be cautious not to interpret this as a focal tear. Rarely the origin of the SL can be injured in this central location, but generally size and shape of the hypoechoic region are changed.

Zone 1B Zone 1B extends from approximately 4 to 7 cm distal to the accessory carpal bone. At this level the slightly crescent-shaped SDFT is on the midline over the oval-shaped DDFT. There are no unique anatomical features of this zone that identify it, and its location is determined by measurement. In all mid-metacarpal zones, as in zone 1A, the medial and lateral borders of the SL and ALDDFT may not be visible from the palmar midline. The transducer must be moved medially or laterally to identify a focal peripheral lesion of one of these structures. At times, the dorsal border of the SL can be difficult

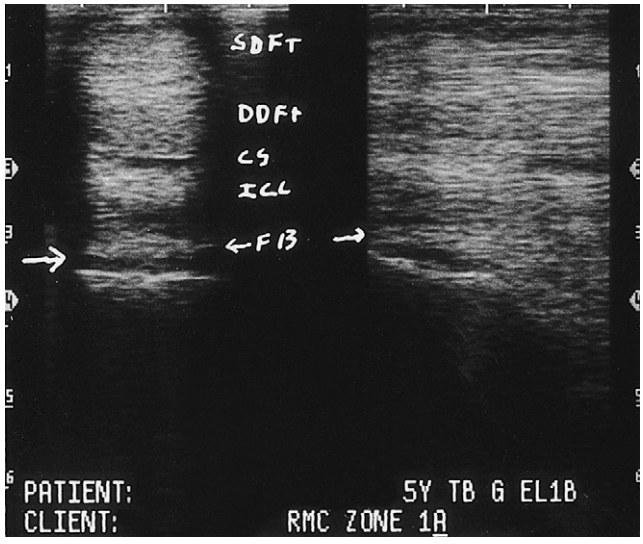


Fig. 16-19 Transverse *(left)* and longitudinal *(right)* (proximal is to the left) images from zone 1A of a forelimb obtained without a standoff pad. There is a ligamentous band (ligamentous segment of origin of suspensory ligament that originates from the palmar carpal fascia *[FB]* and *small arrows)* extending from the palmar aspect of the suspensory ligament, passing palmarly over the anechoic joint recess of the carpometacarpal joint *(large horizontal arrow)* and inserting proximally on the dorsal surface of the accessory ligament of the superficial digital flexor tendon. *CS,* Carpal sheath; *DDFT,* deep digital flexor tendon; *ICL,* accessory ligament of the DDFT; *SDFT,* superficial digital flexor tendon.

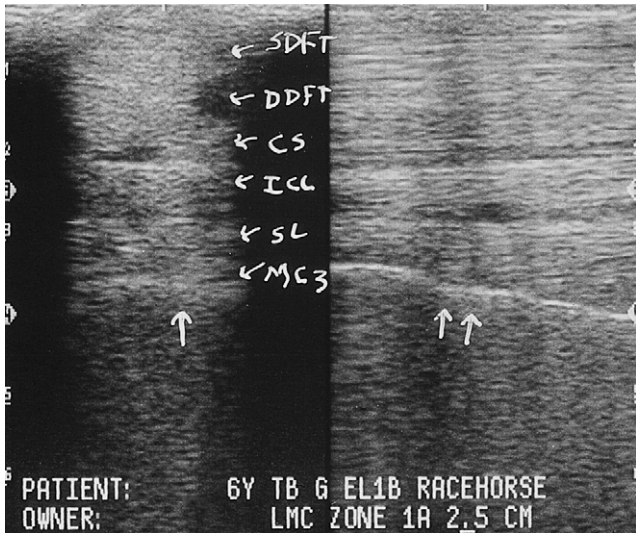


Fig. 16-20 Transverse *(left)* and longitudinal *(right)* (proximal is to the left) images from zone 1A, 2.5 cm distal to the base of the accessory carpal bone, without a standoff pad. There is a firm attachment of the suspensory ligament *(SL)* to the third metacarpal bone *(MC_3)* on the transverse *(single vertical arrow)* and the longitudinal *(double vertical arrows)* views. *CS,* Carpal sheath; *DDFT,* deep digital flexor tendon; *ICL,* accessory ligament of the DDFT; *SDFT,* superficial digital flexor tendon.

to identify because of the density of the periligamentous, fibrous connective tissue. To assist in defining that border, it can be helpful to split the screen and place the longitudinal image adjacent to the transverse image. This technique clearly demarcates the dorsal and palmar borders (Fig. 16-22).

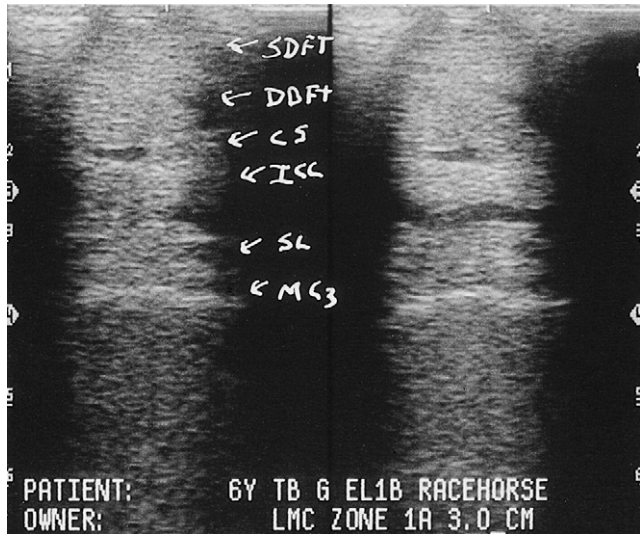


Fig. 16-21 Transverse images at the distal extent of zone 1A (3.0 cm distal to the base of the accessory carpal bone), without a standoff pad. The palmar to dorsal dimension of the suspensory ligament *(SL)* is the greatest for this zone in a normal horse. *CS,* Carpal sheath; *DDFT,* deep digital flexor tendon; *ICL,* accessory ligament of the DDFT, *MC_3,* third metacarpal bone; *SDFT,* superficial digital flexor tendon.

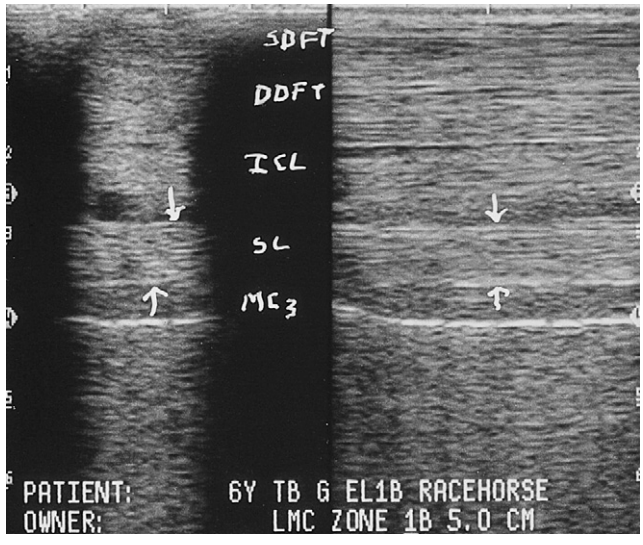


Fig. 16-22 Transverse *(left)* and longitudinal *(right)* (proximal is to the left) images of zone 1B (5 cm distal to the base of the accessory carpal bone) obtained without a standoff pad. The *down arrows* mark the palmar border of the suspensory ligament *(SL)*, and the *up arrows* mark the dorsal border. *DDFT,* Deep digital flexor tendon; *ICL,* accessory ligament of the DDFT, *MC_3,* third metacarpal bone; *SDFT,* superficial digital flexor tendon.

Zone 2A Zone 2A extends from approximately 7 to 10 cm distal to the accessory carpal bone. The same comments apply for this zone as zone 1B.

Zone 2B Zone 2B extends from approximately 10 to 14 cm distal to the accessory carpal bone. One unique feature of this zone is an obliquely coursing nerve located subcutaneously on the palmar surface of the SDFT. This nerve (the communicating branch of the medial palmar nerve) and associated small vessels are a common site for a bandage pinch or "cording" swelling that suggests a primary tendon injury. Overall the

anatomical relationships of tendons and ligaments are similar to zones 1B and 2A. The SDFT and ALDDFT are more crescent shaped and smaller from palmar to dorsal. At this level the carpal sheath still can be identified ultrasonographically.

Zone 3A Zone 3A extends from approximately 14 to 18 cm distal to the accessory carpal bone. Several anatomical features characterize this zone. The first is that in many horses with tenosynovitis, this is the initial zone that incorporates the origin of the digital flexor tendon sheath (DFTS).

The SDFT is crescent shaped and smaller from palmar to dorsal but wider from medial to lateral. The ALDDFT inserts into the DDFT. In transverse images normal crescent-shaped hypoechogenic fiber bundles are seen between the dorsal surface of the DDFT and the palmar surface of its AL. The carpal sheath generally is not appreciated in this zone.

At the region of ALDDFT insertion onto the DDFT, the two structures can clearly be separated ultrasonographically, although grossly they are conjoined. When measuring the CSA of the DDFT, the ALDDFT can be separated from the DDFT, which results in a reduced DDFT CSA, or included with it, which is a more correct CSA determination of the DDFT. The method used is personal preference and results in valid information provided it is consistent.

Zone 3A is just proximal to the bifurcation of the SL. In zone 3A the SL is still a single structure, but the medial and lateral borders become more rounded and the central region may be less echogenic. This is a common site of injury, especially in STB racehorses. At times it can be quite difficult to establish structural borders in acute injury. A longitudinal image viewed adjacent to a transverse may be helpful (Fig. 16-23).

Zone 3B Zone 3B is larger (~5 cm in the average adult horse) than more proximal zones and is the region in which the SL bifurcates into medial and lateral branches. The zone extends from 18 to 23 cm distal to the accessory carpal bone. Anatomical structures include the DFTS; the manica flexoria, a fibrous band extending from the medial and lateral borders of the SDFT and encompassing the DDFT that is associated with a thecal space; and the intersesamoidean space and ligament with associated connective tissue and vasculature. The SDFT decreases in size from palmar to dorsal and widens from medial to lateral. The DDFT becomes ovoid. CSA measurements should be made at identical sites distal to the accessory carpal bone for accurate comparisons between limbs.

The SL branches must be examined individually from palmaromedial and palmarolateral aspects of the limb with a standoff pad. If the ligament is enlarged, no standoff pad is required. The length of a metacarpal SL branch is about 6 cm. To increase data points for CSA comparison, each branch is divided into three, 2-cm sub-zones within zone 3B. For the medial branch, the zone designations are 3B-MP (medial proximal), 3B-MM (medial middle), and 3B-MD (medial distal) (Fig. 16-24). For the lateral branch the zone designations are 3B-LP (lateral proximal), 3B-LM (lateral middle), and 3B-LD (lateral distal). The proximal and middle segments of each branch are oval shaped and the distal segment is more triangular.

Zone 3C Zone 3C extends from 23 to 28 cm distal to the accessory carpal bone and basically includes tissues on the palmar aspect of the metacarpophalangeal joint. The characteristic features of this zone are the presence of the bony shadows of apices of the proximal sesamoid bones (PSBs), the presence of the palmar annular ligament (PAL), and the intersesamoidean ligament (Fig. 16-25).

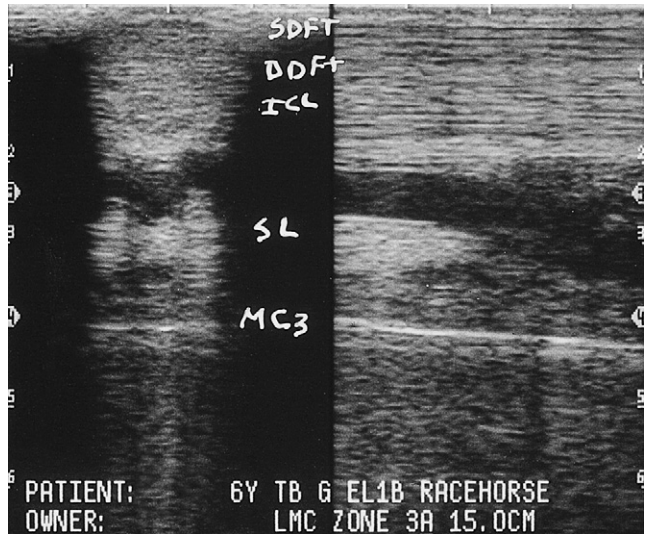


Fig. 16-23 Transverse *(left)* and longitudinal *(right)* (proximal is to the left) images from zone 3A (15 cm distal to the base of the accessory carpal bone), in which the suspensory ligament *(SL)* bifurcates. *DDFT,* Deep digital flexor tendon; *ICL,* accessory ligament of the DDFT, MC_3, third metacarpal bone; *SDFT,* superficial digital flexor tendon.

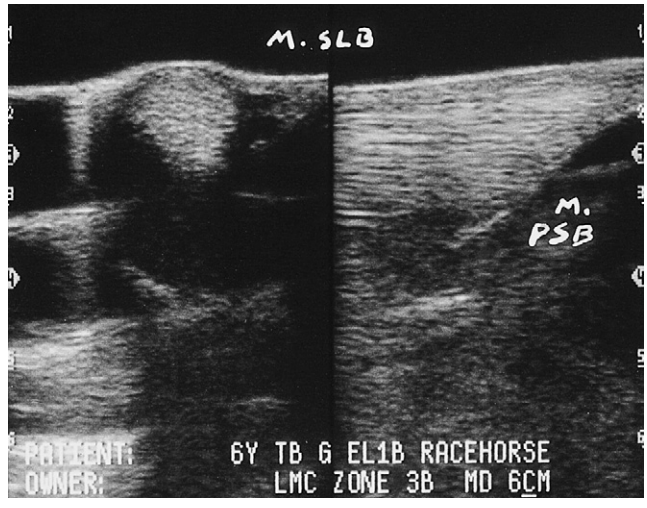


Fig. 16-24 Transverse *(left)* and longitudinal *(right)* (proximal is to the left) images of zone 3B-MD (medial placement of the transducer at the distal extent of the insertion of the medial branch of the suspensory ligament onto the medial proximal sesamoid bone). *M SLB,* Medial suspensory ligament branch; *M PSB,* bone border of the abaxial medial proximal sesamoid bone.

Distal Hindlimb

The anatomical location of an ultrasonographic image can be designated by measuring the distance from a fixed landmark, such as the tuber calcanei or tarsometatarsal joint, or reference to a numbered zone. Most distal limb anatomical features are the same as in the forelimb. However, because of the added length of the SDFT/DDFT and the plantar ligament (long plantar tarsal ligament), the metatarsal zones move up one number.

In the hindlimb, zone 1 includes the plantar aspect of the tarsus. In the adult horse, this is approximately 16 cm in length. Zone 1 is subdivided into two, 8-cm zones, 1A and 1B. The most proximal zone of the metatarsal region is zone 2, which is subdivided into 2A and 2B; the middle one third is zone 3, which is subdivided into zones 3A and 3B; and the distal aspect is zone 4, which is subdivided into zones 4A, 4B, and 4C (Fig. 16-26).

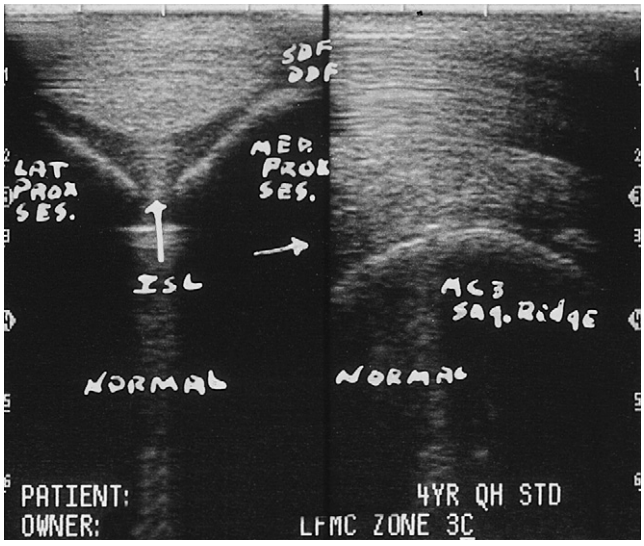


Fig. 16-25 Transverse *(left)* and longitudinal *(right)* images of zone 3C obtained without a standoff pad. Vertical and horizontal *arrows* indicate a normal intersesamoidean ligament. DDF, Deep digital flexor tendon; *ISL*, intersesamoidean ligament; *Lat Prox Ses*, lateral proximal sesamoid bone osseous shadow; *Med Prox Ses*, medial proximal seasamoid bone shadow; *MC_3 Sag ridge*, sagittal ridge of third metacarpal bone.

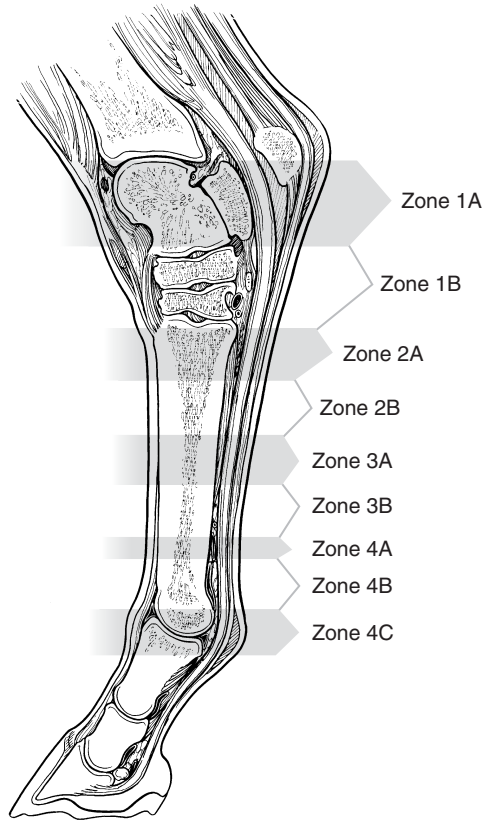


Fig. 16-26 Ultrasonographic zone designations for tarsus/metatarsus.

Hindlimb Zones

Zone 1A This zone extends to 8 cm distal to the tuber calcanei (Fig. 16-27). The SDFT is slightly crescent shaped and located subcutaneously. Deep to the SDFT is the origin of the plantar ligament. Occasionally the subcutaneous bursa or the subtendonous calcanean bursa or both bursae may be seen. The entire width of the SDFT may not be observed on a single midline view, but useful CSA data can be obtained. The extreme medial and lateral borders of the SDFT and the retinacular attachment to the calcaneus may be more visible with slight medial and lateral placement of the transducer. The plantar ligament is located on the midline, firmly attached to the calcaneus, with its origin approximately 3 cm distal to the tuber calcanei. The DDFT and tarsal sheath must be viewed individually by medial positioning of the transducer. The origin of the plantar ligament widens in a plantar to dorsal plane rapidly in this zone. Accurate comparative CSA data require precise anatomical positioning on both hindlimbs. Usually at this level, no visible muscle bundles of the DDFT are seen, but clinicians must be aware of this possibility and not interpret them as a core lesion. At 5 cm distal (zone 1B) to the tuber calcanei, the plantar ligament is roughly triangular shaped and has multiple septated bundles that are all included to measure the CSA (Fig. 16-28).

Zone 1B Hindlimb zone 1B includes the distal half of the tarsus, extending from 8 to 16 cm distal to the tuber calcanei. Multiple images must be obtained from the plantar midline to assess the SDFT, from plantaromedial to assess the DDFT, and from slightly lateral to the midline to assess the plantar ligament. A wedge-shaped fibrous band emanates from the medial border of the SDFT and the fascia of the fibrous tarsal sheath and surrounds the DDFT (see Fig. 16-28). There is a narrow anechoic space representing fluid in the tarsal sheath separating the DDFT and the plantar ligament. The plantar ligament decreases in plantar to dorsal size as it courses distally; it has a firm enthesis to the plantar aspect of the fourth tarsal bone and inserts onto the head of the fourth metatarsal bone (MtIV).

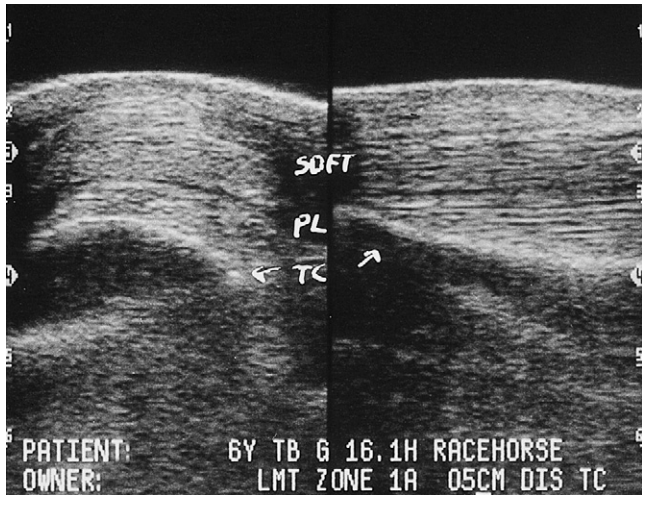


Fig. 16-27 Transverse *(left)* and longitudinal *(right)* (proximal is to the left) images of the plantar tarsus (zone 1A, 5 cm distal to the tuber calcanei) obtained using a standoff pad. *PL*, Plantar ligament or long plantar tarsal ligament; *SDFT*, superficial digital flexor tendon; *TC*, plantar border of the calcaneus.

Zone 2A This is the first metatarsal zone and includes the origin of the SL (~16 to 21 cm distal to the tuber calcanei) and the ALDDFT, which is a rudimentary structure in many horses. The transducer must be slightly medial to midline for complete examination of the DDFT. The SDFT can be assessed from the plantar midline. The origin of the SL can be somewhat confusing on ultrasound images, and this may create

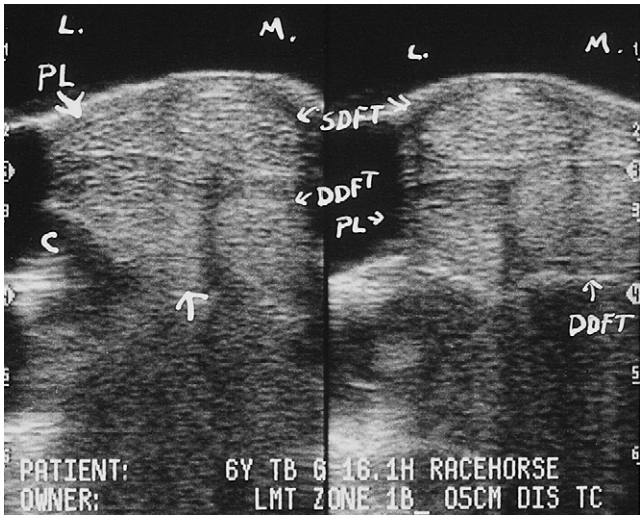


Fig. 16-28 Transverse images from zone 1B, 5 cm distal to the tuber calcanei, obtained using a standoff pad. Medial is to the right. The left image is slightly lateral of midline to highlight the plantar ligament *(PL; between arrows)*, and the right image is slightly medial of midline to highlight the superficial digital flexor tendon *(SDFT)* and the deep digital flexor tendon *(DDFT)*. *C*, Osseous border of the calcaneus. *L*, Lateral; *M*, medial; *PL*, plantar ligament or long plantar tarsal ligament.

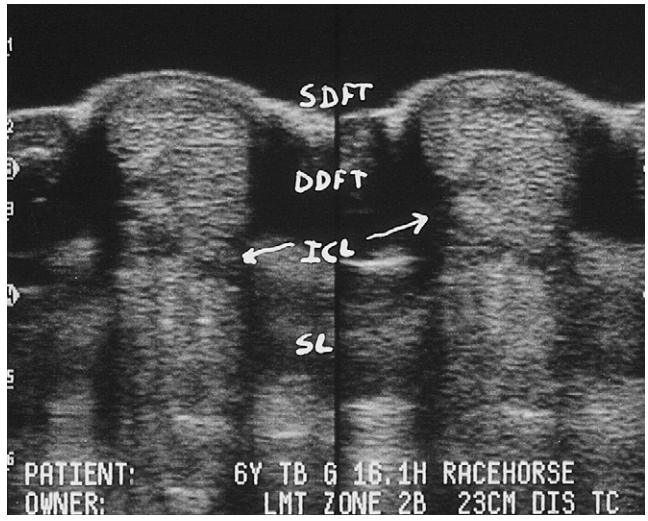


Fig. 16-29 Transverse images of zone 2B, 23 cm distal to the tuber calcanei, obtained using a standoff pad. *DDFT*, Deep digital flexor tendon; *ICL*, accessory ligament of the DDFT; *SDFT*, superficial digital flexor tendon; *SL*, suspensory ligament.

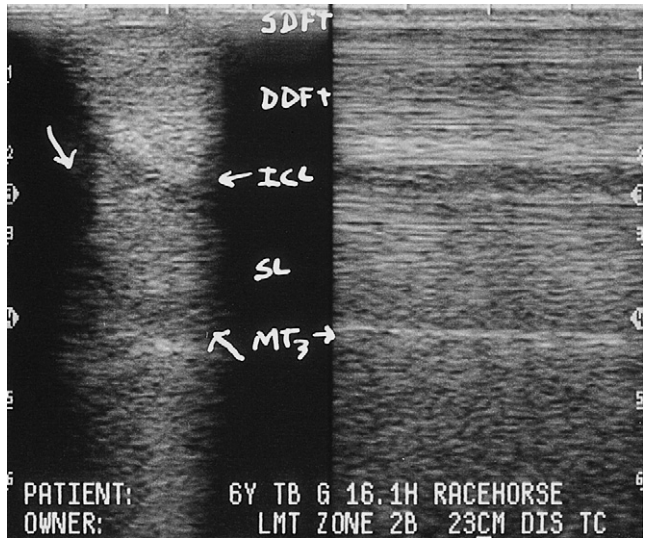


Fig. 16-30 Transverse *(left)* and longitudinal *(right)* images of zone 2B obtained without a standoff pad. The suspensory ligament *(SL)* is located between the large oblique arrows. *DDFT*, Deep digital flexor tendon; MT_3, plantar border of third metatarsal bone; *SDFT*, superficial digital flexor tendon.

a dilemma in detecting subtle injury. The SL is firmly attached to the third metatarsal bone (MtIII) (~4 to 5 cm enthesis) and rapidly increases in plantar to dorsal size within zone 2A. Therefore care must be taken when measuring comparative CSAs to do so at exactly the same level within zone 2A. In longitudinal images the origin of the SL often does not have a tight parallel fiber pattern, which results in a FAS of 1. The large bony surfaces of the second metatarsal bone (MtII) and MtIV obstruct a clear view, and often a linear anechogenic artifact is created by more superficial blood vessels. The origin of the SL begins slightly lateral to midline and thus is not parallel to the plantar surface of MtIII. The SL appears rhomboid from the plantar midline, but if viewed from slightly medial of midline, it appears more rectangular. CSA measurements should be obtained from one view or the other, but not mixed. Comparison of the longitudinal and transverse views of the SL may help in identifying the plantar and dorsal borders.

Zone 2B Hindlimb zone 2B extends approximately 21 to 24 cm distal to the tuber calcanei. The SDFT, DDFT, ALDDFT, and SL can be viewed adequately from a midline and slightly medial positioning of the transducer (Fig. 16-29). At this level the SDFT is slightly crescent shaped, and the DDFT is ovoid and slightly medially placed. The ALDDFT is seen on the dorsal border of the DDFT. The SL is roughly rectangular with an apex toward the plantar lateral aspect (Fig. 16-30). No unique anatomical features identify this zone; it is identified only by measurement.

Zone 3A This zone ranges from 24 to 32 cm distal to the tuber calcanei. The SDFT is slightly crescent shaped and is positioned directly over the DDFT. The SL at this level is roughly rectangular with an apex on the plantar lateral border. Zone 3A has no unique anatomical features.

Zone 3B Hindlimb zone 3B extends from 28 to 32 cm distal to the tuber calcanei. The SDFT is crescent shaped and narrower in the plantar to dorsal plane and wider in a medial to lateral plane, whereas the DDFT is ovoid. This zone has no unique anatomical features.

Zone 4A The hindlimb zone 4A extends from 32 to 36 cm distal to the tuber calcanei. In this zone the ALDDFT inserts into the DDFT, and a normal hypoechogenic artifact may be seen on the dorsal border of the DDFT (Fig. 16-31). This is the most distal zone in which the SL is a single unit. The proximal aspect of the DFTS may be seen.

Zone 4B Hindlimb zone 4B extends from 36 to 43 cm distal to the tuber calcanei and has the same anatomical features as forelimb zone 3B. The hindlimb branches of the SL tend to be longer (~10 cm) and a little larger than the forelimb branches.

Zone 4C This zone extends from 43 to 47 cm distal to the tuber calcanei and has the same anatomical features as forelimb zone 3C.

Palmar/Plantar Pastern Zones and Descriptions

Qualitative assessments are more critical in pastern ultrasonography because some lesions are not accompanied by

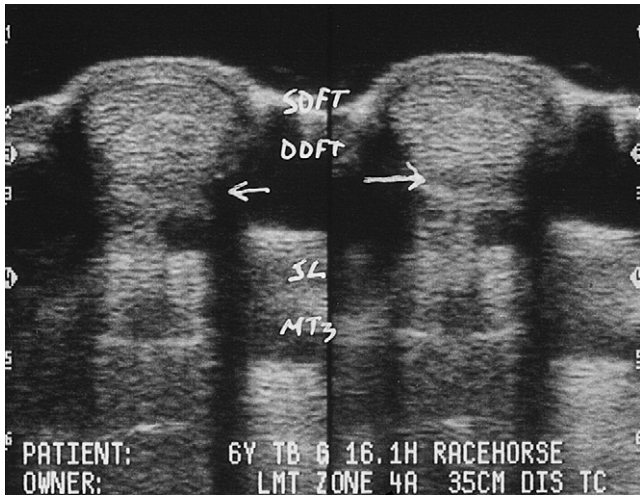


Fig. 16-31 Transverse images from zone 4A, 35 cm distal to the tuber calcanei. There is an artifactual hypoechogenic zone between the dorsal aspect of the deep digital flexor tendon *(DDFT)* and the plantar border of the accessory ligament of the DDFT insertion *(horizontal arrows)*. MT_3, Plantar border of third metatarsal bone; *SDFT;* superficial digital flexor tendon; *SL*, suspensory ligament.

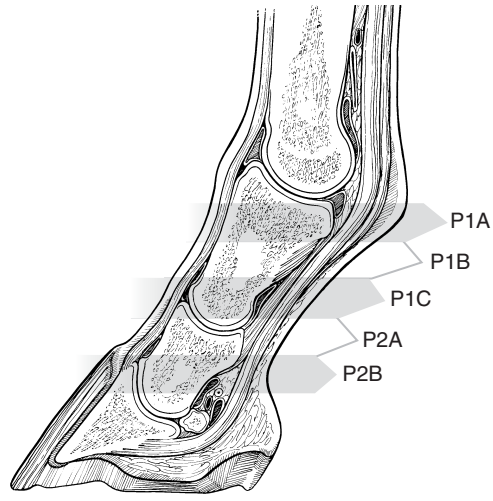


Fig. 16-32 Zone designations for the pastern. See text for zone descriptions.

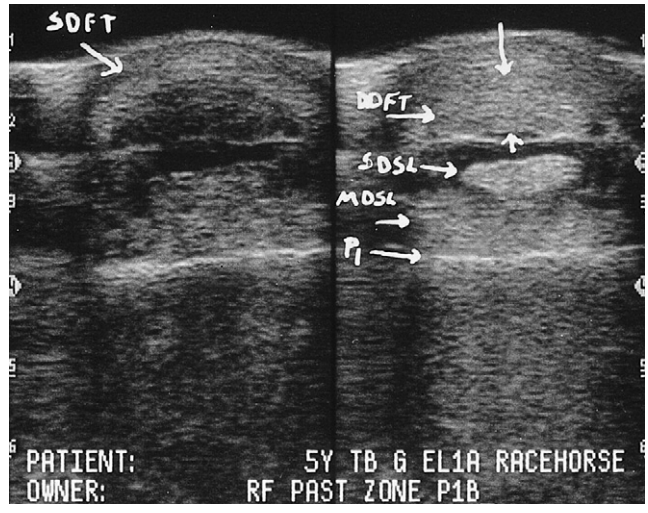


Fig. 16-33 Transverse images of zone P1B obtained using a standoff pad. The left image targets the superficial digital flexor tendon *(SDFT)*. The right image targets the deep digital flexor tendon *(DDFT)*, straight distal sesamoidean ligament *(SDSL)*, and the middle distal sesamoidean ligament or oblique sesamoidean ligament *(MDSL)*. P_1, Palmar border of the proximal phalanx.

significant changes in CSA. If DFTS tenosynovitis is coupled with a subtle primary tendon injury, the tendon may be mildly enlarged. Marginal longitudinal tears of the DDFT require precise qualitative inspection of the lateral and medial borders of the DDFT and the vinculae. Fibrous adhesions of the sheath may be identified but may not be accompanied by an increase in DDFT CSA.[19] It also is more difficult to obtain accurate FAS for the branches of the SDFT and oblique sesamoidean ligament (or middle distal sesamoidean ligament). The distal metacarpal or metatarsal regions should also be examined when assessing the DFTS in the pastern.

The pastern is a complex anatomical structure including many ligaments, joint recesses, vasculature, fibrocartilaginous insertional attachments, entheses, joint surfaces, and two major tendons.[20] The major structures of practical and common clinical concern are the DFTS, SDFT and branches, DDFT, oblique sesamoidean ligament (middle distal sesamoidean ligament), and straight sesamoidean ligament (or superficial distal sesamoidean ligament). The pastern can be arbitrarily divided into four zones, each with unique anatomical features. The pastern is divided into three palmar/plantar zones along the proximal phalanx (P1) and one zone at the proximal interphalangeal joint (Fig. 16-32).

Pastern Zone Designations and Descriptions

Zone P1A Zone P1A extends approximately 1 to 3 cm from the base of the ergot. Anatomical landmarks, such as the ergot, can be used to identify this zone. The SDFT is viewed from the midline and is a crescent-shaped, single-unit structure with very slight enlargement and rounding of the medial and lateral borders. The entire SDFT is included for CSA data. The palmar/plantar surface of the SDFT is covered by the thin proximal digital annular ligament. Dorsal to the SDFT on the midline view lies the ovoid-shaped DDFT. Anechoic fluid in the DFTS is interposed between the DDFT and the triangular-shaped straight distal sesamoidean ligament. Occasionally a fibrous bridge of the middle distal sesamoidean ligament or a small portion of the cruciate sesamoidean ligament (or deep distal sesamoidean ligament) may be identified dorsal to the straight distal sesamoidean ligament. Dorsal to the SDFT on the midline view is a hypoechogenic/anechogenic rounded joint recess of the metacarpophalangeal/metatarsophalangeal joint. The medial and lateral origins of the middle distal sesamoidean ligament are seen as two distinctly separate structures and are more adequately viewed by medial and lateral parasagittal placement of the transducer.

Zone P1B Zone P1B extends from 3 to 5 cm distal to the base of the ergot (Fig. 16-33). The SDFT is still a single unit starting to form rounded medial and lateral branches, which are evaluated by moving the transducer medially and laterally. The DDFT has a bilobed appearance and unless the transducer is perpendicular to the DDFT, central hypoechoic areas are seen in each lobe. The DFTS is seen on the dorsal surface of the DDFT, dorsal to which is the square- or rectangular-shaped superficial distal sesamoidean ligament. The oblique distal sesamoidean ligament is seen as a single structure in close proximity to the palmar/plantar surface of P1, dorsal to the straight distal sesamoidean ligament.

Zone P1C This zone extends approximately 5 to 7 cm distal to the base of the ergot (Fig. 16-34). The bilobed-shaped DDFT lies subcutaneously. The square-shaped or

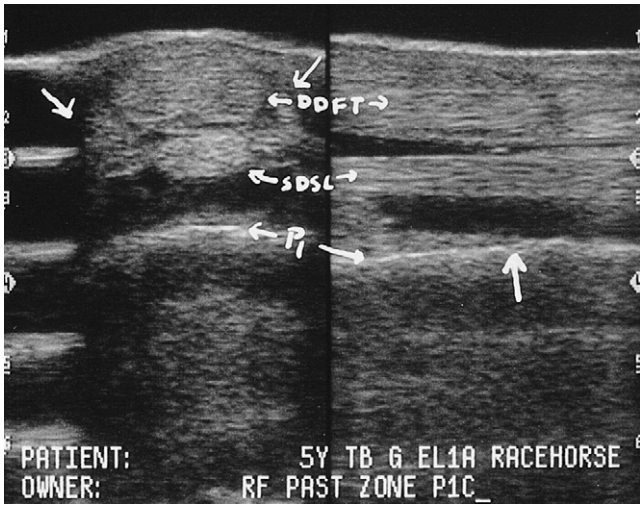


Fig. 16-34 Transverse *(left)* and longitudinal *(right)* images of zone P1C obtained using a standoff pad. The *oblique arrows* indicate the untargeted medial and lateral branches of the superficial digital flexor tendon. The *vertical arrow* indicates the insertion of the middle distal sesamoidean ligament *(MDSL)*. *DDFT*, Deep digital flexor tendon; P_1, palmar border of the proximal phalanx.

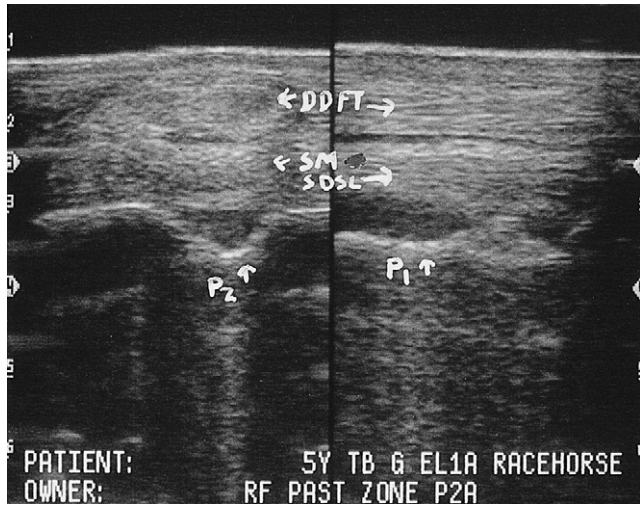


Fig. 16-35 Transverse *(left)* and longitudinal *(right)* images of zone P2A obtained using a standoff pad. *DDFT*, Deep digital flexor tendon; P_1, palmar border of the proximal phalanx; P_2, the palmar border of the middle phalanx; *SDSL*, superficial distal sesamoidean ligament or straight sesamoidean ligament; *SM*, scutum medium.

slightly rectangular-shaped straight distal sesamoidean ligament is on the dorsal surface of the DDFT. The oblique distal sesamoidean ligament is not seen because zone P1C is distal to its insertion. The teardrop-shaped medial and lateral branches of the SDFT are viewed by moving the transducer medially and laterally.

Zone P2A Zone P2A extends approximately 6 to 9 cm distal to the base of the ergot. A thin distal digital annular ligament is closely associated with the palmar/plantar border of the DDFT. The DDFT has a bilobed appearance but is narrower from palmar/plantar to dorsal and wider from medial to lateral. Deep to the DDFT is the middle scutum, the fibrocartilaginous insertion of the straight distal sesamoidean ligament, which may have a heterogeneous echogenicity (Fig. 16-35). Deep to the middle scutum is the proximal interphalangeal joint recess. The medial and lateral branches of the SDFT insert onto the distal aspect of the proximal phalanx and the proximal eminence of the middle phalanx.

Ultrasonography of Proximal Parts of the Limbs

Quantitative ultrasonographic evaluation can be applied to other anatomical areas and the lower limb areas described. These areas do not have zonal designations, and all are identified by measurement from a bony eminence. Many structures contain some muscle fiber bundles that are hypoechogenic, which makes detection of lesions more difficult. Whenever possible, we compare echogenicity, fiber alignment, and CSA between the limbs to determine some quantitative data to support a qualitative diagnosis.

CLINICAL ULTRASONOGRAPHY

With these suggested fundamental principles in terminology and a systematic concept of data collection, the clinician is prepared to examine the patient. A 7-year-old TB gelding racehorse that had raced for 5 years with 80 lifetime starts, 14 wins, and $74,568 in earnings, raced poorly 4 days previously. The next day, the trainer noted peritendonous swelling but no lameness. The horse had never had swelling in this area previously. The horse had received 2 g of phenylbutazone intravenously 24 hours before the race and 2 g immediately after the race. There was heat, slight subcutaneous swelling, and sensitivity and slight enlargement of the SDFT. A core lesion of the SDFT was detected ultrasonographically (Fig. 16-36). This scenario raises several questions. How serious is the injury? Can the horse be treated symptomatically and continue to race? If the horse is rested, will it be able to race again? If the horse is rested, which therapy should be used? How will the degree of healing be determined? Each horse is assessed by subjective evaluation of the ultrasonographic images and quantitative analysis.

Quantitative Analysis

The weakest link, or maximal injured zone, is the level at which the biggest proportion of tendon CSA is reduced in echogenicity, or the zone with the largest increase in CSA when compared with a normal contralateral limb. Total structural size is obtained by summing the CSA from each zone. To obtain the percentage of total extent of injury, add the lesion CSA from all levels with a lesion. The total lesion CSA is divided by the total structure CSA and multiplied by 100. The resulting number provides an estimate of structural compromise. It is this percent of total lesion (%T-lesion) that is used to categorize injury and relates the severity of structure injury to the client.

Quantitative Terms

The following terms are used in reference to injuries:

1. Maximal injury zone (MIZ)
2. Maximal zone cross-sectional area (MIZ-CSA)
3. Maximal injury zone lesion cross-sectional area (MIZ-LCA)
4. Maximal injury zone type or echo score (MIZ-TS) (MIZ-ES)
5. Maximal injury zone fiber alignment score (MIZ-FAS)
6. Total cross-sectional area (sum of included levels; T-CSA)
7. Total lesion cross-sectional area (sum of all levels with a lesion; TL-CSA)
8. Total type or echo score (sum of all levels with a score; T-TS or (T- ES)

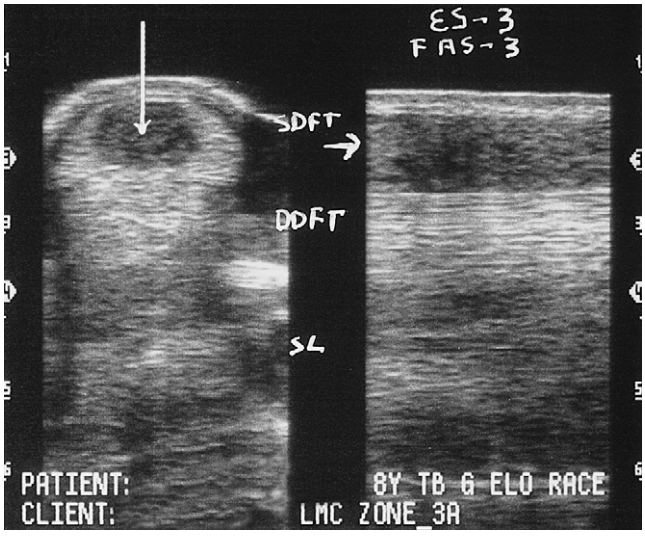


Fig. 16-36 Transverse *(left)* and longitudinal *(right)* images from zone 3A obtained using a standoff pad. The *vertical arrow* indicates a type 3 core lesion of the superficial digital flexor tendon *(SDFT)*. The *horizontal arrow* indicates the target path on the longitudinal image, with a fiber alignment score of 3 *(FAS-3)*. *DDFT,* Deep digital flexor tendon; *ES-3*, echo score 3; *SL*, suspensory ligament.

9. Total fiber alignment score (sum of all level scores; T-FAS)
10. Average fiber alignment score: (total divided by number of levels included; A-FAS)
11. Percent total lesion: ([TL-CSA ÷ T-CSA] × 100 = % T-lesion)

In a normal horse, typical measurements of T-CSA in the forelimb include the following:

- 8 zones of the SDFT = 886 mm^2
- 8 zones of the DDFT = 1001 mm^2
- 5 zones of the ALDDFT = 449 mm^2
- 11 zones of the SL = 1156 mm^2
  - Main body of the SL (5 zones) = 614 mm^2
  - Lateral branch (3 zones) = 258 mm^2
  - Medial branch (3 zones) = 284 mm^2

We find that this quantitative assessment provides practical, useful information that can be used routinely and that lesions can be placed in one of six categories.

Lesion Categories

Category I Category I implies no substantial qualitative or quantitative abnormality. No tendon or ligament fiber bundle lesions are detected, and all zones have lesion and fiber alignment scores of zero. No single zone has a CSA measurement greater than 39% compared with the contralateral limb. This percentage of tolerance more than adequately allows for method error, especially in zones 1A and 3A. However, with precise technique, a variation greater than 25% indicates substantial structural enlargement. Assessment of T-CSA depends on the number of zones included. The larger the number of zones included, the less difference there should be between limbs:

6/7 zones: <15%
11 zones: <12%
5 zones: <17%
3 zones: <20%
2 zones: <25%.

A 9-year-old TB mare competing as a hunter developed new swelling in the right proximal metacarpal region and slight, intermittent lameness. The client was concerned that the tendon was injured, and if so, did not want to continue athletic use of the horse, which would risk further injury. Palpation revealed swelling (2/5), increased skin temperature (2/5), and a painful response to direct digital pressure of the proximal right fore SDFT. Both forelimbs were examined ultrasonographically. No qualitative abnormalities were detected in the normal limb. Subcutaneous fluid was accumulated in the affected limb, indicating edema with or without hemorrhage, but echogenicity of the SDFT was normal. CSA was measured for 7 zones of each limb. Zone 1B was determined the maximal injured zone, and the CSA of the SDFT was 25% larger than the normal limb (i.e., category I) but suggestive of tendon enlargement. The T-CSA difference was only 3%. The SDFT was considered normal, although 2 zones had slightly enlarged CSA (1B was +25% and 2A was +23%). It was recommended that exercise should be restricted to walk and trot for 7 days with daily icing, daily bandaging with alcohol and thick standing bandages, and treatment with phenylbutazone (2 g once daily for 5 days). If the swelling was 100% reduced after this time, then the horse could return to normal athletic use. If no improvement occurred within 7 days or return to work caused a relapse, repeat ultrasonographic evaluation would be indicated. This horse returned to normal use.

Category II No hypoechogenic or anechogenic lesions are detectable, but the affected limb has MIZ-CSA measurements greater than 39%, with or without T-CSA for 6 or 7 zones greater than 14% of the normal limb (i.e., measurable focal or diffuse thickening of the tendon or ligament). Causes of category II injuries may include an unusual developmental difference, a new low-grade tendonitis/desmitis, or an older, currently stable healed tendon or ligament injury. Although generalized thickening may not cause great concern initially, there is evidence that continued use will eventually lead to greater injury. For example, 29% of TB racehorses (n = 13) that continued to train and race with a *new* category II SDFT injury eventually developed a clinically bowed tendon.[21]

A 12-year-old TB gelding competing cross country had recurrent swelling of the SDFT in the mid-metacarpal region that reduced with rest and anti-inflammatory treatment but returned if work was resumed. The horse had a long toe and was shod in smooth-toed, heavy steel shoes. There was slight, diffuse thickening (2/5) of the SDFT with increased skin temperature and marked sensitivity to direct digital pressure in zone 2B (4/5). Ultrasonographic examination revealed subcutaneous fluid and possible SDFT enlargement (Fig. 16-37) in zone 2B. Inflammation of the communicating branch of the palmar nerves was noted, indicating a malpositioned bandage. However, quantitative evaluation indicated a substantial generalized tendon thickening, which is unusual for a focal bandage pinch. The T-CSA was 15% larger than the normal limb (i.e., category II). It was recommended that exercise be restricted to walk with little trotting under saddle for 3 to 4 days a week for 1 month with daily icing and use of a thick-stall bandage with alcohol. Foot length should be shortened and the horse shod with a fully grooved or creased, lighter steel shoe to decrease the strain on the tendon. Five weeks later marked clinical improvement was noted. Ultrasonographic examination revealed that the subcutaneous fluid accumulation had resolved with no qualitative abnormalities of the SDFT. Significant reduction in the T-CSA measurements indicated a resolution of intratendonous inflammation. These findings add credence to the initial diagnosis of low-grade tendonitis. It is difficult to make this diagnosis qualitatively with any confidence. Only use of quantitative analytical techniques allow the clinician to truly appreciate these changes and be confident in a category II diagnosis.

Category III In category III lesions, type 1 or 2 focal hypoechogenic fibers bundles are present, with or without significant MIZ-CSA or T-CSA enlargement. To qualify as a

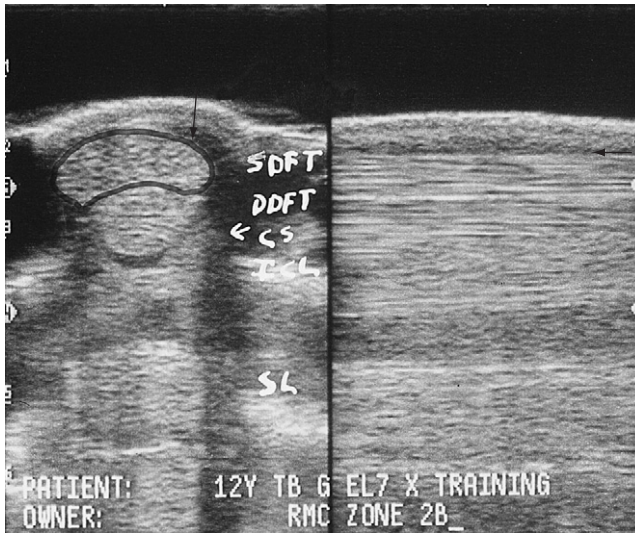


Fig. 16-37 Transverse *(left)* and longitudinal *(right)* images of zone 2B obtained using a standoff pad. The *arrows* indicate subcutaneous fluid accumulation. *CS*, Carpal sheath; *DDFT*, deep digital flexor tendon; *ICL*, accessory ligament of the DDFT; *SL*, suspensory ligament.

category III lesion, the T-TS (T-ES) cannot exceed 3 (i.e., ≤ 2). This abnormality may be an incidental finding, represent minimal tendon or ligament injury, or be the end result of a healed and clinically stable tendon or ligament injury. In these horses, clinical history, physical examination, and serial monitoring help to determine the significance of the ultrasonographic abnormalities.

A 16-year-old Trakhener gelding, used for upper-level dressage, was examined to assess healing of desmitis of the ALDDFT. The horse had been in controlled exercise for many months and the client was anticipating a progressive return to normal athletic use, assuming the ultrasonographic findings were favorable. Moderate thickening of the ALDDFT (3/5) was confirmed ultrasonographically, including a focal area of compromised fiber bundles along the dorsal lateral border of zone 2A (TS = 1), and a generalized alteration in fiber bundle alignment (T-FAS = 4). Given the knowledge that this horse had been rested for many months, a slow return to training was recommended. The ultrasonographic images can be used as a benchmark for evaluation of the stability of the repair as gradual increases in ligament loading commence. The horse eventually returned to upper-level dressage and has been asymptomatic for 2 years.

Even though category II and III injuries do not seem to represent a very serious compromise to a tendon or ligament, they do alert the clinician, trainer, and owner to a subtle problem that requires medical management, and that with early diagnosis is more easily managed. If the decision were made to continue athletic use accompanied with symptomatic therapy, serial ultrasonographic monitoring would be indicated to monitor stability. In this manner, early significant regressions may be detected before overt clinical signs of reinjury occur. These two injury categories are not easily diagnosed with qualitative assessment alone. Quantitative analysis identifies these low-grade abnormalities. However, these two categories often represent a dilemma for the practitioner. The clinical signs are slight, seldom associated with lameness, and often respond quickly to symptomatic therapy. Therefore it is difficult to persuade trainers to rest horses. However, category II and III tendons and ligaments often represent the earliest of ultrasonographically identifiable injury and have the best

Table • **16-1**

Category IV T-Lesions

ZONES USED FOR TOTAL (NO.)	T-LESION (%)*
11	1%-10%
6 or 7	1%-15%
5	1%-20%
3	1%-25%

*All have a total type or echo score ≥ 3.

chance of full recovery with extended rest. Quantitative evidence may help a client appreciate the significance of the injury. Ultimately, regardless of whether the horse is rested or treated symptomatically for a category II or III injury, periodic comparative data will greatly assist the practitioner in advising clients and help the clients to understand the injury.

Category IV A category IV lesion is characterized by focal hypoechogenic or anechogenic lesions with the T-S ≥3, with a specific %T-lesion (Table 16-1). The range of %T-lesion to qualify for category IV varies depending on the number of zones included for determining the "total." The MIZ-CSA or T-CSA may or may not be significantly enlarged. Although this category includes a wide range of injuries, it can be used to document slight injury or a significant compromise of tendon or ligament fiber bundles.

A 5-year-old TB racehorse gelding had a right fore SDFT injury of 6 months' duration. The horse had been galloping daily (exercise level 5) for 2 weeks, but the last time the horse galloped, right forelimb lameness and tendon swelling developed. Physical examination revealed slight swelling (1/5), low-grade lameness (2/5), moderate SDFT thickening (3/5), slight sensitivity to direct digital pressure, and localized heat (2/5) of the right forelimb. Ultrasonographic analysis of the right SDFT in zone 3B displayed a large type 3 anechogenic core lesion (Fig. 16-38). Quantitative data documented a T-CSA of 1243 mm^2. CSA was 67% larger than the left fore SDFT in zone 3B. In addition, bundles of compromised tendon were noted in 6 of the 7 zones. These localized lesions of mixed echogenicity, mostly type 1, probably were the result of the previous injury. The new injury was localized to zones 3B and 3C, with echo scores of 2 and 3. In this analysis of reinjury, all areas of reduced echogenicity were included in the total lesion summation. Additional measurements include %T-lesion of 12%, T-TS of 11, T-FAS of 8, and A-FAS of 1.1.

Category V Category V injuries typically have substantial MIZ-CSA or T-CSA enlargement with focal hypoechogenic, anechogenic, or both types of lesions. T-TS is greater than 3. This category has been arbitrarily associated with moderate tendon or ligament injury. The range of %T-lesion to qualify for category V varies depending on the number of zones included in arriving at the "total" (Table 16-2).

Many horses with category V or VI injuries or re-injuries exhibit lameness in the early stages, but many category III and IV injuries are not associated with lameness.

A 3-year-old Standardbred pacer, colt, developed swelling of the left fore SDFT after racing 1 week earlier and had previously sustained injury as a 2-year-old. The mid-metacarpal region was slightly swollen (1/5) with moderate SDFT thickening (3/5), sensitivity to direct digital palpation (2/5), slight heat (1/5), and slight lameness (2/5). Ultrasonographic examination revealed a type 2, focal, hypoechogenic lesion in the lateral border of the SDFT in zone 2A (Fig. 16-39). Computer analysis of the left fore SDFT documented hypoechogenic or anechogenic lesions along the lateral border in 6 of the 7

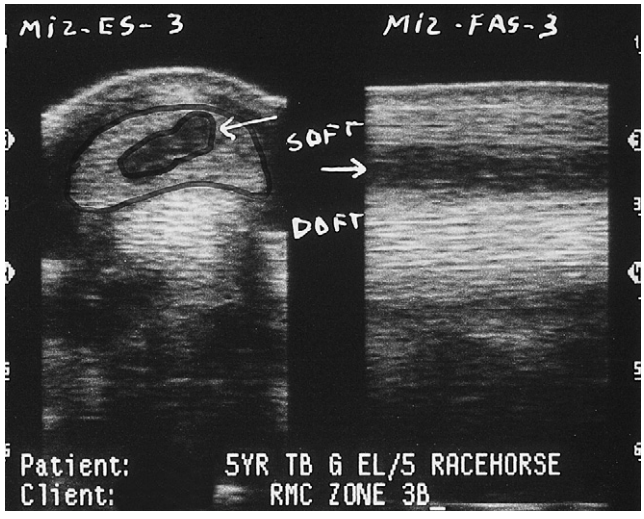


Fig. 16-38 Transverse *(left)* and longitudinal *(right)* images of zone 3B, obtained using a standoff pad. The *arrows* indicate a type 3 core lesion (traced on the transverse image) of the superficial digital flexor tendon *(SDFT)* on both views. The fiber alignment score of the target path of injury is 3. *DDFT,* Deep digital flexor tendon; *MIZ-ES-3;* maximal injury zone type or echo score of 3; *MIZ-FAS,* maximal injury zone fiber alignment score of 3.

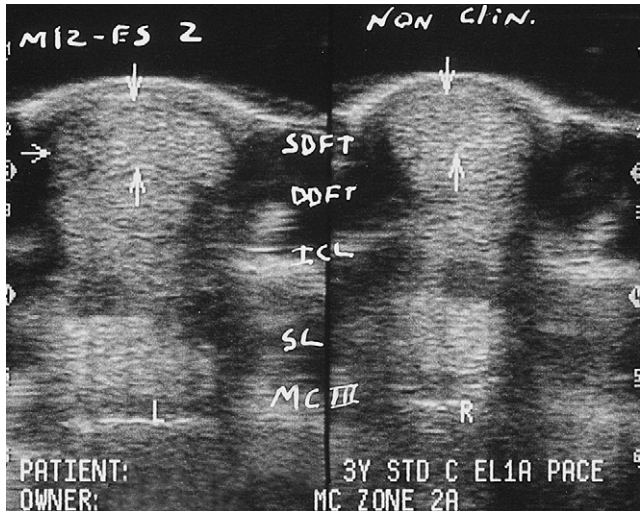


Fig. 16-39 Transverse images of zone 2A obtained using a standoff pad; the clinically abnormal limb is on the left and the clinically normal limb *(non-clin.)* is on the right. The left *(L)* image (lateral to the left) demonstrates a type 2 lateral border lesion of the superficial digital flexor tendon *(SDFT; horizontal arrow)*. *Vertical arrows* mark the palmar and dorsal borders of the SDFTs. The lateral border of the right *(R)* forelimb is to the right. *DDFT,* Deep digital flexor tendon; *ICL,* accessory ligament of the DDFT; *MIZ-ES 2,* maximal injury zone type or echo score of 2.

Table • 16-2

Category V T-Lesion

ZONES USED FOR TOTAL (NO.)	T-LESION (%)*
11	11%-20%
6 or 7	16%-25%
5	21%-30%
3	26%-35%

*All have a total type or echo score ≥ 3.

Table • 16-3

Category VI T-Lesions

ZONES USED FOR TOTAL (NO.)	T-LESION (%)*
11 zones	>20%
6 or 7 zones	>25%
5 zones	>30%
3 zones	>35%

*All have a total type or echo score ≥ 3.

zones (Fig. 16-40). Zone 2A was designated the maximal injured zone because it had the lesion with the largest CSA (37%). The MIZ-CSA of 166 mm^2 was 47% larger than the same zone of the contralateral normal SDFT. T-CSA of the 7 zones was 1179 mm^2, 31% greater than the contralateral SDFT. Additional measurements included T-TS of 11, T-FAS score of 8, and %T-lesion of 22% (i.e., category V). Unlike many newly injured tendons or ligaments with similar T-TS and T-FAS scores, this re-injured SDFT had a difference in these scores. This may serve as a means to identify new versus re-injured structures ultrasonographically.

Category VI Category VI injuries have substantial MIZ-CSA or T-CSA enlargement with more extensive hypoechogenic or anechogenic lesions than category V. The range of %T-lesion to qualify for category VI varies depending on the number of zone used to determine the "total" (Table 16-3).

A 4-year-old TB racehorse gelding developed swelling of the left fore SDFT after a race 6 days earlier. Physical examination revealed slight swelling (2/5), moderate SDFT thickening (3/5), slight sensitivity to direct digital palpation (2/5), slight heat (2/5), and low-grade lameness (2/5). A large type 3 anechogenic lesion of the lateral half of the SDFT was seen in zone 2B, the maximal injured zone (MIZ-LCA 92 mm^2 representing 54% of the tendon CSA). The MIZ-CSA was 114% larger than the contralateral normal zone 2B SDFT (see Fig. 16-15). Six of the 7 zones had hypoechogenic or anechogenic contiguous fiber bundles, with a 65% increase compared with the contralateral SDFT. T-LCSA was 300 mm^2, resulting in a 28% T-lesion. T-TS (14) and T-FAS (14) were the same, a common finding in newly injured tendons, although not acute SL injuries.

Summary

Tendon and ligament injuries can be graded from 1 to 6 to reflect the severity of injury. Advantages of using quantitative assessment in conjunction with clinical findings and qualitative evaluation include the following:

1. Confirmation or addition to a qualitative diagnosis.
2. Improved detection of subtle structural enlargement not accompanied by detectable echogenicity abnormalities, thus reducing interpretive errors.
3. Establishment of an objective means to categorize the severity of an injury, which can be used to determine prognosis for intended use and whether continued use or extended rest is the optimal choice. It also provides baseline information that can be compared with follow-up examinations.

FAS	Zone	Structure SIze (mm^2)	Lesion Size (mm^2)	Type/Echo Score
0	1A	115	5 (3.86%)	1
1	1B	127	17 (12.60%)	1
2	2A	166	61 (36.57%)	2
3	2B	238	83 (34.88%)	3
1	3A	201	1.64 (32.34%)	3
1	3B	173	1.28 (16.24%)	1
0	3C	159	—	
Total 8		1179 (11.79 cm^2)	258 (2.53 cm^2)	11

Maximal injury score (MIZ)	2A
MIZ-cross-sectional area (MIZ-CSA)	166 mm^2 (+41%)
MIZ-lesion cross-sectional area (MIZ-LCA)	61 mm^2 (37%)
MIZ-echo (type) score (MIZ-TS)	2
MIZ-fiber alignment score (MIZ-FAS)	2
Total (7 levels) cross-sectional area (TCSA)	1179 mm^2 (+31%)
Total lesion cross-sectional area (TLCSA)	258 mm^2
Percent total lesion (% T-Hypoechoic)	22%
Total echo (type) score (TTS)	11
Total fiber alignment score (TFAS)	8
Average fiber alignment score (AFAS)	1.1
Present category	V

STAGE			Initial exam		
SWEL	1/5	L	2/5	THIC	3/5
SEN	2/5	H	1/5	T.SH	0/5
ANK. SINK		0/5	FLEX	Neg.	

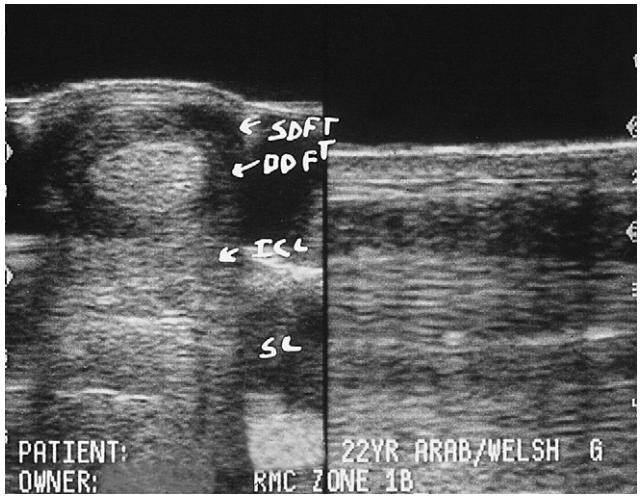
Fig. 16-40 A computer graphic of the left metacarpal region of the same horse as Fig. 16-39 shows seven data points for quantitative evaluation. The superficial digital flexor tendon is 31% larger than the normal limb, has a 22% T-lesion, a T-echo or type score of 11, a T-fiber alignment score of 8, and an average fiber alignment score of 1.1. The maximal injury zone is 2A. The overall category of fascicle compromise is V. *ANK. SINK;* Ankle sinking; *FLEX,* flexion; *H,* heat; *L,* lameness; *SWEL,* swelling; *THIC,* Thickening; *T.SH,* tendon sheath.

4. Enhanced veterinary participation in the rehabilitation of a tendon or ligament injury.
5. Improved client appreciation of the injury and improved compliance for suggested exercise restraint and treatment.
6. Creation of a common ultrasonographic interprofessional language and provision of a valid tool for research purposes to evaluate treatment responses.

Exceptions

As with most attempts to categorize medical injuries, exceptions most always are possible. The following are three common exceptions to quantitative categorization of tendons and ligaments:

1. Diffuse tendonitis/desmitis. This is an uncommon clinical presentation of SDFT thickening, which is seen mostly in 2-year-old TB racehorses and older event horses and show jumpers. The T-CSA measurements indicate substantial enlargement if the contralateral SDFT is normal. However, injury may be bilateral; therefore CSA measurements are compared with normal values for the breed, size, and age of the horse. Two-year-old TB racehorses in training tend to have larger T-CSA values than similar, older horses. In these horses, not only is the SDFT enlarged, but the entire CSA has a subtle reduction in echogenicity that extends through several zones. Although the %T-lesion is high, it does not necessarily indicate severe tendon fiber disruption because the echogenicity score for each zone is type 1 or 2 and the FAS score is 1or 2. This indicates a diffuse tendonitis with little loss of tensile strength. Given time and restricted exercise, documentation with serial ultrasonography indicates that most 2-year-old horses generally do well (Fig. 16-33).
2. If the SDFT or SL is totally disrupted, fiber bundles in adjacent zones may appear artificially more hypoechogenic or anechogenic because of the loss of tension in the structure, which is otherwise known as *relaxation effect*.[22] Clinically, tension is lost and the structure can be moved medially and laterally with ease. Ultrasonography shows a loss of parallel fiber alignment and a decrease in echogenicity. Measurement of CSA is difficult and can even be decreased at the maximal injured zone (Fig. 16-41). The diagnosis of complete rupture of a tendon or ligament is usually readily made clinically.



16-41 Transverse *(left)* and longitudinal *(right)* images of zone 1B obtained with a standoff pad. The borders of the superficial digital flexor tendon *(SDFT)* are not defined, and most of the echoes present represent hematoma. The SDFT was completely ruptured. *DDFT,* Deep digital flexor tendon; *ICL*, accessory ligament of the DDFT; *SL*, suspensory ligament.

3. Total collapse of the SL is characterized clinically by an extreme extension of the metacarpophalangeal or metatarsophalangeal joint (4/5 ankle sink), with little swelling or significant thickening of the SL or SL branch, and no sensitivity to direct palpation. Rupture cannot be palpated, and it appears as though the entire suspensory system has lost its cross-links and stretched, although ultrasonography shows little evidence of fiber tearing. Ultrasonographic images do not reflect the severity of the clinical signs.

Clinical Applications

If clinical evidence of swelling or thickening, with or without lameness, is present, the basic objective of an ultrasonographic evaluation is to determine the cause. Every effort must be made to determine whether tendons and ligaments are normal or abnormal and whether the lameness can be directly attributed to a soft tissue abnormality. Lameness may only be visible at high speeds in a racehorse or during special gait movements in a high-level dressage horse. In some instances the ultrasonographic data are insufficient to confirm the suspected cause, especially in subtle injury, although thorough evaluation and serial ultrasonographic examinations may provide clues.

Incidental abnormalities may be identified that have no bearing on the lameness or represent old, stable injuries of no clinical significance. Careful identification of these abnormalities and serial ultrasonographic monitoring may determine their significance. Sometimes other imaging techniques, such as radiography or nuclear scintigraphy, also are required to completely appreciate an injury process. For example, evaluation of SL insertion injuries should also include radiographic assessment.

Timing of Ultrasonographic Examinations: When to Scan

Timing of the initial ultrasonographic examination is critical. A clinician may be requested to examine a limb within hours of an injury. Peritendonous edema may obscure fiber damage because of acoustic enhancement, and the continued release of destructive enzymes may result in further ongoing fiber injury. Thus an examination shortly after an injury may not reveal the true extent of fiber damage. Ideally, an ultrasonographic evaluation should be delayed until at least 48 to 72 hours after the injury. If an ultrasonographic examination is performed before this time, the time after injury should be recorded, exercise restricted to walking, and symptomatic therapy instituted. Physical and ultrasonographic evaluations should be repeated after an additional 72 hours.

Significant hypoechogenic, anechogenic, or both types of lesions may be seen in an injury less than 72 hours old. The areas of reduced echogenicity may represent a combination of hemorrhage (seroma), edema, and fiber bundle injury. The diagnostic dilemma at this stage is that the clinician cannot accurately determine the relative contribution of each. It should not necessarily be assumed that the hypoechoic areas only represent fiber bundle injury. In most horses this assumption is true, but not always. We administer symptomatic anti-inflammatory treatment and advise complete stall rest (exercise level 0) or limited hand walking (exercise level 1A) for 14 to 30 days, followed by an ultrasonographic re-evaluation. (The exception to this advice would be if tendon splitting surgery were the treatment of choice to attempt to decompress a core lesion). When the horse is re-examined, persistent hypoechoic areas represent fiber bundle compromise, because most inflammatory edema and hemorrhage will have resolved. The severity of the injury can now be determined, and we refer to this as the *baseline evaluation*. The baseline evaluation often reveals the same or increased severity of injury compared with the initial scan (especially in core lesions caused by enzymatic degradation of damaged fiber bundles or pressure necrosis).

As an example, we can compare ultrasonographic images of a SDFT injury after 24 hours and after 22 days. The initial examination determined that the SDFT was 32% larger than the normal contralateral limb and had a 17% T-lesion (category V), a T-TS of 17, and a T-FAS of 16. After symptomatic therapy and a reduction of exercise to exercise level 1A, the baseline scans (22 days later) revealed a 12% increase in size of the SDFT compared with the normal limb, a 13% T-lesion (now category IV), a T-TS of 10, and a T-FAS of 12. The apparent decrease in severity was due to the resolution of the inflammatory response and absorption of the seroma. The baseline data represent the actual severity of fiber bundle compromise and serve as the starting point for rendering prognosis for intended use, treatment programs, and long-term rehabilitation exercise regimens.

Serial Ultrasonographic Examinations as Part of Case Management

Serial ultrasonographic examinations are best performed when it is anticipated that the exercise level may be increased. This justifies the examination to the owner or trainer. In addition to the ultrasonographic assessment, the clinician must always consider the physiological principles of tendon and ligament healing and the necessary physiological time for healing for the severity of an injury before advising exercise level increases. With time and as quantitative ultrasonographic analysis is more commonly used to evaluate tendon and ligament repair, more definitive recommendations will develop concerning convalescent time based on the severity and location of an injury and the intended athletic function of the horse. The same applies to the quality of the repair. Horses destined for light work will be able to perform with a less than perfect repair, but racehorses and other high-level athletes will require optimal repair and a reasonable time to give the best chance for return to athletic use without reinjury.

During rehabilitation a qualitative ultrasonographic evaluation of the clinically injured tendon is done to detect obvious lesions, evidence of re-injury, restricting peritendonous tendon sheath fibrosis, or restriction by the PAL. Objective, or quantitative, ultrasonographic assessments should also be determined.

Guidelines for an optimum chance to return to racing include the following[23]:

1. At least a 60% decrease in category IV %T-lesion and <12% total lesion cross-sectional surface area (T-LCSA) for all categories of severity of injury.
2. At least a 10% to 15% decrease in T-CSA from baseline for all categories of injury (a relatively greater decrease in more seriously injured tendons).
3. At least a 70% decrease in the T-TS (ideally <4; the closer to 0, the better).
4. At least a 75% decrease in the T-FAS (ideally <4; the closer to 0, the better) before advancing to exercise level 5.
5. The horse should meet at least three of the above requirements with only minimal failure of the other criterion.

Nonetheless, even a perfectly healed tendon or ligament injury can be re-injured. Re-injury is especially common in TB racehorses. However, if the repair is of poor quality, re-injury is likely and these horses should be held back from active training until healing has improved. Decision making is more accurate when it is based on ultrasonographic evaluation and consideration of necessary physiological time for intended use than when relying on clinical inspection.

The advantages of veterinary intervention and using quantitative ultrasonographic parameters in case management provide a means to optimize the chances of returning an injured horse to athletic usefulness and are reviewed in this clinical example. A 4-year-old TB racehorse with a left fore SDFT injury was examined 3 days after a race. The maximal injured zone was zone 2B (59% lesion), T-CSA was 1206 mm^2, T-TS was 11, and T-FAS was 16. This was a 49% T-lesion (i.e., category VI). The horse was treated with a series of 3 external blisters and 6 months' pasture rest, a typical theory in the early 1990s. The horse was re-assessed 8 months after injury to determine whether it could return to training (exercise level 5). The tendon was tight and cold. Qualitative ultrasonographic examination indicated improvement, although abnormalities persisted, which were interpreted to be the end result of repair. Thus the trainer was wrongly advised to return to training.

In light of current knowledge, the ultrasonographic data at the time of re-evaluation provided clear indication of an unstable repair. The T-CSA had increased 15%, %T-lesion decreased by only 50%, and therefore was still quite high at 22%. In addition, neither T-TS nor T-FAS met a target decrease of 75%. After 4 weeks of galloping, the horse worked at race speed (breezed) once and became lame again. An ultrasonographic evaluation identified extensive reinjury to the same tendon (i.e., category VI). Thus quantitative analysis of injuries is an extremely beneficial tool to monitor progress of tendon healing.

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CHAPTER • 17

Ultrasonographic Examination of Joints

Jean-Marie Denoix

Ultrasonography has become an essential imaging technique for assessing joint lesions.[1-3] It provides information complementary to radiography but does have some limitations. Ultrasonography requires a precise knowledge of anatomy, not only bone anatomy,[4] and a systematic approach. Examining the joints of the distal part of the limb is physically uncomfortable for the imager and is time consuming: complete examination of a joint may take up to 30 minutes. Quick examination of joints requires several years of daily practice.

The indications for ultrasonographic examination of equine joints include synovial fluid distention, local swelling, pain on passive manipulation of the joint, improvement in lameness after intra-articular or regional analgesia, or positive radiographic or scintigraphic findings. The area to be examined should be clipped, not shaved, and washed with hot water. High-resolution transducers (7.5- to 13-MHz linear probes) and a standoff pad are used for superficial structures. Convex array 2.5- to 5-MHz transducers are preferable for deeper structures. Both left and right joints should be examined to improve sensitivity and specificity. The image quality depends not only on the frequency of the transducer but also on the quality of the machine (from treatment of the signal to display on the monitor) and the skill of the operator in placing and orienting the transducer.

A comprehensive description of all joints cannot be given in this book. The fetlock joint is a model for a general approach to ultrasonography of joints because of its simple anatomy. Some aspects of examination of the stifle and hock also are presented.

FETLOCK

Each aspect of the joint should be examined systematically using 7.5- to 13-MHz linear transducers and a thin standoff pad.

Dorsal Aspect

Fig. 17-1 shows normal ultrasonographic anatomy.[4,5] In normal fetlock joints the articular capsule is echogenic (except if too relaxed), and the articular margins of the proximal phalanx and the condyles of the third metacarpal bone (McIII) are smooth.

Ultrasonography is a useful technique for the differential diagnosis of soft tissue injuries on the dorsal aspect of the fetlock joint. These lesions include subcutaneous swelling or abscess, bursitis of the subtendonous bursa of the extensor tendons, extensor tendonitis, capsulitis, synovial fluid distention of the dorsal recess of the metacarpophalangeal joint (Fig. 17-2), and chronic proliferative synovitis of the proximodorsal synovial fold of this joint (Fig. 17-3). Thinning, fibrillation, and fissures of the articular cartilage of the dorsal and distal aspects of the condyle of McIII can be identified with high-resolution transducers. Subchondral bone lesions can sometimes be detected before they are visible radiographically.

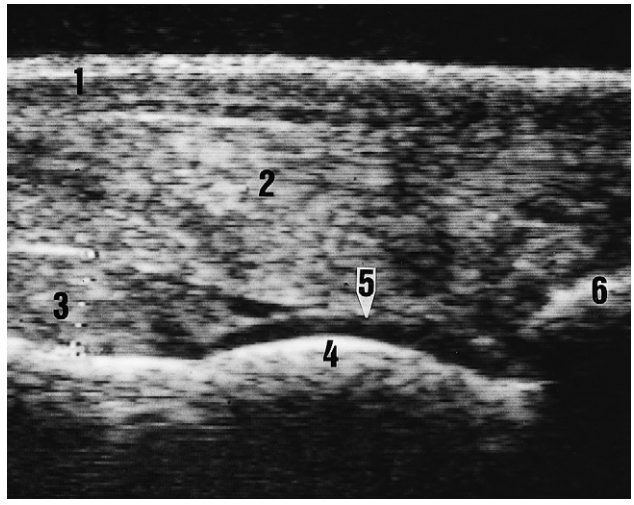

Fig. 17-1 Sagittal ultrasound scan of the dorsal aspect of the fetlock, normal appearance. Proximal is to the left. *1*, Skin; *2*, dorsal capsule; *3*, proximal synovial fold; *4*, subchondral bone of the condyle of the third metacarpal bone; *5*, articular cartilage of the condyle of third metacarpal bone; *6*, proximal phalanx.

Dorsomedial and Dorsolateral Aspects

Examination of the dorsomedial and dorsolateral aspects of the joint is especially useful for complete evaluation of the articular margins, which are smooth in a normal joint. The most common abnormal finding is the presence of periarticular osteophytes. Other injuries are subcutaneous lesions (fibrosis, swelling) and capsulitis.

Medial and Lateral Aspects

The medial and lateral collateral ligaments have superficial and deep layers.[4] If the transducer is parallel to the skin (the ultrasound beam is perpendicular to fiber interface), the superficial layer of the collateral ligament is echogenic and the deep layer is hypoechogenic. Either layer may be damaged (Fig. 17-4). If both layers are affected, joint instability, subluxation, or luxation occur.

Palmar Aspect

A normal fetlock joint has a small amount of anechoic synovial fluid in the proximopalmar recess of the fetlock joint. Abnormal findings observed in affected horses include synovial fluid distention, often associated with enlarged synovial folds, indicative of synovitis (Fig. 17-5).

Echogenic material may be observed in the synovial fluid in any view. Small echogenic spots may represent fibrin or cartilaginous debris. Larger echogenic masses are compatible with osteochondral fragments. These abnormal findings are more easily seen after movement of the joint. A homogenous increase in echogenicity of the fluid is compatible with hemarthrosis or infectious synovitis.

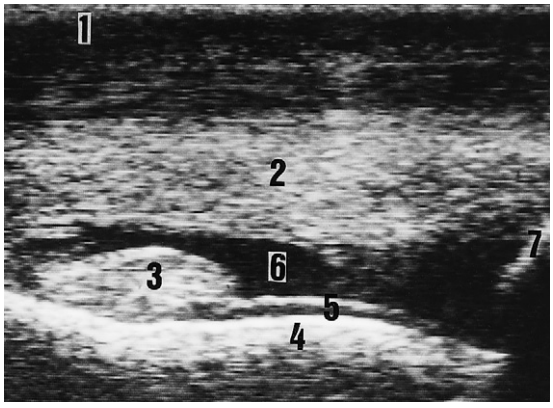

Fig. 17-2 Sagittal ultrasound scan of the dorsal aspect of the fetlock joint. An abnormal synovial fluid accumulation appears between the capsule and the articular surface. The diagnosis is synovial fluid effusion, indicative of synovitis. Proximal is to the left. *1*, Skin; *2*, dorsal capsule; *3*, proximal synovial fold; *4*, subchondral bone of the condyle of the third metacarpal bone; *5*, articular cartilage of the condyle of the third metacarpal bone; *6*, synovial fluid; *7*, proximal phalanx.

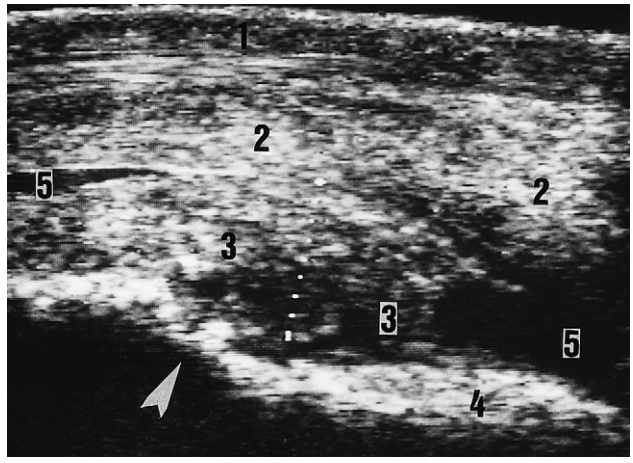

Fig. 17-3 Sagittal ultrasound scan of the dorsal aspect of the fetlock joint. An abnormal hypoechogenic mass appears between the capsule and the condyle of the third metacarpal bone. The proximal aspect of this condyle is irregular, indicating bone lysis (*arrowhead*). The diagnosis is chronic proliferative synovitis. *1*, Skin; *2*, dorsal capsule; *3*, proximal synovial fold tremendously thickened and echoic; *4*, condyle of the third metacarpal bone; *5*, synovial fluid.

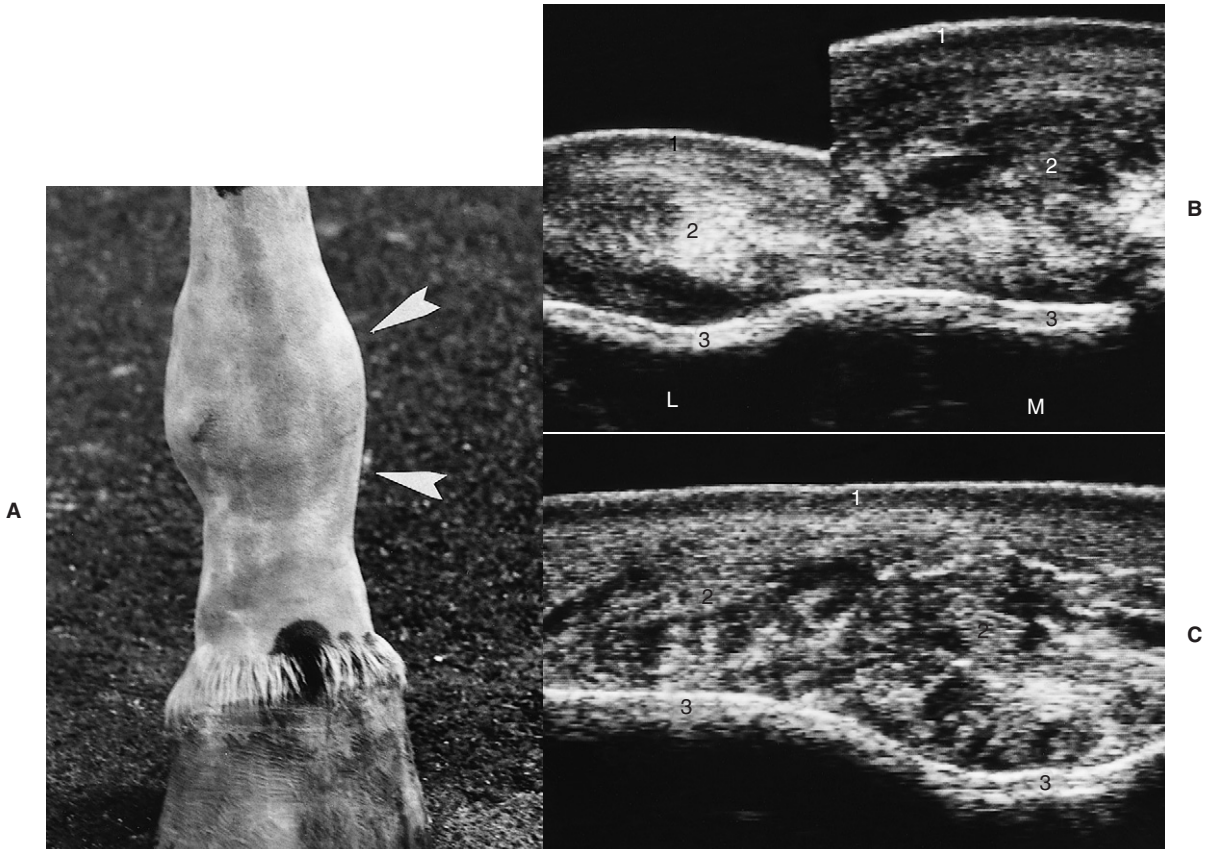

Fig. 17-4 **A,** Physical appearance of an injured hind fetlock joint (*arrowheads* show medial enlargement). Radiography found no bony abnormalities. **B,** Transverse ultrasound scans of the lateral (*L*) and medial (*M*) aspects of the injured fetlock. The size and architecture differ greatly between the normal lateral collateral ligament and the injured medial ligament. **C,** Longitudinal ultrasound scan of the medial aspect of the fetlock shows the thickening and architectural alterations of the medial collateral ligament. The diagnosis is chronic desmopathy of the medial collateral ligament. *1*, Skin; *2*, collateral ligament; *3*, condyle of the third metatarsal bone.

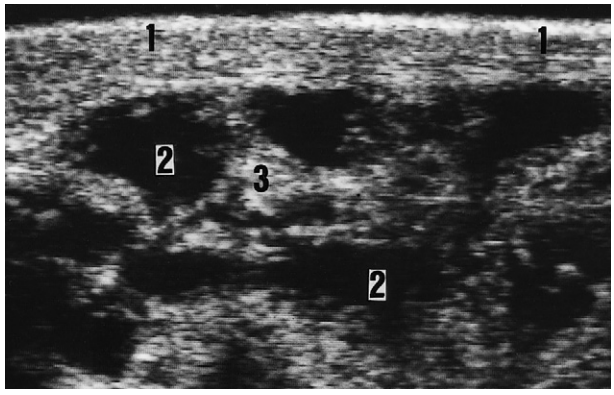

Fig. 17-5 Longitudinal ultrasound scan of the palmarolateral aspect of the fetlock demonstrating a marked fluid distention of the palmaroproximal recess of the fetlock. The diagnosis is synovial fluid effusion indicative of synovitis. *1*, Skin; *2*, synovial fluid; *3*, synovial membrane and villi.

STIFLE

Ultrasonography has considerably improved the knowledge and the diagnosis of soft tissue injuries of the femoropatellar and femorotibial joints.

Femoropatellar Joint

The femoropatellar joint is examined using 5- to 10-MHz linear transducers with a thin standoff pad. The normal patellar ligaments are homogeneously echogenic.[1-3] The articular cartilage is thicker over the lateral trochlear ridge compared with the medial. Each femoral trochlea has a regular hyperechogenic subchondral bone surface.

Medial patellar desmopathy usually is iatrogenic (i.e., caused by medial patellar desmotomy). Desmopathy of the intermediate (middle) patellar ligament is an injury that occurs in athletic horses. Lateral patellar desmopathy often is caused by trauma (Fig. 17-6).

Osteochondrosis of the trochlear ridges is easy to diagnose, and ultrasonography provides information on the extent of the lesion (especially in a lateromedial direction), the size and location of the osteochondral fragments, and the severity of alteration of the subchondral bone. Evaluation of the synovial fluid and membrane may also be useful to assess the consequences of ligamentous and osteochondral lesions in the joint.

Medial and Lateral Femorotibial Joints

Examination of the femorotibial joint requires 5- to 10-MHz linear transducers and a thin standoff pad. In normal joints the medial recess of the medial femorotibial joint is less than 3 cm long in a craniocaudal direction, and the synovial fluid is totally anechoic. The normal medial collateral ligament and medial meniscus are homogeneously echogenic,[1-3] provided that the ultrasound beam is perpendicular to the orientation of the fibers. The normal articular margins of the medial femoral condyle and tibial plateau are smooth and regular.

The medial recess of the medial femorotibial joint can be considered a mirror of the joint, because its size and content are influenced by all joint lesions. Abnormalities include synovial effusion (Fig. 17-7), chronic proliferative synovitis, hemarthrosis (Fig. 17-8), echogenic spots compatible with fibrin, cartilaginous, or meniscal debris (Fig. 17-9), osteochondral fragments, and calcinosis circumscripta. The caudal recess of the medial femorotibial joint can be examined with 2.5- to 5-MHz sector or convex array probes. Distention of this recess always indicates severe femorotibial disease. Distention of the subextensorius recess on the craniolateral aspect of the lateral femorotibial joint can be observed in lateral femorotibial arthropathy or femoropatellar joint lesions.

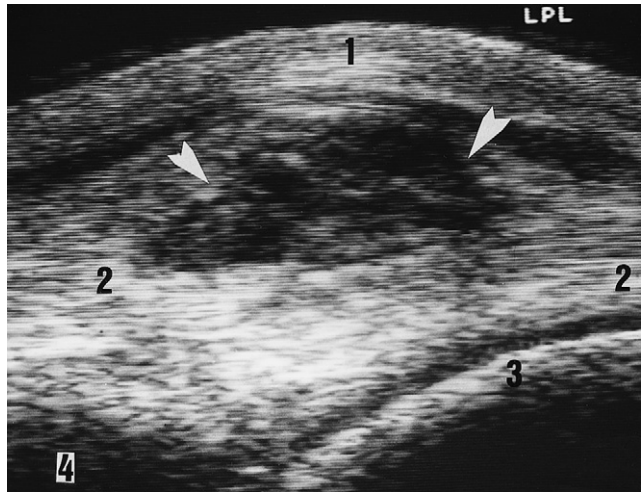

Fig. 17-6 Longitudinal ultrasound scan of the lateral patellar ligament demonstrating localized enlargement. At the site of the lesion (*arrowheads*) the ligament is hypoechogenic and shows severe architectural changes. Diagnosis is traumatic lateral patellar desmopathy. *1*, Skin; *2*, lateral patellar ligament; *3*, lateral ridge of the femoral trochlea (subchondral bone surface); *4*, patella.

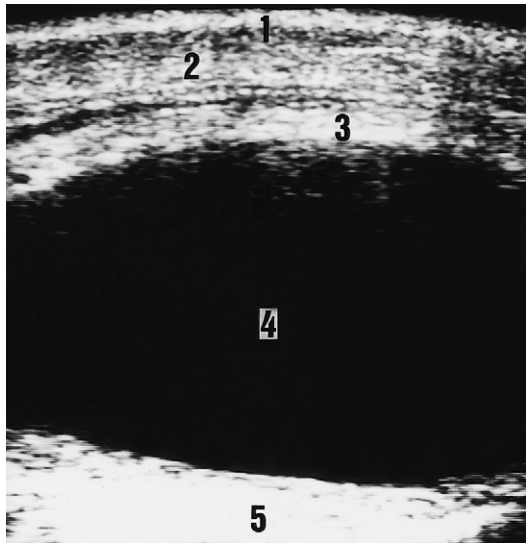

Fig. 17-7 Transverse ultrasound scan of the medial recess of the medial femorotibial joint. Anechogenic synovial fluid has distended this recess. The diagnosis is synovial effusion compatible with synovitis and other potential injuries of the femorotibial joint. *1*, Skin; *2*, medial femoral fascia; *3*, synovial membrane; *4*, medial recess of the medial femorotibial joint; *5*, medial aspect of the distal femur.

Desmopathy of the medial collateral ligament may be identified as disruption in the normal parallel fiber pattern (Fig. 17-10).

Meniscal injuries can be observed alone or with other ligamentous or bone injuries (Fig. 17-11). In our patients, approximately 75% of these lesions are found in the medial meniscus and 25% in the lateral meniscus. A wide range of type and severity of medial meniscal injuries may be seen (Figs. 17-11 and 17-12).

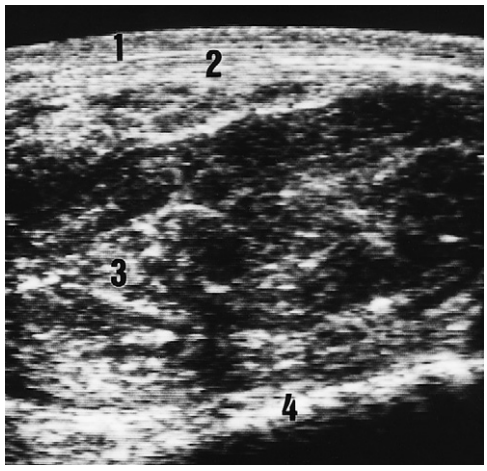

Fig. 17-8 Transverse ultrasound scan of the medial recess of the medial femorotibial joint. Echogenic material distends the recess. The diagnosis is hemarthrosis compatible with other potential ligament or bone injuries of the femorotibial joint. *1*, Skin; *2*, medial femoral fascia; *3*, medial recess of the medial femorotibial joint; *4*, medial aspect of the distal femur.

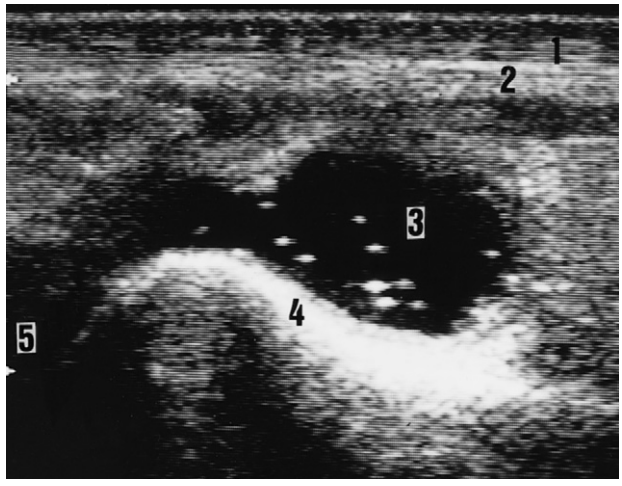

Fig. 17-9 Longitudinal ultrasound scan of the medial recess of the medial femorotibial joint. Echogenic material floats in the synovial fluid. These echogenic spots represent fibrin, cartilaginous debris, or meniscal debris. Proximal is to the right. *1*, Skin; *2*, medial femoral fascia; *3*, medial recess of the medial femorotibial joint; *4*, medial femoral condyle; *5*, medial meniscus (anechogenic because of the orientation of the ultrasound beam).

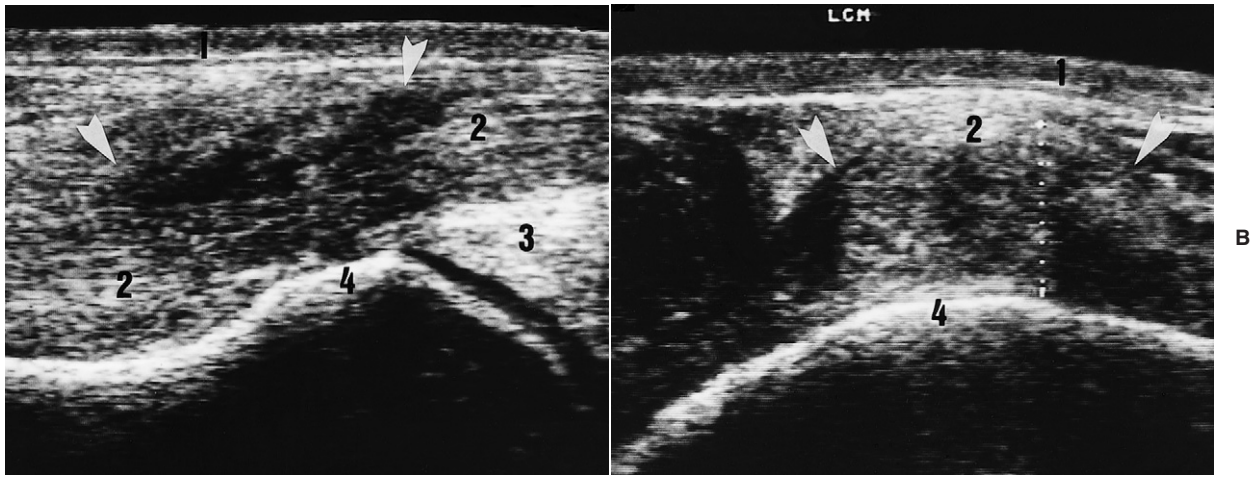

Fig. 17-10 **A,** Longitudinal ultrasound scan (proximal is left) of the medial collateral ligament of the femorotibial joint. This ligament is enlarged and hypoechogenic (*arrowheads*). Diagnosis is traumatic medial collateral desmopathy. **B,** Transverse ultrasound scan of the medial collateral ligament. *1*, Skin; *2*, medial collateral ligament; *3*, medial meniscus; *4*, medial femoral condyle.

If a transducer is held vertically and moved in a caudocranial direction, complete examination of the articular margins of the femoral condyles is possible. Ultrasonography may be more sensitive than radiography in detecting periarticular osteophytes. If the stifle is examined in flexion, the cartilage and subchondral bone surface of the femoral condyles can be imaged.[1-3] Alterations of the subchondral bone surface and echogenicity may be detected.

COLLATERAL LIGAMENTS OF THE HOCK

The causes of soft tissue enlargement of the hock may be difficult to assess clinically and with radiography. This section discusses collateral ligament injuries. The medial and lateral collateral ligaments of the hock are highly echogenic fibrous structures. Each is divided into a long collateral ligament, which inserts distally on the distal tarsus and proximal metatarsus, and a short collateral ligament with two fasciculi: a calcanean fasciculus and a talien one.

Proximal avulsion fracture of the calcanean fasciculus of the medial collateral ligament has been identified (Fig. 17-13). Lesions of this ligament also can be identified in hocks with no radiographic abnormality (Fig. 17-14). In old lesions the long medial collateral ligament remains thickened and may have focal echogenic sites associated with mineralization.

Several types of injury of the lateral collateral ligament have been identified, including desmopathy of the long lateral collateral ligament, avulsion fracture of the short lateral collateral ligament at the proximal insertion on the lateral malleolus of the tibia (distal portion of the fibula), and desmitis of the short lateral collateral ligament associated with sudden synovial distention of the tarsocrural joint capsule.

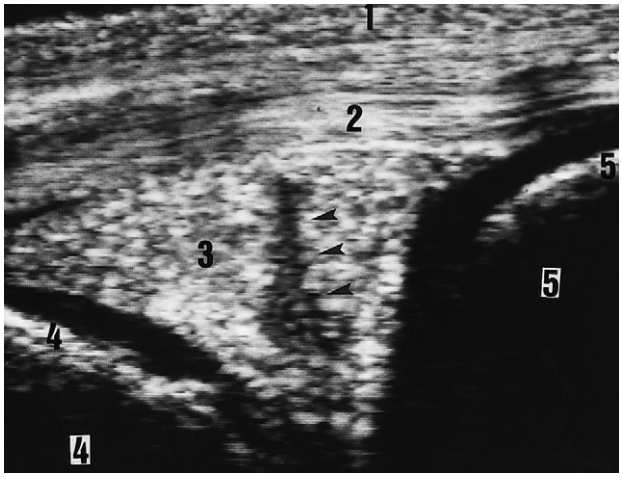

Fig. 17-11 Proximodistal ultrasound scan of the medial aspect of the medial femorotibial joint showing a transverse section of the body of the medial meniscus. An obvious hypoechoic defect (*arrowheads*) is seen. The diagnosis is medial meniscal lesion. *1*, Skin; *2*, medial collateral ligament; *3*, medial meniscus; *4*, medial femoral condyle (with anechoic articular cartilage); *5*, medial tibial condyle (with anechoic articular cartilage).

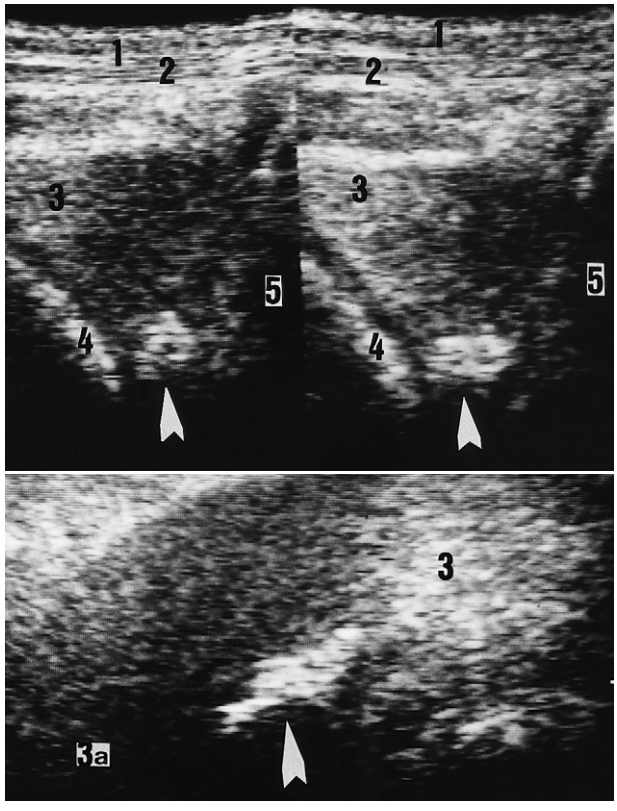

Fig. 17-12 **A,** Proximodistal ultrasound scan of the craniomedial aspect of the medial femorotibial joint showing transverse sections of the cranial horn of the medial meniscus. Hyperechogenic material is present in the deep (axial) part of this horn (*arrowheads*). **B,** Transverse ultrasound scan of the craniomedial aspect of the medial femorotibial joint showing a horizontal section of the cranial horn of the medial meniscus. This scan confirms presence of hyperechoic material in the horn. The diagnosis is that hyperechoic material is a focal site of mineralized metaplasia in a meniscus. *1*, Skin; *2*, medial femoral fascia; *3*, medial meniscus; *3a*, cranial attachment; *4*, medial femoral condyle; *5*, medial tibial condyle.

Fig. 17-13 **A,** Acute medial collateral desmopathy in a 15-month-old filly. Physical appearance of the hock with a medial swelling. **B,** Longitudinal ultrasound scan of the calcanean fasciculus of the medial collateral ligament. This structure is thickened and hypoechogenic with evidence of fiber disruption. An echogenic osteochondral fragment (*arrowhead*) appears close to the medial malleolus of the tibia. The diagnosis is severe desmopathy with avulsion fracture involving the calcanean fasciculus of the medial collateral ligament. *1*, skin; *2*, medial malleolus of the tibia; *3*, long medial collateral ligament; *4*, short medial collateral ligament: calcanean fasciculus *(CALC)*; *5*, medial aspect of the talus *(TAL)*.

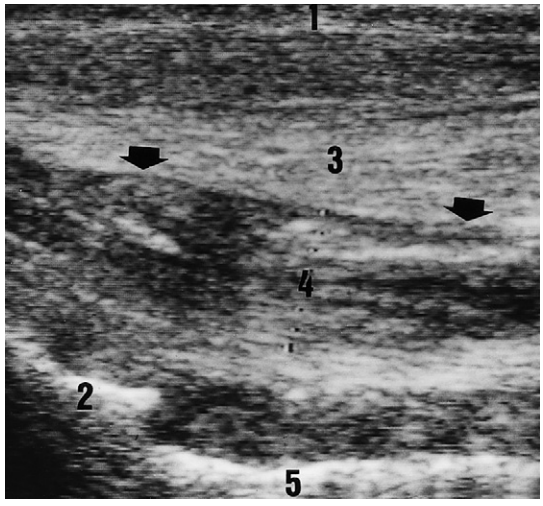

Fig. 17-14 Longitudinal ultrasound scan of the calcanean fasciculus of the medial collateral ligament of a 9-year-old Three-Day Event gelding after injury 3 months previously. This structure *(black arrows)* is thickened and has architectural changes. The diagnosis is desmopathy of the calcanean fasciculus of the medial collateral ligament. *1*, Skin; *2*, medial malleolus of the tibia; *3*, long medial collateral ligament; *4*, short medial collateral ligament: calcanean fasciculus (*arrowheads*); *5*, medial aspect of the talus.

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CHAPTER • 18

Ultrasonography and Orthopedic (Non-Articular) Disease

Virginia B. Reef

The use of diagnostic ultrasonography for evaluation of tendons, ligaments, tendon sheaths, bursae and joints is discussed in Chapters 16 and 17, and patient preparation for other musculoskeletal uses is similar.

SKELETAL MUSCLE

Normal skeletal muscle appears heterogeneous with hypoechogenic muscle fibers laced with and surrounded by echogenic fascia, connective tissue, and fat. In transverse section the normal muscle has a marbled or speckled appearance, which is unique for each individual muscle, as is its striated appearance in longitudinal section.[1,2] A non–weight-bearing muscle appears more echogenic than the same muscle when the horse is fully weight bearing.[1,2] Therefore comparisons between contralateral muscles should be made when the horse is bearing weight evenly.

Traumatic muscle injuries, myositis, or masses infiltrating the muscles usually can be differentiated from each other by ultrasonography.[1] Muscle tears in horses most frequently are seen in the hindlimb and shoulder musculature. The affected muscle or muscles can be identified by tracing the involved muscle from its origin to insertion. Fluid-filled anechogenic areas with hypoechogenic loculations are located within the muscle belly, associated with areas of hemorrhage and muscle fiber tearing (Fig. 18-1).[1,3] As the muscle injury becomes more severe, large areas of interfascial and subcutaneous hemorrhage are detected. The free edge of a completely disrupted muscle may be imaged floating in the anechogenic loculated fluid in the hematoma. Echogenic areas of clot are often imaged within the intramuscular, interfascial, or subcutaneous hematoma. As these echogenic clots become more organized, they may cast an acoustic shadow from the far surface. Partial muscle tears are more difficult to diagnose because the ultrasonographic abnormalities are subtler.[1] The normal striated muscle pattern is lost with increased echogenicity of the injured muscle.[1] Swelling of the affected muscle usually is present. Tears in the muscle fascia or thickening of the muscle fascia or fasciitis also may be detected ultrasonographically.[1]

Ultrasonography is an excellent tool for monitoring the resolution of the hematoma and the healing of the muscle.[1] The affected horse's exercise should be restricted until the hematoma has resolved and the muscle tear has filled in with tissue. As the muscle heals, the fluid becomes more echogenic

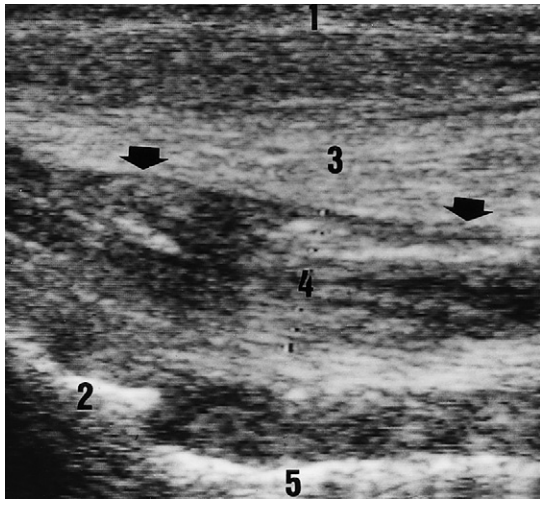

Fig. 17-14 Longitudinal ultrasound scan of the calcanean fasciculus of the medial collateral ligament of a 9-year-old Three-Day Event gelding after injury 3 months previously. This structure *(black arrows)* is thickened and has architectural changes. The diagnosis is desmopathy of the calcanean fasciculus of the medial collateral ligament. *1*, Skin; *2*, medial malleolus of the tibia; *3*, long medial collateral ligament; *4*, short medial collateral ligament: calcanean fasciculus (*arrowheads*); *5*, medial aspect of the talus.

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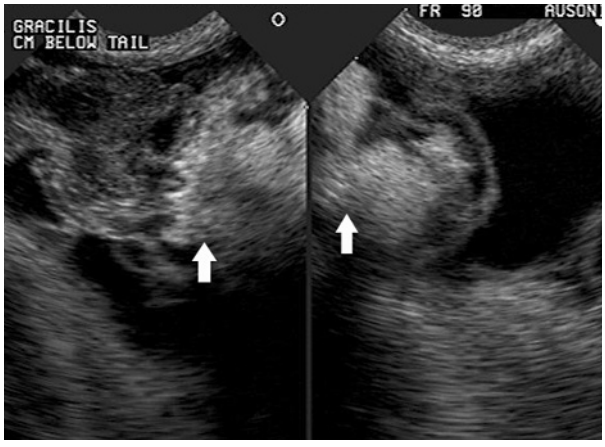

Fig. 18-1 Longitudinal *(left)* and transverse *(right)* ultrasonographic images of a torn gracilis muscle showing small, anechoic fluid-filled areas, hypoechoic amorphous areas, and more normal striated muscle at the periphery of the images. The anechogenic areas and hypoechogenic amorphous area are consistent with a hematoma replacing the muscle. The anechogenic area is the fluid component of the hematoma, whereas the hypoechoic areas *(arrows)* represent the fibrin and clot.

and the area fills in. The healing muscle may become more heterogeneous. Hyperechogenic areas of muscle scarring are seen associated with fibrotic myopathy, especially in the semitendinosus and semimembranosus muscles.[1] As these areas of fibrosis become more organized, acoustic shadowing from the far side of the fibrotic area may be detected. Hyperechogenic areas casting acoustic shadows are seen in horses with an ossifying myopathy and frequently are found adjacent to areas of fibrotic myopathy.[1] Areas of mineralization develop as scattered, pinpoint hyperechogenic areas that progressively become linear and result in acoustic shadowing from the near side of the areas.

Post-anesthetic myopathy results in increased echogenicity of the affected muscle, with loss of the normal muscle striations.[1,2] Muscle edema may result in the muscle appearing less echogenic than normal if the lesion is evaluated early in the course of the disease. Once a large influx of inflammatory cells has occurred, the affected muscle becomes more echogenic. A necrotizing myositis usually results in a more heterogeneous ultrasonographic appearance associated with the inflammatory cell infiltrate and bacterial infection.[1] Cavitation of a severely affected muscle is associated with liquefactive necrosis. Hyperechoic echoes, associated with the local production of gas by bacteria, may be seen with an anaerobic necrotizing myositis.[1]

Muscle tumors are rare; the most common muscle tumor is hemangiosarcoma. Discrete echogenic masses in the muscle or anechogenic loculated, more heterogeneous masses may be detected.[1] Central anechogenic or hypoechogenic areas representing tumor necrosis are identified in large or rapidly growing tumor masses. Primary muscle tumors such as rhabdomyosarcomas are extremely rare. Accurate identification of tumor type in muscle necessitates obtaining an ultrasound-guided biopsy and histopathological evaluation of the tissue obtained.

NERVE

Distinguishing peripheral nerves requires high-resolution images because the majority of nerves are small. Accurate knowledge of the locations of the nerves and the surrounding landmarks is important for identification. The nerves usually are slightly more echogenic than the surrounding soft tissue structures and are round to oval in transverse section.[1] Enlargement of the nerve with an increase or decrease in its echogenicity, with or without heterogeneity in its ultrasonographic appearance, is consistent with neuritis,[1] which may be a primary cause of lameness (see Chapter 83).

PENETRATING INJURIES

Ultrasonographic examination may be helpful in assessing penetrating injuries. The examiner should start at the site of the puncture wound and follow the path left by the penetrating object, a hyperechogenic tubular tract extending into the soft tissues.[4,5] The tract appears hyperechogenic from gas lining the tract. Any foreign material usually is hyperechogenic and casts acoustic shadows in two mutually perpendicular planes. Foreign material is usually irregular in shape, differs in size when imaged in two mutually perpendicular planes, and typically has high acoustic impedance relative to soft tissue.

DRAINING TRACTS

The cause of draining tracts should be determined systematically before a contrast radiographic examination is performed. Usually the source of drainage can be confirmed by ultrasonography if a thorough ultrasonographic examination is performed, eliminating the need to perform a contrast study. The injection of contrast material into the tract also results in the injection of air. This air may limit or prevent evaluation of foreign bodies within the tract because air is a nearly perfect reflector of ultrasound waves. The air that is injected with the contrast material may mix with the fluid within the tract, surround the foreign body, and obscure it from view.

A draining tract should be scanned systemically, keeping in mind that more than one factor may cause drainage. The clinician should start at the skin surface and follow the tract that usually appears as hypoechogenic tissue that is oval to round in transverse section and somewhat tubular in longitudinal section. The draining tract usually is easiest to follow in transverse section, unless the tract is short and straight.[1] The draining tract should be kept in the center of the ultrasound screen as it is followed into the deeper tissues. The tract may be single, or multiple tracts may be present. Often the tract is tortuous, and each branch of the tract should be followed to its source.[1] The source of the drainage at the end of the tract or tracts should be identified. Chronic draining tracts can originate from an area of osteitis or osteomyelitis; a sequestrum, foreign body, or necrotic tissue acting as a foreign body; an abscess; or a synovial structure.[1,4] Once the source or sources of the drainage have been identified, appropriate decisions can be made regarding medical or surgical management.

FOREIGN BODIES

Foreign bodies vary in ultrasonographic appearance.[4-13] The most commonly detected foreign bodies include suture material, wood, plant material, lead, and glass. Sequestered pieces of bone, hoof that becomes embedded in the soft tissues, and necrotic tissue also can create a foreign body reaction. Suture material usually is linear or tubular, hyperechogenic, and casts an acoustic shadow. Wood usually appears as a linear hyperechogenic structure casting a strong acoustic shadow from its near surface.[4,5,13] Plant material tends to appear as small echogenic material that either casts a weak acoustic shadow

or no acoustic shadow. Embedded hoof appears as a hypoechogenic to echogenic structure that often casts no acoustic shadow, or only a weak acoustic shadow is seen. The tubular composition of the hoof wall can sometimes be appreciated during the examination.

The ultrasonographic examination can be used to substantially shorten surgical time by accurately identifying the foreign body locations and identifying the relevant adjacent structures.[4-12,14,15] The surgical approach can be made directly over the foreign body. The foreign bodies should be measured ultrasonographically in two mutually perpendicular planes preoperatively and then compared with the foreign bodies removed at surgery, helping the surgeon to confirm that all foreign material has been removed. Intra-operative localization of the foreign body also can be performed, if localization of the foreign body proves difficult at surgery, aiding the surgeon in its rapid removal.[4,5,8,9,15]

BONE

A bone surface appears as a smooth, uniform-thickness hyperechogenic line that casts an acoustic shadow from its near surface.[1,4,12] Normal bony protuberances appear as bony shelves that are continuous with the parent portion of the bone. The continuity of an irregular bony surface echo can be followed ultrasonographically in a least one plane, differentiating this irregularity from a fracture. Vascular channels create breaks in the normal bony surface echo at the site of vessel penetration.

Fractures

Ultrasonography is useful in the diagnosis of fractures in areas that cannot be assessed radiographically, such as the withers, scapula, humerus, ribs, pelvis, femur and tibia, and in finding fractures that are suspected but have not been detected with routine radiography.[4,15-22] The ultrasonographic diagnosis of fractures depends on imaging the fracture line or fracture fragment in two mutually perpendicular planes.[1] A nondisplaced fracture is seen as a break in the normal hyperechogenic bone surface echo (Fig. 18-2). Detection of a hyperechogenic bony structure distracted from the underlying parent bone in two mutually perpendicular planes is consistent with a displaced fracture fragment. In an acute fracture, anechogenic fluid with hypoechogenic to echogenic loculations in the surrounding soft tissues is present. Echogenic masses of all shapes (frequently oval) within the loculated fluid are consistent with clot. These masses usually cast an acoustic shadow from the far surface as they become more organized. Disruption of the surrounding musculature frequently is seen if a fracture is significantly displaced.

Osteitis and Osteomyelitis

The detection of an extra-articular fluid layer, immediately adjacent to bone and without an intervening soft tissue layer, is diagnostic of osteitis or osteomyelitis, unless the area has sustained recent trauma[1,12] (Fig. 18-3). This ultrasonographic sign can be detected in horses with acute osteitis or osteomyelitis and precedes the detection of radiographic changes by 10 to 14 days. Soft tissue swelling often is present in the tissues adjacent to the osteitis or osteomyelitis. In horses with early osteitis and osteomyelitis the bony surface echo immediately adjacent to the fluid layer usually has a normal ultrasonographic appearance. With increasing chronicity, irregularities are detected in the bony surface echoes that are also detectable radiographically. Thinning of the bony surface echo with scooped-out areas in the underlying bone is consistent with lysis. Irregular hyperechogenic bony spicules correspond to areas of bony proliferative change. Sequestrae are imaged as hyperechogenic fragments that cast an acoustic shadow, are surrounded by anechogenic to hypoechogenic fluid, and are distracted away from the parent bone.[1,4,12] A linear wooden foreign body can mimic the ultrasonographic appearance of a sequestrum.

Ultrasonography is useful for the diagnosis of osteitis or osteomyelitis in horses with fractures where the radiographic signs of fracture healing can be difficult to distinguish from those of infection.[1,4,12] Bony lysis is a component of both fracture healing and infection. Immediately after trauma, or in the immediate post-operative period, fluid is detected adjacent to the bone for several days or longer until the local hemorrhage associated with the original injury or surgical trauma has resolved. A soft tissue layer should then cover the bone in all extra-articular locations.

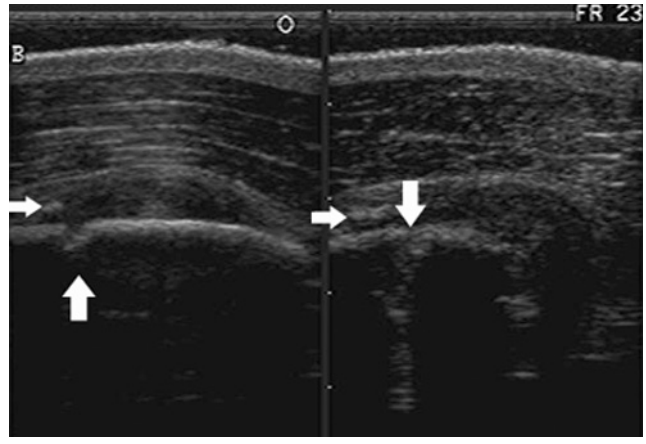

Fig. 18-2 Longitudinal *(left)* and transverse *(right)* ultrasonographic images of a fractured eighteenth rib. The fracture is nondisplaced with the crack clearly visible in the bone in two mutually perpendicular planes *(large arrows)*. A small fragment *(left small arrow)* also is visible at the fracture site in addition to hypoechogenic fluid representing hemorrhage *(right small arrow)*.

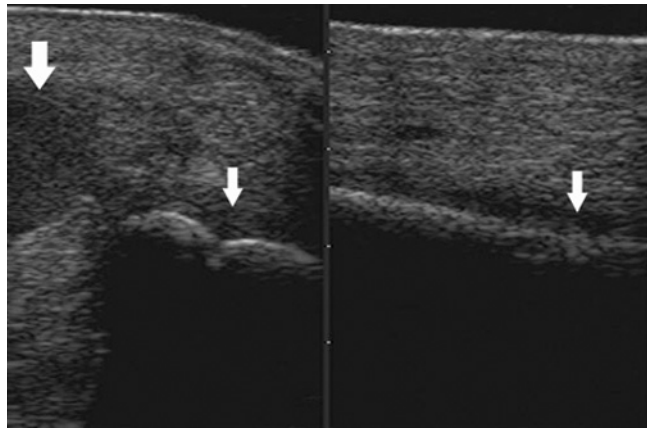

Fig. 18-3 Transverse *(left)* and longitudinal *(right)* ultrasonographic images of the mid metacarpal region of a horse with an acute onset of swelling, fever and lameness associated with osteitis of the second (McII) and third (McIII) metacarpal bones. A large amount of hypoechoic fluid in the subcutaneous tissues extends down to McII and McIII in the mid-metacarpal region. A small hypoechoic fluid layer overlies McII and McIII and is indicative of osteitis *(small arrows)* communicating with a larger subcutaneous area of hypoechoic fluid *(large arrow)*. The echo from McII and McIII is slightly thicker than normal, consistent with the infection of the bone.

The detection of a fluid layer directly overlying the bone in horses with suspected osteitis or osteomyelitis should prompt ultrasound-guided aspiration of the fluid, with or without biopsy of the area and submission of the fluid or tissue obtained for culture and sensitivity testing and histopathological examination (tissue). If surgical curettage of the affected area is chosen. this can be performed at the time of surgery. Horses that receive long-term antimicrobial drugs for osteitis or osteomyelitis should be re-evaluated ultrasonographically before discontinuing the treatment. A soft tissue layer should be present over the affected bone without any intervening fluid.[1] If treatment is discontinued while a fluid layer remains overlying the bone, recurrence of the osteitis or osteomyelitis is likely.

Bone Abscess

A bone abscess may be imaged ultrasonographically as a hypoechogenic, scooped-out area in the cortical and medullary regions of the bones[1] and usually is seen in foals, often associated with *Rhodococcus equi* infections. Large adjacent subcutaneous abscesses may communicate with the bone abscess. A dynamic ultrasonographic examination displacing the fluid within the subcutaneous abscess may help to identify the defect in the underlying cortical bone.

Implants

The cause of a worsening lameness in horses with implants may be difficult to determine from the physical examination and radiographic findings. Ultrasonographic examination can provide valuable information about the implant, bone, and surrounding soft tissue structures. In horses with surgical implants the detection of a fluid layer immediately adjacent to the implant, without an intervening soft tissue layer, is diagnostic of infection.[1] Infection of the implant, infection of the adjacent synovial structures, or problems associated with the implant itself often can be differentiated ultrasonographically. In horses in which fetlock or pastern arthrodesis was performed, worsening of lameness may be caused by infectious tenosynovitis of the digital flexor tendon sheath that can be diagnosed ultrasonographically as hypoechogenic effusion within the sheath. Problems with screw placement can be identified as a probable cause of lameness, such as when a screw is inadvertently located within the deep digital flexor tendon, causing fiber disruption and tendonitis. The various implant materials (plate, screw, wire, or antimicrobial-impregnated polymethylmethacrylate bead) can be differentiated ultrasonographically by characteristic appearances. Thus the most likely cause of the patient's deterioration can be identified and treatment targeted appropriately.

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CHAPTER • 19

Nuclear Medicine

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GENERAL CONSIDERATIONS

Nuclear medicine is a relatively recent advance in diagnostic imaging of the horse, pioneered by Ueltschi[1] in Europe and Devous and Twardock[2,3] in the United States. Seeherman et al.[4] and Lamb and Koblik[5] further demonstrated the value of bone scintigraphy in the evaluation of sports horses.

Bone scintigraphy is a way to reach within the horse and extract clinically useful and relevant information and helps to answer many lameness questions that we previously could not answer. Scintigraphy is used to assess the current status of known radiographic abnormalities, pursue diagnosis in horses with negative or equivocal radiographs, screen horses with obscure forelimb or hindlimb lameness, and evaluate horses with poor performance. However, a gamma camera is not an answer machine, and it is critically important to correlate scintigraphic findings with clinical lameness examination findings. Scintigraphy can play a role as a screening tool but should never replace diagnostic analgesia. In fractious or highly strung horses with hindlimb lameness, scintigraphic examination may be used to provide a diagnosis, but clinical relevance still should be confirmed.

Nuclear medicine involves the in vivo or in vitro use of radioisotopes in the diagnosis and management of clinical disease. Several terms are used synonymously with nuclear medicine, including nuclear scintigraphy, bone scintigraphy, and gamma scintigraphy, and although these terms differ slightly, for horses most clinicians refer to bone scintigraphy, the technique most commonly performed. Bone scintigraphy is highly sensitive compared with radiography. It can detect as little as 10^{-13} g of radiopharmaceutical in bone, whereas changes measured in grams must occur before a lesion can be detected using radiography.[3] Important factors that decrease sensitivity include time from radiopharmaceutical administration to image acquisition, body part to camera distance, shielding, motion, time from injury to image acquisition, ambient temperature and peripheral perfusion, and amount of background radiation. Specificity is low compared with other modalities because disparate diseases can similarly alter blood flow and binding sites in bone. Direct trauma (osteitis) and fracture; stress-related bone injury, including fracture and osteoarthritis; infection (infectious osteitis and less frequently osteomyelitis); osteochondrosis; and neoplasia are in theory difficult to differentiate scintigraphically. Accuracy can be improved by acquiring several images from different perspectives, minimizing factors affecting sensitivity, and knowing the history. For example, a focal area of increased radiopharmaceutical uptake (IRU) in the caudolateral tibial cortex of a 2-year-old Thoroughbred (TB) filly would undoubtedly represent stress-related bone injury rather than a rare bone tumor.

Radiographs depict activity in bone that has already occurred in the past several days to years. Scintigraphy is a *functional* evaluation of bone at the time of imaging. Scintigraphic evidence of bone activity means active bone formation is occurring that might take weeks to be visible radiographically. Therefore a major advantage of scintigraphy is *early detection* of bone injury. Scintigraphy will unlikely accurately reflect changes in bone that occurred longer than 3 to 4 months before imaging. Horses given substantial rest before examination are not good candidates unless the examination is a follow-up to assess healing.

RADIOISOTOPE AND RADIOPHARMACEUTICAL

A radioisotope emits radiation (particles) that is captured using a scintillation camera. Radioisotopes such as ^{111}In are used occasionally, but the most common and useful radioisotope is technetium-99m (^{99m}Tc). ^{99m}Tc, a short-lived (metastable) radioisotope with a half-life of 6 hours, is ideal for radiation safety and animal retention. ^{99m}Tc is excreted almost entirely through the kidneys, so containing and monitoring urine is extremely important. ^{99m}Tc is produced when ^{99}Mo decays to ^{99}Tc. The metastable (^{99m}Tc) radioisotope gives off a gamma ray (140 keV) that is used for imaging. Commercially, ^{99}Mo/^{99m}Tc generators can be purchased for use in large hospitals, but an alternative, cost-effective method is the daily purchase of individual doses, a practice that obviates the need to house generators. Directly from the generator, ^{99m}Tc is in the ionic form of sodium ^{99m}Tc pertechnetate ($Na^{99m}TcO_4$) that can be injected or mixed with a bone-seeking agent or pharmaceutical. Radiation is measured in curies (Ci) or millicuries (mCi), or becquerels (Bq), megabecquerels (MBq), and gigabecquerels (GBq) have been used. One mCi is equal to 37 MBq. The recommended dose of ^{99m}Tc is 0.4 to 0.5 mCi (14.8 to 18.5 MBq)/kg, totaling 150 to 200 mCi (5.5 to 7.4 GBq) per horse. Low doses reduce radiation exposure but prolong acquisition time or may result in inadequate image quality if insufficient counts are obtained. High doses may actually reduce overall radiation exposure by limiting exposure time.

$Na^{99m}TcO_4$ can be injected intravenously directly, but only flow and pool-phase studies can be performed. For most equine studies, $Na^{99m}TcO_4$ is mixed with a pharmaceutical. For bone, ^{99m}Tc is bound to methylene diphosphonate (MDP), or hydroxymethane/hydroxymethylene diphosphonate (HDP/HMDP). MDP is slightly less expensive and is the most common pharmaceutical used worldwide, but we prefer HDP, because the kidneys clear it slightly faster, allowing early acquisition of delayed-phase images. Henceforth MDP and HDP are used interchangeably.

The exact mechanism of binding of ^{99m}Tc-HDP to bone remains unclear. ^{99m}Tc-HDP is thought to bind to exposed sites on the inorganic hydroxyapatite crystal. Binding sites are exposed under normal and pathological conditions in areas of actively remodeling bone or in soft tissues undergoing mineralization.[6,7] ^{99m}Tc-MDP uptake occurs by the processes of chemical adsorption onto, and by direct integration into, the crystalline structure.[8] Other possible mechanisms to account for increased uptake include incorporation into the organic matrix or local hypervascularity.[7] In rat models, ^{99m}Tc was found to be incorporated into the organic matrix rather than the inorganic portion of newly formed bone.[9,10]

Radiopharmaceutical may dissociate with incorporation of ^{99m}Tc and MDP individually into the organic and inorganic phases, respectively.[11] ^{99m}Tc-MDP adsorption might depend on pH and the presence of phosphates, calcium compounds, and other cations.[12]

Accumulation of ^{99m}Tc-MDP is not simply the result from changes in local blood flow, although blood flow is likely increased in sites of actively remodeling bone. Although increased blood flow does not significantly affect a bone scan,[6] *adequate* blood flow is necessary to deliver radiopharmaceutical to available binding sites in bone. Decreased blood flow, caused by infarction or ischemia, can greatly affect a bone scan but is seldom a clinical problem. However, decreased peripheral blood flow in old horses or those imaged in cold weather or on days with high diurnal temperature change can adversely affect image quality.

The most important aspects of ^{99m}Tc-MDP binding relate to timing of the scan and the stage of bone formation or modeling. In actively remodeling bone, osteoclastic activity predominates during bone resorption, whereas osteoblastic activity dominates during bone modeling. Modeling occurs independently or in conjunction with remodeling in cancellous and cortical bone. Histological and scintigraphic findings were evaluated in a rat tibial evacuation model, and ^{99m}Tc-MDP was found to bind to sites of *active* calcification, most prevalent 12 days after injury.[13] ^{99m}Tc-labeled phosphonates were identified during bone formation and ongoing resorption was not necessary for increased uptake to occur.[8] ^{99m}Tc-MDP accumulated in areas of calcification or in fixed bone fragments,[8,14] and accumulation was mediated by osteoblastic activity.[8] The high sensitivity of bone scintigraphy is attributed to increased osteoblast activity that precedes morphological changes visible radiographically.[15] Other mechanisms may exist, however, because positive bone scan results may be seen in human beings with diseases such as osteomalacia, in which high bone matrix turnover and failure of calcification occur.[13]

Site and stage of binding are important from a clinical perspective. Binding sites for ^{99m}Tc-MDP are created by osteoblast activity during bone modeling, and maximal IRU occurs 8 or 12 days after bone injury.[13,16] An acute fracture caused by direct trauma may not be scintigraphically evident for several days. Acute, traumatic injury differs from stress-related bone injury, because the latter, particularly common in racehorses, results from a continuum of bone changes that might lead to stress or catastrophic fractures and osteoarthritis. Microfracture, periosteal callus, and subchondral bone damage precede the development of stress or complete fracture in the dorsal cortex and distal articular surface of the third metacarpal bone (McIII), the third metatarsal bone (MtIII), humerus, tibia, and pelvis.[17-19] In horses with acute lameness from stress-related bone injury, bone scan findings usually are immediately positive because bone modeling is ongoing. In horses with stress-related bone injury a bone scan result is positive long before catastrophic fracture occurs, an important advantage of scintigraphy compared with radiography. In horses with traumatic injury, such as an acute pelvic or other upper limb fracture, a false-negative scan may result from lack of modeling. Other factors resulting in false-negative results include distance, shielding, high background activity, and motion. For example, a horse developed acute hindlimb lameness during hospitalization, and suspecting a pelvic fracture involving the acetabulum, clinicians performed scintigraphic examination on day 2, but the scan result was negative. On day 9 a faint IRU appeared consistent with fracture.

In horses with stress-related bone injury and traumatic injury a considerable decrease in radiopharmaceutical uptake occurs within 6 to 8 weeks after injury. Decreased intensity after fracture varies with the specific bone and fracture type. The ideal time to image is 10 days to 8 weeks after injury.

IMAGING EQUIPMENT

Scintigraphy can be performed in two ways, but the most common and useful method is acquisition of two-dimensional images with a gamma camera. Alternatively, a handheld probe is used to acquire count density computer-generated charts, or graphs are created to determine IRU. Known as *probe point counting*, this form of scintigraphic examination was first introduced in 1984.[20,21] Some probes are like miniature gamma cameras built with a single photomultiplier tube. Crystal probe detectors are more expensive than the photomultiplier detectors, but they are small and easily used on the body surface, or per rectum in horses with suspected pelvic fracture.[21] Probe point counting is done with a substantially lower radioisotope dose, minimizing cost and radiation exposure. Probe point equipment is inexpensive. Because the probe is placed directly on the skin surface, body part to probe distance is minimal. A gamma camera is often difficult to get close to body surface, a problem that can prolong acquisition time and decrease image quality. A good correlation appears between results of probe point counting and gamma camera imaging in acute injuries in young horses, but in older horses the technique has limited sensitivity. However, quantitative information is only available as a histogram, and an actual image allowing qualitative assessment is not obtained.

Gamma camera images are obtained in analog or digital form, and permanent hard copies of a two-dimensional scintigraphic image are generated. In people, one or several gamma cameras operating simultaneously can be used to generate a cross-sectional image similar to computed tomography (CT) or magnetic resonance imaging (MRI). This is known as single photon emission computed tomography (SPECT) and photon emission tomography (PET). Although SPECT and PET imaging are theoretically possible in anesthetized horses, we are not aware that the techniques have been performed.

A reconditioned gamma camera is perfectly satisfactory, less expensive than new equipment, and durable, often being useful for at least 7 to 10 years. Gamma cameras are either large field of view (LFOV) or small field of view (SFOV) based simply on the size (Figs. 19-1 to 19-3). LFOV cameras can be rectangular or circular in shape, but crystals for rectangular cameras are more costly. When adapting equipment for use in horses, an important consideration is how to move the camera safely and easily to the horse and vice versa. Most standard gantry (support structure of the gamma camera) designs are unsuitable for equine imaging, because good-quality images can be obtained only by having the body part close to the camera. The number of gamma rays reaching the detector is inversely proportional to the square of the distance (inverse square law). To quickly obtain dorsal and lateral, and lateral and plantar images of the forelimbs and hindlimbs, respectively, the camera must be able to be lowered below floor level (see Fig. 19-3). Alternatively, the horse must be positioned on an elevated platform. Limbs can be held manually near the camera, but this practice increases radiation exposure to personnel and should be avoided whenever possible. Suitable gantries are commercially available or can be custom made. A lead collimator is also required.

IMAGE ACQUISITION

For flow and pool-phase images, horses are sedated and positioned in front of the camera and ^{99m}Tc-HDP is injected intravenously. Delayed-phase images are then obtained 2 to 4 hours later. Scintigraphic images are produced when the horse, now the radiation source, emits gamma rays from normal and abnormal bone. The gamma rays must pass through overlying tissues and traverse the distance between the body part and the

Fig. 19-1 The nuclear medicine facility at New Bolton Center, University of Pennsylvania, has large field of view and small field of view cameras. A custom-made central column overhead gantry holds the rectangular large field of view camera, which can be lowered below floor level to image the distal extremities.

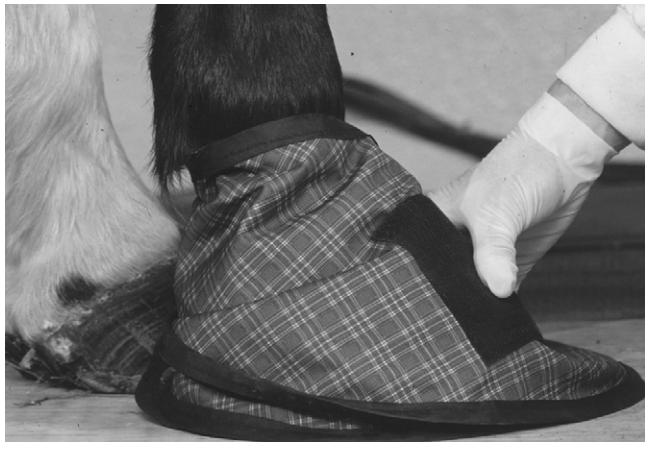

Fig. 19-2 The small field of view camera in the floor allows easy acquisition of solar images of the foot. After a wooden cover is applied, the horse stands over the small field of view camera for the solar image. A lead-lined wrap placed around the coronary band region shields the foot and camera from radiation emanating from the more proximal aspects of the affected limb.

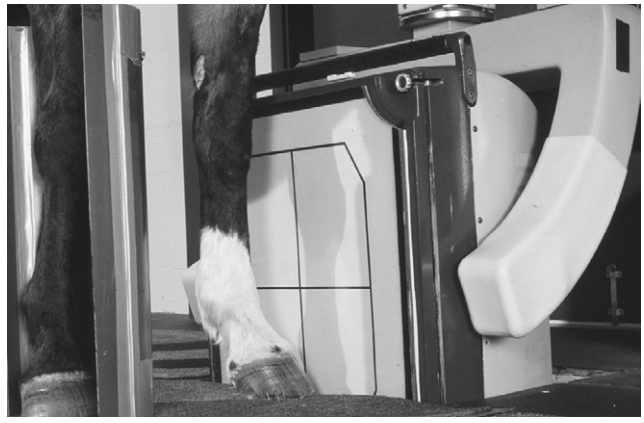

Fig. 19-3 Construction of a recess or pit into which the large field of view camera can be lowered allows imaging of the distal extremities with minimal movement of the horse. The lateral aspect of the hoof touches the face of the camera (clear polycarbonate protects the collimator). This position minimizes distance, improves image quality, and decreases acquisition time. A curved, lead-lined shield protects the camera and left forelimb from radiation emitted by the right forelimb.

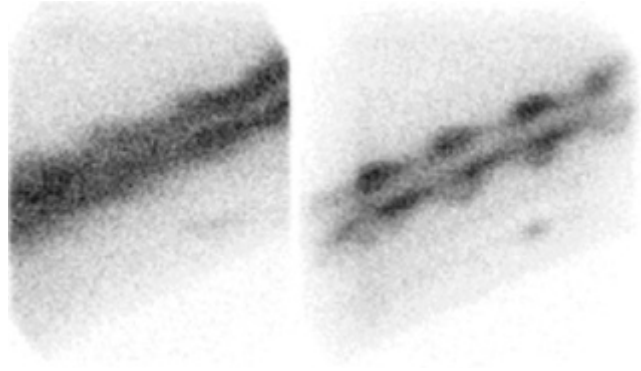

Fig. 19-4 Original uncorrected (*left*) and corrected (*right*) right lateral images of the cervical vertebrae. Motion correction allows acquisition of more counts and reduces the harmful effects of motion on image quality, particularly of the upper limbs and axial skeleton.

camera. An area of IRU emits more gamma rays than does adjacent normal bone and contributes more counts to the scan. Sites farther from the camera, those being shielded by overlying soft tissue or adjacent bone, or those in areas of high background activity may not be visible, because they may not contribute enough gamma rays. Gamma rays strike a sodium iodide crystal, and scintillations produced are detected and amplified by photomultiplier tubes that subsequently transmit information through electronic circuits. The image is displayed on an oscilloscope or sent directly to a film processor, called analog imaging. Alternatively, information is transmitted to a computer, stored digitally, manipulated, and subsequently sent to a film processor or printer.

An important recent advancement is the ready availability of user-friendly computer programs to acquire, analyze, store, and process images. Systems based on Apple, Windows, and Unix are currently available and are straightforward. Motion correction software is the latest, exciting advance that improves upper limb and axial skeleton image quality by negating the effects of motion and allowing higher count numbers than with conventional software (Fig. 19-4). Modern software also allows post-processing of images, for example, masking out the bladder, which might otherwise steal counts.

Images are obtained in either static or dynamic mode. Static images, most commonly obtained, are acquired using a predetermined number of counts per image. For example, because motion and soft tissue covering are limited in the distal limbs, good-quality images can be obtained using 100,000 to 150,000 counts per image. In general, increasing counts per image improves image quality, but to acquire more counts, time and motion become factors. Motion correction can add flexibility.

A fundamental principle of image interpretation is comparing images of one limb with those of the contralateral limb, but the clinician should keep in mind that both limbs, and for that matter all limbs, can be abnormal. To compare limbs accurately using this technique, body part to camera distance and camera position relative to the limb must be standardized between limbs. Time to acquire each image ranges from 30 to 90 seconds depending on radioisotope dose, type and age of horse, and ambient temperature. Time rather than count number can be standardized, assuming that limb perfusion

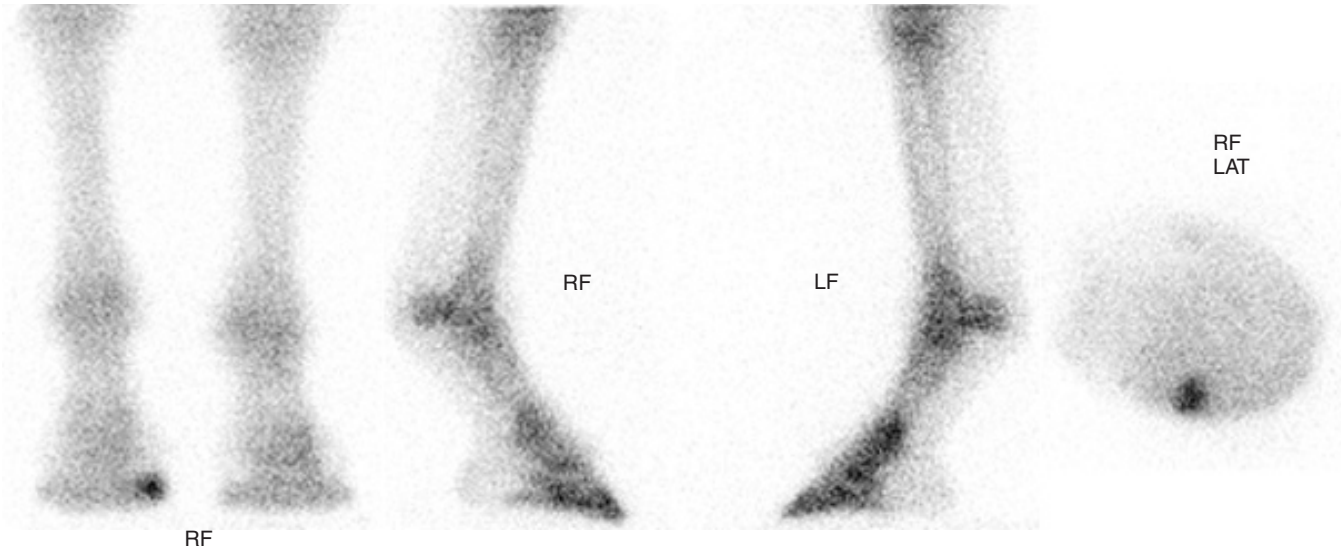

Fig. 19-5 Delayed-phase scintigraphic images of a horse with a fracture of the medial aspect of the right forelimb (*RF*) distal phalanx. The lateral *(LAT)* view of the right forelimb does not show the area of increased radiopharmaceutical uptake, and little difference is noted between both front feet. The dorsal and solar images, however, clearly show increased radiopharmaceutical uptake. It is extremely important to obtain more than one scintigraphic view. *LF,* Left forelimb.

is adequate and symmetrical. For example, lateral images of both metacarpal regions are obtained for 90 seconds and compared.

The number of views to obtain is based on the body part being imaged, but usually at least two views are required. In some areas this may not be possible, but the clinician should be aware that false-negative results can occur. A dorsal scintigraphic image only gives detailed information about the dorsal aspect of the limb, and abnormal palmar regions might be missed. The image and information are not the same as depicted in a dorsopalmar radiographic view. Similarly, areas of IRU on the medial side of the limb might not be visible on a lateral image. Areas more distant from the camera contribute substantially less radiation than those closer because of the inverse square law. Bone interposed between a medial lesion and the camera can effectively shield the site. A lateral view of a right forelimb failed to reveal a fracture of the distal phalanx (Fig. 19-5). Radiographic examination and dorsal and solar scintigraphic images, however, revealed an incomplete fracture that we completely missed in the lateral scintigraphic view. Additional views may be necessary for accurate diagnosis.

We prefer to localize lameness, because this allows for detailed examination of a specific area. For instance, routine screening views of the front digit include lateral and dorsal delayed images, but in horses in which lameness is abolished using palmar digital analgesia, pool-phase images and lateral, dorsal, solar, and occasionally medial, palmar, and flexed delayed images are obtained. Diagnostic accuracy can be improved by taking many views, and pinpointing a lesion using scintigraphy can allow a focused radiographic, ultrasonographic, CT, or MRI study to be performed. When augmenting the routine dorsal and plantar images of the metatarsophalangeal joint with flexed lateral and sometimes flexed dorsal and medial views, instead of saying, "There is IRU in the fetlock joint," the diagnostician can say, "There is focal IRU involving the distal, plantarolateral aspect of MtIII," a much more accurate description (Fig. 19-6).

Dynamic acquisition can be used before motion correction in delayed images or to evaluate blood flow. One- or 2-second per frame images are generated sequentially and can be evaluated individually or combined into a single composite image, with or without motion correction. First-pass angiography can be used to assess blood flow in the aorta, iliac, and femoral arteries[22] in horses with suspected thromboembolism (Fig. 19-7) or to assess blood flow in the distal limb.

Image Quality

Factors that contribute to poor image quality are related to the horse and to processing. Analog (old way) and digital (new way) processing provide good-quality images provided the equipment is working properly. Regular quality control is absolutely essential to maximize image quality and avoid artifacts. A cracked or poor-quality collimator, sodium iodide crystal damage, malfunction or incorrect tuning of photomultiplier tubes, age, and general condition of refurbished equipment can cause artifacts, gradual deterioration in quality, or completely shut down the process. The dose of radioactivity administered, the number of counts acquired, image distance (distance from body part to camera surface), motion, and background radiation are most critical. As many gamma rays as possible from the affected bone must reach the crystal without motion to maximize image quality.

Number of Counts

Image acquisition, principles of using count numbers as opposed to time, and the importance of standardizing images between limbs were discussed earlier. One limb may overall be hotter than the contralateral limb and count faster on individual images or contribute more counts when combined in images of both limbs. Horses with unilateral lesions in the digit, such as those with distal phalanx fractures and soft tissue injures, appear to have compensatory IRU normally in the rest of the limb. Our experience suggests that horses with distally located lesions in the affected limb often have more prominent normal uptake in that entire limb, but others suggest that the non-lame contralateral limb may be hotter because of increase in weight bearing.[23] An area of intense IRU present with a condylar fracture or stress-related bone injury may overwhelm the contribution of normal bone, making it difficult to see, a phenomenon called *count stealing.*

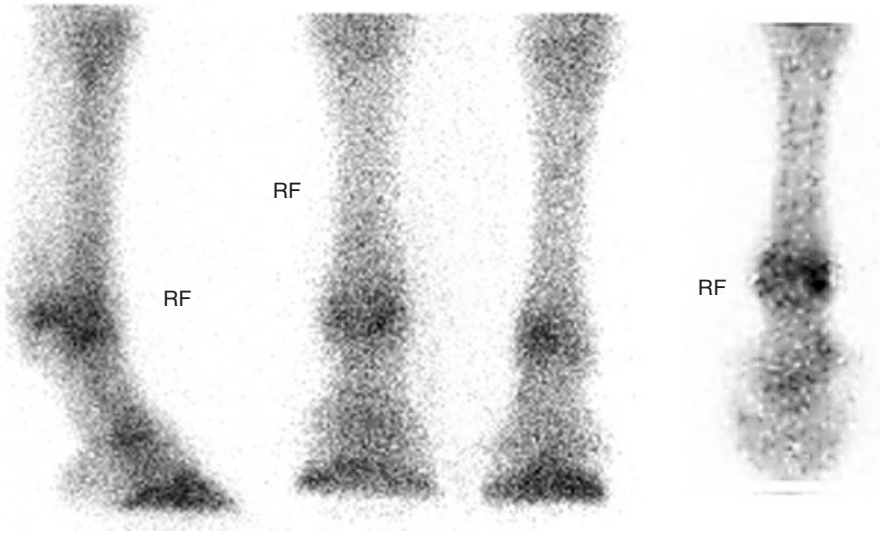

Fig. 19-6 Lateral *(left)*, dorsal *(middle)*, and flexed dorsal *(right)* delayed-phase scintigraphic images of the right forelimb *(RF)*. The flexed dorsal image best shows the focal area of increased radiopharmaceutical uptake involving the distal, medial aspect of the third metacarpal bone. Although increased radiopharmaceutical uptake appears in other views, the flexed dorsal image allows differentiation of increased radiopharmaceutical uptake involving the third metacarpal bone and the proximal phalanx.

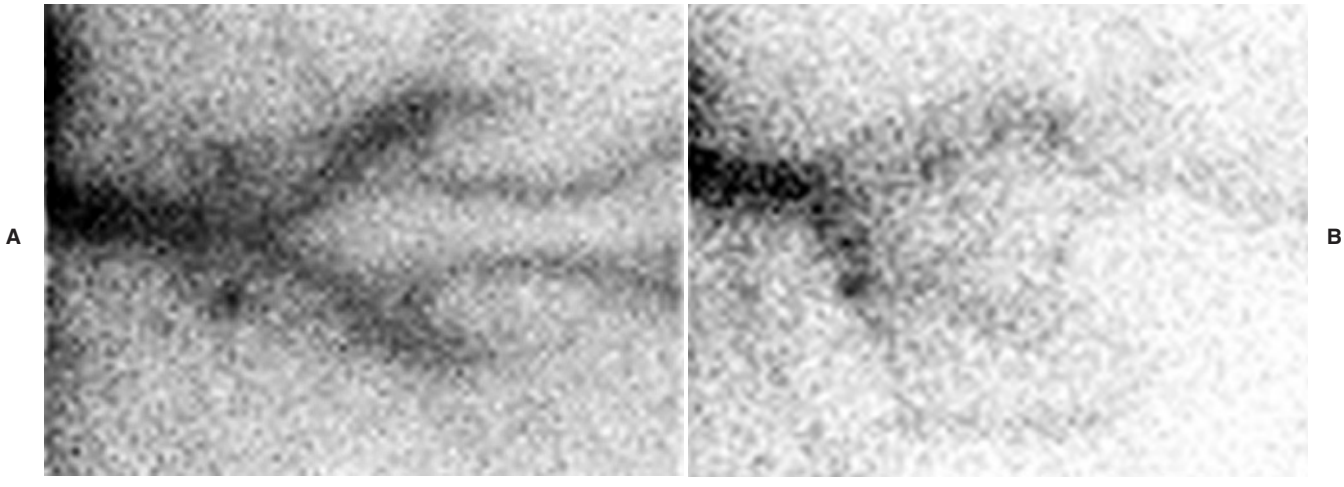

Fig. 19-7 Composite image generated after dynamic acquisition of 32 sequential images (2 seconds per frame) with the gamma camera positioned over the terminal aorta and branches. Imaging started 20 seconds after administration of ^{99m}Tc-hydroxymethane diphosphonate in the external jugular vein. **A,** In a normal horse the external iliac arteries and a linear caudal course of the internal iliac vessels show a V-shaped divergence. **B,** In this horse with aortoiliac thromboembolism, the direction and contour of the external iliac arteries is disrupted and the internal iliac vessels lack recognition.

Distance

Having the affected body part too far from the camera is likely the most important factor contributing to false-negative bone scan findings. Osteoarthritis and subchondral bone cysts involving the medial femorotibial joint can be completely missed unless a caudal view is obtained simply because the lesions cannot contribute enough counts to the scan to be visible. Distance is too great. Pelvic images are dramatically and negatively affected by distance. Even if the camera is resting on the skin of the rump, some bones are still many centimeters away.

Shielding

Shielding is important in radiation safety, but it can be a negative factor in image quality and interpretation. Shielding helps or harms image quality and interpretation. High background radiation reduces image quality, because unwanted tissues steal counts. Pool-phase images of the thoracolumbar spine and pelvis are rarely informative because within 1 to 2 minutes after injection, radiopharmaceutical in the kidneys and urinary bladder creates such high background levels of radiation that soft tissue lesions are missed. Delayed-phase pelvic images are similarly affected by urine retention in the

bladder. Bladder uptake causes poor image quality by creating high background levels of radiation and by superimposition on the sacrum in dorsal images. To avoid this problem, the bladder should be catheterized or a diuretic should be given. Lead shields can be placed on the skin between the bladder and camera and count subtraction techniques can be used to take away bladder counts, but maximizing image quality by initially limiting radioactive urine is best.

Internal shielding can be a problem for interpretation. Gamma rays originating from a lesion distant to the camera must traverse bone, interposed soft tissue, and air before striking the camera. In a lateral view of the stifle, gamma rays originating from subchondral bone of the distal medial femur must traverse the thick lateral condyle, dense ligaments, and other soft tissues, all the while competing with gamma rays from other sources. Intuitively it would seem that counts would be additive, meaning a lesion medially would add to the counts normally originating from the lateral aspect, clearly showing the abnormal area. Acquiring images with sufficient counts reduces the risk of false-negative scan results. Bone stacking, when bones are superimposed, may explain normal areas of IRU visible in a lateral view of the coxofemoral joint. However, often bone interposed between a lesion and the camera seems to provide unwanted shielding. Overlying skeletal muscle may also provide unwanted shielding in pelvic images. Asymmetrical muscling caused by gluteal atrophy can explain mild differences in IRU between left and right sides of the pelvis.

Motion

Two sources generate excess motion: movement of the camera or the horse. Swaying of cameras hung by hoists or chains and movement induced by the horse leaning on the camera result in poor image quality. In the distal limbs, horse movement is usually not a factor and acquisition times are short. Horses sway minimally when scanning the distal limbs, but sway excursion is large in the upper limbs because of a longer pendulum effect from the anchor point (feet). Sedation may accentuate the effect. Motion detracts greatly from upper limb and axial skeleton image quality. We use detomidine (0.008 to 0.01 mg/kg) and butorphanol (0.006 to 0.075 mg/kg), a combination that allows optimal acquisition while minimizing motion. Manual support on each side and resting the horse's head on a stationary object partially negates the tendency of the horse to sway and drift. Motion correction software is useful in improving image quality but cannot compensate for all movement.

Background Radiation

High background radiation contributing to count stealing and handling bladder interference have been discussed. Shielding the limb or body being imaged with custom-made lead-lined barricades (see Fig. 19-3) or placing drapes on the horse or around the body part (see Fig. 19-2) helps isolate the region being examined. Old horses, large breed, and fat horses and those with extensive swelling or fibrosis in the distal limbs often have high background activity in soft tissues, which inevitably results in poor-quality delayed images.

Poor-Quality Bone Uptake

Image quality in large breed horses such as TB crosses, Warmbloods, and Draft horses or in old horses may be poor despite following accepted protocol. Average image quality in large breed horses may be worse than in young racehorses for many reasons, but age and type of exercise might explain some differences. Large breed performance horses tend to be older than the racehorse population, lack normal physeal activity, and often have dense soft tissues and large bones in the distal limbs. Exercise type and level in this type of horse is much different than in racehorses. A relative lack of stress-related bone injury in subchondral and cortical bone limits the number and type of scintigraphic changes seen.

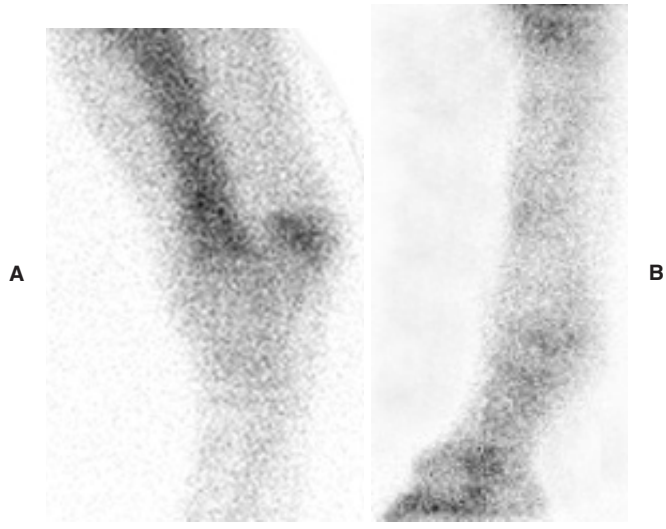

Fig. 19-8 **A,** Delayed-phase image of an aged Warmblood gelding shows reasonable quality bone upake in the proximal limb and diffuse moderate increased radiopharmaceutical uptake in the distal tibia. **B,** This delayed-phase image is consistent with the pool-phase scan of the tarsus and distal extremity. This abnormal pattern of poor bone uptake commonly appears in horses scanned in cold weather or in large breed horses.

Image quality in all types of horses may be compromised in areas with cold weather or great diurnal temperature differences, especially in fall, winter, and early spring.[24-26] Generally reduced uptake of radiopharmaceutical occurs distal to the antebrachium and crus, either bilaterally symmetrical or asymmetrical. Delayed-phase images taken above the carpus and tarsus have high background radiation but can be interpreted. A compensatory area of intense IRU may occur in the distal radius and tibia. Distally, from the carpus and tarsus to the sole of the foot, images resemble pool-phase images even when acquired 3 hours after injection (Fig. 19-8). Occasionally a different pattern appears with compensatory focal intense IRU in the distal radius and tibia, relative photopenia of the carpal and tarsal bones, and reduced to normal or interspersed (spotty) uptake in the metacarpal/metatarsal regions and digit. This phenomenon is not related to renal clearance, radiopharmaceutical affinity, or dose of radioisotope but appears to be caused by poor peripheral perfusion.[26] Normal horses appear to be able to shunt blood away from the periphery, presumably a homeostatic mechanism involving high sympathetic tone during times of abrupt drop in ambient temperature. Other related factors that might increase sympathetic tone are dehydration, stress, and transporting the horse shortly before injection of radiopharmaceutical. High sympathetic tone may reduce blood flow in the distal extremity, and the radioisotope may be unable to reach available binding sites in bone. Blood supply must be *adequate* to ensure distribution of radiopharmaceutical to sites of osteoblast activity. Persistence of radiopharmaceutical in soft tissues and vessels in the distal extremity and occasional photopenia of the carpal, tarsal, or other bones cannot be explained.

We prefer that all horses be hospitalized the night before examination to avoid the problem of poor-quality bone uptake and maximize image quality. This may reduce stress and improve hydration status. Keeping the distal limbs warm with bandages, and when the temperature is cold, applying a

body blanket to maintain core temperature may help. In a recent study, Dyson et al.[26] found that exercise, but not warming, before injection was most important in improving peripheral blood flow and maximizing image quality in all types of horses. Horses are lunged or trotted on a treadmill for 15 to 20 minutes before injection. When we suspect poor-quality scans may result, we administer acetylpromazine (15 to 20 mg per adult horse) before injection. If poor-quality images result despite following these procedures, the procedures should be repeated the next day.

RADIATION SAFETY

Rules for radiation safety and licenses to obtain and house radioactive materials differ for each state and country. In Pennsylvania a license to use radioactive materials must be obtained from the federal Nuclear Regulatory Commission (NRC). Horses are quarantined for 24 hours after injection and can then leave with instructions to avoid unnecessary contact with them for an additional 36 hours. Radiation levels at the horse's body surface range from 0.4 to 0.7 mR/hr at 24 hours after injection, well below the trigger level of 2.0 mR/hr established by the NRC. Horses are kept in defined stalls in which solid waste removal (stall drains are plugged; shavings are used for bedding) is used. Waste is kept for 60 hours, at which time radiation levels do not differ from background.

We practice the concept of ALARA (as low as reasonably achievable) and attempt to maximize distance from horse to handler and minimize total imaging time. Persons handling radioactive materials wear body and ring badges, and all those in contact with horses wear body badges. Total yearly radiation exposure for full-time technicians is approximately 500 mR/yr (450 horses scanned each year). Daily monitoring of selected sites and weekly wipe tests are performed. Nuclear medicine techniques in horses can be done safely, within NRC or other supervisory guidelines, without undue contamination or exposure.

BONE SCAN PHASES

The bone scan is divided into three phases named for the tissues in which the majority of radiopharmaceutical resides at that time. Flow-phase (phase I, vascular phase) images are obtained for 1 to 3 minutes after injection, during which time radiopharmaceutical resides in blood vessels. First-pass studies of the heart and major central and peripheral vessels can be performed (see Fig. 19-7). The flow phase appears to last longer in horses than in people, because radiopharmaceutical persists in distal limb veins often for greater than 10 to 15 minutes, a finding that can complicate interpretation and potentially decrease sensitivity of pool-phase images. Pool-phase (phase II, soft tissue phase) images are classically obtained between 3 and 15 minutes after injection, during which time radiopharmaceutical resides in the extracellular fluid pool. Pool-phase images are used to evaluate soft tissues such as tendons, ligaments, tendon sheaths, and bursae. A common misconception is that many distant soft tissues can be evaluated, but the short time interval dictates careful selection of sites for pool-phase imaging. Delayed-phase (phase III, bone phase) images are obtained between 2 and 4 hours after injection, during which time radiopharmaceutical resides in bone and unbound drug has cleared the kidneys. Although information gained in the flow or pool-phase images is useful, delayed images are the mainstay of equine imaging. A three-phase bone scan involves acquisition of flow, pool, and delayed images, but most commonly only delayed images, or delayed and selected pool-phase images, are obtained. An area of IRU visible in all three phases could indicate fracture or infection, but in horses it generally indicates fracture. Pool- and delayed-phase images should be carefully evaluated to determine if areas of IRU are the same or different. In our experience, early pooling of radiopharmaceutical usually represents early bone uptake. Pool- and delayed-phase images can be superimposed, or region of interest analysis can be used to determine size and position of IRU. In the proximal palmar/plantar metacarpal/metatarsal region, uptake of radiopharmaceutical can occur in only bone, only soft tissue, or both, and prognosis in horses with suspensory desmitis or bony injury can vary with tissue involved. Fractures are evident in delayed images, but sometimes they are visible as early as 20 to 30 seconds after injection or as progressive IRU in all phases. In horses with suspensory desmitis without bony involvement, pool-phase images may indicate positive findings in horses with acute injuries but are often negative, whereas delayed-phase image findings are often negative or equivocal. Differentiating soft tissue injury from that of bone is particularly important in the foot. Bruises, laminar tearing, and sub-solar abscesses should cause positive results on pool-phase images, but negative or equivocal delayed-phase images. In horses with long-standing infection or inflammation, osteitis of the distal phalanx can cause IRU in delayed images, complicating differentiation of involvement of the two tissues. Conversely, early bone uptake in horses with osteitis or fracture of the distal phalanx may prompt suspicion of soft tissue involvement as well. Although scintigraphy is considered highly sensitive, negative scan findings occur in horses thought to have severe soft tissue lameness in the foot. In pool-phase images, areas of intense uptake at the dorsal and palmar/plantar aspects of the coronary band are normal, making interpretation of uptake in important structures such the deep digital flexor tendon (DDFT), navicular bursa, and distal interphalangeal joint difficult.

Pool-phase images of the shoulder, thoracolumbar, and pelvic regions are often requested, but we have found them unrewarding. Radiopharmaceutical retention in large vessels, early (within 1 minute) intense uptake in the renal pelvis and urinary bladder, and problems such as distance, shielding, and motion all reduce probability of finding inflamed soft tissue. Pool-phase images may be useful in evaluating *acute inflammation* of the DDFT, navicular bursa, superficial digital flexor tendon, suspensory ligament, and digital flexor tendon sheath but may provide negative or equivocal findings if disease is chronic and active inflammation is only mild. Images should be obtained within 4 to 6 weeks after injury to identify lesions within these structures accurately. Pool-phase scintigraphic images appear to be less sensitive and specific than ultrasonographic images.

SCAN INTERPRETATION

Qualitative assessment is the most common and useful method to interpret scintigraphic images. Quantitative assessment such as region of interest analysis (comparing the region of one limb with another after considering background values), first-pass, or gaited studies and linear profile studies are easily performed but are time consuming. However, in mature competition horses quantitative assessment may be essential to confirm relatively small differences in radiopharmaceutical uptake that can be clinically significant. Images are qualitatively evaluated for *location*, *intensity*, and *character* of radiopharmaceutical uptake, and areas of increased (hot spots) or decreased uptake are noted.

Location of Increased Radiopharmaceutical Uptake

Areas of IRU should be accurately identified and located. Is the area of IRU located in cortical or subchondral bone?

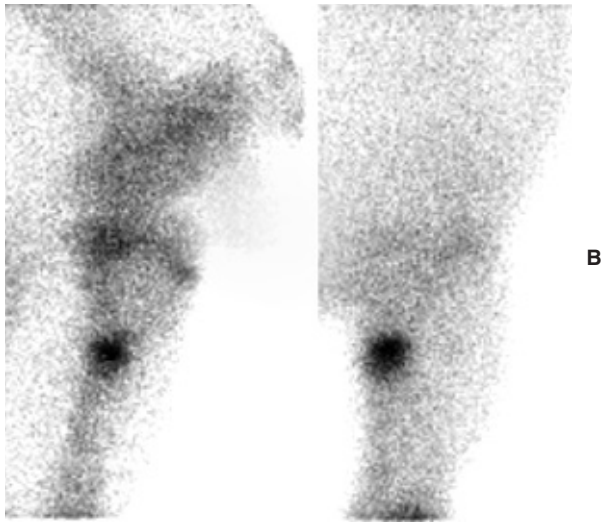

Fig. 19-9 **A,** Lateral and **B,** caudal delayed-phase images of the tibia showing focal intense increased radiopharmaceutical uptake in the medullary cavity of the tibial diaphysis associated with an enostosis-like lesion. Using only the lateral scintigraphic view, the increased radiopharmaceutical uptake could be confused with a laterally located tibial stress fracture.

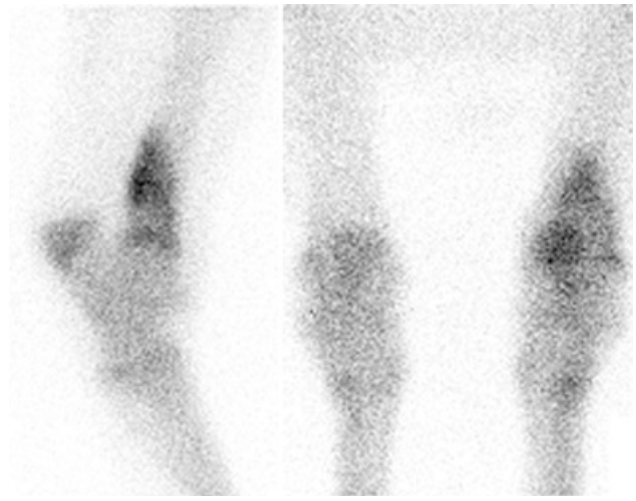

Fig. 19-10 Lateral (*left*) and caudal *(right)* delayed-phase images of a Thoroughbred racehorse with a caudolateral tibial stress fracture. In the caudal view, increased radiopharmaceutical uptake involving the lateral cortex clearly appears, an important difference from that seen in horses with enostosis-like lesions.

Cancellous (subchondral) bone located in the epiphyses is normally more active than is bone in the diaphyses, because these regions of bone are larger and have more active bone turnover. A small area of mild IRU in subchondral bone may not be relevant, whereas the same area in the mid-diaphysis of the tibia or McIII would indicate stress-related bone injury. Any area of IRU, even if small (1 to 2 pixels in width or length), in the cortex of a long bone is abnormal, but not all areas of IRU are clinically significant.

Is the area of IRU on one or both sides of the joint? Scintigraphic evidence of early osteoarthritis appears as a focal area of IRU on one side of a joint (see Fig. 19-6). Involvement of opposing articular surfaces, determined by evaluating several views, is a negative prognostic sign. If located in the diaphysis, is the area of IRU in cortical or medullary bone? At least two views are needed to determine location in the diaphysis. Enostosis-like lesions can appear similar to stress-related bone injury (see Chapters 15, 38, 41, and 46), but prognosis and management decisions differ (Figs. 19-9 and 19-10). A rare finding, an authentic fibular fracture, can be differentiated from enostosis-like lesions, soft tissue mineralization, or tibial stress fractures using lateral, caudal, and oblique views. Incidental findings such as an area of IRU associated with calcinosis circumscripta can be spectacular.

Intensity of Increased Radiopharmaceutical Uptake

Areas of IRU are described as mild, mild-moderate, moderate, moderate-intense, or intense. When analyzed using region of interest analysis, areas of IRU must have between 10% and 15% increased count density to be visually recognized as abnormal compared with surrounding bone or the contralateral limb. When both limbs are abnormal, an obvious difference may not be visible. Experience is critical for accurate image interpretation. Because time from injury to imaging is inversely proportional to intensity, IRU associated with fracture may not be intense but only mildly abnormal. Scintigraphic examination in horses suspected of having pelvic fractures may be critical for diagnosis, but expecting to see intense IRU is unrealistic, because distance, shielding, and motion complicate image quality. Pelvic fractures are usually not as obvious as fractures in the distal limb until muscle atrophy is pronounced. Cursory examination of images or placing undue importance on changes in intensity, without considering the nature of uptake, is dangerous. Even if mild and small, focal IRU should always pique the examiner's interest. Images should be evaluated before horses leave the imaging area, because additional 90° or 180° flexed or other views of the area might give important information. Commonly, advanced radiographic changes may be seen in bone when IRU is only mild, whereas negative or equivocal radiographic changes appear in bone when IRU is intense. Radiographic evidence of fracture often lags behind scintigraphic changes, an important advantage of using scintigraphy for early diagnosis. Conversely, scintigraphic activity may abate long before radiographic changes subside.

Character of Increased Radiopharmaceutical Uptake

Focal IRU means well-localized bone modeling exists, such as that seen with fracture or other substantial bone injury. Even without radiographic confirmation, focal IRU should be considered consistent with fracture, particularly in horses in which lameness has been localized to the specific region. In any horse, focal IRU indicates active bone modeling, but intensity of IRU varies with time between injury and scan. Differentiation between focal and diffuse IRU is important. For example, the dorsal cortex of McIII of TB racehorses undergoes a spectrum of bone changes caused by high-strain cyclic fatigue that can be seen scintigraphically and radiographically. Periostitis (bucked shins) can be differentiated from dorsal cortical fracture scintigraphically, even if accompanying radiographic findings are negative (Fig. 19-11). Fracture or traumatic osteitis accounts for most focal areas of IRU, but infectious osteitis or osteomyelitis can look similar. Labeled white blood cell studies using ^{111}In or ^{99m}Tc are useful to differentiate infectious processes from other bone injury and are particularly useful when evaluating potential infection after surgery. *Diffuse* IRU appears when a large area of cortical, subchondral bone, or soft tissue is abnormal, but in horses with diffuse IRU, radiographic changes usually are not found. *Generalized* IRU is another term used synonymously with diffuse IRU. A moderate, generalized IRU associated with both sides of a joint may be an incidental finding.

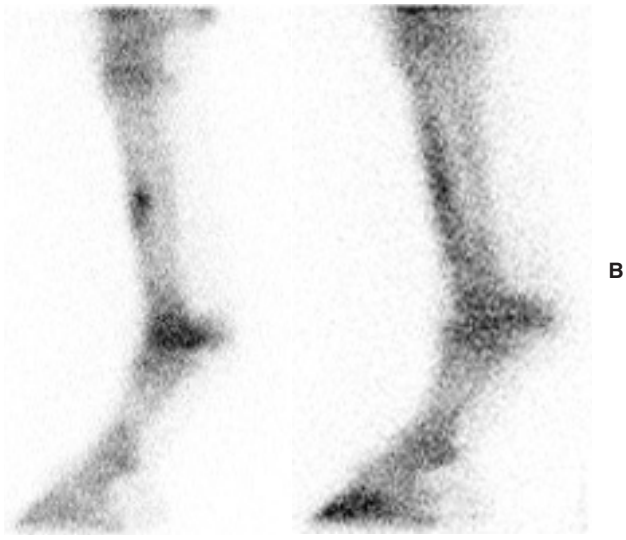

Fig. 19-11 Lateral delayed-phase scintigraphic views. **A,** A horse with a dorsal cortical fracture of the third metacarpal bone with focal, mild-moderate increased radiopharmaceutical uptake. **B,** A horse with periostitis (bucked shins) with diffuse increased radiopharmaceutical uptake of the dorsal cortex.

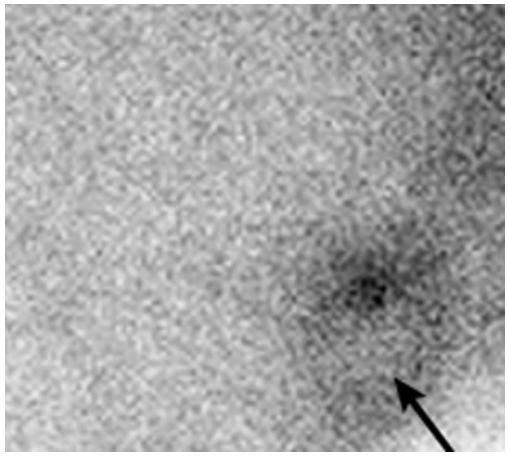

Fig. 19-12 Pool-phase image of a horse with a sub-periosteal abscess involving the olecranon process in which photopenia appears in the area of fluid accumulation (*arrow*). Surgical drainage and antibiotic administration successfully resolved the infection.

Photopenia is an unusual scintigraphic finding. Theoretically, if blood supply is not adequate during flow and pool-phases, radiopharmaceutical concentrations sufficient to delineate normal or abnormal bone are not achieved. Bone infarct, abscess, intense resorption, or sequestration might cause photopenia. Resolution of scintigraphic equipment is not sufficient to see small areas of resorption in dense cortical or subchondral bone, and even if possible to resolve technically, modeling in surrounding bone would obscure, or steal, counts. No known equine disease involves only bone resorption without modeling. Photopenia is seen rarely when portions of bone are surgically removed or displaced out of view. Bone photopenia normally appears in pool-phase images performed using ^{99m}Tc pertechnetate, because early bone uptake cannot occur. One horse with a sub-periosteal abscess of the olecranon process had photopenia in pool-phase images that was thought to be caused by fluid accumulation and compression of nearby vessels (Fig. 19-12).

False-Positive Findings

Few false-positive bone scans occur if the clinician is aware of normal areas of IRU and background radiation (bladder, large vessels) is considered. Normally, cortical bone is less apparent than cancellous bone (subchondral), but both are adequately visible in good-quality images. Physeal activity in young horses is pronounced, but normal and many variations exist (Fig. 19-13). Physeal activity decreases with age, but IRU at the distal radial and tibial physes persists for years. In a study of distal radial physeal closure, radiographic closure (24 to 32 months) preceded decrease in scintigraphic activity by several months (mean, 42 months).[27] In lateral pelvic images the coxofemoral joint (bone stacking of proximal femur and acetabulum) and tubera coxae are always prominent. Additional normal hot spots include the lateral aspect of the metatarsophalangeal joint in plantar views, a bi-lobed area in the proximal tibia in a lateral view of the stifle, the distal lateral femur and proximal tibia in a caudal view of the stifle, the proximal humerus, the proximal lateral aspect of the radius (radial tuberosity), the pastern joints, the dorsal articular facets of the sixth and seventh cervical vertebrae, the dens, and many places in the skull, such as the temporomandibular joint. In some sport horses such as jumpers, hunters, event horses, timber horses, and driving horses, focal mild to intense IRU is found on the dorsal (most common) or palmar cortex of the proximal phalanx in one to four limbs (Fig. 19-14). The Editors have been unable to attribute lameness to dorsal cortical IRU and feel it is likely to represent a normal area of modeling, but in 6 of 23 horses lameness was attributed to this site.[28] In another study of jumping, hunting, and event horses, IRU of the proximal phalanx was considered to be clinically important, but clear differentiation of dorsal cortical IRU from that of the proximal and distal aspects of the bone was not made.[29] Careful clinical examination and infiltration of local anesthetic solution may help to differentiate this site from other, much more common sources of pain.

In dorsal pelvic images the bladder is often superimposed on the sacrum or other pelvic bones and can be mistaken for abnormal IRU. Additional oblique, hemipelvic, or caudal transverse pelvic views should always be obtained. The kidneys are commonly, if not always seen, in delayed images. The right kidney is more prominent than is the left, because it has a more superficial location and normally accounts for approximately 60% of total glomerular filtration rate.[30] Diagnosis of renal dysfunction should only be made if gross anatomical derangement is seen and should be confirmed using specific renal function studies and ultrasonographic evaluation.

Peri-vascular injection or extravasation of radiopharmaceutical can cause obvious, intense IRU in soft tissues at the injection site. If a large volume of radiopharmaceutical is sequestered, distribution is slow and poor-quality delayed images result. Extravasated radiopharmaceutical can be taken up by caudal cervical lymph nodes, causing interesting but inconsequential IRU. Other non-osseous areas of IRU may be incidental (calcinosis circumscripta, other sites of mineralization, nerve block sites) or clinically important (skeletal muscle).

False-Negative Findings

A false-negative scan occurs when the image(s) fails to identify an existing lesion. False-negative scans usually result from failing to obtain appropriate views, obtaining poor-quality images, or incorrectly interpreting images. Failure to perform diagnostic analgesia and interpret the clinical relevance of scintigraphic changes may lead to apparent but not authentic false-negative results. Problems with distance, shielding, and motion likely account for many false-negative findings. A false-negative result is possible if a horse with acute pelvic fracture

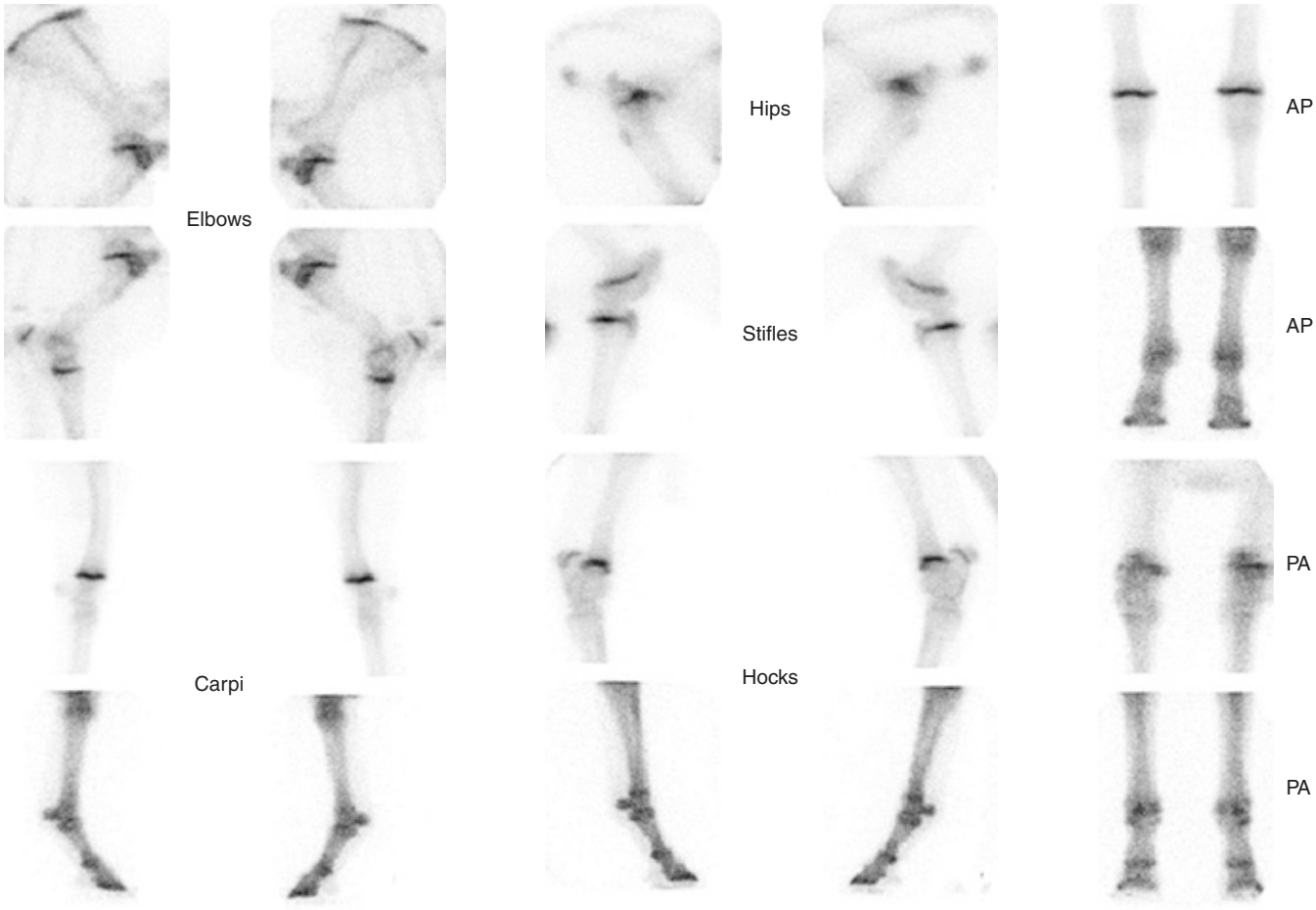

Fig. 19-13 Delayed images showing intense physeal uptake in a yearling. These areas are normal but may erroneously be interpreted as abnormal (false positive) or obscure nearby lesions causing false-negative scans. *AP,* Anteroposterior; *PA,* posteroanterior.

is scanned within 7 to 10 days after injury. False-negative scan results rarely occur in horses with stress-related bone injury, because bone changes are long-standing and resorption and modeling occur simultaneously. False-negative results have been reported in people undergoing chronic corticosteroid therapy and in those with disseminated metastatic disease, the elderly, and those with osteomalacia or renal osteodystropy.[31] Bone resorption, high osteoclastic activity, and slowed bone turnover may occur with these diseases, although bone modeling usually is present to a certain extent, ensuring a positive result.

Accuracy can be greatly improved by obtaining as many as possible standard and special scintigraphic views (see Fig. 19-5). Knowledge that the lameness is localized to a specific site can help the clinician carefully evaluate this region by obtaining all possible views, and such targeting can reduce the chance of a false-negative result.

Negative Bone Scan Results

Owners and veterinarians look for scintigraphy to provide the answer to the nagging lameness or performance issue, but in many instances a scan result is truly negative. Every bone scan provides useful and interesting clinical information, regardless of whether the finding is positive or a diagnosis can be made. Negative findings are useful in horses in which an increase in exercise is planned, such as in a racehorse with a suspected hairline or stress fracture that is entered in an upcoming race. Negative findings in any type of sport horse examined for

Fig. 19-14 Delayed image of the distal part of a forelimb of a jumper showing focal moderate increased radiopharmaceutical uptake in the dorsal cortex of the proximal phalanx. This is a normal finding in non-racehorses and may involve one to four limbs.

poor performance or a suspected obscure lameness can help clarify the picture. Negative findings on delayed images support clinical information that pain is likely originating from soft tissues. In horses with hindlimb lameness, negative scan findings often lead us to suspect the stifle may be a source of pain, because delayed images of this region may not be highly sensitive. Findings in horses with chronic proximal suspensory desmitis also are often negative.

Osteochondrosis in Delayed Images

We have identified areas of IRU in weanlings, yearlings, and older horses with osteochondritis dissecans or osseous cyst-like lesions, but scintigraphic evidence of bone modeling can be difficult to detect, or it is missed. We have heard anecdotally that some believe osteochondrosis cannot be successfully identified scintigraphically, but our experience suggests otherwise. Intense physeal uptake in weanlings and yearlings is the most important factor in correctly identifying osteochondrosis. Count stealing is certainly important, but so is the possibility that bone modeling around osteochondritis dissecans or osseous cyst-like lesions is either minimal or only present at certain stages. Subchondral bone cysts of the distal femur are generally scintigraphically active, but images must be interpreted carefully, and a caudal view of the stifle is mandatory. Osteochondritis dissecans of the shoulder joint involving the glenoid or proximal humeral head usually is scintigraphically active. We have identified several 2- and 3-year-old Standardbreds (STBs) and Warmbloods with large abaxially located extra-articular or small intra-articular plantar process osteochondritis dissecans fragments, or both, of the proximal phalanx in which bone modeling could be seen scintigraphically. Interestingly, in TB racehorses focal IRU in this region is common but does not usually result from plantar process fragmentation. Osteochondritis dissecans of the lateral trochlear ridge of the distal femur and cranial intermediate ridge of the distal tibia is rarely scintigraphically evident.

Artifacts

Urine contamination is particularly prominent in the caudal and plantar aspects of the hindlimbs in fillies. Urine contamination of the bottom of the feet is common, unless boots are applied to the horse before imaging, a procedure that is mandatory if the feet are of specific interest. Alternatively, urine can be washed away and horses can be re-scanned. Damage to the collimator, crystal, maladjustment of the photopeak for ^{99m}Tc, deterioration in flood quality, and electrical problems can produce artifacts. Incidental scintigraphic findings have been discussed.

Nerve Blocks

It is reasonable to assume that injections of any type may interfere with interpretation of scintigraphic images. Local or intra-articular injections likely produce short-lived inflammatory changes that may be visible on pool- or delayed-phase images, but we have found this to be an overrated problem. Low and high palmar analgesic techniques resulted in IRU seen in pool-phase images for up to 17 days after injection, but little effect was seen after palmar digital and abaxial sesamoid techniques. No effects of injections were seen in delayed images. The authors concluded that although effects were mild, pool-phase images should be delayed for 3 to 4 days after palmar digital blocks and 2 weeks after low and high palmar techniques.[32] We have not found it difficult to interpret pool-phase images done 24 hours after blocking, if we are aware the horses have been blocked. At least two scintigraphic views will clearly show persistence of IRU at the subcutaneous injection sites and not in important, deeper soft tissues. In most horses, little to no IRU can be seen in pool-phase images and no effect occurs on delayed images with one exception. After fibular (peroneal) and tibial nerve blocks, pool and delayed images show persistence of activity at the fibular block site, and it is extremely important to know the blocking history and obtain at least two scintigraphic views. Increased radiopharmaceutical uptake is likely caused by local myositis (Fig. 19-15). Intra-articular injections do not affect delayed images, but they may interfere with pool-phase imaging. If pool-phase images are critical, evaluation of a joint should be delayed for at least 2 weeks.

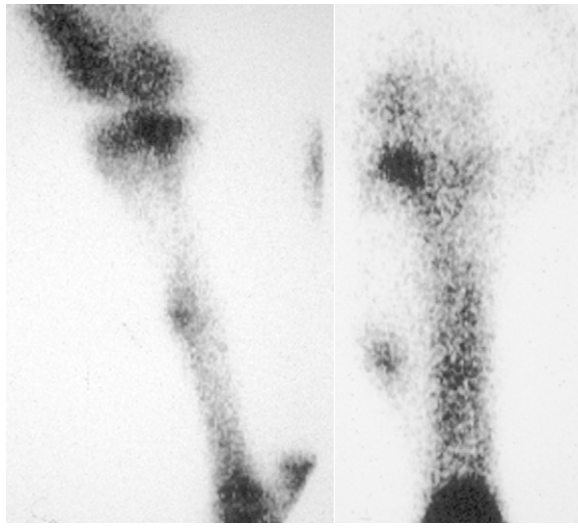

Fig. 19-15 Lateral (*left*) and caudal *(right)* delayed-phase images showing focal increased radiopharmaceutical uptake in what appears in the lateral view as the cranial tibial cortex. In the caudal view, increased radiopharmaceutical uptake clearly involves soft tissue. Cranial tibial stress fractures are not known to occur, but this horse had a fibular and tibial nerve block the day before these images were taken.

INDICATIONS AND CASE SELECTION

Bone scintigraphy is a valuable clinical tool but, like all other modalities, has limitations. Scintigraphy is useful in *early diagnosis* before injuries become radiographically apparent, a finding especially true in horses with subchondral stress-related bone injury, because scintigraphy may identify the problem months or years before radiographic findings are positive. Once familiar with scintigraphy the clinician can become comfortable diagnosing fracture without adjunct radiographic confirmation. Although fractures are common, scintigraphic findings of other less obvious lesions must be differentiated and defined. The high expectations placed on scintigraphy to provide the lameness answer can be partially met by careful consideration of clinical characteristics of lameness and case selection. We have listed, in decreasing order, the types of clinical situations in which scintigraphy is likely to provide the most information to optimize the chance of answering that tough lameness question. Most important, horses should be lame at the time of the examination, and when possible lameness should be localized to a defined region in one or more limbs. Targeted imaging is invariably much more productive than whole-body screening.

Scintigraphy is most valuable in horses with unilateral or multiple limb lameness that has been localized using diagnostic analgesia but in which radiographic findings are negative, equivocal, or confusing. In this group of horses, lameness at the time of scintigraphic examination allows for the distinct

possibility that bone modeling subsequent to injury will be detected, if indeed bone is the source of pain. Key to accurate interpretation and obtaining all necessary views is localizing the source of pain. Scintigraphy then is most useful in horses that are *lame at the time of examination and for which lameness has been localized*.

Horses that are lame at the time of examination but in which lameness has not been localized comprise the next best group. The importance of lameness at the time of examination cannot be overemphasized. We often scan horses after periods of rest or on returning to training, but if lameness is not apparent, determining clinical relevance of findings is difficult. A subset of horses in which the lower limb has been ruled out by careful diagnostic analgesia makes even better candidates. These are horses in which an upper limb problem such as a humeral, tibial, or pelvic stress fracture may be suspected. These horses are *lame, but although the definitive source of pain has not been identified, the lower limb has been ruled out*.

Horses with an accurate history of high-speed lameness but that are not currently lame are in the next category. These are usually racehorses with stress-related bone injury and strong histories to support that lameness is indeed the cause of poor performance. Performing whole-body scans is most useful in these horses, because compensatory lameness is often present. Lameness may be subtle or difficult to detect during routine lameness examination, and diagnostic analgesia would be difficult to perform. However, this group comprises young, light horses undergoing intense training and racing in which high-strain cyclic fatigue is maximal. In this group then are *racehorses without obvious baseline lameness but in which stress-related bone injury is suspected*.

Scintigraphy is *least* likely to yield a diagnosis in show horses with nebulous histories of gait abnormalities and poor performance and with problems that can be perceived only by the rider. These horses usually are not lame, may have equivocal or negative manipulative test results, and may be difficult individuals with which to work. Diagnostic analgesia may be difficult to perform or interpret. In some horses, selective analgesic techniques may improve the feel of the horse and in some, bilaterally symmetrical lameness may be diagnosed by abolishing pain in one limb and observing lameness in the contralateral limb. Whole-body scintigraphic screening can be frustrating and unrewarding for the clinician and referring veterinarian, but it is occasionally helpful. High-quality images are essential for recognizing subtle abnormalities, for example, in the proximal palmar/plantar metacarpal/metatarsal region, in the foot, thoracolumbar spine, or pelvis. A negative scan result can help direct attention to soft tissues and adjunct evaluation using chiropractic techniques or recommend conditioning and exercise intensity changes.

Scintigraphic examination has been used as part of a comprehensive purchase examination, but results must be carefully interpreted and clinical relevance established. Although unusual, this adjunct imaging procedure usually is requested in high-profile, expensive, upper-level sport horses and results can often be confusing. Finding an upper-level horse of any type without any scintigraphic changes would be unusual, mirroring radiographic findings in these horses.

KNOWLEDGE GAINED FROM SCINTIGRAPHIC EXAMINATION OF THE LAME HORSE

Scintigraphy is a powerful tool for examining a lame horse by which we have been able to give answers, pinpoint diagnoses, study disease progression, and learn about pathogenesis of many lameness conditions. Although we cannot discuss every scintigraphic finding in the lame horse, a few select issues and areas should be reviewed.

Stress-Related Bone Injury in Cortical and Subchondral Bone

Recent studies using scintigraphy and clinical and pathological findings support the concept of a continuum of adaptive and non-adaptive responses of bone leading eventually to fracture (stress or complete) and osteoarthritis.[17-19,33,34] Microfracture and callus formation in pelvic bones and the humerus, and prodromal lesions and microfracture in ipsilateral and contralateral limbs of horses with condylar fractures, indicate that pathological changes preceded fracture.[17-19] Pathological changes appeared histologically in subchondral bone of distal McIII/MtIII and the third carpal bone that preceded overlying cartilage damage, indicating a continuum of changes lead to fracture or osteoarthritis in racehorses.[33] Scintigraphic findings in the metatarsophalangeal joint of STBs indicated the existence of focal IRU of distal MtIII was a common scintigraphic finding in horses in which lameness was localized to this region, but in which radiographic findings were negative or equivocal.[34] A continuum of subchondral bone changes was proposed to account for the later development of fracture or osteoarthritis, but early changes could only be seen scintigraphically, not radiographically (Fig. 19-6).[34] Common areas for stress-related bone injury in subchondral bone include distal McIII/MtIII, proximal sesamoid bones, and the third carpal bone (see Fig. 19-16), but we feel strongly that a continuum of stress-related bone injury could account for development of fractures of the distal phalanx and osteoarthritis of the distal interphalangeal joint.[35] Abnormal IRU is common in the lateral aspect of the distal phalanx in the left forelimb and medial aspect of the distal phalanx in the right forelimb, the same sites in which fracture of the distal phalanx is most common in racehorses in North America (Fig. 19-17).[35,36] We know of at least two horses that subsequently developed articular palmar process fracture in these locations.[35] In non-racehorses, a common finding in those in which lameness is abolished using palmar digital analgesia is focal IRU in subchondral bone of the distal phalanx, corresponding to radiolucent defects and other radiographic evidence of osteoarthritis of the distal interphalangeal joint (Fig. 19-18). Observations in racehorses and non-racehorses led us carefully to evaluate medial to lateral hoof balance, because many of these horses were high on the affected side, strongly suggesting mechanical forces may be involved in early stress-related bone injury.

Scintigraphic examination has allowed us to demonstrate clinically that a continuum of cortical and subchondral bone changes exist before the development of fracture or osteoarthritis. This process is better understood and accepted in cortical bone but also occurs in subchondral bone and explains why horses can show lameness without obvious signs of synovitis, because pain originates from subchondral bone, without obvious overlying cartilage damage. Cortical and subchondral bone undergo *stress remodeling*, the term used to describe the process by which exercised bone is modeled (new bone) and remodeled (resorption and replacement). Bone adaptation is triggered by changes in number of strain cycles and the magnitude, rate of change, and distribution of strain.[37-39] Adaptive bone remodeling is normal, but exercise may lead to non-physiological strains, or during remodeling, resorption may outpace replacement, and both situations may lead to a non-adaptive response in which damage to cortical or subchondral bone occurs, eventually leading to fracture or osteoarthritis.[4,34,35,40] Lameness appears to develop long before radiographic evidence of bone damage exists, but during this time scintigraphic examination often reveals focal areas of moderate or intense IRU.

In summary, many common equine lameness conditions, including catastrophic and stress fractures and many forms of osteoarthritis, result from a continuum of cortical and

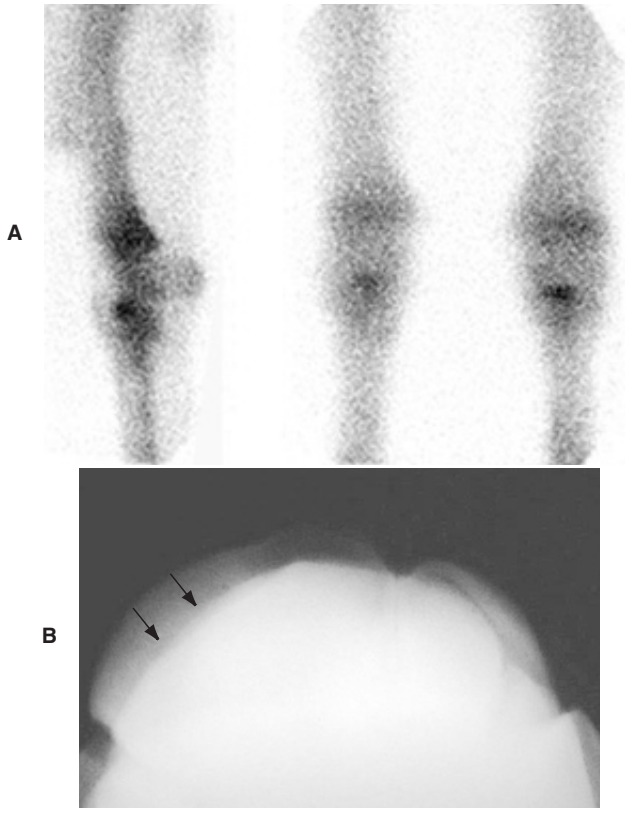

Fig. 19-16 **A,** Lateral (*left*) and dorsal (right forelimb is right) delayed-phase scintigraphic views of the carpus of a Standardbred with stress-related bone injury of the radial facet of the third carpal bone. Focal increased radiopharmaceutical uptake in the radial facet is a common scintigraphic finding in horses with lameness abolished by middle carpal analgesia but in which other clinical findings are equivocal. **B,** Proximodorsal-distodorsal (skyline) radiographic view of the same horse showing sclerosis *(arrows)* of the third carpal bone, a finding consistent with non-adaptive subchondral remodeling.

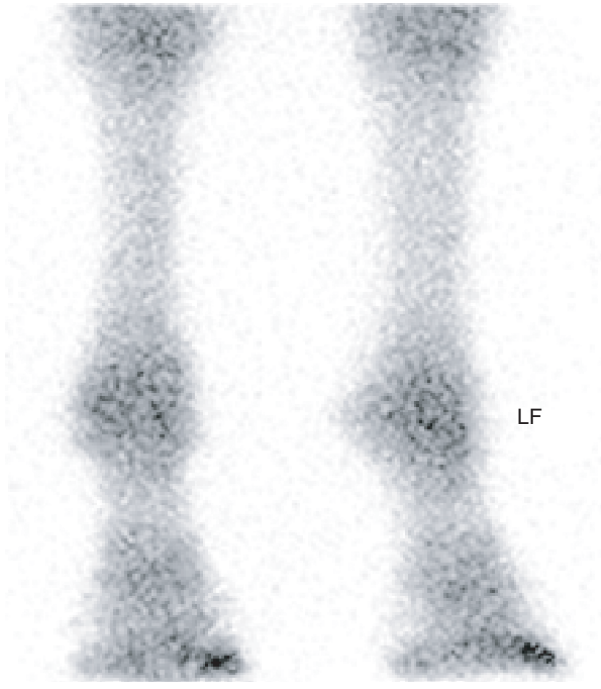

Fig. 19-17 Dorsal delayed scintigraphic view of the distal forelimbs in a racehorse showing a common finding of increased radiopharmaceutical uptake in the lateral left forelimb *(LF)* and medial right forelimb distal phalanx. Stress-related bone injury in these locations likely leads to later development of fracture or osteoarthritis.

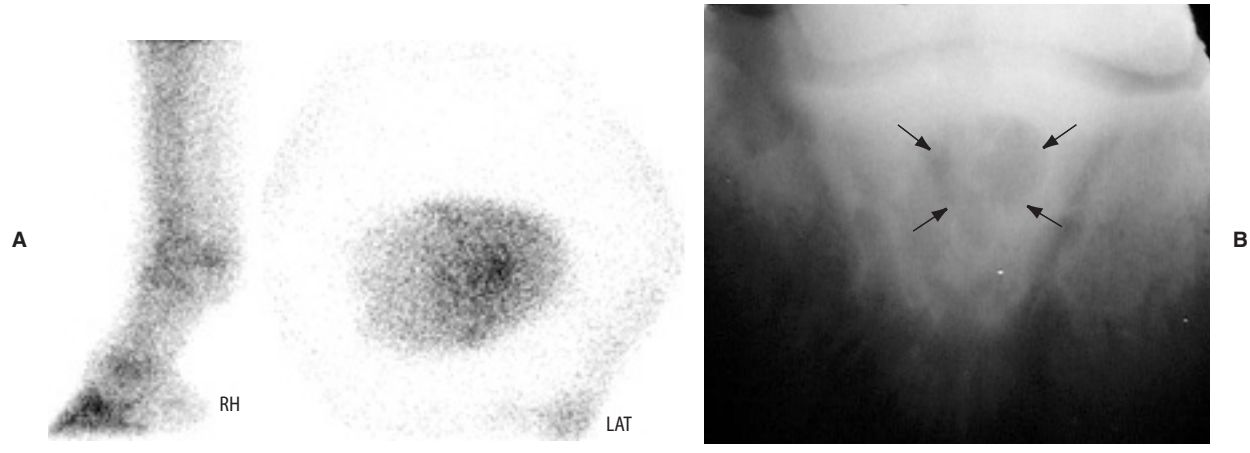

Fig. 19-18 **A,** Dorsal delayed scintigraphic view in a Warmblood showing focal increased radiopharmaceutical uptake in the subchondral bone of the distal phalanx. **B,** Dorsopalmar radiographic view showing subchondral radiolucency *(arrows)* consistent with long-standing trauma and osteoarthritis of the distal interphalangeal joint. *RH,* Right hindlimb; *LAT,* lateral.

subchondral bone changes occurring in response to exercise. Scintigraphic examination is the only way to provide early, accurate diagnosis. Single-event injuries occur from direct trauma or acute overload of bone and soft tissue material properties, but they are in the minority in most sport horses with stress-related bone injury. We find it interesting that in the forelimb, with the exception of the distal phalanx, most stress-related bone injuries involve the medial aspect, whereas in the hindlimb most changes involve the lateral aspect of the limb. This finding is consistent in TB and STB racehorses and must reflect loading of the limb and commonality of movement, rather than the expected effects of counterclockwise racing.

Confusing or Equivocal Radiographic Changes

We have often been able to pinpoint diagnosis in horses with confusing or equivocal radiographic changes using scintigraphy. Although racehorses with commonly seen areas of stress-related bone injury fall into this category, we have been able to accurately identify sources of pain in other types of sport horses. Navicular disease formerly was a common default diagnosis in horses in which lameness was abolished by palmar digital analgesia. However, scintigraphic examination has allowed us to define 15 to 20 different possible sources of pain in horses with this blocking history. Navicular disease is still a common finding and accounted for part or all of lameness in 65% of non-racehorse sport horses.[35] Central areas of IRU are most common, and often conventional radiographic views yield negative or equivocal findings. The proximopalmar distopalmar (skyline) view was most useful in identifying subtle radiographic changes and should be obtained routinely (see Chapter 30). However, navicular disease should not be the default diagnosis, because osteoarthritis of the distal interphalangeal and proximal interphalangeal joints, stress remodeling or stress reaction of the distal phalanx, soft tissue pain, and a myriad of other diagnoses can be made. Differentiation of lameness in the proximal palmar metacarpal region from that of the carpus can sometimes be difficult from overlap of diagnostic analgesic techniques, but scintigraphy has proved valuable in correctly identifying horses with avulsion injury at the origin of the suspensory ligament from those with lameness of the middle carpal joint (Fig. 19-19). Similarly, lameness associated with the tarsus can be clearly differentiated from that of the proximal metatarsal region. Scintigraphic examination has identified horses with incomplete fractures of the talus, distal tibial subchondral bone injury, fractures of the central and third tarsal bones, and osteoarthritis of the centrodistal and tarsometatarsal joints. In young racehorses the predominant scintigraphic and radiographic changes are seen involving the dorsolateral aspect of the tarsus, unlike what was once thought, that the dorsomedial or medial aspect was most commonly involved (see Fig. 109-7). Focal or diffuse areas of IRU are found in the thoracolumbar dorsal spinous processes and may account for clinical signs of back pain, poor performance, or gait restriction but occur only in horses that are ridden (Chapter 54). We have not identified areas of IRU in the STB racehorse. A rare scintigraphic finding in horses with acute forelimb lameness or with acute back pain and respiratory distress is fracture or other injury to a single or two adjacent vertebrae in the mid-thoracic region.

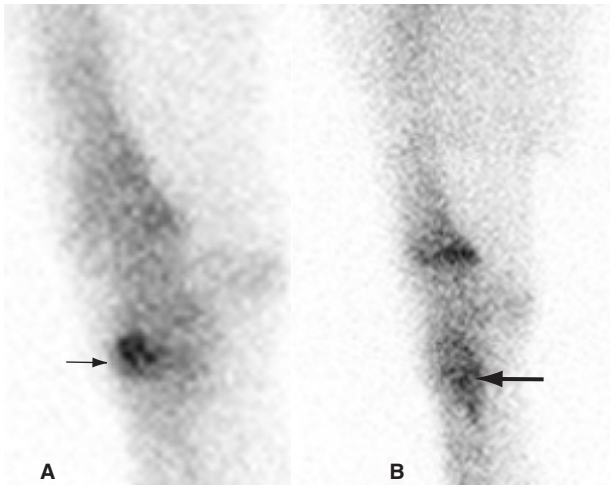

Fig. 19-19 A, Lateral delayed scintigraphic view of horse with lesion involving the third carpal bone *(small arrow)*. **B,** Same view of another horse with an avulsion fracture of third metacarpal at the origin of the suspensory ligament *(large arrow)*. High palmar and middle carpal joint analgesic techniques improved lameness in both horses.

Lameness of the Pelvis

Good-quality scintigraphic images of the pelvis can obviate the need to obtain radiographs with horses under general anesthesia. Image quality can further be improved using motion correction software (see Fig. 19-4). Horses with IRU of the coxofemoral joint often have distant areas of IRU, a scintigraphic finding that worsens prognosis for future soundness. In two horses in which scintigraphy identified single or multiple areas of IRU, fracture of the pubic symphysis, not identified in either horse scintigraphically, was found at necropsy examination in addition to those areas properly identified.[41] Although horses seemingly have one predominant source of lameness, with pelvic injury scintigraphic identification of additional sites is an important negative prognostic sign. False-negative results are also possible. Pelvic stress fractures, fractures or enthesopathy of the third trochanter and tubera ischii, sacroiliac injury, direct trauma to the tubera coxae, and fractures of the tail head and caudal sacrum have been identified as causes of obvious to subtle signs of hindlimb lameness (see Chapters 51 and 52).

Damaged Skeletal Muscle

Damaged skeletal muscle behaves like damaged bone. Areas of IRU in skeletal muscle appear in *delayed images* and not in pool-phase (soft tissue) images. We have observed two distinct patterns of IRU in skeletal muscle. Most commonly, generalized IRU in the gluteal and epaxial muscles appears in racehorses and is thought to be consistent with previous exertional rhabdomyolysis. Occasionally, IRU appears concomitantly in the latissimus dorsi muscle or rarely in the triceps muscle. In these horses, creatine kinase and aspartate aminotransferase enzyme concentrations usually are only mildly elevated. In the original report, IRU in skeletal muscle was seen 24 hours after horses underwent strenuous exercise on a high-speed treadmill, and the authors suggested that imaging should be performed within 24 to 48 hours of exercise.[42] Our experience is different, however, because we have seen generalized IRU of skeletal muscle in horses that have trained at least 7 to 10 days before scintigraphic examination. Of 129 horses with scintigraphic abnormalities of the pelvis, 34 had IRU of skeletal muscle.[41] Lameness associated with IRU of skeletal muscle was not apparent, but abnormal IRU in muscle was thought to explain a portion of the horse's poor performance.[41] However, not all horses with previous exertional rhabdomyolysis have scintigraphic abnormalities.

In the less common second form, IRU appears in individual muscles or portions of muscles. The hindlimb is most commonly affected, and individual portions of the biceps femoris, semitendinosus, or gluteal muscles are abnormal. Although this form is most common in racehorses, occasionally epaxial

or gluteal IRU is identified in non-racehorse sport horses. Although horses may show pain on palpation of the affected region, we have not been able to identify these areas as the cause of hindlimb lameness. Elevated levels of creatine kinase and aspartate aminotransferase are not found. Horses with this form of IRU in muscle usually are lame in the ipsilateral hindlimb but from a distally located site. We have speculated that muscle strain or tearing may have occurred from altered limb carriage resulting from the primary lameness condition. Rarely, we have identified IRU in a forelimb involving subclavius, pectoral, or triceps muscles.

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CHAPTER • 20

Computed Tomography

José M. García-López

Tomography was not fully developed until the end of the 1960s and early 1970s but was first considered in the 1920s when radiologists were trying to develop alternative options to avoid some of the drawbacks of radiographic imaging: a way to determine the depth of pathology within the body and a means to look past the bones.[1,2] Godfrey Hounsfield, an electrical engineer working for the British Electrical & Musical Industries Ltd. in 1970, was responsible for the development of the first computed tomography (CT) scanner.

EQUIPMENT AND PHYSICS

A CT scanner is made up of three major components. The gantry houses the x-ray tube and radiation detectors (Fig. 20-1). It can be angled from 15° to 30°, which becomes useful for imaging areas such as the skull.[1,2] In human and small animal medicine, a table is attached to the gantry, which positions the animal within it in precise increments. Such tables are becoming more readily available for horses (Luminys Table, Universal Medical Systems, Solon, OH).[3] The gantry is connected to a computer system and the operating console. In the console, information such as the kilovolt peak (kVp), milliampere seconds (mAs), and slice thickness can be manipulated in addition to the contrast to attain optimum image visualisation by adjusting the brightness in the screen.

A CT image is made of many cells, called *pixels*, each of which is assigned a number and later displayed as a brightness level on the video monitor. The numerical information stored in each pixel is known as a Hounsfield unit (HU) or CT number, which is relative to the attenuating power of pure water.[1,2,4] The pixel is a two-dimensional representation of a corresponding tissue volume. The tissue volume (voxel) is determined by the pixel size and the slice thickness, which usually ranges from 1 to 10 mm. HUs range from –1000 (air) to +1000 (dense bone) for each pixel; water has a HU of 0, whereas soft tissues and fat have a range of 0 to 100 and –100, respectively.[1,2] This represents enormous gradient in the gray scale and an incredible amount of wasted information, because a video screen can only display approximately 256 HUs (shades of gray) out of the possible 2000.[1,2] To display the image in the monitor, the imager selects a HU that is approximate to the average HU of the body tissue (bone or soft tissue) being examined in the study. With this information the scanner's software is instructed to assign one shade of gray to each of the 128 HUs above and below the HU established for the body tissue.[1,2] This middle number is referred to as the *window level*, and the range of HUs is referred to as the *window width*. A window level for bone is usually approximately +200 and for soft tissue it usually ranges from +20 to +40.[1,2]

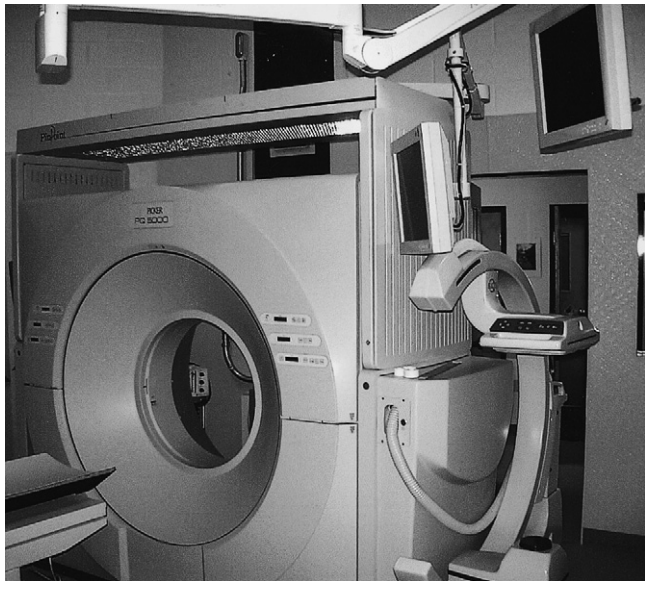

Fig. 20-1 Picker PQ 5000 Helical CT scanner. (Courtesy Tufts University School of Veterinary Medicine, North Graton, Mass.)

Several generations of CT scanners have evolved. The first, which was the original scanner developed by EMI, had a pencil-thin x-ray beam and single paired detector. The scan time was approximately 25 minutes, up to 5 minutes per slice. The second-generation scanners had the primary objective of increasing the speed of the procedure. They had multiple detectors, and the x-ray beam was fan shaped. Although these machines used a translate-rotate mechanism similar to the first-generation scanners, fewer linear scans were needed because of the increased number of detectors, and rotation of the beam increased from 1° to up to 30° between scans. This resulted in a substantial decrease in image acquisition time. The third-generation scanners introduced a rotate-rotate mechanism, which permitted the x-ray tube and detector to rotate concentrically about the patient, rather than the linear translations used in the first- and second-generation machines. The x-ray beam had a wider, fan-shaped configuration (30° to 60°), and a pre-patient collimator was developed that controlled the thickness of the beam, therefore determining the slice thickness. The number of detectors also was greatly increased (288 to 700), which further decreased the scan time to approximately 1 second. Unfortunately, one major problem with third-generation scanners was the creation of ring artifacts from faulty detectors. Partially because of this factor, fourth-generation scanners were developed that used a rotate-fixed mechanism in which only the tube, and not the detectors, rotates about the patient. Since 1990, thanks in part to great strides made in CT technology, helical scanners were developed to further improve the quality of the images and the speed of the study. The slip-ring technology allows continuous rotation of the tube, allowing for simultaneous translation and data acquisition. This results in a dramatic increase in speed, requiring only 20 to 40 seconds for an entire study rather than the 4 to 6 minutes with conventional CT. The increase in speed also results in less mis-registration, fewer motion artifacts, and production of

overlapping images without additional radiation exposure.[1,2] It also allows for manipulation of the position and spacing of axial slices after data acquisition. Disadvantages of the helical scanner include an increased demand on the x-ray tube and the fact that image reconstruction can take additional time and cannot be viewed immediately.

COMPUTED TOMOGRAPHY IN EQUINE SPORTS MEDICINE

Diagnostic imaging is fundamental for the diagnosis of equine lameness. Conventional radiography displays a three-dimensional object in two dimensions. A 30% to 50% variation in bone density is required because it is radiographically apparent.[3,5] In addition, problems such as summation of bony densities and superimposition of different contours can result in a failure to detect conditions such as subchondral bone cysts and small hairline fractures (Fig. 20-2, *A*).[5] In common conditions such as navicular syndrome and osteoarthritis of the distal tarsal joints (bone spavin), the clinical presentation and the radiographic appearance of the area often correlate poorly. The addition of ultrasonography and nuclear scintigraphy has resulted in improved diagnosis.[6-8] CT offers the ability to evaluate structures deep to gas-containing organs, as well as deep to bone, which are inherent limitations of ultrasonography.[3] Nuclear scintigraphy has been invaluable in the evaluation of lameness, but although it is generally a very sensitive diagnostic tool, it does not have great specificity (Fig. 20-2, *B*). This lack of specificity sometimes can result in an inaccurate or incomplete idea of the cause of the lameness. The location of the problem may be identified but not the cause. Such shortcomings can be avoided with CT because it offers extraordinary detail and a three-dimensional representation of the area examined (Fig. 20-2, C).

Over the past decade the potential value of CT and magnetic resonance imaging (MRI) has been recognized.[9-23] CT is generally indicated for the evaluation of bony structures and, to a lesser extent, soft tissue, although MRI provides a clear advantage over CT for examination of soft tissues.[10] Applications for CT include the examination of joints for the assessment of osteoarthritis, subchondral bone disease, osseous cyst-like lesions (see Fig. 20-2), and complex fractures.*

Several reports have already shown the benefits of CT imaging of the distal phalanx and navicular bone.† Lesions in the proximal and distal surfaces of the navicular bone, as well as in its medullary cavity (e.g., erosions, changes in cortical thickness, increased medullary sclerosis, synovial invaginations, and small fractures) can be more adequately identified using CT.[9] Radiography underestimated the extent of the degeneration of the navicular bone, which included prominent synovial invaginations along the distal border, cyst formation, and significant erosion of the flexor surface.[9] It was concluded that bony changes in the navicular bone were better assessed using CT than MRI.[9] Abnormalities of the flexor surface of the navicular bone in both normal and lame horses were more accurately determined using CT than radiography.[17,25] A trend developed toward over-interpretation of changes seen radiographically on the flexor surface of the navicular bone when compared with CT images.[17] The normal appearance of the deep digital flexor tendon in the distal pastern and within the hoof capsule has been described.[26] Thus CT also may provide additional information about other causes of palmar foot pain.

Maladaptive bone disease or stress-related bone injury, sometimes referred to as *bone bruising*, is a condition that affects a large number of young racing Standardbred and Thoroughbred racehorses.[6,27] Nuclear scintigraphy has demonstrated that the metacarpophalangeal and metatarsophalangeal joints and the third carpal bone are commonly affected.[6,13-16,27] However, in many horses no radiographic changes are

*References 5, 9, 17, 18, 20, 21, 23, 24.
†References 9, 17, 18, 20, 22, 25.

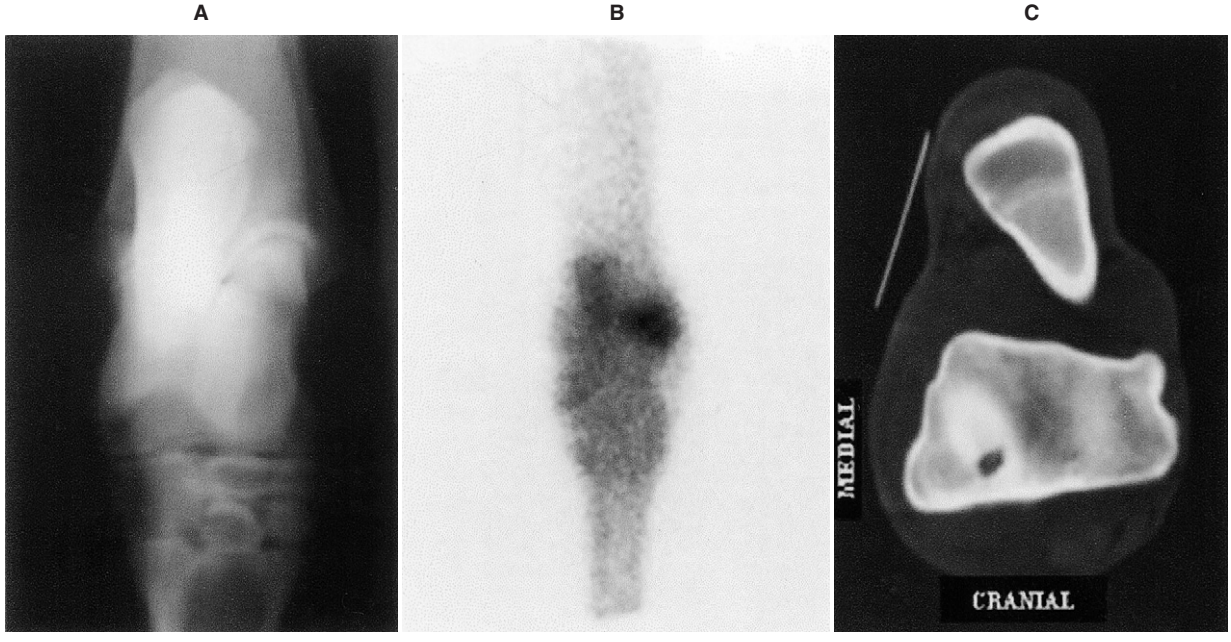

Fig. 20-2 Comparison of imaging modalities in the diagnosis of an occult cyst-like lesion in the medial malleolus of a horse. **A,** Dorsoplantar radiographic projection fails to identify any abnormalities. **B,** Plantar delayed-phase scintigraphic projection of the same area shows a focally intense area of radiopharmaceutical uptake in the region of the medial malleolus. **C,** Cross-sectional CT images of the distal tibia reveals a hypodense area with a sclerotic rim (cyst-like lesion) just proximal to the medial trochlear groove and a mild periosteal reaction on the cranial aspect of the distal tibia.

detectable and thus the actual state of the subchondral bone is poorly defined. CT was used to examine the effects of training or exercise on bone mineral composition of the distal aspect of the third metacarpal or third metatarsal bones in young horses.[15,16,28] This produced a substantial increase in subchondral bone sclerosis on the palmar/plantar aspect of the condyles, which can be a manifestation of subchondral bone disease and a possible prelude to condylar fractures.[15] The use of CT may be helpful in better defining stress-related bone injury and determining appropriate management strategies.

CT also has been used to identify occult osteochondral defects in the metacarpophalangeal joint, describe the microvasculature of the proximal sesamoid bones (PSBs), and characterize the axial border of the PSB in horses suffering from osteomyelitis.[19,23,29] Occult subchondral bone cysts in regions such as the distal tibia, talus, and proximal phalanx have been identified.[5,24] Occult osseous cyst-like lesions were identified in the tarsocrural joint in 11 horses (see Fig. 20-2).[24] CT was the only imaging modality that consistently identified the presence of a cyst-like lesion. CT permitted both identification of the lesion and a means of planning surgical access.

The disadvantages of CT include the need for general anesthesia, inability to image proximal to the distal third of the radius and tibia, the production of artifacts deep to dense bone as well as around metal objects (e.g., implants), and cost for the client.[3] The latter is arguable, because in most horses the cost for the study, including general anesthesia, is comparable to that of nuclear scintigraphy.

Currently the United States has approximately six CT facilities, and a larger number of facilities are available in continental Europe, both in academic institutions and private practices. It is likely that the number of centers offering CT will increase in the coming years, and hopefully our diagnostic capabilities will increase.

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CHAPTER • 21

Magnetic Resonance Imaging

Chris Whitton, Rachel C. Murray, and Sue J. Dyson

Until recently, magnetic resonance imaging (MRI) of horses has been limited to cadavers because of the difficulty of positioning equine limbs in long, narrow magnets. Advances in equipment design have made MRI of live horses a reality.

PHYSICS OF MAGNETIC RESONANCE IMAGING

The creation of MR images is a complex process, and this is a simplified description. The image is produced by exciting hydrogen nuclei in the body at a specific resonance frequency within a static magnetic field and then detecting the energy released as the nuclei relax. The area to be imaged is placed within a static magnetic field. Magnetic field strength is generally divided into high field (1 to 1.5 tesla [T]), mid-field (0.5 to 1 T), and low field (<0.5 T). Superconducting magnets generate high fields, whereas resistive or permanent magnets generate low fields. The hydrogen nuclei within the tissues align with the magnetic field. The equipment then applies radiofrequency pulses specific for hydrogen nuclei from a radiofrequency coil placed around the area of interest. The hydrogen nuclei absorb the radiofrequency pulses and change alignment. Nuclei that are not aligned with the radiofrequency coil in turn produce a radiofrequency signal that is detected by the coil. The strength of the signal depends on the sequence of radiofrequency pulse or pulses applied and the amount of fat or water within the tissue. The most abundant sources of hydrogen nuclei are water and fat, so most signal in MRI is derived from these.

Creation of a three-dimensional (3D) image requires that the signal be positioned in space. This is achieved by using three gradient coils that change the intensity of the static magnetic field in different directions, thus affecting the intensity of the signal based on the position of the hydrogen nuclei. The resulting images can be constructed mathematically by computer software using Fourier transformations, with signal intensity shown in gray scale. High signal areas are shown as white, and low signal areas are shown as black. MR images can be acquired as two-dimensional slices of varying thickness, with the signal averaged across the slice of tissue imaged, or as 3D data sets. Three-dimensional acquisition allows thinner slice thicknesses and improves signal to noise ratio, and the data can be shown either as 3D reconstructions or as slices in any plane.

EQUIPMENT SUITABLE FOR IMAGING HORSES

The major factor limiting the use of MRI in horses has been an inability to position areas of interest in magnets designed for people. For high-quality images the area to be examined must be placed in the imaging portion of the magnet, close to its isocenter. High-quality image production requires creating a uniform magnetic field. This and the need for high-strength magnetic fields initially resulted in the production of closed, long, narrow cylindrical-bore magnets. However, high-field, short-bore magnets with flared ends are now available and have been successfully used to image up to and including the carpus and tarsus and head and cranial neck of adult horses (Fig. 21-1). Open magnets also have been produced for the medical market and use a lower field magnet, but the problems with creating a uniform magnetic field mean that the image quality tends to be inferior.

Radiofrequency coils designed for the medical field can be used in equine imaging, although imaging could ultimately be optimized by design of specific radiofrequency coils for equine extremities. In live horses, successful imaging of limbs up to and including the carpus and tarsus has been achieved using a human extremity (or knee) coil, whereas a torso array coil designed for the human torso has proved practical for imaging the head.

PRACTICALITIES OF IMAGING HORSES

The use of high-strength magnetic fields creates special problems for MRI in horses. To acquire images, the area of interest must be within the center of the magnet and must be immobile for the duration of each scan sequence; thus general anesthesia is required. Loose ferrous material close to the magnet is hazardous, and fixed ferrous material can produce imaging artifacts. A height-adjustable anesthetic table made of nonferrous material is required as are an MR-compatible anesthetic machine, ventilator, and anesthetic monitoring devices.

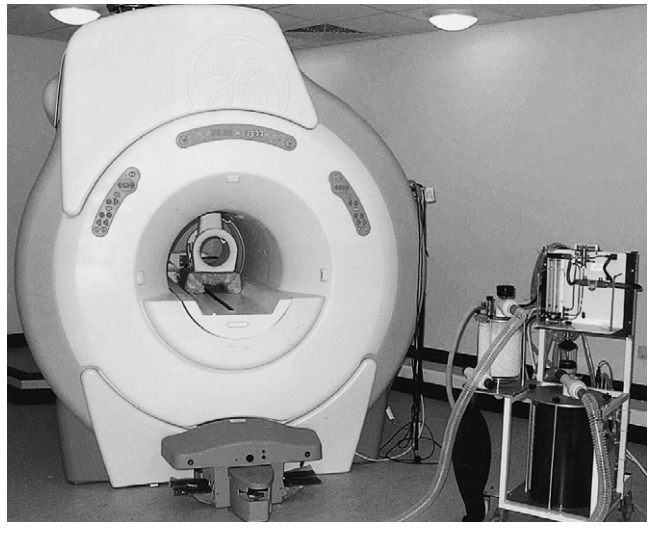

Fig. 21-1 The 1.5-T GE Echospeed (General Electric Medical Systems, Milwaukee, WI) magnet at the Animal Health Trust, Newmarket, is used for MRI of live horses. Located in the bore of the magnet is a radiofrequency coil suitable for use on the equine distal limb. Both limbs are placed into the magnet bore. A specially shaped pad allows the coil to be placed only over the limb being imaged and in the center of the magnet.

Before anesthesia is administered, any shoes and associated nails must be removed from the horse and debris must be cleaned from the area of interest. Any clipping or preparation of the horse that requires metal equipment must be performed before entering the imaging area. Placing the area of interest in the radiofrequency coil and then positioning it in the isocenter of the magnet requires a well-trained team.

SEQUENCES FOR ORTHOPEDIC IMAGING

The pulse sequence used to create the MR image describes the sequence of radiofrequency pulses applied during an MRI study and the associated application of magnetic field gradients. Numerous pulse sequences can be used to highlight different tissues and various pathological changes. These may be T1 or T2 weighted, depending on the detection of T1 or T2 relaxation of hydrogen nuclei. On T1-weighted images, tissues with fast T1 relaxation such as fat show high signal intensity (bright). On T2-weighted images, tissues with slow T2 relaxation such as water show high signal intensity. The relative weighting of the image determines the signal intensity of and contrast between different tissues. In general, structures with a high water content are bright on T2-weighted images, and those with a high fat content are bright on T1-weighted images. T1-weighted images show anatomy well, whereas T2-weighted images are more likely to demonstrate pathological conditions as reflected by increased water content.

A variety of sequences is available, including basic spin echo (SE) and fast spin echo (FSE) sequences, gradient echo (GRE), spoiled gradient echo, and inversion recovery. New sequences are continually being developed. In a live equine patient, time under anesthesia may be critical in preventing post-anesthetic complications, and thus it is important to aim for short acquisition times while maintaining image quality. More than one sequence may be used for each diagnostic procedure to maximize the information provided.

The conventional spin echo sequence is acquired as a two-dimensional image and may be either T1 or T2 weighted. However, acquisition times are long and impractical for live horse imaging. Fast spin echo sequences can be used to reduce acquisition times while maintaining signal to noise ratio. The reduced acquisition times make this sequence a practical option for live horse scanning. Although a fast spin echo sequence potentially reduces coverage compared with a spin echo sequence, this does not seem to cause significant problems clinically. The weighting of fast spin echo is similar to that of spin echo, except that the fat remains bright on T2-weighted images.

The purpose of the gradient echo sequence is to increase the speed of the scan, and therefore this sequence has significant clinical application in the horse. In addition, fast acquisition using the gradient echo sequence has the advantages of allowing 3D acquisition (showing higher detail) and increased sensitivity to the magnetic effects of hemorrhage. These advantages make the gradient echo the optimum sequence for equine orthopedic imaging, but increased susceptibility to inhomogeneities in the magnetic field occurs with a risk of magnetic susceptibility artifacts. A basic gradient echo sequence is largely T2 weighted, whereas the spoiled gradient echo produces a largely T1-weighted image. Clinically the gradient echo sequence appears useful for detecting pathological conditions of the cartilage, tendon, and bone, whereas the spoiled gradient echo sequence appears most useful in defining anatomy, showing cartilage and subchondral bone morphology and detecting cartilage surface defects. A flip angle of approximately 30° produces the best images.[1]

Sequences using fat suppression increase the sensitivity of T2-weighted images to edema in bone. Short inversion recovery sequences are the most useful in orthopedic imaging and can be used to improve subtle T1 contrast (e.g., between articular cartilage and synovial fluid) that is usually overshadowed by the far greater difference in signal intensity between fat and most other tissues on T1-weighted images.

Specialist techniques can enhance aspects of the image and reduce artifacts. Magnetization transfer has been used in human imaging to enhance visibility of articular cartilage. The magnetization transfer pulse selectively saturates tightly bound protons within tissues and reflects the equilibrium between freely mobile protons and protons constrained by macromolecules within the same tissue. Articular cartilage, hyperplastic synovium, and muscle show marked magnetization transfer effects, whereas fat and joint fluid remain relatively unaffected, thus increasing the contrast between structures within a joint.[2]

INTERPRETATION OF MAGNETIC RESONANCE IMAGES

An understanding of MRI, a detailed knowledge of anatomy, and considerable experience are required to interpret MR images reliably. Lesions that may be detectable using MRI but are difficult to detect with other imaging modalities include ligament and tendon injuries (within the hoof capsule), occult fractures, articular cartilage damage, and subchondral bone remodeling. For best assessment of structures, images are routinely obtained and analyzed in three planes (sagittal, dorsal, and transverse). Interpretation is improved by assessing T2-weighted, T1-weighted, and fat-suppressed images at each site.

In T1-weighted images, fat has a high signal intensity (bright) and water has a low signal intensity (dark); therefore the bone marrow of the medullary cavity of bones is bright, with darker synovial fluid (Fig. 21-2). Cortical bone and other mineralized tissue is dark (nearly black). T2-weighted images

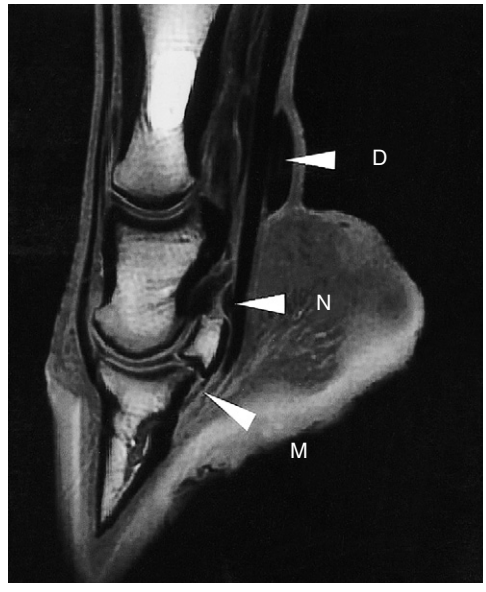

Fig. 21-2 Sagittal slice of the distal limb using a T1-weighted spoiled gradient echo sequence. The high signal in the medullary cavity of the phalanges and navicular bone is caused by fat. No signal appears in the deep digital flexor tendon, straight sesamoidean ligaments, and dense bone. Increased signal in the most distal portion of the deep digital flexor tendon is probably a magic angle effect (*M*). Synovial fluid is dark in the navicular bursa (*N*), distended digital flexor tendon sheath (*D*), and proximal and distal interphalangeal joints, but cartilage shows a relatively brighter signal.

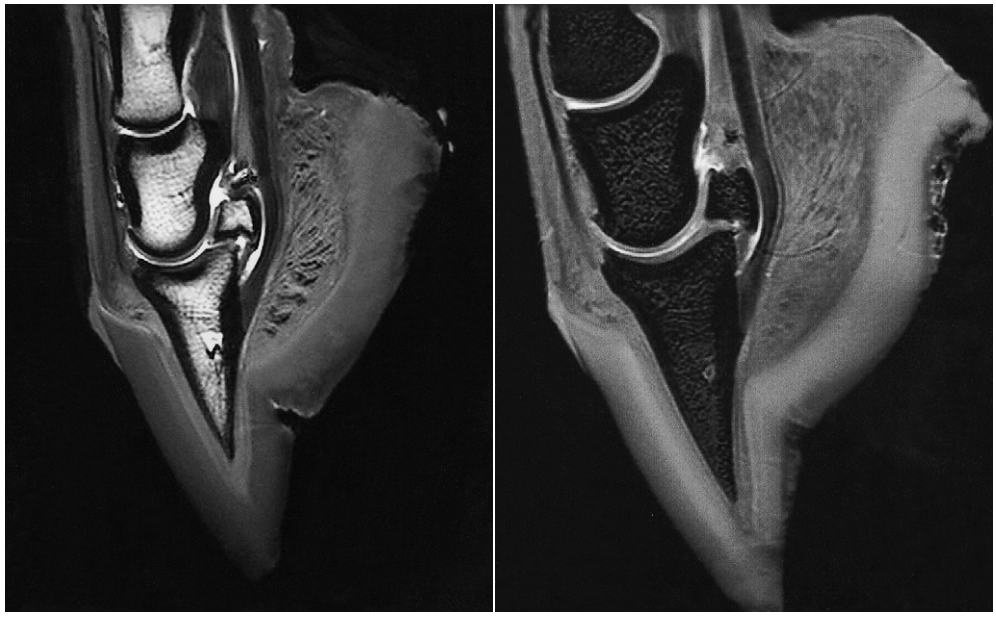

Fig. 21-3 Sagittal slices of the distal limb using a T2* gradient echo sequence. **A,** Three-dimensional T2* gradient echo shows a relatively bright signal associated with medullary fat of the phalanges and navicular bone, but on this sequence fluid is bright (compare with Fig. 21-2). Synovial fluid in the navicular bursa and joints is bright, but cartilage exhibits a relatively low signal. **B,** Fat-saturated, 3D T2* gradient echo shows the absence of fat signal in the medullary bone and improved contrast between articular cartilage and surrounding structures.

produce a high signal in water and a lower signal in fat, and thus synovial fluid appears white because of its high water content. Although T2 spin echo sequences show bones as dark throughout, using the more clinically applicable fast spin echo and gradient echo sequences, fat maintains relatively high signal intensity so bone marrow remains relatively bright, unless fat is suppressed (Fig. 21-3). On T2-weighted images, cartilage has low signal intensity. In all images, dense tissues such as subchondral bone and tendon have low signal intensity.

Although cortical bone produces low signal, MRI is extremely useful for detecting pathological conditions of the bone using either T1- or T2-weighted images. Fractures show as defects in bone outline and structure and may show as lines of increased signal on T2-weighted images (Fig. 21-4). The surface contour of bone can be accurately assessed, allowing the detection of osteophytes, which are better delineated by MRI than by conventional radiography,[3] and periosteal and endosteal new bone (Fig. 21-5). Trabecular architecture changes can be maintained.[4] Subchondral bone irregularity appears best on standard (non–fat-suppressed) images.

MRI is currently the most sensitive technique for detecting osteonecrosis, osteomyelitis, and trauma, including bone contusion and non-displaced fractures.[4] Pathological conditions of the bone are detected as a local increase in fluid content. In clinical imaging of horses, fat-suppressed and standard T2-weighted sequences must be compared. The fluid signal in medullary bone often is obscured by the high signal produced by fat on standard T2-weighted images, and therefore fat suppression techniques are needed to reduce interference from the fat signal. The fluid signal is frequently referred to as bone *edema*, although it reflects bone necrosis, inflammation, trabecular microdamage, and fibrosis as well as bone edema.[5,6] The ability to detect edema within bone as an increased signal on T2-weighted images means that MRI is extremely sensitive for detecting subtle bone injury. In people, subchondral bone injury, referred to as *bone bruising,* is commonly observed with acute cruciate ligament and meniscal injury and is not detectable by any other means. In horses, bone edema on MRI has been observed adjacent to large fractures and subchondral bone edema at sites of trabecular microdamage, hemorrhage, and edema (Fig. 21-6) and in the navicular bone at sites with histological evidence of edema or bone necrosis.[7]

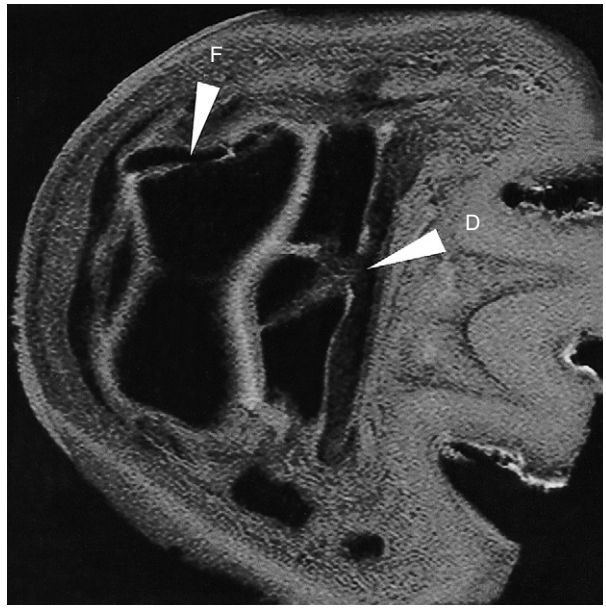

Fig. 21-4 A transverse 3D T2* gradient echo image of the distal limb showing a comminuted fracture of the navicular bone and fracture of the middle phalanx that had been detected radiographically (*F*), and laceration of the medial branch of the deep digital flexor tendon (*D*), which is interposed between the fracture fragments of the navicular bone.

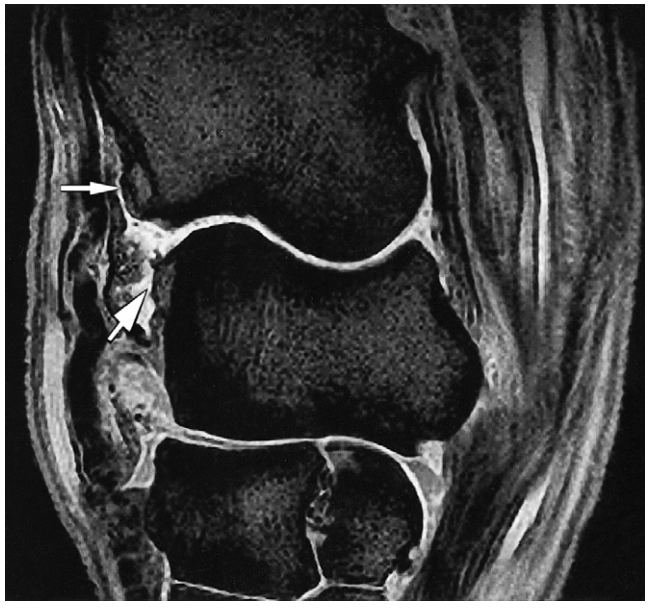

Fig. 21-5 A sagittal T2* gradient echo slice of a carpus demonstrating osteophyte formation on the distal radius and proximal radial carpal bone in the antebrachiocarpal joint (*arrows*). Synovial effusion of the antebrachiocarpal and middle carpal joints occurs with synovial proliferation on the dorsal aspect of the antebrachiocarpal joint.

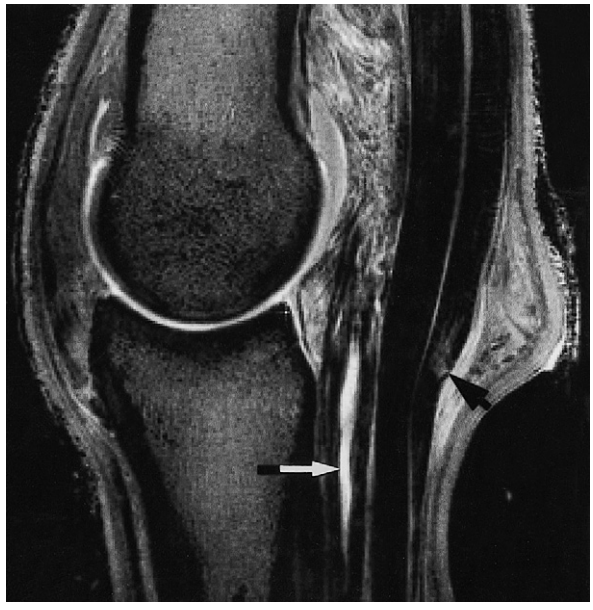

Fig. 21-7 A sagittal T2* gradient echo slice of a fetlock of a horse with lameness that was alleviated by intra-articular analgesia of the metacarpophalangeal joint. A linear area of high signal appears in the straight sesamoidean ligament (*white arrow*). Increased signal within the superficial digital flexor tendon (*black arrow*) corresponds to an area of cartilage metaplasia.

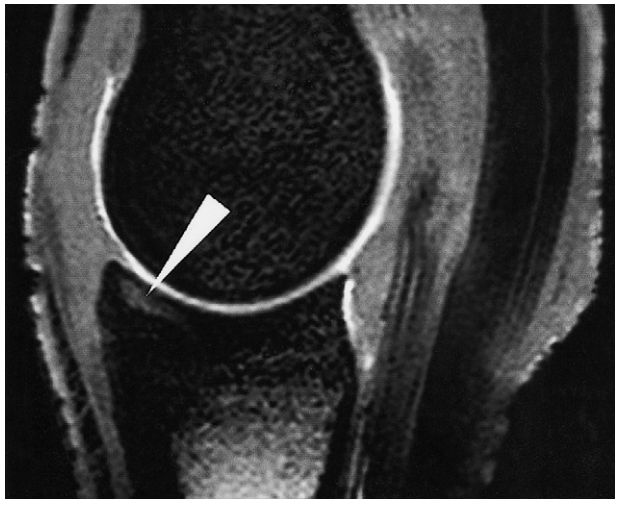

Fig. 21-6 Sagittal fat-saturated, T2* gradient echo slice of metacarpophalageal joint showing an area of bright (fluid) signal in the subchondral bone of the proximal phalanx (*arrow*), representing a pathological condition of the bone. Histopathological examination revealed subchondral bone edema, inflammation, and trabecular microdamage.

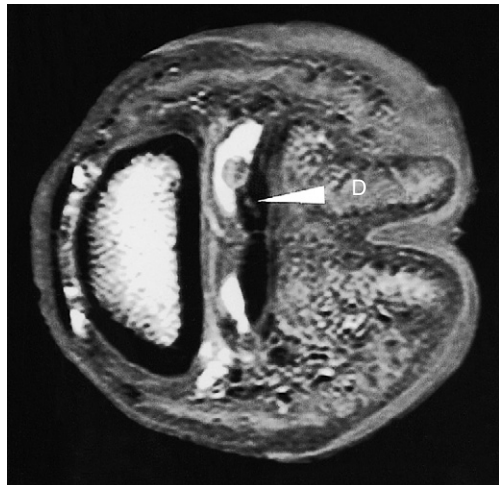

Fig. 21-8 A transverse 3D T2* gradient echo slice of the distal limb of a horse with lameness that was alleviated by intra-thecal analgesia of the navicular bursa. There is an area of increased signal within the medial branch of the deep digital flexor tendon (*D*), with adjacent granulation tissue protruding into the navicular bursa.

Like cortical bone, tendons and ligaments generally have low signal intensity on T1- and T2-weighted images and so appear nearly black. Identifying the interface between dense collagenous tissues and bone may therefore be difficult. As with bone, tendon and ligament injury is identified by an increase in signal (Figs. 21-7 and 21-8). In acute stages of equine tendon and ligament damage, focal pathological conditions tend to appear as areas of bright signal on T1- and T2- weighted images with detectable local swelling. In the inflammatory phase, dark linear signal separated by areas of high signal may represent damaged fibers surrounded by cellular infiltration. During the later phases of healing and fibrosis, signal intensity tends to decrease but remains higher than that of normal tendon.[8] These changes must be differentiated from normal areas of increased signal. Areas of cartilage metaplasia in tendons often occur at sites subject to compressive forces such as the deep digital flexor tendon (DDFT) at the level of the metacarpophalangeal joint (see Fig. 21-7). Increased signal may also be observed when the collagen fibers of a ligament or tendon are placed at an angle of 55° to the

static magnetic field. This is termed the *magic angle effect* and is an artifact (see Fig. 21-2). Changing the position of the limb within the static magnetic field confirms this as an artifact.

Muscles contain less collagen than tendons and therefore have intermediate signal intensity on T1- and T2-weighted images.[4] Muscular inflammation appears as increased signal intensity on T2-weighted images related to elevated water content (edema) and loss of collagen. Fibrosis lowers signal intensity on T2-weighted images.

Although low-field MR systems tend to produce poor images of articular cartilage, use of high-field systems has demonstrated that articular cartilage can be assessed accurately using MRI. Articular cartilage has a complex appearance on MRI because of the different collagen orientation and composition of layers present in hyaline cartilage, and imaging is prone to artifacts.[9] However, improvements in pulse sequence design and image interpretation have eliminated several of these problems. Commonly, T2-weighted fat-suppressed fast spin echo and gradient echo sequences are used clinically to detect pathological conditions of cartilage in people.[10,11] This is also supported by experience with equine cartilage, where use of fat-suppressed 3D gradient echo sequences, with minimal thickness, contiguous slices, maximizes information obtained in the shortest time.[1] Use of fat-suppressed images not only improves contrast in the cartilage but also highlights subtle areas of subchondral bone edema, which may draw attention to a defect in the overlying articular surface.[12] MR findings suggesting cartilage degeneration include signal abnormality and contour or thickness abnormalities. On T2-weighted images the low signal of cartilage and the high signal intensity of synovial fluid give clear delineation of cartilage fluid interfaces. Pathological conditions can be detected as altered signal intensity. Loss of collagen can lead to focal signal loss, whereas increased signal intensity may indicate matrix damage.[4]

Detailed images of the synovium allow assessment of synovial proliferation and synovial distention (see Fig. 21-5). Synovial villi and evidence of adhesion formation are easily observed, which is particularly useful for assessing the synovial structures within the foot, including the navicular bursa, where clinical experience has indicated the significance of navicular bursa distention, synovial proliferation, and adhesion formation detected by MRI.

ARTIFACTS

As with any imaging technique, MRI is prone to production of artifacts that may confuse the interpreter. *Ghosting* (repeated picturing of a structure throughout the image) originates from movement during the acquisition of data because of respiration or pulsatile movement of vessels. This can cause problems in equine patients because respiratory chest movement causes large displacements of the upper forelimb during imaging. Motion can be minimized by weighting the limb with sand bags or reducing respiratory excursion by avoiding mechanical ventilation. Alternatively, techniques such as respiratory compensation or triggering and flow compensation can be used. Anything metallic can severely distort the image, which is known as *a magnetic susceptibility artifact* and is more prominent in gradient echo sequences. Shoes and residual nails must therefore be removed from feet, and dirt must be cleaned from the sulci of the frog. Wounds may be contaminated with fine metal particles, and these particles may be detected by MRI after apparently remaining embedded for a prolonged duration.[13] *Chemical shift artifact* occurs only at high field strengths and occurs when fat and water that are adjacent in the patient are shown farther apart on the image. If part of the patient is just outside the field of view but produces a signal detectable by the receiver coil, this may be shown incorrectly within the field of view, known an *aliasing*. This problem can be combated by using "no phase wrap" or "no frequency wrap" techniques in the relevant phase or frequency direction. The *Gibbs effect* (truncation artifact) results from under-sampling of data and occurs when bright or dark lines appear parallel and adjacent to borders of abrupt intensity change, for example, a line of low signal is incorrectly shown running through a high intensity area. *Partial volume effects* result from structures passing obliquely through the slice of tissue being imaged. Reducing the slice thickness minimizes this effect. The *magic angle effects* appear when collagen fibers are located at an angle of 55° to the static magnetic field. This is most important in ligaments and tendons, although it has been reported to occur in cartilage. Increasing the echo time reduces the effect, which is less evident on T2-weighted images.

INDICATIONS FOR MAGNETIC RESONANCE IMAGING IN EQUINE LAMENESS

MR imaging is time consuming, and the need for general anesthesia limits the number of sites that can be imaged because of time constraints and the practicalities of moving a horse within the scanner. It is therefore important to localize the site of lameness with clinical examination and nerve blocks before imaging and to rule out differential diagnoses with other diagnostic techniques. Because of anatomical variations between horses, imaging of both limbs is recommended to provide a comparison. The detail detectable by MRI means that the significance of potential abnormalities observed needs to be evaluated in the light of the clinical presentation. MRI is most useful when lameness has been localized to a site with no detectable radiographic or ultrasonographic changes. In some situations, because of the non-specificity of local analgesic techniques, the clinician may be directed toward a structure that is not the cause of the lameness. The ability of MRI to examine all tissues in an area at once may identify an adjacent structure as the cause of lameness. In the horse illustrated in Fig. 21-7, intra-articular anesthesia of the metacarpophalangeal joint alleviated the lameness. No significant radiological abnormalities were identified. MRI demonstrated a core lesion within the straight distal sesamoidean ligament. No significant abnormalities of the metacarpophalangeal joint were detected at post-mortem examination.

MRI is also particularly useful for lameness localized to the foot, where use of ultrasonography is limited by the hoof capsule. DDFT lesions in the foot are easily identified with MRI (see Fig. 21-8). MRI may also be indicated where scintigraphy shows changes but no radiological abnormality is detected and further information is required, such as the extent of subchondral bone injury. Using MRI, palmar heel pain has been shown to encompass a variety pathological entities, including lesions of the DDFT, distal sesamoidean impar ligament, medulla and flexor cortex of the navicular bone, and navicular bursa and osteochondral lesions of the distal interphalangeal joint.[14]

Because of advances in ultrasonographic technology the identification of pathological conditions within structures such as tendons and the suspensory ligament can be readily achieved in the standing horse. However, MRI is extremely sensitive to subtle changes in these structures, and except for magic angle effects, fiber alignment is not a problem as with ultrasonography. Therefore MRI is likely to be of great benefit when ultrasonographic examination findings are equivocal. In particular, use of MRI to evaluate proximal suspensory desmitis should be considered because of the difficulty in accurately diagnosing and monitoring this condition. Fig. 21-9 shows a

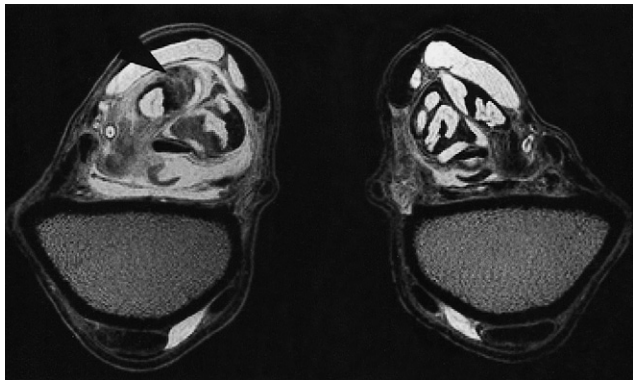

Fig. 21-9 Transverse T2* slices at the level of the musculotendonous junction of the superficial digital flexor tendon of the left *(shown on the left)* and right forelimbs. There is enlargement of the left superficial digital flexor tendon and its accessory ligament. Normal muscle tissue (areas of high signal) is replaced by an area of low signal consistent with scar tissue (*arrow*).

lesion within the musculotendonous junction of the superficial digital flexor tendon that was suspected with ultrasonography but was difficult to demonstrate conclusively. The lesion is easily identified with MRI.

Imaging of joints allows differentiation of degrees of cartilage damage and osteoarthritic change. Subchondral bone structures and pathological conditions can be identified with MRI, and the presence of bone edema can indicate active damage at this site compared with cartilage erosions without marked subchondral bone damage. Evidence of inflammation is seen clearly with increased quantities of synovial fluid, synovial proliferation, and capsular thickening. Small mineralized particles, such as bone fragments that are invisible radiographically, can be detected by MRI.

ADVANTAGES AND DISADVANTAGES COMPARED WITH OTHER IMAGING TECHNIQUES

Advantages

MRI is more versatile than most other imaging modalities in the ability to provide images sliced in many planes and 3D images in a variety of orientations. Like ultrasonography and radiography, MRI has the ability to provide anatomical information, but MRI also has a physiological sensitivity like that of nuclear scintigraphy.

MRI is the only method presently available that can assess all tissues during a single examination. Ultrasonography is extremely useful for imaging soft tissues but is impractical in a number of areas, most importantly the foot. MR technology allows imaging of those soft tissue structures that are inaccessible with ultrasonography. Radiography shows mineralized tissues but is less sensitive than MRI because each image is obtained through the full thickness of the area, and only the orientations selected can be assessed, so small abnormalities may be missed. In contrast, MR images can be obtained in any plane with no loss of image quality, and data can be assessed in slices so that the image is not limited to a summation through the entire structure. MRI has been shown to be more sensitive to pathological conditions of bone and tendons than radiography and ultrasonography, respectively.

Like MRI, computed tomography (CT) can be used in horses for accurate assessment of the 3D distribution of pathological conditions of the bone. Some soft tissue detail is also demonstrated with CT but with inferior contrast. Articular cartilage can be assessed only with CT by use of invasive arthrography. Arthroscopy is the standard of reference for the evaluation of articular cartilage, but it cannot assess deep chondral lesions and lesions of the subchondral bone.

Disadvantages

The greatest disadvantages of MRI are the expense of the equipment and the requirement for general anesthesia. MRI is limited to areas that can be placed within the magnetic field, and thus the caudal neck, trunk, and proximal limbs of adult horses cannot be imaged. MRI provides a huge amount of information, so interpretation is time consuming.

FUTURE DIRECTIONS

The high cost of MRI has been the major limiting factor in its use in equine orthopedics. Because of the technical difficulties in producing high-field magnets, the price of MRI equipment will not likely decrease dramatically in the near future. However, developments in low-field equipment are interesting: low-field image quality is improving rapidly, and purchase and running costs are significantly cheaper than for high-field units. Dedicated low-field units have found a place in human orthopedics for identifying gross lesions of menisci and cruciate ligaments and subchondral bone injury. Open magnets offer much greater flexibility in design. Open magnets for imaging the limbs of standing horses have been proposed, but the low field strength achievable with such magnets would severely limit the image quality and resolution. Movement also significantly degrades image quality, further limiting the development of MRI of the standing horse. Motion correction software could conceivably improve image quality.

Major improvements in the accessibility of the imaging area of high-field magnets have occurred in recent years, but further developments are limited. High-field units remain the gold standard in MRI, and these devices will become more common in referral institutions and will greatly improve the diagnosis of orthopedic conditions in horses. Other potential MR techniques that could have applications in the future of equine orthopedic imaging are spectroscopy to look at metabolite distribution, diffusion studies, angiography, and use of contrast agents.

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CHAPTER • 22

Gait Analysis for the Quantification of Lameness

Alicia L. Bertone

The quantification of equine gait and lameness can be achieved by several methods that assess kinematics (limb position in time and space) and kinetics (direction and magnitude of forces upon limb loading). Integration of these data permits the calculation of impulses (average loading forces/stride or phase of stride), net joint moments (torque around a joint), and joint power (rate of mechanical energy generation and absorption across the joint).

KINEMATICS

Kinematic analysis of gait includes measurement of joint angles, stride length, duration of stance phase, and head excursion and can be extrapolated to estimate forces of loading[1,2] or combined with force plate data to calculate joint power and moments.[3-6] Although kinematic studies can be performed in the field, control of outside influences on gait, gait velocity, and camera relationship to the limbs is difficult. Therefore most studies are performed with the horse on a treadmill so that velocity can be controlled and with several cameras simultaneously so that all limbs can be assessed. Three coordinates (X, Y, and Z) are analyzed to define the position of the limb in space. A high-speed video camera capable of 60 frames/sec is adequate for assessing equine stride-timing characteristics at slow trotting velocities (3 m/sec), but a higher speed camera is preferable for higher gait velocities (up to racing speeds of 18 to 20 m/sec) or detailed limb/hoof placements.[7]

The most popular systems include a camera, computer, and light-emitting or light-reflecting markers that can be resolved by the camera based on location, reflection pattern, color, or a combination of these factors. Data acquisition software can be customized, but most systems have a method to control the duration of data collection (time or stride number) and automatically average values over the defined data acquisition phase for a predefined number of variables. Collection and averages continue as long as the program is activated. Data usually are displayed in real time on a monitor as a "stick figure" of the markers to allow marker recognition and tracking. Data can be immediately stored and later used for analysis and data printing. Data sets can be immediately deleted if other factors invalidate the trial, such as loss of a marker or failure of marker detection, errors in the treadmill, head shaking, or gait velocity variations, such as the horse skipping. Stride stance phase, joint angles (the joint number limited by the number of markers the system can distinguish simultaneously), and head excursions are analyzed. One marker must serve as a reference marker for calculations and usually is placed on the scapula. Placement of other markers on long bones, hoof, and head (atlas) allows calculation of joint angles and head excursion (nod), respectively. Typically, 30 seconds to 2 minutes of data are collected (representing approximately 30 to 120 strides) for 5 to 10 trials, which are averaged to produce 1 valid trial. Immediate printing of valid data for storage in the experimental or clinical record is recommended.

System accuracy and repeatability are currently limited by camera placement, marker diameter (~6 cm), marker movement with skin, marker resolution (markers too close, or of indistinguishable shade or reflection), and marker placement consistency from trial to trial. The camera must be placed at the center of stride length and at a distance that allows collection of the entire movements of all markers to avoid distortion of image and miscalculation of values. Background camera images of handlers, treadmill railings, and floor surfaces should be minimized and made as homogenous as possible. Control of light reflection with directed halogen lamps, a light-controlled treadmill environment, and black covers to reduce background reflection greatly assist. A protocol should be established at each testing center. White and multicolored horses can present problems. Clipping of hair and use of glue-on hook-and-loop fasteners (Velcro; Velcro USA Inc., Manchester, NH) can assist with repeatable marker placement on successive days. An established protocol system and technical assistance that can effectively train horses for the treadmill and operate the cameras and computer are essential for reliable data comparison among trials and horses.

For quantification of equine lameness, several landmark papers identified findings that moved kinematics closer to practical use in the diagnosis of lameness and related the findings to classical lameness scores. Head and withers excursion (head nod) were the only parameters evaluated that significantly changed after lameness was induced to a grade 3

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(American Association of Equine Practitioners scoring system) by carpal amphotericin B injection.[8] Head excursion was a reliable indicator of lameness in horses with navicular disease.[9] Fetlock extension angle also decreased significantly. However, gait analysis with a single 60-Hz (frames/sec) camera may not provide the resolution necessary to consistently identify small changes in kinematic variables, such as fetlock angle, that occur with change in lameness. In our gait laboratory, analysis of more than 296 independent, blinded kinematic trials (approximately 35,000 measured strides) of 12 variables associated with lameness scores ranging from 0 to 4, resulted in low correlation coefficients ($r \cong 0.4$). Using fetlock extension angle as the example, the high coefficient of variation in fetlock angle measurements from stride to stride (over 30 seconds to 2 minutes) relative to the percent mean change in fetlock angle as horses went from lame to sound (range 1% to 6%) resulted in low sensitivity for kinematic analysis of fetlock angle to detect lameness change. Improvements resulting in greater sensitivity, specificity, and repeatability, such as smaller marker size, control of marker motion relative to true centers of joint motion,[10,11] and incorporation of measures of limb acceleration and deceleration into programs,[12,13] will promote kinematic lameness quantification. Use of simultaneous collection of right and left limb data allows for enhanced sensitivity because joint angles in the contralateral limb change in the opposite direction to the lame limb. This doubles the measured difference between limbs in lame horses. Problems with variation in skin marker and camera placement from side to side produce additional variability that affects these measurements.[12] With forelimb lameness, load may be shifted to hindlimbs complicating relative limb measurements. Assessment of gait symmetry should analyze all four limbs simultaneously. Analysis of bilateral forelimb lameness or concurrent forelimb and hindlimb lameness is not straightforward. Until these issues are resolved, kinematic gait analysis will probably remain a second choice to kinetic analysis for the quantification of lameness.

Analysis of clinician scoring of lameness and kinematic quantification has exposed the limited ability of clinicians, especially those who are inexperienced, to grade mild lameness consistently. This highlights the need for improvement in methods to quantify lameness accurately. However, in the author's and editors' view an experienced trained clinician is currently more sensitive than kinematic assessments and offers the flexibility of observation in many conditions and gaits. This is further supported by the close correlation of trained clinician lameness scores and kinetic analysis (see the "Kinetics" section).

Kinematic analysis also has been used for measurement of activity indices,[14] dimensions of growth,[15] estimates of flexibility,[16] accelerometry,[12,13] and joint moments and joint power.[3-6] Joint moments, joint power, and applied forces[17] can be calculated by the simultaneous measurement of kinematic parameters and force plate recordings. Measurement inaccuracies of each system may limit the power of this type of analysis, but theoretically these calculations could provide another index that could be correlated to lameness and may be used to detect joints receiving most of the torque and absorbing most of the energy during various sports. This may provide insight as to which joints are likely to be injured and how to adjust shoeing, riding surface, and gait during or after injury to assist with healing, rehabilitation and prevention of re-injury. These areas of equine locomotion science are expanding and are expected to assist with lameness quantification and detection.

KINETICS

Weight-bearing profiles that either directly measure the direction and magnitude of force on loading, or can be used to extrapolate these forces, can be obtained with hoof strain gauges,[17,18] instrumented horse shoes,[19-21] force plates,[1,2,22-26] and pressure-sensitive shoes.[27] Currently marketed and published systems for equine kinetic quantification include force plates and pressure-sensitive systems.

Force plate quantification of ground reaction force (GRF) is the current gold standard for objective quantification of lameness in horses and dogs.[1,2,23-25,28-30] Peak vertical GRF decreases with increased lameness scores.[1,2] In our gait laboratory, analysis of 150 blinded force plate valid trials (1200 individual measurements), associated with lameness scores ranging from 0 to 4, resulted in high correlation ($r > 0.85$) of peak vertical GRF with subjective lameness score. These correlations were significantly higher than for kinematic gait variables evaluated using the same lipopolysaccharide-induced arthritis model of lameness.[31] Variability among force plate trials is low, and reduction in peak forces range up to 50% for lameness of grade 4. This large percentage change of peak forces associated with change in lameness and low standard deviation among repeated measurements provides a sensitive system that can detect small percentage changes in lameness associated with treatment, surgery, or time. Standard GRF patterns and variation in normal and lame horses have been described and have confirmed the high repeatability of force plate data provided velocity is controlled within a narrow range.[1,2,25] Measurement of gait velocity has been incorporated into data acquisition systems using wall-mounted photoelectric detection.[1] Velocity is immediately displayed at the end of the force plate pass, and pre-established cut-off ranges can determine validity of the trial.

Force plate equipment uses quartz crystal sensors and at least four sensing elements in the corners of an approximate 1-m × 0.5-m plate that is mounted in the ground to be level with a 10-m lead-in and lead-out runway. Quartz crystals respond to changes in shape and generate an electrical charge (piezoelectric effect) oriented to the external force. Three orthogonal GRFs are measured: mediolateral, craniocaudal or braking/ propulsion, and vertical. The voltage produced is directly proportional to the applied force. The electrical output of the crystals is collected by electrodes, and data from the four corners of the plate are integrated to determine orthogonal GRFs. Although other strain-sensing elements exist and can be accurate, piezoelectric systems are highly sensitive and accurate over a large measuring range, do not require recalibration even after years of use, do not require an external energy source, and are not damaged by overloading. These practical advantages provide significant incentive for continued use.

The installation of force plates requires forethought for maximal benefit and ease of use. Force plates should be secured in a concrete foundation. The sensors are encased in a heavy metal mounting plate. The selection of a site for the force plate should consider the following factors. The surroundings should be non-distracting to minimize the number of passes necessary to collect a valid trial. At least 10 m of space should be allowed on either side of the plate for adequate room for the horse to establish a stable speed and uniform stride rhythm. A 20-m long roll-out thin rubber carpet is placed over the force plate so that the horse does not avoid it, and to provide a non-slip surface, a path for the horse to follow and easy clean-up. The mat must be cut around the outside border of the force plate so as not to influence the measurement of forces in any of the three planes. Wall-mounted sensors for stable velocity measurements that can feed directly into the computer are useful. A sliding windowed door to the area housing the computer should be positioned to allow direct visibility of the force plate so that observers are hidden. The limb must be placed on the force plate to complete a valid trial. The limb to be measured should land squarely inside the edges of the plate without any

Box • **22-1**

Camera, Data Acquisition, and Analysis Software System Reported in the Literature

Camera system	*Manufacturer*	*System power*
Ektapro 1000	Eastman Kodak Co.; Rochester, N.Y.	1000 Hz[7]
CODA-3	Charnwood Dynamics; Loughborough, England	300 Hz[22,32]
16 MM-1PL	Photo-Sonics Inc., Burbank, Calif.	300 Hz[19]
Locam 164-5 DC	Red Lake Laboratories; Santa Clara, Calif.	~250 Hz[4,22]
Peak Modus Video System	Peak Performance Technologies; Englewood, Colo.	60 Hz or 180 Hz
Selespot II	Selcom, Sweden	120 Hz[33]
Panasonic AG-450 series Super-VHS camcorder	Panasonic Corp.; Seacaucus, N.J.	60 Hz[9]
TI-23A videocamera	NEC Corp.; Tokyo, Japan	60 Hz[7]
Sony FX-700 series	Sony; Tokyo, Japan	50 Hz[34]

Data Acquisition and Analysis Systems

System	*Manufacturer*
Peak Performance Technologies System	Peak Performance Technologies; Englewood, Colo.
ProReflex	Qualysis; Savedalen, Sweden[35]
Ariel Performance Analysis System	Ariel Dynamics, Inc.; Trabuco Canyon, Calif.[9]
VP 320	Motion Analysis Corp.; Santa Rosa, Calif.[32,33]
TrackEye System	Innovativ Vision AB; Linkoping, Sweden[36]
Columbus Instruments System	Columbus Instruments Corp.; Columbus, Ohio

of the hoof landing off the plate. Typically the ipsilateral hindlimb strikes the plate after the forelimb, and this is readily identifiable on the force-time curves. If the contralateral forelimb strikes the plate, the curves must be immediately identified to avoid later confusion. Use of a large plate for small animals makes it difficult to identify the limb of interest on the generated curves. The observer should immediately delete any trial that did not meet the velocity requirements, limb strike position, or curve pattern indicating stride interference. Handler physical conditioning and practice is critical to maximize efficiency of valid trial collection. The horse must pass the photoelectric detectors at a predefined velocity, and the horse must trigger both the force plate and photocell. It may take as few as 5 passes or as many as 50 passes to achieve a valid trial. An acceptable valid trial is usually composed of 5 to 10 measurements that meet all the criteria. Statistical power calculations can be customized from the data collected at each testing center to ensure that adequate numbers of measurements are made to comprise a valid trial. Software is provided with the system to calculate normalized force (newtons per kilogram of body weight). Typical parameters evaluated include peak (maximum) force, average force, impulse (force over time), loading rate (slopes) and unloading rate. These can be calculated for each of the three directional forces: vertical, craniocaudal, and mediolateral. If reduction of the data to a single value that reflects lameness is desired, peak vertical force should be used.

Instrumented horse shoes have continued to evolve to provide a method to analyze weight-bearing forces in successive strides or on a treadmill.[17-21,27] These systems may prove to be repeatable and accurate at quantifying lameness, as initially reported.[27] Further studies verifying repeatability over days of use, after reapplication of shoes, and in clinical cases will be important for these systems to gain acceptance for most practical applications in quantification of equine lameness. Proof of sensor durability, evidence of correlation with the entire range of lameness scores, including no lameness, and use of kinematics to document the changes in gait associated with the weight and bulk of the system on the limbs are required. Telemetric control of these systems would provide a substantial advantage because data acquisition would not be limited to a treadmill. Systems reporting pressure across the entire hoof contact surface offer a potential advantage of quantifying the force distribution within the hoof.

FUTURE USES

Future directions for the quantification of lameness include the incorporation of telemetry to allow use in natural settings and avoidance of wires attached to limbs that probably affect gait. Hoof-based systems that are accurate, but not bulky or heavy, and a single system that directly measures three-dimensional forces and the distribution within the hoof are required. A simplified data acquisition and analysis system that can integrate all four limbs simultaneously also is needed. See Box 22-1 for a list of camera, data acquisition, and analysis software systems.

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CHAPTER • 23

Arthroscopic Examination

Mike W. Ross

Arthroscopic surgery is arguably the most important advance in management and one of the most important in diagnosis of equine joint disease. Arthroscopic surgery has been a mainstay in managing joint disease since the early 1980s and has mostly replaced arthrotomy. An effective equine surgeon cannot lack extensive arthroscopic surgical experience, and lameness diagnosticians must understand indications and limitations of the technique. Innovators have used the same instruments for bursoscopy and tenoscopy (see Chapter 24).

ADVANTAGES AND DISADVANTAGES OF ARTHROSCOPIC SURGERY COMPARED WITH ARTHROTOMY

Arthroscopic surgery offers several advances compared with arthrotomy; however, this type of surgery also has some disadvantages. Both are discussed in the following text.

Advantages

Improved Visibility Improved visibility during arthroscopic examination allows for evaluation of most of the joint compared with the limited view provided by arthrotomy. Even long arthrotomy incisions rarely provide added visibility because overlying capsule, retinaculum, and sheaths make retraction difficult. During arthroscopic examination, cartilage at locations distant to the primary lesion site, synovium, and intra-articular soft tissue structures such as ligaments can be examined. For instance, newly described disease conditions involving intercarpal ligaments were not known before arthroscopic examination existed.

Reduced Trauma and Morbidity Arthroscopic examination is less traumatic and causes less morbidity. Arthroscopic examination allows surgery to be performed through small incisions and requires less surgical exposure and damage to overlying soft tissues, upholding a time-honored principle of limiting trauma. Little pain occurs after arthroscopic surgery, and complications such as wound dehiscence and seroma formation are minimal. Horses often show pronounced lameness for several days after arthrotomy. After arthroscopic examination and surgery, owners and trainers expect horses to return to full work soon after surgery, an idea that is fueled by widespread reports of human athletes returning to professional sports quickly after arthroscopic surgery. A general misunderstanding is that incision size is the limiting factor. Although arthrotomy does cause short-lived lameness after surgery, the fear that it will delay onset of training is unfounded, because the underlying lesion dictates recovery time. Although not recommended, horses with mild conditions such as osteochondritis dissecans, horses with effusion but no lameness, and horses that receive prophylactic arthroscopic surgery can resume training a few weeks after arthroscopic examination or arthroscopic surgery. Arthroscopic surgery can be performed within weeks before a sale, and if hair was not clipped, surgical sites are barely noticeable.

Better Cosmetic Results Improved cosmesis is a definite advantage. Small incisions cause less fibrous tissue formation, and incisions are difficult to identify by 2 to 3 months after surgery. Generally the instrument portal (incision), the site through which the arthroscopic instruments are passed and lavage is performed, suffers more trauma than does the arthroscopic portal, and swelling, reactions, and fibrous tissue production are more common.

Earlier Functional Capability Earlier return to function was once believed to be an advantage of arthroscopic surgery. However, incision size has little to do with lameness except within the first few weeks after surgery. Lameness observed in horses returned to work 4 to 6 weeks after arthroscopic surgery for substantial articular lesions has nothing to do with incision size. When horses undergoing arthroscopic surgery were given only a brief rest, results were unsatisfactory. Incisions heal from side to side, not end to end, and incision size has no effect on articular healing. Lesions must heal as completely as possible before full work can begin, a process that often takes several months. Type and location of lesions are important, because horses with osteochondritis dissecans not involving weight-bearing surfaces can start training within 2 to 3 weeks of surgery, if necessary, whereas those with lesions in critical sites, such as the typical carpal osteochondral fragment, must be given 3 to 4 months of rest.

More Versatility Improved versatility is a definite advantage because joints considered inaccessible, such as the coxofemoral joint, can be evaluated. Techniques such as repair of third carpal slab fractures, distal third metatarsal (MtIII), or third metacarpal (McIII) fractures and those in other joints and resurfacing techniques can be done using arthroscopic surgery.

Fewer Complications Arthroscopic surgery has fewer complications. Arthrotomy of the scapulohumeral, femorotibial, femoropatellar, cubital, and tarsocrural joints has been associated with dehiscence, seroma formation, and infection, but arthroscopic surgery of these joints is safe.

Disadvantages

Expensive Instrumentation Expense of surgical instrumentation is a disadvantage because start-up costs are high, but basic instrumentation is no more costly than routine surgical instruments. Although arthroscopic surgery can be done without video equipment, video arthroscopy is valuable for archiving images. It is much more comfortable than direct view arthroscopy (looking directly through the eyepiece), because surgery is performed using a comfortably positioned monitor. It allows the procedure to be viewed by others and is valuable in maintaining aseptic technique, because during direct view arthroscopic surgery, breaks in technique often occur.

Lack of Surgical Experience Experience necessary to learn arthroscopic surgery is a disadvantage. Arthroscopic surgery requires skill in stereotactic techniques and orienting and operating without looking at the surgical site, and surgeons must be able to use instruments properly and safely while looking at a monitor. Poor technique may result in iatrogenic

damage or extravasation of large amounts of lavage fluid that can severely compromise distention of the joint capsule and thus ability to see adequately within the joint. Experience cannot be gained by simply attending a weekend course, but by working long hours with cadaver specimens and observing experienced surgeons. Initially, arthroscopic surgery can be time consuming until experience is gained, but with experience arthroscopic surgery is faster than arthrotomy.

Equipment Problems Equipment failures can be a disadvantage, and because arthroscopic examination depends on electricity, a generator should always be available. Instruments are not made to withstand forces applied to cut, grasp, and debride equine bone and, along with the arthroscope, may break.

Improper Case Selection Case selection is the most important disadvantage. Poor case selection can make arthroscopic surgery difficult and disappointing. Although much information can be gained by arthroscopic examination of any joint, prognosis should be carefully considered when operating on joints in which extensive osteoarthritis exists. Inadequate communication before surgery may leave owners and trainers with too high expectations of results. Often they have little appreciation for the magnitude of cartilage damage and osteoarthritis, and they only see or hear about the chip fracture(s) visible radiographically. Owners and trainers tend to want to do something, but removal of osteochondral fragments from a carpus or fetlock joint with hopeless osteoarthritis can be time consuming, cause instrument failures, and worse, often results in a poor outcome. The fragment size that can be easily removed during arthroscopic surgery is limited, and the instrument portal must be enlarged to accommodate such fragments unless ostectomy is performed. The fragment must be located in the joint, a decision that must be made before surgery, because arthroscopic surgery is of little benefit if the fragment is not in the joint. In the metatarsophalangeal joint, large plantar process fragments from the proximal phalanx are often located extra-articularly and cannot be removed arthroscopically. In the stifle joint, fragments in the distal aspect of the femoropatellar joint may appear radiographically as if they are in the femorotibial joint. Rare fragments in the caudal pouches of the medial and lateral femorotibial joints can be difficult to retrieve, especially if the precise location cannot be determined radiographically before surgery. Highly mobile fragments in a large joint such as the femoropatellar joint can prove challenging to locate.

PRINCIPLES, INSTRUMENTATION, AND TECHNIQUE

Surgical technique, basic and advanced instrumentation, and approaches are well described elsewhere,[1] and in-depth discussion is beyond the scope of this textbook. The basic principle of arthroscopic surgery is to use a rigid 4-mm endoscope to evaluate and perform intra-articular surgery through small incisions. Limited arthroscopic surgery was previously performed through a single incision, and instruments were passed through a second slot in the arthroscopic cannula. Versatility was severely compromised. Triangulation, a versatile technique in use today, uses two distant portals so the arthroscope and instruments create two sides of a triangle. For example, during routine tarsocrural arthroscopy the arthroscopic portal is dorsomedial and the instrument portal is dorsolateral. This allows the tips of the instruments to be in the surgical field from distant points and obviates the need for the arthroscope to be close to the lesion. The end of the arthroscope is angled 25° to 30°, and a different view is obtained by simply rotating it. The image must be positioned on the screen to optimize special orientation and facilitate stereotaxis.

Arthroscopic surgery can be performed with basic instruments, the most important of which are rongeurs (Ferris-Smith), grasping forceps, bone curettes, lavage cannulae, intra-articular blades, and probes. Arthroscopic surgery depends on the availability of a suitable light source and monitor, digital camera, and recording devices. A fluid delivery system is mandatory, and pumps or other methods to pressurize fluid are preferred to gravity flow systems. Many common lesions removed by arthroscopic surgery are located on the joint margin, and visibility often is compromised when joint capsule and synovium collapse or when bleeding is excessive. Carbon dioxide insufflation is sometimes used. Lasers are used rarely. Motorized equipment is useful but expensive. Abrasion arthroplasty units include various motorized burrs to remove and smooth cartilage and bone surfaces and are used in horses with large lesions such as osteochondritis dissecans lesions of the lateral trochlear ridge of the femur. Synovial resectors are alternating, motorized, rotating blades that are helpful in removing synovium and fibrous tissue and are particularly valuable for removal of osteochondral fragments from the dorsal aspect of the distal interphalangeal joint, apical sesamoid fracture fragments, and fragments from the lateral malleolus of the distal tibia and when synovectomy is indicated.

The division between diagnostic and surgical arthroscopy is now artificial because the same instruments are used for both. Diagnostic arthroscopy, that is, arthroscopic examination, generally refers to evaluating cartilage, bone, and soft tissues to gain information for diagnosis and prognosis. The client must be warned that although valuable information is always learned, arthroscopic examination may provide little therapeutic benefit. However, I believe a tendency exists to under-sell arthroscopic examination, because a positive intervention cannot be made unless the joint is evaluated, and much can be learned about lesions with early arthroscopic examination. This is particularly true in the stifle, carpus, and fetlock joints. Arthroscopic examination is indicated in horses with lameness localized to a joint, but in which radiographic findings are negative or equivocal. Scintigraphic evidence of subchondral bone injury is common in these horses, and defects in overlying articular cartilage can be debrided and attempts at resurfacing performed arthroscopically. Scintigraphy and ultrasonography have low sensitivity in the complex stifle joint, and arthroscopic examination should be encouraged. Arthroscopic examination may uncover proliferative synovitis, intersesamoidean ligament injury, meniscal or cruciate lesions, and intercarpal ligament injury. Intercarpal ligament injury often is associated with osteoarthritis or osteochondral fragments and can cause hemarthrosis and presumably joint instability[2,3] (Fig. 23-1). However, owners and trainers must also be made aware that in some joints, such as the distal interphalangeal and femorotibial joints, anatomical constraints limit inspection of all structures.

Changes in articular cartilage are the most important information gained from arthroscopic examination. Prognosis was found to worsen in proportion to the extent of articular cartilage damage in the carpus,[4] but all joints are similarly affected. Surface area and depth of damage are important. Widespread damage affecting many cartilaginous surfaces is usually worse than well-defined lesions extending into subchondral bone. Cartilage damage is graded as mild (<20% damage to cartilage and subchondral bone at the primary lesion site), moderate (involvement of apposing bone, but <30% total cartilage and subchondral bone damage of primary and apposing sites), severe (≤50% of cartilage and subchondral bone damage of primary and apposing sites), and global (extensive cartilage damage visible on most articular surfaces) (Figs. 23-2 to 23-4). If cartilage lesions do not extend past the zone of calcified cartilage, healing after surgery will be minimal.

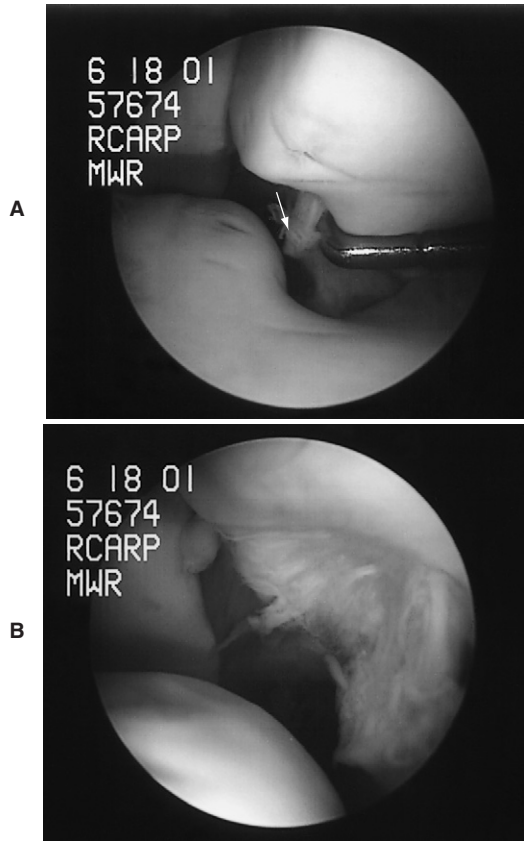

Fig. 23-1 A, Intra-operative arthroscopic image of the left middle carpal joint showing tearing of the medial palmar intercarpal ligament *(arrow)*. Proximal is uppermost, medial is right, and the arthroscopic probe is between the radiocarpal and third carpal bones. The torn medial palmar intercarpal ligament appears as it courses from the third carpal bone to the radial carpal bone. Tearing of the medial palmar intercarpal ligament causes hemarthrosis, a condition previously thought to be idiopathic without arthroscopic examination. **B,** When the medial palmar intercarpal ligament is torn, the palmar aspect of the middle carpal joint can be examined, a finding that is impossible if the ligament is intact.

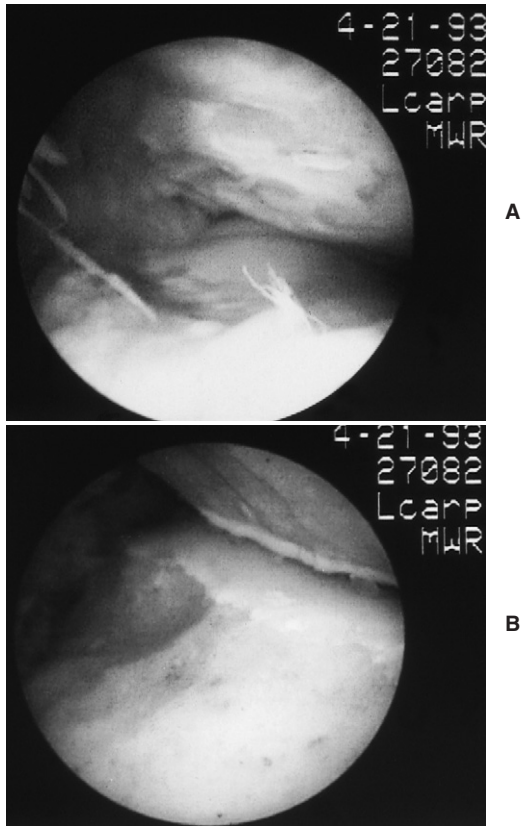

Fig. 23-3 Intra-operative arthroscopic image of the right middle carpal joint showing moderate cartilage damage of the radial carpal (*top*) and third carpal (*bottom*) bones before (**A**) and after (**B**) debridement. The exposed subchondral bone shows considerable damage.

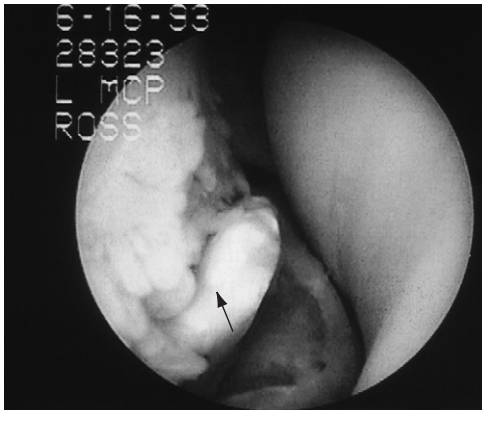

Fig. 23-2 Intra-operative arthroscopic image of the dorsal aspect of the fetlock joint (dorsal is left, proximal is top) showing a large osteochondral fragment *(arrow)* from the dorsomedial aspect of the proximal phalanx. Mild cartilage damage surrounds the base of the fragment with mild scoring of the distal, medial third metacarpal condyle.

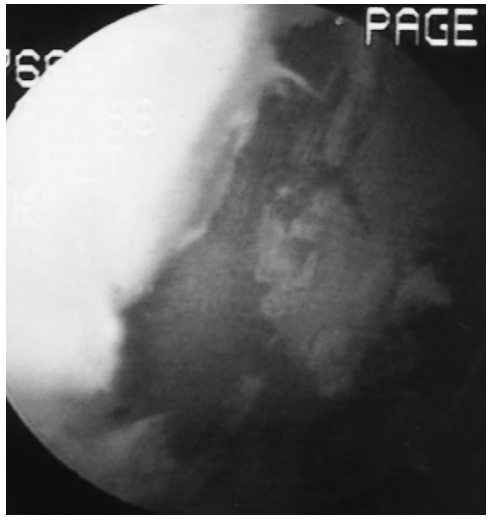

Fig. 23-4 Intra-operative arthroscopic image of the palmar aspect of the right metacarpophalangeal joint showing complete loss of articular cartilage of the lateral proximal sesamoid bone. Not shown are complete full-thickness cartilage and subchondral bone loss on the palmar, medial condyle of the third metacarpal bone indicating severe, near global cartilage loss.

Occult (not radiographically apparent) osteochondral fragments may be diagnosed by arthroscopic examination. Recognition requires inspection and probing, if necessary, after synovial resection. Common sites include the medial aspect of the intermediate carpal and lateral aspect of the radial carpal bones and small fragments involving the proximodorsal aspect of the proximal phalanx. Unexplained tarsocrural effusion (bog spavin) is commonly caused by osteochondritis dissecans fragments of the distal, medial malleolus of the tibia, and although fragments may be suspected radiographically, the presence is confirmed and they are removed arthroscopically. Occult or radiographically suspicious subchondral defects of the third carpal bone, distal medial femoral condyle, and distal MtIII and McIII can be confirmed and debrided.

Most arthroscopic surgery is performed to remove the radiographically apparent osteochondral fragment(s). Grading cartilage damage and subchondral bone damage along with removing one or more osteochondral fragments is important in assessing prognosis. Discovery of severe cartilage damage in horses with few radiographic changes is common, especially in the carpus and fetlock joints. Whenever possible, all aspects of the joint should be evaluated. In racehorses, although cartilage damage and osteochondral fragmentation are often seen dorsally, the palmar/plantar aspect of the metacarpophalangeal joint or metatarsophalangeal joint often has extensive cartilaginous score lines or large, full-thickness areas of cartilage loss on medial McIII and the medial proximal sesamoid bone (PSB) and on the lateral MtIII and lateral PSB. In non-racehorse sport horses, changes are most prominent dorsally. Lesions of the patella, sometimes with fragmentation, found during arthroscopic examination explain why some horses with large osteochondritis dissecans lesions of the lateral trochlear ridge of the femur perform poorly. Arthroscopic examination is useful in horses with infectious arthritis to evaluate articular surfaces and synovium, identify and remove foreign material, facilitate removal of fibrin, and perform much more effective and complete lavage than is achieved by through-and-through lavage. Arthroscopic examination usually is reserved for horses with long-standing infections and those with extensive fibrin accumulation, but the Editors strongly promote its use for acute infection as well. I have not found a good correlation between arthroscopic and ultrasonographic identification of fibrin early in the disease process (amount of fibrin over-estimated by ultrasonography). Arthroscopic examination can facilitate drain insertion, synovectomy, and debridement or removal of osteochondral defects. Arthroscopic portals can be left open to facilitate drainage after surgery, but this procedure should be used only in horses with refractory infections.

SURGICAL PROCEDURES

The specific indications and surgical procedures are discussed in other chapters. Dorsal and palmar/plantar, medial and lateral, and cranial and caudal approaches have been described; the approach, choice of portals, fluid delivery, and instrumentation vary among joints and surgeons. Positioning the horse on the operating table is important to allow access to the involved joint(s) and repositioning may be necessary. Lateral and dorsal recumbency are used successfully, and choices are based on the surgeon's experience and the number of joints affected. Although standing arthroscopy of the fetlock and other joints has been described,[5] I prefer to use general anesthesia.

Minimally invasive surgery such as arthroscopic surgery continues to be developed in horses. Standard procedures include fragment removal, debridement and curettage, partial synovectomy, and incision of adhesions. Reduction, fragment removal, and screw placement in horses with condylar fractures of McIII or MtIII can be done using arthroscopic surgery, obviating the need for arthrotomy. A technique to insert one or more cortical bone screws to repair frontal slab fractures of the third carpal bone was an early advance that has led to use of a similar technique to repair radial carpal and ulnar carpal bone slab fractures, PSB fractures, and other unusually located articular fractures.[6]

Cartilage Resurfacing

Recently cartilage resurfacing techniques in experimental and in a limited number of clinically affected horses have been used. Repair strategy involves creating access to stem cells, growth factors, and blood in subchondral bone to assist in cartilage repair or transplantation (grafting) of tissues, osteochondral grafts, or cells (chondrocytes). Partial- or full-thickness cartilage defects that do not penetrate the subchondral plate heal incompletely or not at all. An important principle in cartilage repair is to curette through the layer of calcified cartilage and perforate the subchondral plate to allow influx of healing elements. Extensive curettage seems to be self-defeating, because exposure of large areas of denuded subchondral bone is associated with a poor prognosis and often is found in horses with naturally occurring osteoarthritis. Unfortunately, many defects already involve complete erosion through subchondral bone, at least around and apposing the primary lesion site. A technique known as *microfracture* of the subchondral plate has been introduced and involves using a 45° or 90° orthopedic awl (micropick) to create small perforation sites into subchondral bone in the area of cartilage damage.[7,8] Studies have shown that removal of the calcified cartilage layer alone, or perforation of the layer with the microfracture technique, improves the amount and quality of repair tissue, but this tissue lacks the biomechanical and histological character of hyaline cartilage.[9] The procedure is currently being used, but objective results compared with routine procedures have not been published.

Nixon[10] has considerable experience in transplantation resurfacing techniques and is focusing current efforts on using autogenous fibrin laden with insulin-like growth factor-1 and chondrocytes harvested from neonatal foals. An arthroscopic technique using a two-component system was used to implant the graft in experimental and clinically affected horses with defects in the carpus and subchondral cystic lesions of the distal medial femoral condyle and in the fetlock joint. Early results appear promising.

A technique called *mosaic arthroplasty* involves using arthroscopic procedures to harvest osteochondral pegs (grafts) from the trochlear groove of the distal femur and implant them in distant recipient sites.[11] Grafts harvested and implanted using special instruments have better congruence than previously reported osteochondral grafting techniques, most using donor sites in the sternum. Mosaic arthroplasty has been used to manage osteochondral defects in the fetlock joint and to attach large osteochondral flaps (osteochondritis dissecans) on the lateral trochlear ridge of the femur using pegs as biological pins.[12] Results appear promising, but the technique is difficult.

Post-Operative Care

Aftercare instructions differ depending on the joint(s) involved, severity of cartilage damage, surgeon's experience, economic factors, and competition schedules. Bandage and wound care, administration of anti-inflammatory analgesic and anti-microbial therapy, and suture removal are routine. Decisions about rehabilitation time should be based on extent of the damage and location but often are made based on the perceived need for a quick return to training and performance. Intra-articular therapy with hyaluronan and polysulfated glycosaminoglycans (PSGAG) is often recommended, and

although such therapy may be beneficial to reduce inflammatory changes in joints early after surgery, little evidence exists that repair is augmented. I generally recommend intra-articular hyaluronan 14 and 28 days after surgery in horses with mild or moderate cartilage damage and PSGAG, administered intra-articularly 3, 5, and 7 weeks after surgery, for horses with severe or global cartilage damage. Treatment with PSGAG given intramuscularly beginning 2 weeks after surgery (once weekly for 8 weeks) is recommended.

COMPLICATIONS

The infection rate after arthroscopic surgery is low, but not zero, and is generally less than other soft tissue and orthopedic procedures performed in the same hospital. Poor case selection and failure to remove the intended fragments are the most common important complications. Intra-operative radiographs should be obtained if any doubt exits about fragments (number and size) removed. Extravasation of fluid, most commonly at the instrument portal but occasionally at the arthroscopic portal, occurs frequently and is of little concern. Fibrous tissue formation at the instrument portal is common if a large amount of cartilage and bony debris is flushed from the joint or removal of several osteochondral fragments required extensive manipulation. Prolonged drainage from any portal should be managed as a potential infectious arthritis. Synovial fistulae occur rarely, but they may require repair. Damage to overlying tendons may cause esthetically unpleasing tenosynovitis that usually responds to injections, but occasionally repair of synoviosynovial fistulae is necessary.

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CHAPTER • 24

Tenoscopy and Bursoscopy

Eddy Cauvin

The advantages of arthroscopy as a diagnostic and therapeutic approach to joints[1] have prompted the development of applications for other synovial cavities. *Tenoscopy* is the term used to describe endoscopy of synovial tendon sheaths, usually using a rigid arthroscope.[2] *Bursoscopy* is used for endoscopy of bursae.

Tenoscopy has many advantages. Traditional approaches to tendon sheaths, requiring long incisions over highly mobile areas, are associated with substantial post-operative risks including wound dehiscence and ascending sheath infection.[3] These techniques are invasive, time consuming, and offer limited visibility of tendovaginal structures.[2,3] Endoscopic approaches to the carpal tunnel in people have been described[4,5] and have provided substantial improvements in terms of decreased morbidity, scarring, and loss of function compared with open techniques.[4,6] Tenoscopy in the horse was first described for the examination of the digital flexor tendon sheath (DFTS).[1,2] Since the early 1990s, other applications have been described.[7-11]

GENERAL PRINCIPLES OF TENOSCOPY AND BURSOSCOPY

Equipment

Standard athroscopic equipment is used, including arthroscope, sleeve, and obturators; arthroscopic cannulae; probes; grasping forceps; and Ferris-Smith rongeurs. Sharp tenotomes should be available. A standard 4.0-mm, 25° to 35° forward angle arthroscopic telescope is adequate for most sheaths and bursae, although thinner endoscopes may be useful for extensor sheaths. Light source and video camera apparatuses are as for arthroscopy. Motorized synovial resectors are particularly useful, because debridement or synovec-

although such therapy may be beneficial to reduce inflammatory changes in joints early after surgery, little evidence exists that repair is augmented. I generally recommend intra-articular hyaluronan 14 and 28 days after surgery in horses with mild or moderate cartilage damage and PSGAG, administered intra-articularly 3, 5, and 7 weeks after surgery, for horses with severe or global cartilage damage. Treatment with PSGAG given intramuscularly beginning 2 weeks after surgery (once weekly for 8 weeks) is recommended.

COMPLICATIONS

The infection rate after arthroscopic surgery is low, but not zero, and is generally less than other soft tissue and orthopedic procedures performed in the same hospital. Poor case selection and failure to remove the intended fragments are the most common important complications. Intra-operative radiographs should be obtained if any doubt exits about fragments (number and size) removed. Extravasation of fluid, most commonly at the instrument portal but occasionally at the arthroscopic portal, occurs frequently and is of little concern. Fibrous tissue formation at the instrument portal is common if a large amount of cartilage and bony debris is flushed from the joint or removal of several osteochondral fragments required extensive manipulation. Prolonged drainage from any portal should be managed as a potential infectious arthritis. Synovial fistulae occur rarely, but they may require repair. Damage to overlying tendons may cause esthetically unpleasing tenosynovitis that usually responds to injections, but occasionally repair of synoviosynovial fistulae is necessary.

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CHAPTER • 24

Tenoscopy and Bursoscopy

Eddy Cauvin

The advantages of arthroscopy as a diagnostic and therapeutic approach to joints[1] have prompted the development of applications for other synovial cavities. *Tenoscopy* is the term used to describe endoscopy of synovial tendon sheaths, usually using a rigid arthroscope.[2] *Bursoscopy* is used for endoscopy of bursae.

Tenoscopy has many advantages. Traditional approaches to tendon sheaths, requiring long incisions over highly mobile areas, are associated with substantial post-operative risks including wound dehiscence and ascending sheath infection.[3] These techniques are invasive, time consuming, and offer limited visibility of tendovaginal structures.[2,3] Endoscopic approaches to the carpal tunnel in people have been described[4,5] and have provided substantial improvements in terms of decreased morbidity, scarring, and loss of function compared with open techniques.[4,6] Tenoscopy in the horse was first described for the examination of the digital flexor tendon sheath (DFTS).[1,2] Since the early 1990s, other applications have been described.[7-11]

GENERAL PRINCIPLES OF TENOSCOPY AND BURSOSCOPY

Equipment

Standard athroscopic equipment is used, including arthroscope, sleeve, and obturators; arthroscopic cannulae; probes; grasping forceps; and Ferris-Smith rongeurs. Sharp tenotomes should be available. A standard 4.0-mm, 25° to 35° forward angle arthroscopic telescope is adequate for most sheaths and bursae, although thinner endoscopes may be useful for extensor sheaths. Light source and video camera apparatuses are as for arthroscopy. Motorized synovial resectors are particularly useful, because debridement or synovec-

tomy using hand-operated instruments can be tedious in large tendon sheaths.

Surgical Principles and Techniques

The basic principles of arthroscopy are also valid in tendon sheaths and bursae. The sheath usually is distended with fluids to facilitate insertion of the cannula. However, this is not necessary for the carpal and tarsal sheaths, which are not approached through distended pouches.[9,10] It is generally recommended that the portal be created with a scalpel and the cannula inserted using a blunt, conical obturator to avoid damaging the tendons. A thorough knowledge of the normal endoscopic anatomy of the sheath is paramount for several reasons. First, all the surfaces are covered by synovium and look alike, making identifying the structures seen difficult. Second, normal anatomical structures such as vinculae (adhesion-like formations carrying blood vessels to the tendon from the parietal sheath), endotenon (reflection of the synovial membrane, which forms a continuous band attaching the tendon to the sheath along its length), and synovial folds are apparent. These should not be damaged, because they participate in the blood supply of tendons within the sheathed portion.[12-14]

Triangulation techniques are applied for instruments using separate portals. These should be created as close as possible to the lesion, although the shape of the sheath often dictates the position of the portals.

TENOSCOPY

Tenoscopy of the Digital Flexor Tendon Sheath

The DFTS is the most common site of tenosynovitis. Endoscopy is indicated as a diagnostic procedure to examine lesions of the surfaces of the deep digital flexor tendon (DDFT), superficial digital flexor tendon (SDFT), and parietal surface of the sheath. High-definition ultrasonography allows non-invasive examination of the sheath, its contents and peripheral tissues, and provides more accurate information about the internal architecture of tendons.[15] However, differentiating some adhesions, tears, and superficial fraying of the tendons may be difficult ultrasonographically.[16] Tenoscopy is useful for debridement of masses, such as villonodular synovitis-like lesions and other lesions within the sheath, adhesiolysis, removal of debris, and synovectomy in infectious tenosynovitis.[2,16,17] Recently a technique for desmotomy of the palmar annular ligament under endoscopic control has been described to avoid inadvertent damage to the tendons, manica flexoria, and other peritenovaginal structures.[18]

The advantages of tenoscopy over traditional open surgery are similar to those recognized for arthroscopy over arthrotomy. They include decreased morbidity and more rapid return to normal function of the sheath and reduced risks of complications, such as wound breakdown, infection, fibrosis, and ankylosis.

Anatomy

The DFTS is organized primarily around the DDFT, which it completely surrounds from the junction between the third and distal quarters of the metacarpal or metatarsal region to the level of the proximal interphalangeal joint, before tapering dorsally to the DDFT to the proximal border of the distal sesamoid bone.[12,13,15,19] At the level of the metacarpophalangeal joint the DFTS also surrounds the SDFT, except for a wide mesotenon over the palmar/plantar aspect of the tendon. At this level the DFTS is bound dorsally by the proximal scutum, a fibrocartilage covering the palmar (plantar) surfaces of the proximal sesamoid bones (PSBs) and intersesamoidean ligament, and palmarly by a tough transverse ligament, the palmar (plantar) annular ligament (PAL), thus forming a non-elastic canal through which both flexor tendons run independently. In the pastern region the SDFT separates into two branches and is no longer within the sheath. The sheath is bound dorsally by the distal sesamoidean ligaments and palmarly (plantarly) by two broad digital annular ligaments.

The proximal pouch bulges when distended proximal to the PAL and PSBs and contains a number of long villi in normal horses. Several small, subcutaneous pouches appear between the insertions of the PAL and digital annular ligaments abaxially and over the palmar (plantar) aspect of the DDFT in the mid-pastern region. A number of vascular structures are found, including two large vinculae forming a V-shaped adhesion-like structure between the dorsal surface of the DDFT and the dorsal sheath wall in the proximal pastern. Densely packed villi often are found in that area. The wide palmar mesotenon prevents examination of the palmar aspect of the SDFT in the fetlock region. Immediately proximal to this level, the manica flexoria forms a smooth, thin membrane originating from the axial surfaces of the SDFT and surrounding the DDFT dorsally.

Surgical Technique

The technique for tenoscopic exploration of the DFTS has been described in detail.[2] The term *palmar* is used in the following description for either palmar or plantar. The horse is placed in lateral recumbency with the affected limb uppermost for a lateral approach or lowermost for a medial approach. The DFTS is distended with 10 to 20 ml of physiological solution through a needle inserted in the palmar aspect of the mid-pastern region. Overdistention of the sheath is avoided because it causes flexion of the digit. A 5-mm longitudinal incision is made through the skin, immediately distal to the PAL, lateral or medial to the DDFT, and 1 cm palmar to the neurovascular bundle, which must be carefully avoided. A stab incision is made into the sheath, taking care to avoid damaging the DDFT, and the cannula, with a conical obturator inserted in a proximal direction, between the DDFT and dorsal sheath wall (Fig. 24-1). The sheath is lavaged through an 18-gauge needle inserted in the proximal pouch. The obturator is

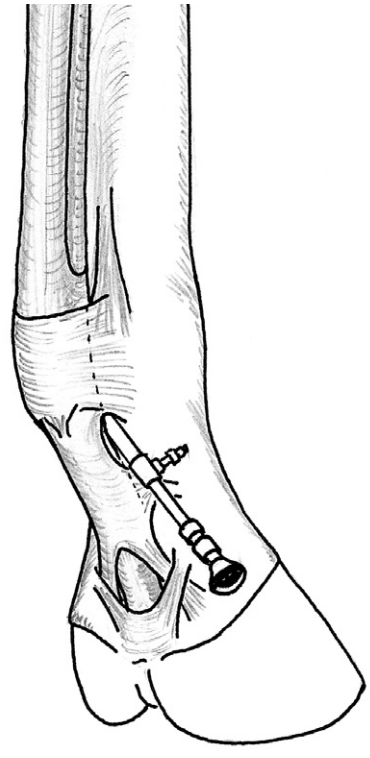

Fig. 24-1 Digital sheath tenoscopy. The arthroscope is inserted lateral to the deep digital flexor tendon, immediately distal to the annular ligament.

replaced with the endoscope, and examination is carried out from proximal to the level of the portal by rotation and gradual withdrawal. The proximal pouch is examined, followed by the abaxial aspects of the two tendons. Flexion of the fetlock joint allows insertion of the endoscope between the SDFT and DDFT without damage to the manica flexoria. The endoscope is finally rotated around the SDFT on the side of the portal to inspect the mesotenon. Examination of the opposite side is possible by flexion of the fetlock and rotation of the arthroscope window palmarly.

The endoscope is then pushed across the sheath, between the DDFT and dorsal sheath wall, to avoid exiting the sheath. The endoscope is then re-directed distally. The distal part of the sheath is examined by gradual withdrawal of the endoscope. The vinculae are visible in the proximal pastern region, between the dorsal surface of the DDFT and dorsal wall (Fig. 24-2). Palmarly and farther distally, bifurcation of the SDFT branches forms a manica-like ring around the DDFT (Fig. 24-3).

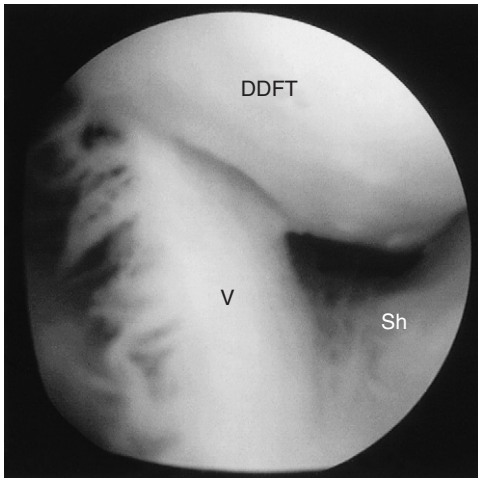

Fig. 24-2 Tenoscopic view of the dorsal aspect of the deep digital flexor tendon (*DDFT*) in the proximal pastern region, showing one of the vinculae (*V*) between the deep digital flexor tendon and dorsal sheath wall (*Sh*).

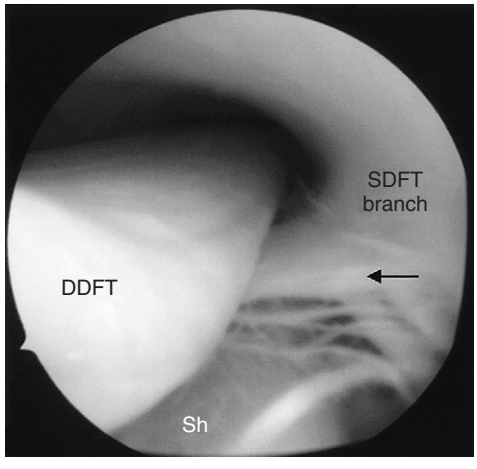

Fig. 24-3 Tenoscopic view of the palmar aspect of the deep digital flexor tendon in the proximal pastern region, showing the manica-like ring (*arrow*) surrounding the deep digital flexor tendon, between the two superficial digital flexor tendon (*SDFT*) branches (within the abaxial sheath walls). The dorsal sheath wall (*Sh*) is rich in long villi.

Debridement of potential lesions is carried out through separate instrument portals made as close as possible to the lesion to allow for adequate triangulation. At the end of surgery the sheath is lavaged through a large-bore cannula and the skin incisions are closed. It is recommended that a pressure bandage be applied for 5 to 7 days postoperatively. In horses with infection with substantial debris accumulation, it is possible to place a drain, exiting in the distal palmar recess through a separate incision.

A technique for transection (desmotomy) of the PAL by endoscopic control has been described,[18,20] using a desmotomy kit designed for palmar carpal ligament transection in people.[4,5] This permits adequate examination of the sheath before PAL transection and prevents inadvertent damage to the tendons and manica flexoria. This is not described here because standard percutaneous techniques are generally adequate.

I have used an alternative technique for digital tenoscopy, using an endoscopic portal made proximal to the PAL, dorsolateral to the DDFT. This technique allows adequate examination of the whole sheath, is useful to examine lesions in the proximal phalangeal region, and provides improved triangulation when surgically treating such lesions. Using a wound as an initial endoscopic portal is often possible, although creating a new endoscope portal and using the wound for instruments is usually preferable after examination is completed.

Post-Operative Care

At the end of surgery the sheath is emptied of fluids and the skin portals are closed with staples or sutures of the surgeon's choice. Damage to the neurovascular bundle by needles and sutures is carefully avoided. The horse recovers with the limb in a pressure bandage from foot to distal carpus (or tarsus) to prevent sheath swelling and to reduce motion. Post-operative antibiotics are only necessary in horses with infectious tenosynovitis, but systemic non-steroidal anti-inflammatory drugs (NSAIDs) should be given for several days after surgery. The horse should be restricted to box rest for 3 days, and then it may be walked out in hand to decrease restrictive adhesion formation. Box stall rest with controlled exercise should be continued for 2 weeks until the sutures are removed, and then at the surgeon's discretion exercise is increased progressively. Hyaluronan has been advocated as an adjunctive therapy[2,17] but should be avoided if infection is present.

TARSAL SHEATH

Injuries to the tarsal sheath are relatively common (see Chapter 77). Open surgery has been associated with a high rate of post-operative complications,[3,10,21] but early results indicate that the morbidity may be greatly reduced with tenoscopy.[10]

Anatomy

The tarsal sheath is the sheath of the lateral digital flexor tendon, the largest of the two tendons (lateral and medial digital flexor tendons), the fusion of which in the proximal metatarsal region forms the deep digital flexor tendon (see Chapter 77).[10,19,22] It is paramount to recognize the anatomical location of the plantar nerves and vessels that pass within the plantar flexor retinaculum and the presence of a continuous mesotenon, passing longitudinally along the plantaromedial aspect of the lateral digital flexor tendon. However, accidental damage to these structures does not appear to have major consequences.

Surgical Technique

A technique allowing examination of the whole sheath has been described.[10] Prior distention of the sheath is not necessary. The medial edge of the sustentaculum tali and medial

insertion of the retinaculum are located, and an 8-mm vertical incision is made in the skin and underlying fascia, 8 mm plantar to the edge of the bone at the point where the tendon changes direction over the sustentaculum tali. The absence of blood vessels in the retinaculum deep to the incision is ascertained using hypodermic needles before making a 5-mm stab incision through the retinaculum. Care is taken to avoid damage to the underlying lateral digital flexor tendon. The endoscope sleeve and conical, blunt obturator are inserted dorsal to the lateral digital flexor tendon in a proximal direction. The obturator is replaced with the endoscope, the sheath is distended moderately with fluids, and examination is carried out as described for the DFTS by rotation of the window of the arthroscope and gradual withdrawal. After examination of the proximal half of the sheath the endoscope is re-directed into the distal pouch. The proximal pouch is large and lined by a thin wall (Fig. 24-4, *A*). Muscle fibers from the lateral digital flexor muscle and separate head of the tibialis caudalis muscle may be seen by transillumination through the synovial membrane. Within the tight, rigid canal formed by the sustentaculum tali and retinaculum, the fibrocartilage appears glistening white, and small vessels are seen within the parietal membrane plantarly and in the mesotenon. In the distal half of the sheath a fold of synovial membrane forming a small, blind pouch medially is seen dorsomedial to the lateral digital flexor tendon at the level of the tarsometatarsal joint (Fig. 24-4, *B*). Distal to this fold, the sheath continues as a cylindrical, blind recess dorsal to the tendon and contains small villi. The separate sheath of the medial digital flexor tendon is not visible from the tarsal sheath.

If a wound exists, most commonly at the plantaromedial edge of the sustentaculum tali, it may be used as an endoscope portal. Instruments may be inserted as required close to lesions to allow for triangulation. Needles are used to determine the optimal location for the instrument portal.

Carpal Sheath

Tenosynovitis of the carpal flexor tendon sheath (carpal sheath) is relatively rare in horses but can cause severe lameness associated with carpal canal syndrome (see Chapter 76). Wounds leading to contamination of the sheath are rare but possible. Common causes of non-infectious tenosynovitis include lesions of the SDFT in the carpal region, fractured accessory carpal bone, osteochondromata of the caudal distal radius, and sprain injuries to the sheath,[23,24] although other causes may exist.[25] Effusion may be secondary to lesions outside the sheath, such as desmitis of the accessory ligament of the DDFT (ALDDFT), accessory ligament of the SDFT, limb edema, and unassociated wounds. Ultrasonography is the technique of choice for investigating carpal sheath distention, but adhesions and subtle fraying of the tendons may not be visible.[24,26] Radiography is also mandatory to rule out fractures and osteochondromata.[23-25,27]

Tenoscopy may be useful as a diagnostic tool if lameness is associated with pain localized to the carpal sheath by intrathecal analgesia but where no lesions are visible with other imaging methods. The main indication for tenoscopy of the carpal sheath is debridement and repair of structures within the sheath.

Anatomy

The anatomy of the sheath has been described elsewhere in detail.[14,18,26] Briefly, the sheath is organized around the DDFT from the distal caudal antebrachium, 4 to 7 cm proximal to the accessory carpal bone, to the metacarpal region. The sheath extends around the DDFT and SDFT in the carpal region, where it is enclosed within a tight canal (the carpal canal) formed by a thick palmar carpal flexor retinaculum palmarly and medially, the accessory carpal bone laterally, and the palmar carpal ligament dorsally. At this level the SDFT is attached palmarly to the sheath wall by a thick mesotenon that contains the median artery and medial palmar nerve. A thinner mesotenon links the DDFT to the SDFT medially. Proximal to the canal a large pouch occurs dorsal to the flexor muscles, bulging laterally and medially caudal to the radius if distended. Distal to the carpometacarpal joint the sheath continues as a blind sack between the DDFT and ALDDFT and bulges dorsolaterally to the DDFT in the proximal third of the metacarpal area. The lateral palmar nerve and palmar veins pass within the retinaculum.

Surgical Technique

Several techniques have been described depending on the lesions identified before surgery. Removal of osteochondromata of the caudal distal radius has been described using an approach through the distended proximal pouch.[8] With the horse in lateral recumbency with the affected limb uppermost the arthroscopic portal is made between the lateral digital extensor and ulnaris lateralis tendons, 3.5 cm proximal to the

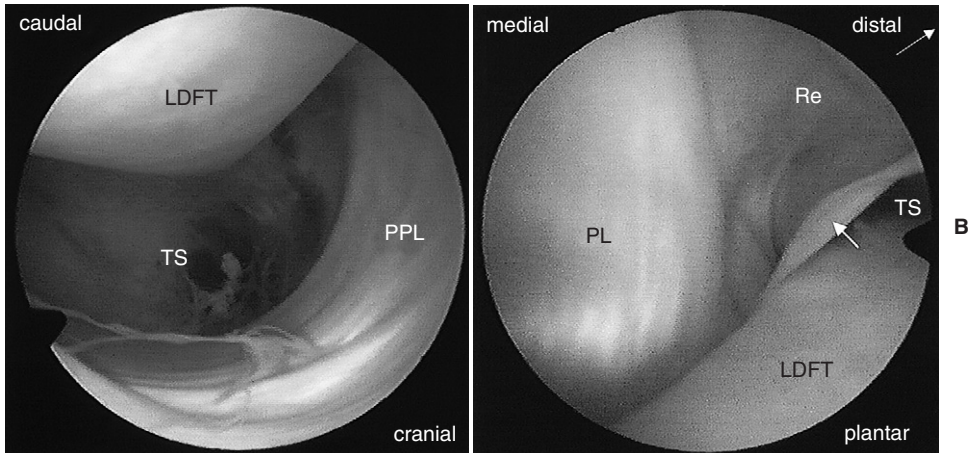

Fig. 24-4 Tenoscopic views of the tarsal sheath. **A,** Proximal pouch of the tarsal sheath (*TS*) showing the cranial aspect of the lateral digital flexor tendon (*LDFT*) and the proximal plantar ligament (*PPL*) thickening of the plantar capsule of the tarsocrural joint. **B,** Distal recess of the tarsal sheath (*TS*). At the tarsometatarsal joint the end of the plantar ligament (*PL*) is visible. A synovial fold (*white arrow*) forms a small recess dorsomedially (*Re*).

distal radial physeal scar, after distention of the sheath. The instrument portal is made on the same side as the arthroscope portal, 2 cm distal to it. A medial approach has also been described.[7]

These techniques do not permit examination of the distal two thirds of the sheath. Therefore another technique has been developed to facilitate endoscopic examination of most of the sheath.[9] The horse may be placed in lateral recumbency with the affected limb uppermost, or in dorsal recumbency with the affected limb suspended loosely, so that the carpus is slightly flexed. An 8-mm longitudinal skin incision is made without prior distention of the sheath 1.5 to 2 cm distal to the distal border of the accessory carpal bone along the lateral aspect of the DDFT. A medial approach is not recommended because examination is restricted by the mesotenons and the risk of injury to the median artery and medial palmar nerve. A needle is used to ensure that no vessels are present in the fascia deep to the incision. The incision is extended with a scalpel through the fascia into the sheath. The arthroscope is inserted proximally, between the DDFT and dorsal wall of the sheath. The proximal pouch is large and contains small villi. The radial head of the DDFT forms a conical prominence in the medial aspect of this pouch. Rotation of the endoscope window and manipulation around the tendons allows examination of the DDFT and SDFT surfaces, caudal surface of the distal radius, and fibrocartilaginous sheath wall in the carpal canal. Minimal fluid pressure should be used to avoid flexion of the carpus. The endoscope may be inserted between the tendons to view the surfaces and the lateral aspect of the mesotenon, covered by short, thin villi. Redirecting the endoscope distally permits examination of the distal recess. A normal, longitudinal synovial fold appears dorsomedially in the sheath in the distal carpal region.

Instrument portals are made where necessary to allow for adequate triangulation. In particular, improved triangulation is obtained for removal of osteochondromata of the distal caudal radius with this method. Major anatomical structures should be taken into account. Post-operative care is as described for other sheaths.

Other Tendon Sheaths

Most tendon sheaths may be examined by tenoscopy, including extensor tendon sheaths in the dorsal carpal and tarsal regions. However, no published reports indicate the use of tenoscopy for these smaller synovial cavities, possibly because acceptable results have been obtained with traditional surgical exposure.[28] I have attempted tenoscopy of the carpal extensor sheaths experimentally in isolated limbs, using a 4-mm, 30° forward endoscope inserted medially or laterally to the tendons, proximal to the carpus. This technique provides acceptable examination of the sheath and tendon surfaces, but little movement is possible because of the tight extensor retinacula.

BURSOSCOPY

Reports of bursoscopy in horses are limited,[11,29] but it is probable that the technique would yield similar advantages to tenoscopy in horses with masses or infection in normal or acquired bursae. Surgical exposure and debridement of hygromas, capped hocks, and metacarpophalangeal subtendonous bursae are often associated with wound dehiscence and chronic infection.[30] However, I am unaware of published data describing bursoscopic examination in these locations. A technique for bursoscopy of the calcaneal bursa of the SDFT has been described and advocated for diagnosis and treatment of injuries to this structure and is probably most useful in horses with infectious bursitis.[29]

Bursoscopy of the Intertubercular Bursa

Bursoscopic surgery has also been used for the management of infectious intertubercular (bicipital) bursitis,[31] and a report describes its use as a diagnostic method in a horse with traumatic bicipital bursitis.[11] The horse is placed in lateral recumbency with the affected limb uppermost. The endoscope is introduced through a craniolateral skin portal, made over the point of the shoulder, immediately cranial to the lateral tubercle of the humerus. The bursa is distended with 40 ml of fluid. The skin incision is continued through the brachiocephalicus muscle and into the bursa, and the endoscope sleeve and conical obturator are inserted in a caudoproximal direction. A technique using two separate portals has been described to improve visibility.[32] The first portal is made into the distal recess through an incision immediately proximal to the deltoid tuberosity. For the second portal, an incision is made cranioproximal to the lateral humeral tubercle to view the proximal half of the bursa. Experience with these techniques is currently limited, but they should provide similar advantages to tenoscopy, including easier access to the sheath, improved visibility, and decreased post-operative morbidity. This needs to be confirmed by more extensive reviews.

Bursoscopy of the Navicular Bursa

Endoscopic examination of the navicular bursa (bursa podotrochlearis)[33] recently has been described as an alternative to the traditional streetnail procedure.[34,35] It is the technique of choice for treating infectious navicular bursitis.

The main indication for bursoscopy of the navicular bursa is as a surgical approach for treating a contaminated or infected bursa.[33] Other indications include diagnostic examination in horses with pain localized to the palmar aspect of the foot but with no lesions identifiable using other imaging modalities and the debridement of adhesions between the navicular bone and the dorsal surface of the DDFT.

Surgical Technique

Standard arthroscopic equipment is used, although a thinner, 4-mm diameter or less, endoscope is more practical. The horse is placed in lateral recumbency with the metacarpal or metatarsal region supported and the digit moving loosely. A medial approach with the affected limb lowermost or a lateral approach with the limb uppermost may be used. A vertical, 5-mm skin incision is made along the abaxial border of the DDFT, 1 cm proximal to the cartilage of the foot and palmar/plantar to the neurovascular bundle. The cannula, with a conical obturator, is inserted distally and slightly axially, dorsal to the DDFT. The obturator is replaced with the endoscope. Rotation of the endoscope allows examination of the whole bursa, including the smooth dorsal aspect of the DDFT and palmar fibrocartilage of the navicular bone. The collateral (suspensory) ligaments of the navicular bone, blending over the proximal surface of the navicular bone into the fibrocartilage and the impar ligament, in the distal recess of the bursa, are covered by synovium with thin villi. The larger proximal pouch and the T-ligament are also covered by synovium.

A wound in the sole, sulci, or frog can be used for insertion of cannulae or instruments into the bursa. Some enlargement of the tract using curettes or a motorized synovial resector may be required but should be minimal to limit damage to the DDFT. If access through the wound is difficult, or to reach a lesion that cannot be accessed this way, a similar approach to that used for the endoscope may be made on the opposite side of the limb.

The bursa is lavaged and any lesions are debrided sharply. Partial synovectomy may be carried out if necessary. The use of motorized synovial resectors is recommended. At the end of the procedure the opening of a sinus tract on the solar surface of the foot may be enlarged and debrided to avoid abscess formation. Debridement of the defect through the

DDFT should be minimal. An aminoglycoside antibiotic may be placed in the bursa, and the skin incisions are closed routinely. A bandage including the whole hoof and extending to the proximal metacarpal or metatarsal region is applied.

Post-Operative Management and Results

Post-operative use of antibiotics and NSAIDs is at the surgeon's discretion. The horse can be walked out in hand after 2 or 3 days, and exercise can be gradually resumed after healing of the wound is complete, in the absence of complications.

Complications of infection include recurrence, extension of the infection into adjacent synovial structures, and osteitis or fracture of the navicular bone.[33,35] Contamination of the distal interphalangeal joint or DFTS may be treated during the initial surgery by an endoscopic approach using the same skin portal.[33] If infection recurs, lavage through needles or bursoscopy may be performed, and an open approach through the solar surface may be used, but the prognosis should then be considered graver.

The prognosis for horses with infectious navicular bursitis using this approach[33] appears to be greatly improved compared with the more invasive streetnail procedure.[35] Seventy-five percent of horses returned to the initial use after bursoscopy versus 31.5% using the open technique, although the criteria used to judge success may be slightly different.[33] Bursoscopy is considered to be the technique of choice for infectious navicular bursitis, although I recommend using a streetnail approach for the salvage of horses with chronic bursitis with extensive damage to the podotrochlear structures.

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CHAPTER • 25

Thermography: Use in Equine Lameness

Tracy A. Turner

DEFINITION

Thermography is the pictorial representation of the surface temperature of an object.[1,2] This non-invasive technique is used to detect superficial inflammation and therefore may be helpful in the early diagnosis of some causes of lameness. The examination must be performed in a draft-free room protected from sunlight. The hair coat must be of uniform length. Clipped areas or regions that have been bandaged or had topical applications produce erroneous results.

Heat dissipates through the skin by radiation, convection, conduction, or evaporation.[2] Skin temperature is generally 5° C cooler than body core temperature (37° C). Skin derives its heat from the local circulation and tissue metabolism.[3] Tissue metabolism is generally constant; therefore variation in skin temperature is usually caused by changes in local tissue perfusion. Normally, veins are warmer than arteries because they are draining metabolically active areas. Venous drainage from tissues with a high metabolic rate is warmer than venous drainage from normal tissues.

The circulatory pattern and the relative blood flow dictate the thermal pattern that is the basis for thermographic interpretation.[2] The normal thermal pattern of any area can be predicted based on vascularity and surface contour. Skin overlying muscle is subject to temperature increase during muscle activity. The midline of the back, the chest, and between the rear legs and the ventral midline are generally warmer than the rest of the trunk. Heat over the limbs follows the cephalic and saphenous veins.[1,2]

SPECIFIC USES TO DETERMINE INJURY

Foot

Thermography may provide information concerning laminitis, palmar foot pain, sub-solar, or sub-mural abscesses, corns, and other inflammatory conditions of the hoof, especially if the results of clinical and radiographic examinations are inconclusive.[1,2,4]

Normally the coronary band is the warmest area of the limb, and diagnosis of inflammation can be difficult in this location.[1] All hooves should be compared thermographically. A difference of more than 1° C between hooves may be significant. However, in the Editors' experience, there may be quite marked differences in foot surface temperatures in normal horses, especially in cold ambient temperatures. If all feet are affected, the hoof temperature should be compared with that of the area between the bulbs of the heel.

A change in thermal pattern of the hoof wall is useful in recognizing laminitis (see Fig. 118-3, *B*). Generally the coronary band is 1° to 2° C warmer than the remainder of the hoof. As the hoof begins to approach the temperature of the coronary band, this indicates an inflammatory problem. Thermography can be helpful for monitoring a limb at risk to develop laminitis from excessive load bearing because of severe lameness in the contralateral limb. It may be possible to detect inflammation in the contralateral foot before lameness is evident. Preventive therapy can then be instituted sooner, and hopefully before the laminitis is irreversible. Results of thermography may be affected by existing preventative measures used in high-risk patients such as ice baths, and if laminitis is bilateral, a difference will not be detected.

I have never identified a consistent thermal pattern associated with palmar hoof pain, but I have recognized reduced blood flow in some horses.[5] The foot is thermographically evaluated before and after exercise. A normal horse has a 0.5° C increase in foot temperature after exercise, but about 50% of horses with palmar foot pain syndrome have no increase temperature in the palmar aspect of the foot, possibly from low blood flow or other factors. Focal inflammatory conditions of the hoof such as abscesses, bruises, and fractures are characterized by focal areas of increased temperature that correspond to the site of injury (Fig. 25-1; Color Plate 1). Exercise intensifies the area of increased temperature.

Joints

Most joints are best evaluated from the dorsal aspect.[4,6] A normal joint is cool compared with the surrounding structures, but the hock joint has a vertical area of increased temperature medially corresponding to the saphenous vein. With inflammation, an oval (or circular in distal joints) area of increased temperature is centered over the joint and is widest

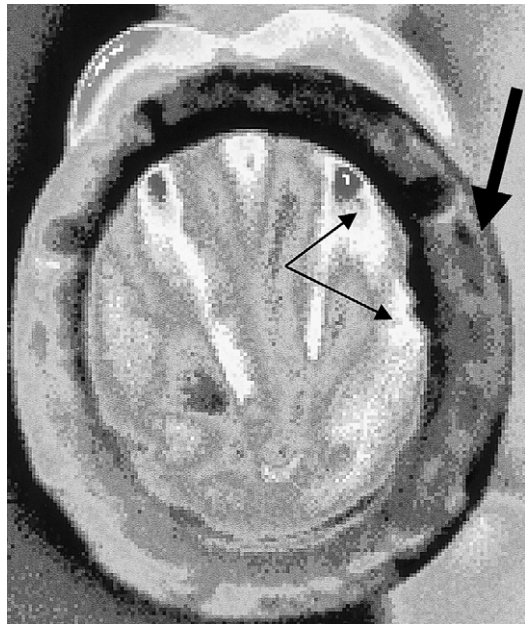

Fig. 25-1 Thermal image of the sole of a horse's foot showing asymmetrical heat in the sole of the hoof (*small arrows*). The shoe on that side (*bold arrow*) is colder, indicating less friction on the shoe, probably as a result of avoiding the injured sole.

horizontally, medial to lateral. The areas of joint capsule attachment tend to be hotter, but the center of the joint is relatively cooler. This may be the result of joint swelling or pressure and subsequent loss of microcirculation. Thermography does not appear reliably useful to detect joint damage, because joint temperature is influenced by chronicity and inflammatory changes affected by the amount of cartilage damage and the presence or absence of osteochondral fragments. However, thermal patterns of joints changed 2 weeks before lameness because of osteoarthritis; therefore by detecting inflammation before clinical signs are evident, training programs can be modified to reduce the risk of more serious injury.[6]

Long Bones

A bone must be superficial to affect skin temperature.[7] Bones that are heavily covered with muscle cannot be accurately assessed by thermography. Thermography may be useful for evaluation of the bucked-shin complex (see Chapter 104) or stress fractures of the radius or tibia. Thermographic changes may precede radiographic changes by up to 2 weeks.[4]

The bucked-shin complex is categorized into three grades.[8] Grade 1 is characterized by eliciting pain on palpation of the third metacarpal bone (McIII), but with no detectable radiographic abnormality. Grade 2 is characterized by pain and radiographic evidence of subperiosteal callus. Grade 3 is characterized by pain and radiographic evidence of a stress fracture. Grades 2 and 3 may be indistinguishable, and radiographic confirmation of a stress fracture may not be possible for 2 to 3 weeks. Thermal variations between grades 2 and 3 may result in earlier diagnosis. Grades 1 and 2 are characterized by an area of increased temperature 1° to 2° C warmer than the surrounding tissues over the dorsal aspect of McIII. Grade 3 disease has a more widespread area of increased temperature, 2° to 3° C warmer than the surrounding tissues, and this is usually visible on the lateral and medial views, in addition to the dorsal view.

Tendons

Normal flexor tendons have elliptical isothermic zones that are bilaterally symmetrical.[9,10] The lowest temperature is centered over the palmar aspect of the tendons, and the peripheral areas near the carpus and fetlock are approximately 1° C warmer.

Acute tendonitis causes an area of increased temperature over the site of the tendon lesion[10] and may be demonstrated up to 2 weeks before physical evidence of swelling and pain (see Fig. 118-4). Thus tendon lesions of potentially clinical significance can be identified, and adjustments in the training protocol can be made to prevent further damage to the tendon.

As a tendon heals, the thermal pattern becomes more uniform but remains abnormally elevated compared with normal tendon.[10] As the lesion heals and scar tissue is deposited, the skin over the injured area may actually show a decrease in temperature, whereas the remaining neovascularized tendon continues to have increased thermal emissions. During the assessment of healing the thermal changes do not correlate well to the structural reorganization of the tendon matrix as assessed by ultrasonography.[11] The thermal pattern diffuses so an area of increased temperature no longer is apparent, but an overall increased thermal emission from the damaged tendon may occur compared with normal. Mechanical stress proximal to an injury can aggravate existing tendon damage, and thermography can be used to detect new injury so that changes in management are made accordingly.

Ligaments

Ligament injuries appear similar to tendon injuries, with an area of increased temperature centered over the injured area. However, somewhat paradoxically, proximal suspensory desmitis may be associated with an area of increased temperature over the proximodorsal aspect of McIII. Thermography is most useful in correlating whether heat is associated with a sensitive ligament, especially the suspensory ligament (SL). Horses often resent palpation of the body of the SL. Thermography can determine if associated inflammation exists (Fig. 25-2; Color Plate 2). Thermography can detect whether inflammation of the SL is associated with an exostosis on the second or fourth metacarpal bones.

Muscles

Identification of damaged muscles may be difficult clinically. Thermography may be able to locate an area of inflammation associated with a superficial muscle or muscle group and may identify atrophy before it is clinically apparent.[12]

Muscle inflammation is usually identified as an area of increased temperature in the skin directly overlying the affected muscle.[12] Rarely, swelling and edema in the affected muscle is severe enough to inhibit blood flow through the muscle; the injured muscle is seen as an area of decreased temperature. The right and left sides must be compared and are normally nearly identical. Consistent variations from side to side indicate muscle damage at either the area of increased temperature or decreased temperature.

The most common cause of muscle inflammation is muscle strain. A classification of first-, second- or third-degree strain

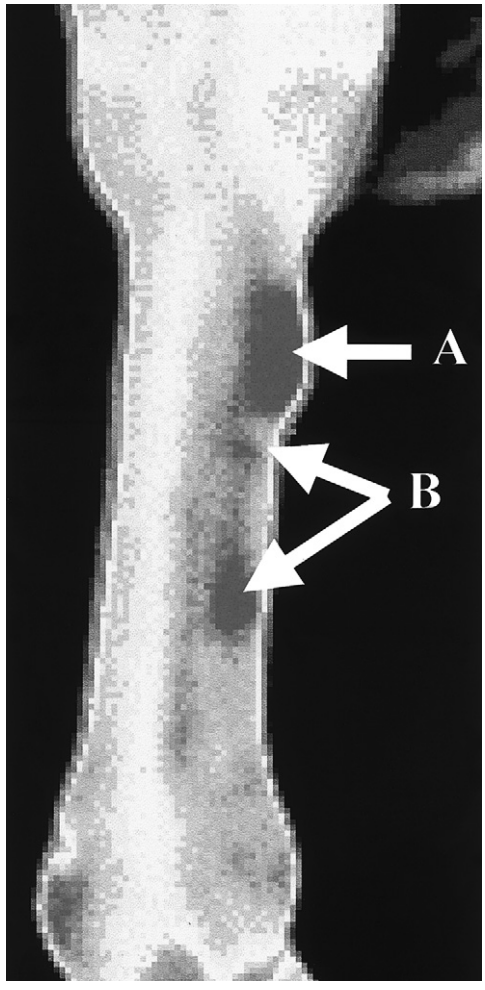

Fig. 25-2 Thermal image of the left medial metacarpal region showing an active splint (*A*) that is causing secondary inflammation in the suspensory ligament distal to the injury (*B*).

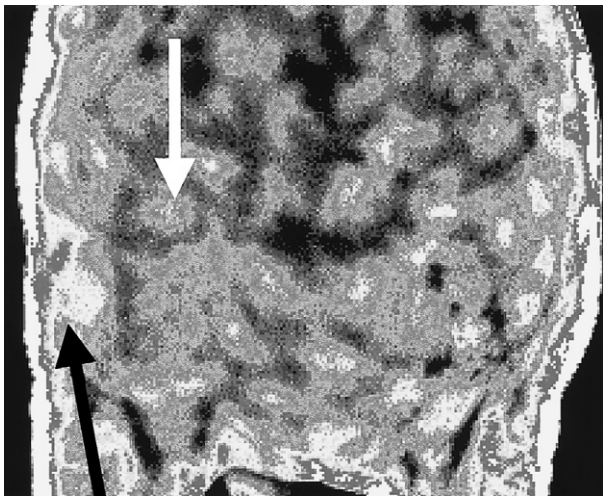

Fig. 25-3 Thermal image, front view, of a horse's chest showing increased heat over the lateral aspect of the right shoulder (*black arrow*) and decreased heat over the right bicipital tendon *(white arrow)*. Ultrasonography showed increased fluid in the intertubercular (bicipital) bursa. The decreased heat over the muscle was presumed to be due to lack of use because of pain.

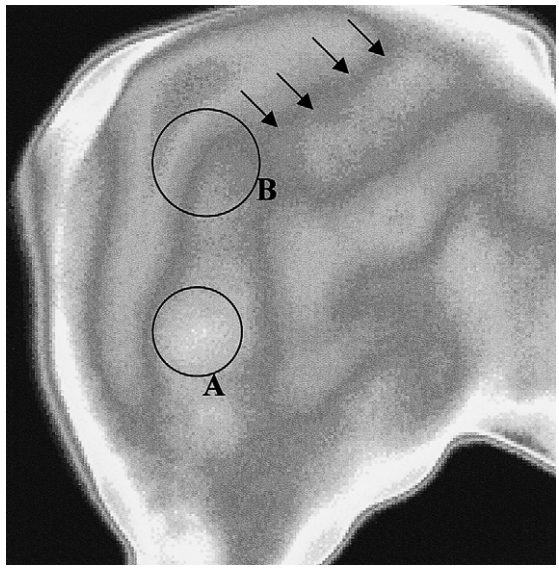

Fig. 25-4 Thermal image of the lateral view of the right hip region showing increased heat over the third trochanter (*A*) and greater trochanter (*B*). In addition, the body of the gluteal muscle has increased heat (*small arrows*). This condition was diagnosed as a gluteal muscle strain.

injuries, described in human athletes, has been applied to horses.[12] Muscle strains have not been commonly documented in the forelimb, but I have identified pectoralis and shoulder extensor muscle injuries (Fig. 25-3; Color Plate 3). Croup and caudal thigh myopathies are better recognized. Croup myopathies are strains of the longissimus, the origin of the gluteus medius (level of the sacroiliac), the body of the gluteus medius, and the insertion of the gluteals on the greater and the third trochanters of the femur (Fig. 25-4; Color Plate 4). Caudal thigh myopathies consist of injuries to the biceps femoris, semitendinosus, or semimembranosus muscles. Injuries to the biceps femoris and semimembranosus most commonly are mid-body muscle strains, but semitendinosus injuries usually occur at the musculotendonous junction.

Vertebral Column

Injuries to the vertebral column are characterized by areas of increased temperature, decreased temperature, or root signatures. Results of thermography may suggest the need for radiography if a luxation, subluxation, or fracture is suspected.

The thermographic evaluation is performed from above the animal (top line view) for the thoracic, lumbar, and sacral vertebrae. Images made from the left and right sides are used to evaluate the cervical vertebrae. Injuries generally are located along the midline. The thermal area directly corresponds to the injury. Areas of decreased temperature noted over the vertebral column, unlike other areas, are not seen in horses with chronic injuries and may suggest more recent injuries associated with marked swelling, which may affect the autonomic nerve supply. *Root signatures* are linear increases in temperature that follow nerve roots from the vertebral column. They theoretically occur because of irritation to the local sympathetic nerves. They can involve the entire sympathetic trunk where an entire side will have an increased temperature, such as occurs in Horner's syndrome.[13]

Sacroiliac subluxations have been associated with a distinct thermal pattern, with a focal area of decreased temperature between the two tubera sacrale. This correlates with ultrasonographic evidence of sacroiliac ligament damage.[14]

The use of thermography for assessing saddle fit is discussed in Chapter 118.

EDITORS' NOTE: In our experience, the results of thermography do not always correlate with the results of other diagnostic techniques, and therefore, while recognizing the potential value of the technique, consider that results must be interpreted with care.

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CHAPTER • 26

Clinical Investigation of Foot Pain

Kevin Keegan and Sue J. Dyson

Many aspects of clinical examination are discussed in Chapters 2 to 8 and are not repeated here. This chapter considers some specific aspects in more depth.

DETAILED EXAMINATION OF THE HOOF

Visual Examination

The hoof is specialized epidermis that responds to stress and tissue damage by altering growth. The gross appearance of the hoof therefore provides visible clues to abnormalities of the entire foot. Potential lameness can be anticipated by early recognition of hoof abnormality. Overall size and shape of the hoof and the character of the hoof wall, sole, frog, and coronary band should be noted. The veterinarian should inspect the hoof from many directions, from a distance and close up, and palpate a clean hoof wall, sole, and coronary band. The veterinarian should note broken-back or broken-forward hoof wall-to-pastern axes, under-run heels, sheared heels, contracted heels, and excessively small feet, which are discussed in detail in Chapters 4, 27, and 28.

The white line is the junction between the tubular horn of the sole and the soft horn (epidermal laminae) of the hoof wall and is a potential space. The white line should parallel the weight-bearing surface of the hoof wall and should be of uniform thickness (approximately 3.5 mm) throughout its curvature. Mechanical trauma, nutritional dysfunction, excessively wet or polluted environments, microorganism invasion, or underlying physical or metabolic dysfunction, such as with laminitis, may cause horn damage and cleft formation within the white line. Poor or abnormal hoof growth for any reason increases vulnerability. A widened white line is evidence of hypertrophic laminar growth in response to epidermal injury. Metaplastic differentiation of damaged epithelial cells at the laminar surface creates friable, granular horn within the white line, a condition called *seedy toe.* Cleft formation is more common with a widened white line and in the seedy toe condition, predisposes the sensitive tissues of the foot to contamination.

Evaluation of the foot should consider the ratio of the horse's weight to overall foot size. Heavy horses with small feet are more prone to develop foot-related lameness conditions and generally have more guarded prognoses. A crude formula has been created that relates body weight to weight-bearing surface area of the foot. The weight-bearing surface area of the foot can be estimated by measuring the circumference of the foot just below the coronary band. Performance horses with body weight to single foot surface area above 5.5 kg/cm^2 have an excessive weight/area ratio. Decreasing body weight by feed manipulation or increasing weight-bearing surface area of the foot by shoeing should be recommended for large horses with small feet.

Evaluating Hoof Wall Surface

The hoof wall is a keratinized avascular, stratified squamous epithelium. Two different types of gross structure may be seen during close examination and palpation of the hoof wall. Small, slender lines that run parallel to the long axis of the hoof are *horn tubules.* Horn tubules are normal hoof wall structures that propagate distally from a basal layer of cells at the coronary band. Irregular, raised, or recessed transverse lines (rings) that run parallel to the coronary band are evidence of dysfunction in hoof wall growth. Laminar hyperplasia occurs in response to injury. Substantial ring formation in a single foot indicates previous abnormality or stress to that limb. Changes in nutritional status, activity level, environmental conditions, or other physiological stress, such as septicemia, cause rings to appear in all feet. Rings move distally as the hoof wall grows.

Width of rings correlates roughly with the duration of previous stress or hoof-related abnormality, and the distance of rings from the coronary band gives some indication of how long ago the stress or abnormality occurred. An increase in the distance between rings at the heel compared with the toe (diverging rings) indicates slower relative hoof wall growth at the toe, previous laminar damage, and diminished blood supply to the dorsal regions of the foot.

In a normal horse within a normal environment the hoof wall grows distally at a rate of approximately 8 to 10 mm per month or about 0.14 to 0.2 mm per day. In horses with laminar inflammation this increases to about 0.25 mm per day.[1] In a study of Lipizzaner horses the rate of growth for control animals and those given biotin supplements was 7 mm in 28 days, resulting in a renewal period for the entire hoof wall of about 11 months.[2] Other estimates put complete hoof wall regeneration at 12 months, 6 to 8 months, and 4 to 5 months for the hoof wall at the toe, quarter, and heel, respectively.[3]

Palpation

Hoof wall temperature may vary between limbs in a normal horse and may partially reflect environmental temperatures. The hoof wall may be unusually warm in association with a sub-solar abscess or laminitis. Assessing the amplitude of the pulses in the medial and lateral digital arteries can be an excellent aid in identifying foot pain, particularly associated with a sub-solar abscess, sub-solar hemorrhage, nail bind, or laminitis. Assessment is easy in most horses but difficult in heavy cobs and draft breeds in which thick skin and a thick, long hair coat may make accurate palpation impossible. Diffuse filling in the metacarpal and pastern regions may reflect severe inflammation in the distal part of the limb caused by sub-solar abscessation. Careful palpation of the coronary band may reveal a softer region, from a long-standing sub-solar abscess, through which pus could potentially drain. Assessing the coronary band is also important in laminitis, because depression at the coronary band may be the first sign of sinking (distal displacement) of the distal phalanx.

The foot should be palpated bearing weight and not bearing weight. With the limb picked up the stability of the bulbs of the heel can be assessed by grasping each bulb with the left and right hands and determining whether movement occurs between the two, indicating sheared heels. Firm pres-

sure should be applied over the distal palmar aspect of the pastern to determine whether pain can be elicited, which may reflect injury of the deep digital flexor tendon (DDFT).

Hoof Tester Application

A complete examination of the foot includes a thorough and proper application of hoof testers to the hoof capsule. Many different kinds of commercially and custom-made hoof testers are available. The most useful are those that are adjustable, to enable the user to apply them across differing widths of the foot, and large enough to allow the user to grip and apply pressure to both handles with one hand. For complete evaluation the hoof testers should be applied across the heels, from one frog crura to the opposite hoof wall at the quarter, and along the periphery of the foot from the sole to the hoof wall. When applied across the heels, the contact points of the hoof testers should be centered approximately between the coronary band and the bottom of the hoof. Force of application should be strong enough to move the heels slightly axially. When applied from the frog to the quarters, the contact points of the hoof testers should be at the middle of the frog and midway between the coronary band and the bottom of the foot at the quarter. Force of application should be strong enough to depress the frog noticeably. When applied across the sole and hoof wall, the force of application should be strong enough to incur slight indentation in the solar surface. A positive response is interpreted when the foot is continually withdrawn in synchrony with consistent application of pressure. In horses with a very compressible thin sole, careful comparison should be made with the responses in each foot. Hoof testers are helpful for localizing foot abscesses, identifying nail bind or prick and sub-solar bruising, and checking preliminary suspicion of acute laminitis, distal phalangeal fracture, and navicular disease, provided that the foot is not excessively hard. However, in one study, only 11% of animals with navicular disease responded to the use of hoof testers. Percussion can be applied to the hoof wall or the sole using hoof testers or a hammer and can also be valuable in identifying a subsolar abscess, nail bind or prick, laminitis, laminar tearing, and an unstable hoof wall crack or a fracture.

OTHER PROVOCATIVE TESTS

Frog Pressure Test

Direct pressure is applied to the frog and underlying structures during the frog pressure test. The horse is forced to stand on a hard object placed under the frog by the veterinarian picking up the opposite limb. The object should be firm, small enough to prevent sole and hoof wall contact, but large enough to avoid trauma to the frog. The wooden handle of a farrier's hammer is well suited for this. The horse is forced to stand on the object for 1 to 2 minutes and then observed for lameness at a trot. This test is valuable for assessing horses with suspected navicular disease, navicular bursitis, distal DDFT pain, and bruising of the sensitive frog tissues. Horses that respond more positively to this test than to the reverse wedge test are presumably more adversely influenced by ground impact and may benefit more from application of shock-absorbing pads to the foot than by changing the dorsal hoof wall angle.

Reverse Wedge Test

The reverse wedge test applies tensile stress to the DDFT, and indirectly, compressive force to the navicular bursa and bone. The reverse wedge test also tenses the navicular suspensory ligaments. An object is placed underneath the toe; the handle of a farrier's hammer works well. The horse is forced to stand with the toe elevated for 1 to 2 minutes by the veterinarian picking up the opposite limb and is then observed for lameness at a trot. Some horses with navicular disease show increased lameness. Horses that respond with increased lameness to this test, but not to the frog pressure test, presumably have a better chance of responding favorably to heel elevation or extension than to the addition of shock-absorbing pads to the foot.

Distal Limb Flexion Test

For the distal limb flexion test the distal limb is elevated with as little carpal flexion as possible. The toe of the foot is grasped and pulled proximally, while the distal metacarpal or metatarsal region is stabilized. This stresses all of the distal limb joints and the soft tissue structures on the palmar/plantar aspect of the distal limb (distal sesamoidean ligaments, flexor tendons, and navicular bursa). Withdrawal of the limb during flexion, following 1 minute of continuous flexion, may reflect pain or dysfunction in any of these distal limb structures. The distal limb flexion test greatly depends on evaluator technique and therefore is not very specific.[4]

NERVE AND JOINT BLOCKS

Localization of lameness to the foot is facilitated by perineural or intra-thecal analgesia of the distal limb (see Chapter 10). The abaxial sesamoid nerve block may be used to determine quickly if the foot is the source of pain, whereas the palmar/plantar digital nerve block is used to isolate more specifically the source of pain. Intra-articular analgesia of the distal interphalangeal joint and intra-thecal analgesia of the navicular bursa are used to localize the source of pain to these specific areas. Each of these techniques is described briefly, but care must be taken in interpretation because no block is specific. A combination of techniques probably gives the most information.

Palmar/Plantar Digital Nerve Block

The palmar/plantar digital nerves may be blocked anywhere along the length between the distal aspect of the proximal sesamoid bone (PSB) and the proximal extent of the cartilage of the foot. Deposition of local anesthetic solution more distally can be expected to be more specific than deposition more proximally and is less likely to block dorsal branches inadvertently, thereby desensitizing the entire foot, but this has not been substantiated. The needle (20- to 25-gauge) is inserted subcutaneously directly over the nerve. It is advisable to use small amounts of local anesthetic solution (1 to 1.5 ml) to decrease the likelihood of inadvertent analgesia of dorsal branches. Many more structures than previously thought are affected by palmar/plantar digital analgesia.[5]

A successful palmar/plantar digital nerve block desensitizes some of the skin surface along the back of the heel bulbs but should not desensitize the skin over the dorsal coronary band, although it often does. The area of heel desensitization varies from horse to horse and can be limited to a small area between the heel bulbs or encompass the entire area on the palmar surface of the distal limb distal to the level of needle insertion. In the hindlimb, medial and lateral dorsal metatarsal nerves may also supply plantar structures distal to the metatarsophalangeal joint.[6] Therefore in the hindlimb, additional subcutaneous deposition of local anesthetic solution may be necessary over the dorsal pastern at the same level, but this also desensitizes dorsal structures of the foot, negating the utility of the block for lameness diagnosis. Careful checking for effectiveness and area of desensitization is important for proper evaluation of lameness. A horse may be somewhat apprehensive after the needle insertions and frequently picks up the limbs randomly when approached. The clinician should ask the person holding the horse to cover its eye.

Checking the blocked limb from the contralateral side is useful after first touching the contralateral limb. This gives the horse the impression that the veterinarian will now be manipulating that limb. If the block is successful, the horse will not withdraw the limb when the skin is touched lightly or pressed hard with a blunt point, such as a key or tip of a pair of artery forceps. If local anesthetic solution is deposited immediately around the nerve trunk, the block is effective within a few minutes. Deposition of local anesthetic solution a few centimeters from the nerve trunk may be effective after a longer period. Effectiveness for lameness reduction should not be finally decided until at least 10 minutes after injection.

Some evidence indicates that a palmar digital nerve block changes the biomechanics of the blocked and contralateral unblocked limbs, even in a normal horse.[7,8] Presumably, the horse's proprioceptive ability is partially affected, but the change is slight and unlikely to affect subjective evaluation of lameness.[7,8]

A biaxial palmar digital nerve block is most frequently used to diagnose navicular disease in the forelimb but also improves lameness from many other causes, but the diagnosis of navicular disease should not be based only on a decrease in lameness subsequent to this block. Many horses with navicular disease also do not respond completely to the palmar digital nerve block because of concurrent flexor tendon, suspensory ligament, distal interphalangeal joint, distal phalanx, or shoulder area pain. Uniaxial palmar/plantar digital nerve block may be used to confirm lameness caused by uniaxial injury or infection of the cartilage of the foot (sidebone, quittor), distal interphalangeal or proximal interphalangeal joint exostoses, or nonarticular palmar process fracture of the distal phalanx.

Abaxial Sesamoid Nerve Block

The abaxial sesamoid nerve block is performed distal to the dorsal branching from the main trunk of the digital nerve over the abaxial surface of the PSB. Although the block is performed distal to the dorsal branching, the dorsal branches are desensitized because of the close proximity of the dorsal branches to the main nerve trunk at this location. A successful abaxial sesamoid nerve block desensitizes most of the skin distal to the fetlock joint. Occasionally horses retain some dorsal skin sensitivity, especially in a hindlimb. It is important to recognize that with severe pain associated with a sub-solar abscess, a fracture of the distal phalanx, or laminitis, apparent desensitization of the foot may in some horses have no effect or only a partial effect on lameness. It is also important to be aware that local anesthetic solution, through local spread and proximal diffusion, may alleviate pain at the level of the block or proximal to it.

Distal Interphalangeal Joint Block

Intra-articular analgesia of the distal interphalangeal joint is not specific, and peri-articular structures of the foot are also desensitized, presumably because of direct analgesia of the palmar/plantar digital nerves because they lie near the abaxial surfaces of the joint. Local anesthetic solution also diffuses into the navicular bursa in levels sufficient to desensitize intrabursal tissues, improving lameness caused by primary navicular bursal inflammation[9] or navicular disease. However, up to 20% of horses with navicular bone pain have a negative response to intra-articular analgesia of the distal interphalangeal joint.[10] Many approaches are used, including a lateral approach, which is easier to perform when the limb is not bearing weight (see Chapter 10).[11]

Navicular Bursa

A variety of techniques have been described for injection of the navicular bursa (see Chapter 10). The needle should ideally be directed toward the middle third of the navicular bone, from proximally to distally, to avoid inadvertent penetration of the palmar pouch of the distal interphalangeal joint or the digital flexor tendon sheath (DFTS). Retrieval of synovial fluid generally means that the needle is in the distal interphalangeal joint or the DFTS. The response to intrathecal analgesia should be assessed within 5 minutes of injection. Occasionally if lameness is not improved, a horse may appear transiently much lamer after intra-bursal analgesia, probably because of injection of excessive volume (>4 ml). Intra-thecal analgesia of the navicular bursa can improve pain associated with the bursa, the navicular bone, the distal sesamoidean impar ligament, the collateral ligaments of the navicular bone, and sometimes the DDFT and the distal interphalangeal joint.

RADIOGRAPHIC EVALUATION

Because slight radiographic changes can denote serious problems, good radiographic technique and patient positioning are vital. Proper preparation of the foot is important, because prominent radiographic artifacts may be created by the uneven surfaces of the sole and frog or by radiodense dirt and gravel packed into the bottom of the foot. To eliminate such artifacts, the shoe should be removed and the foot trimmed and the frog sulci packed with clay or modeling compound (e.g., Play-Doh; Hasbro, Inc., Pawtucket, R.I.) before radiographs are obtained.

Equipment and Technique

Because of the small size of the equine foot, good-quality radiographs can be obtained with small portable x-ray machines. Inconsistent power supplies in the field, exacerbated by excessively long extension cords, may compromise the quality of radiographs. A length of 7.5 m is acceptable, but a cord longer than 15 m can cause a loss of up to 30% of the electrical energy supplied to the x-ray machine. A standard radiographic technique for evaluation of the foot using a portable machine and a standard 400 speed film-screen system would be 80 kVp, 10 mA, for 0.06 to 0.08 seconds or 100 kVp, 10 mA, for 0.04 to 0.06 seconds at a film focus distance of 50 cm.

Views: Positioning and Evaluating Images

A standard radiographic series of the equine foot includes lateromedial, dorsopalmar, dorsoproximal-palmarodistal oblique (upright pedal; DPr-PaDio), and palmaroproximal-palmarodistal oblique (PaPr-PaDio) views. Two DPr-PaDiO (upright pedal) views should ideally be obtained, one exposed to evaluate the margins of the distal phalanx and the other well collimated and exposed to assess the navicular bone. Alternatively, views of the distal phalanx and navicular bone can be obtained with the horse standing on the cassette using dorsal 65° proximal-palmarodistal oblique views, but this results in more distortion. Flexed oblique (dorsolateral-palmaromedial and dorsomedial-palmarolateral oblique) views are useful for evaluating the articular margins of the interphalangeal joints and the palmar processes of the distal phalanx.[12]

Oblique dorsopalmar views obtained with the horse bearing weight are also useful for assessing the cartilages of the foot and palmar processes of the distal phalanx and for differentiating the source of mineralization proximal to the navicular bone.

Lateromedial

The foot should be positioned on a block high enough to raise the bottom of the foot off the floor so that the radiographic beam can be directed at the coronary band and parallel to the floor, with the lower edge of the cassette beneath the level of the sole. The limb should bear weight, which may be facili-

tated by elevating both feet on blocks or by picking up the opposite limb during radiographic exposure. To assess rotation or sinking of the distal phalanx, taping a metal strip on the dorsal aspect of the hoof wall, extending from the level of the coronary band distally, is helpful. However, if the hoof capsule is excessively distorted in shape, this can be misleading. A thumbtack placed in the apex of the frog gives positional relationship between the frog and the tip of the distal phalanx, which may be helpful for planning proper placement of a heart-bar shoe for the treatment of laminitis or for helping to determine position of break over for shoeing purposes. Softer exposure factors should be used when assessing the orientation of the distal phalanx compared with evaluation of the architecture of the distal phalanx and navicular bone.

Examining a hind foot may be easiest by positioning the foot on a higher block, with only the toe bearing weight, taking care not to rock the foot medially or laterally, because a horse may be reluctant to allow the foot to be held flat on the block.

The slope of the dorsal hoof wall capsule should be the same as the slope of the dorsal aspect of the distal phalanx. Rotation of the distal phalanx has occurred if the angle of the dorsal surface of the distal phalanx with the ground is greater than the corresponding angle of the dorsal hoof wall. This may occur secondary to laminar or horn damage as in laminitis and white line disease, respectively. If the hoof wall is excessively long and distorted, it may give a false impression of rotation of the distal phalanx, and radiographic assessment should be repeated after radical trimming of the foot. Distal displacement of the distal phalanx within the hoof wall capsule is evaluated by measuring the space between the dorsal hoof wall and the dorsal aspect of the distal phalanx relative to the palmar cortical length of the distal phalanx. A ratio greater than 28% is abnormal and is evidence of distal phalangeal distal displacement within the hoof capsule (sinking).

In a normal foot the solar border of the distal phalanx slopes, with the palmar processes located slightly more proximally than the toe. In some horses the solar border is horizontal, or the palmar processes are actually lower than the toe. This may be associated with palmar heel pain, especially in a forelimb. In the lateromedial view the thickness of the sole can also be evaluated. Horses with particularly thin soles seem prone to "footiness."

The lateromedial view is useful for detecting osteochondral fragmentation of the extensor process of the distal phalanx, modeling of the articular margins of the distal interphalangeal joint or navicular bone, and mineralization within the DDFT or navicular suspensory ligament. The shape of the navicular bone can be assessed, and the presence of entheseous new bone, trabecular sclerosis, and erosion of the flexor surface can be detected in some horses.

Dorsopalmar

The foot is positioned on a block, and a cassette placed palmar to it, perpendicular to the ground. The x-ray beam is directed perpendicular to the limb, parallel to the ground, at the level of the coronary band. The dorsopalmar view is useful for assessing mediolateral foot balance, mediolateral symmetry of the phalanges, and interphalangeal joints and for detecting modeling of the articular margins. Mineralization or ossification of the cartilages of the front foot (sidebone) is common and unusually incidental and is more prominent in large horses, especially in working breeds, than in lighter horses used for racing and riding.[13,14] Ossification of the cartilages of the foot increases rapidly in the first 2 to 3 years of life. Extensive ossification of the cartilages of the foot reaching the level of the proximal interphalangeal joint is unusual and a potential cause of pain and lameness. There is limited evidence that incomplete fusion of the cartilages to the distal phalanx may be clinically significant.[15]

Dorsoproximal-Palmarodistal Oblique View (Upright Pedal)

The toe of the foot to be examined is placed in the V of a purpose-designed block, with the dorsal wall of the foot slightly in front of the vertical. The cassette is positioned vertically against the sole. A horizontal x-ray beam is used, centered on the coronary band. Avoid over-flexion of the fetlock by bringing the block forward so that the navicular bone is positioned proximal to the distal interphalangeal joint, so its distal border can be evaluated. Ideally two exposures should be used, first a softer exposure so that the margins of the distal phalanx can be assessed and then a higher exposure, using a well-collimated x-ray beam, to evaluate the navicular bone. In a well-positioned view the proximal articular and flexor margins of the navicular bone can be seen separately.

Examining a hind foot is easier by positioning the toe of the foot on a horizontal block, holding the x-ray cassette vertically on the dorsal aspect of the foot, and directing the x-ray beam from plantar to dorsal.

The DPr-PaDiO view is useful for evaluating the margins and internal architecture of the distal phalanx, although nondisplaced fractures, especially of the palmar processes, may not be detectable. Considerable variation exists between horses in the regularity of the solar margin of the bone, the size and number of the vascular channels, and the size and shape of the crena at the toe, but most horses have bilateral symmetry between left and right limbs. This view is also valuable for evaluating the shape of the navicular bone, the contour of the proximal articular margins, the presence of entheseous new bone along the proximal border, or mineralized fragments distal to the bone and assessing the number, size, and shape of radiolucent zones along the distal or proximal borders of the bone or within the medulla. A fracture of the navicular bone is usually clearly evident in this projection, but care should be taken not to confuse an artifact created by a frog cleft.

Palmaroproximal-Palmarodistal Oblique

The PaPr-PaDiO view can be used to assess the navicular bone and palmar processes of the distal phalanx. Ideally the x-ray beam should be well collimated to the region of interest. The two techniques for obtaining this view both involve the horse standing on the cassette, which ideally should be protected by a non-radiodense cassette tunnel or hardboard. With the first technique the limb to be examined is positioned caudal to the contralateral limb to extend the metacarpophalangeal joint and increase the angle of the pastern. The exact position depends on the conformation of the distal part of the limb, but the aim is to avoid superimposition of the palmar aspect of the metacarpophalangeal joint over the navicular bone. The x-ray machine is placed under the horse's abdomen and the x-ray beam is centered between the bulbs of the heel, perpendicular to a tangent to the heel bulbs. The angle of the x-ray beam is directed by the conformation of the foot and in a well-conformed foot is 45° to the horizontal.

The x-ray beam should be tangential to the flexor surface of the navicular bone. With a low collapsed heel the angle of the x-ray beam should be reduced accordingly to as little as 35°. Conversely, with an upright foot conformation the angle should be increased to approximately 50°. The angle of the x-ray beam is critical because artifacts are readily created, including an impression of reduced corticomedullary demarcation, sclerosis of the medulla, and an abnormal contour of the flexor surface. Alternatively, the foot to be examined is placed on a 10° wedge, and the contralateral foot is picked up. The x-ray beam is centered similarly, at a 30° angle to the horizontal for a normally conformed foot.

These views are more difficult to obtain in small, overweight ponies, and are generally easier to perform in forelimbs than hindlimbs. The PaPr-PaDiO (skyline) view of the navicular bone

permits evaluation of the body of the bone without superimposition over other structures, if correctly positioned, and is most sensitive for evaluating trabecular architecture, demarcation between the medulla and the dorsal and flexor cortices, and thickness, regularity of outline, and opacity of the flexor cortex. In a normal horse a clear definition exists between the radiopaque flexor cortex and the more radiolucent medulla, which has a well-defined trabecular pattern, with small lucent zones representing the distal border synovial invaginations. The flexor cortex varies in width between horses and tends to be thinner in a horse with a clubfoot. There may be a normal crescent-shaped radiolucent area within the flexor cortex at the sagittal ridge.

The view is invaluable for detecting medullary sclerosis, decreased corticomedullary demarcation, increased thickness of the cortex, and an irregular outline of the flexor cortex caused by erosions, often associated with adhesions of the DDFT, or by new bone formation. Communication of a radiolucent zone within the body of the bone with the flexor surface can be determined.

Flexed Oblique Views of the Interphalangeal Joints

The toe of the foot to be examined is placed in a V-shaped block. The x-ray beam is directed horizontally, centered at the coronary band midway between the dorsal wall and the heel. The cassette is place perpendicular to the x-ray beam. Dorsal 30° to 45° lateral (or medial)–palmaro (plantaro) medial oblique and dorsal 30° to 45° medial (or lateral)–palmarolateral oblique views are best for evaluating the lateral and medial palmar processes, respectively, whereas a 60° angle is preferable for assessing the articular margins of the interphalangeal joints.

Weight-Bearing Oblique Views of the Distal Phalanx

If a fracture of the distal phalanx is suspected but cannot be identified in any of the preceding views, it may be worthwhile obtaining dorsal 45° lateral-palmarodistal medial oblique and dorsal 45° proximal 45° lateral-palmarodistal medial oblique views of the lateral and medial palmar processes, respectively. These views are obtained with the horse standing on the cassette. Obviously the degree of obliquity can be varied. These oblique views are useful to evaluate the articular surface, subchondral bone, and dorsomedial and dorsolateral aspects of the distal phalanx.

Contrast Studies

If a horse has sustained a penetrating injury to the solar surface of the foot and a tract can be identified, its extent is best determined by insertion of a metal probe and obtaining a lateromedial radiographic view. Injection of radiopaque contrast agent can also be invaluable for determining communication with the distal interphalangeal joint, the navicular bursa, or the DFTS.

SCINTIGRAPHIC EVALUATION

Most scintigraphic imaging of the foot is performed using ^{99m}Tc–methylene diphosphonate (^{99m}Tc-MDP; see Chapter 19).[16] Flow, pool, and delayed (bone) phase images of each foot can be obtained using dorsal (or plantar), lateral, and palmar (solar) images. Diagnostic scintigraphic images of the distal part of the limbs depend on adequate blood flow, and to avoid poor uptake of the radiopharmaceutical into bone associated with poor blood flow, it is useful to lunge the horse at trot and canter for 15 minutes before injection of the radiopharmaceutical and bandage the distal part of the limbs for the previous 15 hours.[17] Scintigraphy is more sensitive but less anatomically specific than radiography for detecting pathological conditions of the bone. Scintigraphy can help identify many different bone-related abnormalities of the foot and is most useful to detect, or eliminate from suspicion, early navicular disease or an acute distal phalangeal fracture.

Navicular disease may be difficult to diagnose unequivocally radiographically except for severe or end-stage disease. False-positive (low specificity) and false-negative (low sensitivity) results may occur.[18-22] Scintigraphy is more sensitive than radiography and may be helpful in differentiating navicular pain from non-specific palmar heel pain.[11] Although false-positive results may be due to confusion with radiopharmaceutical uptake in the cartilages of the foot in a lateral view, or in the distal interphalangeal joint in a solar view, horses without foot pain may have a high uptake of the radiopharmaceutical in the navicular bone.[23] False-negative results are not common. Interpretation is more accurate using quantitative analysis of radiopharmaceutical uptake and also interpreting results in the light of responses to analgesia of the distal interphalangeal joint and navicular bursa.[23] Fractures of the distal phalanx are common injuries but may not be detectable radiographically in the acute phase. Scintigraphy provides earlier diagnosis, with intense focal uptake of radiopharmaceutical. Solar views are most sensitive. Intensity of uptake decreases with time, but evidence of a fracture may persist up to 1 year after injury. Because some distal phalangeal fractures commonly heal by fibrous union and the horse still returns to successful function, scintigraphy may be useful to determine when to return to exercise.

Scintigraphic imaging of the feet for laminitis has primarily been performed as part of research studies to evaluate digital blood flow, using radiolabeled platelets or macroaggregated albumin.[20,21] One study, using blood pool imaging after intravenous injection of ^{99m}Tc-dithylene-triamine penta-acetic acid (similar to ^{99m}Tc-MDP), found significantly lower dorsal laminar activity in horses refractory to treatment with heartbar shoeing.[22] Scintigraphy may be useful for identifying peripheral distal phalangeal injury and inflammation consistent with pedal osteitis[24] and subchondral bone injury.[25] Uptake in the region of the DDFT in lateral pool phase images occurs with DDF tendonitis.[23,26] Less commonly, focal increased radiopharmaceutical uptake has been identified at the insertion of the DDFT in bone-phase images, in association with a distal DDFT lesion identified using magnetic resonance imaging (MRI).[26]

FLUOROSCOPY

Fluoroscopy is designed to evaluate dynamic characteristics, gives poor image detail, and is a potential radiation safety hazard. Image-intensified fluoroscopy is the only legal technology allowed in veterinary practice, is not recommended for use in routine veterinary practice, and should only be used after specialized training. Fluoroscopy is useful for monitoring positioning of the needle for injection of the navicular bursa.

DIAGNOSTIC ULTRASONOGRAPHY

The horny hoof capsule limits ultrasonographic evaluation of the foot. Limited access is available using either a convex array or sector transducer through the bulbs of the heel[27] or the frog.[28] This allows limited evaluation of the DDFT, the navicular bursa, and the proximal and palmar surfaces of the navicular bone. However, a transducer positioned between the bulbs of the heel is not parallel to the fibers of the DDFT; therefore artifacts are readily created. Access through the bulbs of the heel or the frog is principally restricted to the sagittal midline. The frog must be soft enough to penetrate, which may require soaking it for several days. A curved bed for the transducer must be created to ensure good contact. The ease with which this can be done is determined by the shape and consistency of the frog. The collateral ligaments of

the distal interphalangeal joint and the chondrocompedal and the chondrocoronal ligaments can be assessed from the dorsolateral and dorsomedial aspects of the distal pastern.[29] Lesions of the soft tissue structures on the palmar aspect of the pastern may cause lameness that is alleviated by perineural analgesia of the palmar nerves and may be present without detectable swelling or pain on pressure. Therefore the palmar aspect of the pastern should be examined routinely (see Chapters 16 and 83).

MAGNETIC RESONANCE IMAGING

MRI of the foot is discussed in detail in Chapter 21. MRI has been useful for identification of lesions of the DDFT; navicular bursa and bone; distal sesamoidean impar ligament, phalanx, and laminae; and the cartilage and subchondral bone of the distal interphalangeal joint.[26,30] MRI has the potential to greatly increase our knowledge of the causes of foot pain, but it requires general anesthesia.[30]

COMPUTED TOMOGRAPHY

Computed tomography (CT), like MRI, requires general anesthesia but permits evaluation of the bony and soft tissue structures of the foot. CT may be more sensitive than radiography in determining lesions of the navicular bone[31,32] and has proved valuable for identification of lesions of the DDFT within the hoof capsule.[33]

GENERAL BIOMECHANICS

An appreciation of the biomechanics of the foot is helpful to understand foot lameness and corrective trimming and shoeing (see Chapter 27). Computer-assisted kinematic analysis of gait and force plates has been used to study foot and limb movement and the forces acting on the foot. This, together with detailed anatomical modeling of the foot and distal limb, allows estimation of the forces acting on or through internal structures of the foot that are not directly measurable using such non-invasive techniques.[34,35]

Five opposing forces act on the distal phalanx within the hoof capsule: tension and shear at the foot-laminar junction, DDFT tension, extensor tendon tension, body weight applied down through the middle phalanx, and ground reaction force (GRF). Stresses at the foot-laminar junction have important implications in the development and treatment of laminitis. Tension in the DDFT has implications concerning navicular disease. Extensor tendon tension is minimal and active primarily during the swing phase of the stride. The amplitude and distribution of GRF to a large extent determines the stresses at the foot-laminar junction and in the DDFT tendon.

A stride begins at hoof-strike and ends at the next hoof-strike of the same limb. In normal horses at the walk, the heels strike the ground first, followed by the ground surfaces of the bars, quarters, and toe. The time between heel and toe impact is 9 to 15 ms.[36] At a slow trot this interval is much shorter, too fast to be definitively determined by the naked eye, and for most practical purposes the foot lands flat. Heel-first landing is obvious in the hind feet.[37] At hoof-strike the impact force is not maximal, because peak vertical GRF occurs at midstance.[38] However, at hoof-strike, peak initial foot and limb deceleration may reach 100 g (1 g = 9.8 m/sec^2).[36] After impact the foot then vibrates at high frequency for a short time. Most of the damping of the vibration after impact is provided by the interface between the hoof wall and the distal phalanx.[39,40] Therefore diminishing impact force and vibration is most important when addressing treatment of foot disorders involving the hoof wall laminae. Metal shoes in general increase the amplitude and frequency of hoof wall vibration after impact.[41,42]

The period during the stride in which the hoof is in contact with the ground is defined as the *stance phase* and is subdivided into *cranial* and *caudal phases*. The cranial stance phase begins with hoof-strike and ends at midstance, when the limb axis is perpendicular to the ground and the weight of the trunk is directly over the foot. Vertical GRF on the foot is maximal at midstance.[43] The center of vertical GRF is cranial to the center of rotation of the distal interphalangeal joint, creating an opposing lever arm to DDFT tension. At the trot, DDFT tension is maximal near midstance, corresponding approximately to the instant of maximum vertical GRF. However, at midstance, strain in the navicular suspensory ligament is negative, that is, it is relaxed.[44]

Vertical forces on the equine foot at a walk have been measured at about 4000 N.[45] Because trunk mass is medial to the limb, at midstance the center of maximum vertical GRF on the foot is slightly medial of midline. Initial impact on the lateral wall, followed by peak force occurring medially, causes many horses to develop low and flared lateral, but high and upright medial hoof walls. Reducing the effects of maximum vertical GRF is an important consideration in corrective trimming and shoeing.

The foot also experiences substantial horizontal GRF. Horizontal GRF is initially deceleratory or braking. In the forelimb, at about midstance, this horizontal braking force changes to become a horizontal propulsive force of approximately equivalent magnitude. The transition from braking to propulsion occurs much earlier in the forelimb than in the hindlimb.[46] In hindlimbs the horizontal propulsion is of greater magnitude than the horizontal braking; thus the forelimbs primarily perform support, whereas the hindlimbs perform support and propulsion.

Breakover occurs at the end of stance, beginning at heel-lift and ending at toe-lift. During breakover the hoof rolls about 45°. The position of the center of vertical GRF applied to the bottom of the foot increases and moves rapidly toward the toe during breakover. During breakover the strain in the navicular suspensory ligament is maximal.[44] Asymmetric hoof impact, high peak vertical GRF at midstance, and the rapid shift of vertical GRF to the toe during breakover all play important roles in the understanding of the treatment of certain foot problems.

During weight bearing the thinner, more elastic hoof wall at the heel expands. Hoof wall expansion creates tension in the laminae and periosteum of the distal phalanx.[47,48] As a result the distal phalanx descends and rotates in a clockwise direction around the toe (the dorsal border moves palmarly). Application of a shoe tends to reduce strain on the hoof wall.[9] The vasculature of the foot, predominantly the venous sinuses, augments the protective mechanisms of hoof wall expansion by providing additional hydraulic shock absorption. Increasing weight-bearing pressure on the frog has inconsistent effects, sometimes increasing heel expansion and sometimes causing contraction of the heels.[49]

The swing phase of the stride begins at toe-lift and continues until next hoof-strike. *Mid-swing* is defined as the halfway point in the foot flight pattern. Mid-swing can be estimated by the position of the contralateral limb, which will be at midstance. In a sound horse, hoof trajectories are individual and variable.[50] Some horses have relatively parabolic arcs, with maximum hoof height occurring at mid-swing, and some have more skewed paths, with maximum hoof height occurring during the first half of the swing phase. Many horses exhibit a flipping up of the toe of the foot just before hoof strike as the foot prepares to land flat-footed or slightly heel first. This toe elevation at the end of swing is decreased in horses with low

hoof wall angles, and some horses with substantially low hoof angles land abnormally, toe first. The pattern of hoof flight is used by some equine practitioners to detect lameness and by many lay performance judges to assess athletic ability.[51,52] In one study, horses judged to be of inferior ability had lower maximum hoof flight arcs during swing. All shoeing, because it adds weight to the foot, will increase maximum hoof height during swing.[50,53]

BIOMECHANICS AND LAMENESS

Two general questions of interest concern foot biomechanics and lameness. The first asks whether specific measurements can be used to isolate lameness to the foot. Most gait changes associated with weight-bearing lameness result from decreased vertical GRF, regardless of the specific location of pain within the limb. No biomechanical indicators specific for foot lameness have been identified. The second question asks what foot parameters can be used to indicate the horse's general mechanical response to lameness. It seems reasonable to assume that a lame horse spends less time bearing weight on a lame limb, but many studies have shown that in horses with mild to moderate lameness, stance-phase duration is actually increased with lameness.[54] Therefore, it follows that if total stride duration is not changed, then swing-phase duration of the lame limb decreases with lameness. Presumably, a reduction in maximum vertical GRF can be accomplished by spreading the force out over a longer period of weight bearing.[55] The stance phase duration does not begin to decrease until lameness becomes severe in nature and the horse is unable to maintain forward trunk velocity. In horses with unilateral lameness, maximum vertical GRF is lower in the lame limb compared with the sound limb, and the braking component of horizontal ground reaction is shorter and decreased in amplitude.

Equine practitioners commonly contend that protraction, or the length of the cranial phase of the stride, decreases in horses with lameness. A decrease in hindlimb protraction has been objectively measured in horses with induced tarsal lameness and this is easily seen at the trot.[56] This contention, however, has not been confirmed by objective studies of other lameness conditions, and a slight increase in protraction has been measured in some studies of equine forelimb lameness.[54,57,58] Decrease in limb protraction is not a good general indicator of lameness in horses. However, in the Editors' experience in the clinical situation, horses frequently appear to have a shortened cranial or sometimes caudal phase of the stride. Stride length is not significantly decreased until lameness is severe.[58] The appearance of decreased stride length with lameness may be visual deception caused by decreased swing-phase duration or altered relative positions of the trunk, limb, and foot during lameness.

Absolute hoof height during the swing phase of the stride is a poor parameter for quantitative evaluation of lameness. Classic textbook descriptions describe low hoof flight arcs to be characteristic of lameness.[59] Electromyographic measurements of forelimb muscles suggest that the swing phase is entirely passive, resulting from elastic energy released from the flexor tendons at the end of stance.[60] Because this passive recoil would be reduced in a lame limb, absolute hoof height during the swing phase of the stride should also be reduced. However, kinematic studies of horses with lameness of the forelimb have failed consistently to document this. Additionally, lower trunk height (and therefore lower hoof height) after lame limb lift-off is frequently offset by increased joint flexion necessary for the limb to clear the ground during the forward progression of the stride.[56] Therefore maximum hoof height during swing is not consistently changed with lameness.

During breakover, compressive force concentrates at the toe and navicular suspensory ligament tension increases. Although breakover is difficult to measure accurately because of its short period, like total stance, it increases in duration with mild to moderate lameness caused by navicular disease. Changes in breakover duration have been studied more extensively in association with changes in trimming and shoeing.

For a discussion of the biomechanics of trimming and shoeing, see Chapter 27.

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CHAPTER • 27

The Foot and Shoeing

FOOT BALANCE, CONFORMATION, AND LAMENESS

• Andrew Parks

Athletic injury usually results from imposition of repetitive stresses that exceed the capacity of the tissues. The magnitude of stresses and hence the likelihood of injury frequently depend on balance and conformation. Therefore balance and conformation are extremely important in maintaining optimal limb function and limiting athletic injury.

Conformation describes shape, in this case, the shape of the distal equine limb and conveys the size and relative proportions of the limb. Balance embraces both the conformation and function of the hoof, conformation because it describes the shape of the hoof, and function because it describes the way the hoof relates to the skeletal structures of the limb and the ground at rest and at exercise. Balance is divided into geometric (static) balance and functional (dynamic) balance.

Balance and conformation are both three-dimensional concepts. Balance usually is divided into three planes: frontal, sagittal, and transverse. Balance in the frontal plane is called *mediolateral balance* and in the sagittal plane is called *dorsopalmar balance*.

To understand how balance and conformation affect stresses that cause injury it is necessary to consider the function of the distal limb and then examine how it changes with conformation and balance. Therefore consideration must be given to the musculoskeletal system, the hoof and the ground, and the interfaces. There are some substantial differences between the front and hind feet. Because almost all research and documented clinical observation is related to the front feet, all discussion in this chapter refers to the front feet unless specifically stated otherwise.

The hoof is the interface between the musculoskeletal system and the ground. The hoof functions both as an extension of the distal phalanx, as a lever about the distal interphalangeal joint, and as an entity in itself. As part of the integument the hoof behaves differently to the structures of the musculoskeletal system, both in its manner of constant growth and its biomechanical properties. As the hoof capsule is constantly worn at the ground surface, it is replaced by the germinal epithelium of the coronary band and the sole. In nature and in an appropriately trimmed foot, there is an approximate balance between growth and loss of the hoof capsule so that the growth rings are parallel.[1-3] The exact mechanism by which hoof growth is regulated is unknown, but several factors are known to influence it: season, inflammation, nutrition, and topical irritants.[4] Growth of the wall also is inversely related to pressure on the coronary band. Hoof wall growth proximal to a hoof wall resection or horizontal grooving of the hoof wall appears accelerated, whereas the immediately adjacent hoof wall growth may be retarded.[5] This effect may be mediated by an effect on the vasculature of the coronary band.

During normal hoof growth the hoof wall migrates distally in relation to the distal phalanx by active separation and reformation of desmosomes as the primary epidermal lamellae move past the secondary epidermal lamellae.[6] The distal growth of the hoof wall under normal loading patterns is approximately even around the circumference of the hoof, and the position of the coronary band in relation to the distal phalanx is static. However, in response to locally increased and decreased loads within the hoof wall, migration of the coronary band proximally and distally in relation to the distal phalanx is superimposed on the normal pattern of hoof wall migration, suggesting that a whole segment of the wall can displace distally and even proximally by movement within the lamellae.

The stiffness of the hoof wall changes radially, and the outer stratum medium is stiffer than the inner stratum medium but is much less stiff than bone throughout. The stiffness of the hoof wall at the toe and quarters is similar, but the difference in thickness indicates that the quarters are more flexible.[7] The hoof wall sustains strains greater than bone, but under normal circumstances it operates within its elastic range at a fraction of its yield capacity.[8] The stiffness of the hoof wall increases with increased strain rate.[9] The hoof wall is viscoelastic. It responds to a rapidly applied force in an elastic deformation so that it returns to its original form rapidly; however, to a slowly applied force, it deforms in a manner so that when the force is removed, it returns to its original form slowly.[10] Because of its physical properties the hoof wall is more fracture resistant than bone,[11] but because it is a much less stiff material than bone, it will bend and shear more readily. The biomechanical properties of the soft tissues between the hoof capsule and distal phalanx are less well understood, but the periosteum of the distal phalanx fails before the junction between the epithelial and dermal lamellae.[8] The lamellar junction is much less stiff than the hoof wall[12]; dorsally the lamellae are oriented perpendicular to the tangent to the hoof wall, but at the quarters they are in a more palmar direction.[13]

FOOT FUNCTION

At Rest

At rest a horse bears approximately 28% to 33% of its body weight on each forelimb. The exact roles of the wall, sole, and frog in weight bearing are undetermined. Weight bearing has traditionally been viewed with a horse on a flat firm surface so that the weight-bearing surface is the full circumference of the wall and the immediately adjacent sole, although the weight is not evenly distributed around the perimeter of the foot. Studies on feral horses indicate that the toe and quarters are worn so that if the horse stood on a flat firm surface, the weight would be transmitted through the wall at both heels and the junction of the toe and quarter biaxially, although there is some variation related to the terrain on which the horse has lived.[14] The dorsal toe and mid-quarters would not bear weight. Domestic horses that were allowed to wear the feet "naturally" at pasture and then stood on different surfaces showed remarkably different loading patterns.[15] When stood on a firm surface, greatest contact was at the medial and lateral heels and just medial and lateral to the dorsal toe, comparable to feral horses. When stood on sand, the greatest

contact was with the central sole, and the total contact area was approximately four times greater for horses standing on sand than on a flat firm surface.

Any part of the ground surface of the foot is potentially weight bearing. Each point of contact that bears weight transmits that force to the ground, although the pressure at each point varies. The sum of all the forces from all points of contact is called the ground reaction force (GRF). It is represented as a vector, with a magnitude and direction, and a location or point of force, which is also called *the point of zero moment*. In the stationary horse this force is vertical and located slightly medial to the dorsal third of the frog. However, in the naturally worn hooves of feral horses the distance between the toe and the point of force is likely to be reduced because of the greater wear at the toe. In either case, the GRF is dorsal to the center of rotation of the distal interphalangeal joint with a resultant moment about the joint. This moment is opposed by an opposite moment created by tension in the deep digital flexor tendon (DDFT). The GRF acting through the phalanges creates a moment about the metacarpophalangeal joint that is opposed by tension in the digital flexor tendons and the suspensory ligament (SL).

At Exercise

Stride Phases

Initial contact At exercise the stride is divided into several phases: initial contact, impact, stance, breakover, and flight.[16] The foot should move in a sagittal plane parallel to the longitudinal axis of the horse.[5] In an exercising horse the GRF changes in magnitude, point of force, and direction with and within the phases of the stride. The GRF is separated into components in three axes: *X* (mediolateral), *Y* (craniocaudal), and *Z* (vertical). At a walk, trot, or gallop the initial contact most frequently is heel first,[17,18] either one heel (usually lateral) before the other or both heels simultaneously.[19] However, some horses may land flat-footed, and the propensity to do so increases with increasing speed.[19] When the heels do strike first, the foot is flat within 1% to 2% of the stride duration.[20] Toe-first landing is rare.[19,20] It has been suggested that the position of the foot at landing is determined by proprioceptive reflexes that optimally orient the position of the distal phalanx before impact, regardless of the length of toe or angle of the foot.[21]

Impact phase The impact phase is characterized by oscillations in the GRF centered on the heels that last for approximately 50 ms.[16] The oscillations are associated with the highest rate of loading during the stride; thus the greatest likelihood of injury is during the impact phase. The vertical velocity and acceleration are greater in the forelimbs than in the hindlimbs, which explains the greater concussion and likelihood of lameness in the fore limbs.[20] Significant damping of the impact oscillations occurs within the hoof, the two distalmost phalanges, and the associated articulations.[22-24]

Stance phase The stance phase extends from the end of impact until the onset of breakover. At a walk the vertical GRF is biphasic, with peaks either side of the middle of the stride, but at the trot there is a solitary peak approximately halfway through the stride.[25,26] For most of the stance phase the GRF is slightly medial to the dorsal third of the frog.[27] The force is absorbed and energy is stored by the flexor tendons and SL as the metacarpophalangeal joint extends[28] so that the maximal GRF coincides with maximal extension of the metacarpophalangeal joint.[17,29] At the walk, forces in the superficial digital flexor tendon (SDFT) and DDFT peak before the peak in the GRF, but the force in the accessory ligament of the DDFT (ALDDFT) peaks during the second half of the stride as the distal interphalangeal and metacarpophalangeal joints extend.[30,31] The GRF in the dorsopalmar direction is negative during the first half of the stride as the limb decelerates. The foot continues to slide forward after initial impact until arrested, at 6% of the stride duration in the forelimb[32] and 23% in the hindlimb[33] in trotters on a dirt track. The fore foot bounces more on impact, whereas the hind foot slides more.[20] During the second half of the stride the horizontal GRF becomes positive as the limb accelerates to provide propulsion. The balance of propulsion and retardation is such that the forelimbs contribute more to retardation and the hindlimbs to propulsion.[26] Faster gaits create higher GRFs and greater strains in the hoof wall,[8] DDFT, SDFT, and SL.[34] Under in vitro loading conditions, hoof wall strains increase with load as strain field epicenters develop around the circumference of the hoof, at the junction of the middle and distal thirds of the hoof, regardless of load.[35]

As the distal phalanx is loaded during the stance phase, it descends within the hoof capsule and the palmar processes rotate ventrally.[36] The sole flattens and spreads as the heels expand,[2,37] more so distally than proximally.[8] At the same time, the dorsal hoof wall flattens and rotates palmarly to parallel movement of the distal phalanx. Frog contact with the ground during exercise appears to be variable,[24,37] but frog pressure does not appear to play a role in hoof expansion,[24] although it must function as an effective expansion joint to permit movement of the sole ventrally and the heels abaxially. It is hypothesized that the digital cushion, in conjunction with the cartilages of the foot, participates in dissipating energy during impact through a hydrodynamic mechanism.[38,39] The distal interphalangeal joint passively flexes[40] and the metacarpophalangeal joint extends. During the second half of the stride the distal interphalangeal joint extends[40] and the tension in the ALDDFT increases.[31] Tension in the collateral sesamoidean and distal sesamoidean impar ligaments also increases,[40] and pressure on the navicular bone increases.[41] The metacarpophalangeal joint flexes. The point of action of the GRF moves toward the toe toward the end of the stance phase.[16]

Breakover phase Breakover begins when the heels start to lift off the ground and ends when the toe leaves the ground. The point of breakover is the most dorsal part of the hoof or shoe in contact with the ground as the heel begins to lift off the ground. From the instant the heels and sole have left the ground, the GRF is concentrated at the toe. Tension in the ALDDFT peaks,[30] and increased strain in the dorsal hoof wall[8] causes the distance between the heels to be narrower than at rest. The horizontal forces between the ground and the hoof at the toe during the latter part of the stance phase and breakover are associated with the final stages of propulsion.

Flight phase The flight phase begins at maximal retraction of the limb and the foot reaches maximal height soon thereafter. A second peak in height occurs just before maximal protraction. The limb retracts slightly before impact to decelerate the limb as the distal phalanx is optimally aligned for impact. The deceleration of the forward movement of the foot is important in reducing the stresses of impact.[42] The stresses on the distal limb during the flight phase are low, because the distal joints flex and extend passively following movement of the upper limb during protraction.[16]

Stride Characteristics

The time and motion characteristics of the stride are important in determining the animation qualities of the gait and the speed of the horse. Higher movement of the foot and greater flexion and extension of the joints represent greater animation. Long retraction with a high starting point is considered desirable, and longer strides are associated with greater speed. Maximum stride frequency is inversely related to speed index.[43-45]

The ground surface affects the angle of the hoof to the ground during the stride, the duration of the stride, and the absorption of impact energy. On a flat, firm surface the plane

of the hoof is the same as that of the ground, but on a surface such as sand, the angle of the hoof with the ground increases gradually during the stance phase of the stride.[34] This rotates the plane of the sole so that it is more perpendicular to the vector of the GRF, which appears to aid traction and propulsion. Ground footing has been divided into three types: dense hard surfaces, surfaces with friction damping such as sand, and structural damping surfaces such as wood chips.[46] Friction damping occurs through displacement of small particles, whereas structural damping occurs through viscoelasticity of the particles. The duration of impact oscillations is related to the hardness of the surface. Harder surfaces are associated with a longer duration of impact oscillations than soft surfaces and less energy absorption.[47] A loose cushion on the surface reduces the peak impact force.[48] Racetracks with a hard surface result in faster race times, but horses are more likely to suffer injury associated with the increased energy of impact.[48] Ground footing also affects stride and swing duration, which are both longer on an elastic, softer surface than a hard, firm surface.[49] At a walk, strain in the DDFT and ALDDFT is lower on sand than on a hard, flat surface,[34] such that it resembles the effect of a heel wedge.

OPTIMUM BALANCE AND CONFORMATION

Optimum function should intuitively demand optimum conformation and balance. Practitioners have inherited many empirical notions that often are based on what types of conformation and balance do not work and cause problems. Generally these ideas predate modern motion analysis and therefore relate to conformation and static balance. Modern techniques that allow dynamic evaluation of function have supplemented and sometimes contradicted the geometric approach.

Static Balance and Conformation

Viewed from the lateral aspect, the foot-pastern axis should be straight; that is, the dorsal hoof wall should be parallel to the dorsal surface of the pastern, and the angle of the heel should approximate that of the dorsal hoof wall (Fig. 27-1). The angle of the dorsal hoof wall and the foot-pastern axis to the ground is variable, but it frequently is cited between 50° and 54° in forelimbs and approximately 3° steeper in the hindlimbs.[50] Other studies report higher or lower means and variable relationship between the angles of the dorsal hoof wall of the forelimbs and hindlimbs.[51,52] In domestic horses the length of the hoof wall has been approximately linked to the weight of the horse (7.6 cm for 360 to 400 kg; 9.3 cm for 430 to 480 kg; 8.9 cm for 520 to 570 kg).[50] In feral horses the toe length ranges from 6.7 to 8.9 cm[14,51] but is independent of weight.[51] In domestic horses with either trimmed or shod hooves, the length of the heel should be approximately one third that of the toe,[2,52] but in feral horses it varies with the terrain.[14] An imaginary line that bisects the third metacarpal bone (McIII) should intersect the ground at the most palmar aspect of the weight-bearing surface of the heels.[53,54]

On a lateral radiographic view the dorsal hoof wall should be 14 to 20 mm thick depending on breed[55-57] and parallel to the dorsal surface of the distal phalanx. The angle made by the distal solar border of the distal phalanx with the ground ranges from 2° to 10°.[55,57,58] The center of rotation of the distal interphalangeal joint should be vertically above the center of the ground surface of the foot.[58,59]

Viewed from the dorsal aspect, a line bisecting the metacarpal region should bisect the phalanges and foot, so that the foot is approximately symmetrical, including the mass of foot on either side of the line, the heights and angles of the walls.[1,3,54,58] The medial quarter is frequently steeper so that the medial wall is shorter than the lateral wall.[2,60] A line drawn between any two comparable points on the coronary band should be parallel to the ground, and a vertical line bisecting the McIII bone should be perpendicular to a line drawn across the coronary band or the ground surface of the foot[61] (Fig. 27-2).

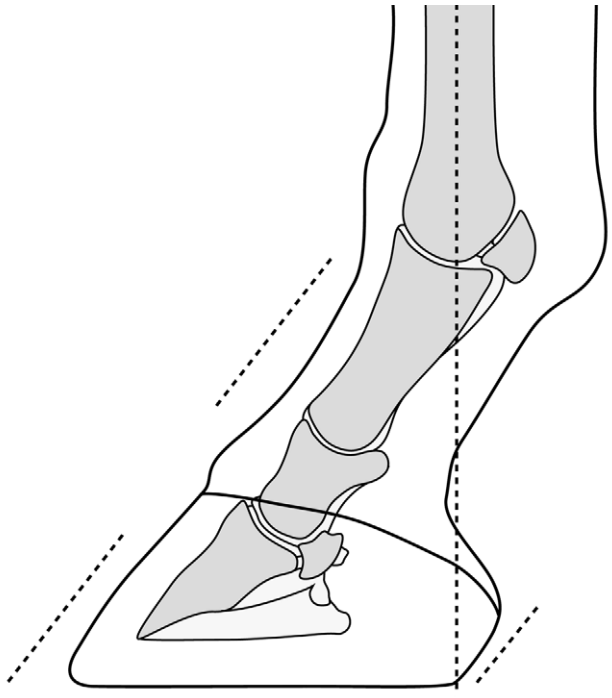

Fig. 27-1 Traditional guidelines defining normal dorsopalmar static or geometric balance. The dorsal hoof wall should be parallel to the dorsal aspect of the pastern and to the hoof wall at the heels. A line bisecting the third metacarpal bone should reach the ground at the weight-bearing parts of the heel.

On a dorsopalmar radiographic view the center of the distal interphalangeal joint should be centered over the ground surface of the foot. The articular surface and the distal border of the distal phalanx should be parallel to the ground, and the interphalangeal joint spaces should be symmetrical.

Viewed from the ground surface the width and length of the hoof capsule of the fore foot should be approximately equal, although it may be slightly wider than it is long.[51,60] The hind foot is invariably slightly longer than it is wide. The point of breakover is best assessed from the ground surface and should be located at the center of the toe. The ideal location for breakover in the dorsopalmar axis is disputed. In a traditionally trimmed and shod horse, breakover is positioned where the line of the dorsal hoof wall intersects the ground, although it may ideally be located more palmarly. Using the position of the hoof wall as the reference point, breakover is between the dorsal margin of the hoof and the white line.[62] Alternatively, breakover is 2.5 to 3.8 cm dorsal to the apex of the frog or 0.6 cm dorsal to the dorsal margin of the distal phalanx.[14,63] The relationship of the longitudinal axis of the frog to the underlying distal phalanx is relatively constant compared with the rest of the ground surface of the hoof. The medial and lateral aspects of the ground surface of the foot are symmetrical about the central axis of the frog,[3,54,62] although slight asymmetry, the lateral sole being about 5% wider than the medial sole, may be beneficial.[60] The latter is compatible with an even coronary band and a steeper medial wall. The size of the foot should be proportional to the weight of the horse.[51,52] The sole should be concave. The frog width should

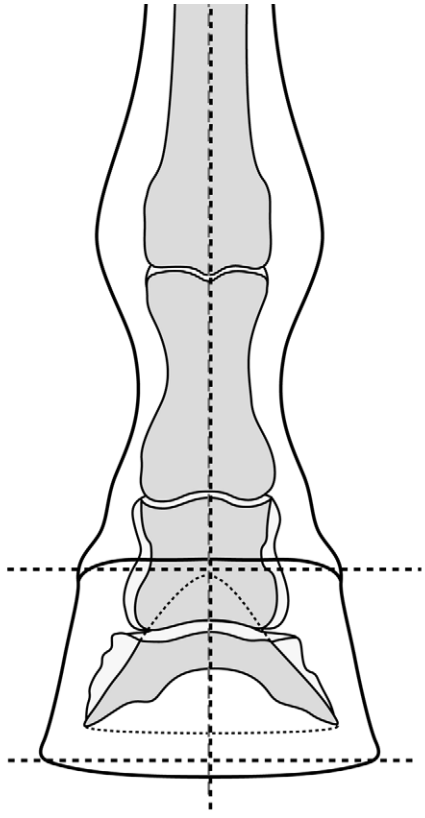

Fig. 27-2 Traditional guidelines defining normal mediolateral static or geometric balance. A single line should bisect the metacarpal region and phalanges. A line drawn across any two comparable points on the coronary band, or on the weight-bearing surface of the foot, should be perpendicular to the axis of the metacarpal region.

be at least 50% to 67% of frog length,[52,60] and the weight-bearing surface of the heels should coincide with the widest part of the frog.[52]

Dynamic Balance

Current definitions of dynamic balance describe the placement of the foot at initial impact. The foot is said to be in dynamic mediolateral balance when both heels contact the ground simultaneously[3,61] and breakover occurs at the center of the toe.[61] The foot is said to be in dynamic dorsopalmar balance when either the heels land slightly before the toe, or the toe and the heels contact simultaneously.[3] However, both these observations are a function of observation frequency. The fewer observations made per unit time, the more likely the foot is to appear in balance. The more frequently the observations are made, the less likely the foot is to appear in dynamic balance. By increasing frequency of observation, it appears that one heel commonly lands before the other.[19] It is likely that the scope of dynamic balance will expand in the future to incorporate the magnitude, location and direction of the GRF, distribution of stresses within the hoof capsule during the stride, and dynamics of breakover.

IMBALANCE AND POOR CONFORMATION

Balance and conformation cannot be considered in isolation, because poor conformation predisposes a horse to developing imbalance, and an imbalanced foot may cause a horse to stand as if it has poor conformation.[1] The effects of imbalance have been examined by experimentally inducing imbalance and by clinical observation. To understand the effects of imbalance it is simplest to consider the consequences of deliberately imbalancing the foot. Poor conformation cannot be altered under experimental conditions, and its effects must be assessed by comparison between horses with different conformation.

Mediolateral

Mediolateral imbalance is caused by either poor trimming of a horse with good conformation or poor conformation causing excessive stress on one side of the foot so that it grows slower than the other side. Inappropriate shoe placement can promote imbalance. If a shoe is set too much to one side or the other, or if the shoe is rotated so that the shoe covers less of one heel than the other, it alters the mediolateral stress on the hoof capsule.[64] A single heel calk causes elevation of one heel; the foot tilts and rotates and may contribute to interference.[64]

Mediolateral imbalance can be induced by applying a wedge pad to elevate the medial or lateral side of the foot or trimming the foot unevenly. The coronary band is no longer parallel to the ground, or perpendicular to the sagittal axis of the limb. The horn tubules of the dorsal hoof wall are no longer oriented in the sagittal plane. Dorsopalmar radiographic views demonstrate that the distal interphalangeal joint space is narrower on the side of hoof elevation and the middle phalanx slides to the lower side.[40] In addition, the condyle of the middle phalanx on the elevated side of the foot moves palmarly, in effect causing the distal phalanx to rotate on the middle phalanx so that the dorsal margin of the distal phalanx rotates away from the elevated side[40] (Fig. 27-3), but the horse has the appearance of being toed toward the elevated side.[1]

The GRF shifts toward the elevated wall.[65] In foals, compressive strains were immediately increased in the lateral cortex of McIII and decreased in the medial cortex by elevation of the lateral wall with a wedge.[66]

The immediate dynamic effects of mediolateral imbalance result in a greater frequency of mediolateral asymmetrical footfall, the lengthened side landing first.[19] The location of the GRF displaces abaxially toward the lengthened side of the foot.[19,67,68]

Prolonged mediolateral imbalance affects the relationship between the hoof capsule and the distal phalanx, causes distortion of the hoof capsule, and alters hoof wall growth.[69] If a foot is trimmed unevenly, so that one wall is longer than the other, the longer wall grows more slowly than the shorter wall. The longer wall develops a flare and the shorter wall becomes underrun.[1,69] In more severely affected horses the coronary band bulges abaxially to create a lip at the proximal margin of the hoof wall on the elongated side. The horse breaks over on the shorter side of the toe. It is my impression that the solar margin of the distal phalanx partially realigns with the ground surface, either because the wall actually migrates proximally or because distal movement of the hoof wall is inhibited, resulting in a net proximal displacement of the coronary band in relation to the distal phalanx. Ultimately, prolonged mediolateral imbalance causes remodeling of the phalanges because of redistributed stresses according to Wolff's law.

Rotational deformities and angular deformities may mimic each other. In most rotational deformities, it appears as if the metacarpophalangeal joint is the most proximal joint to be rotated, but it is not uncommon for the carpus to appear rotated. With rotational deformities, the McIII is vertical and the observer can rotate around the limb until the metacarpal and phalangeal axis are correctly aligned unless the rotation occurs within the phalangeal axis. In horses with angular deformities that occur proximal to the fetlock, including base-wide or base-narrow conformation, McIII may not be vertical

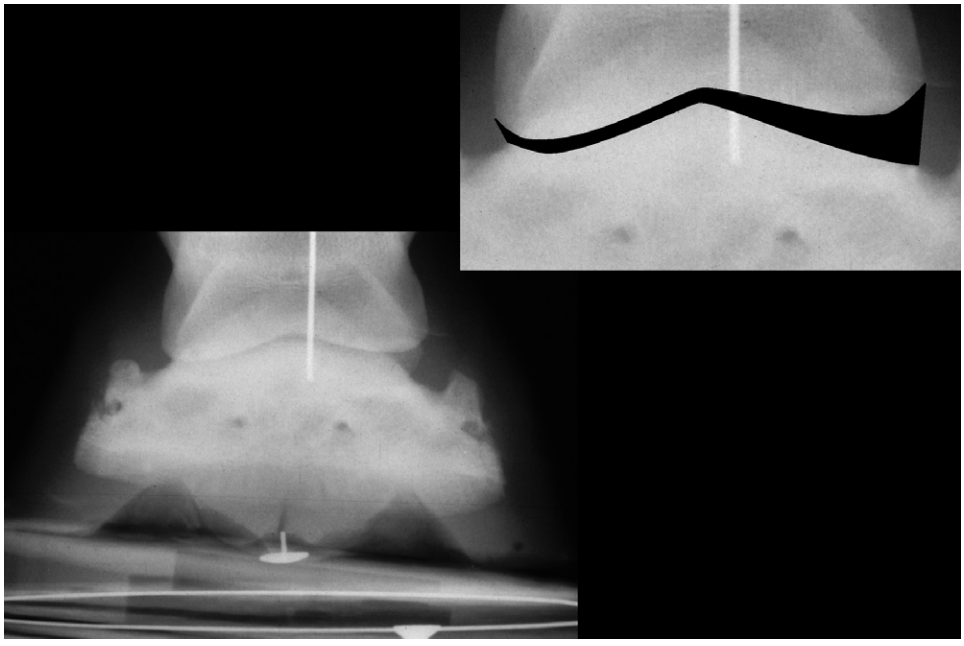

Fig. 27-3 Mediolateral imbalance causes misalignment of the articular surfaces and rotation of phalanges in relation to each other. Deliberate elevation of one wall causes the articular surface of the distal phalanx to tilt, but it does not tilt as much as the ground surface of the foot, indicating that there is accommodation within the viscoelastic structures of the foot. The distal articular surface of the middle phalanx is displaced away from the elevated side of the foot. The inset in the upper right corner shows the joint space (outlined in black) on the elevated side of the foot *(left)* is narrower than the lower side *(right)* of the foot, caused by compression of the distal interphalangeal joint surface on the left side.

and there may not be a view point from which the metacarpal and phalangeal axis are correctly aligned.

Rotational deformities, such as toe-in and toe-out conformation, alter the position of the ground surface of the foot in relation to the mid-sagittal vertical axis of the limb. Toe-in conformation causes the foot to wing out during the flight phase of the stride,[1] and if the limb does not deviate from a vertical axis, the foot lands on the lateral heel quarter and breaks over at the lateral toe. Toe-out conformation causes the foot to wing in during the flight phase of the stride. The foot lands most frequently on the later heal quarter, as with toe-in conformation. Breakover is reported to occur at the medial toe, but it is less consistently directed than breakover in horses with toe-in conformation. Angular deformities, including base-wide, base-narrow conformation, and varus and valgus deformities also alter the position of the ground surface of the foot in relation to the ideal vertical axis of the limb. If an angular deformity (e.g., valgus) causes the foot to be lateral to a plumb line dropped from the shoulder, the stresses on the medial hoof are increased, the medial wall growth decreases as the lateral wall growth increases, and the medial wall becomes underrun as the lateral wall develops a flare. With varus deformity the opposite is true because the limb deviates medially. Rotational and angular deformities may occur in combination, complicating the picture.

Dorsopalmar

Dorsopalmar imbalance has several causes. A broken-back foot-pastern axis may follow poor trimming, either leaving the toe too long or trimming the heels too short. A shod horse wears its heels against the shoe while the toe wears very little, so the hoof angle changes up to 3° over 8 weeks.[2] This change must be allowed for by trimming the toe slightly more than the heels, because even trimming around the foot causes a gradual decrease in the hoof angle.[3] Using too small a shoe or leaving a shoe on too long causes the palmar ground contact to move dorsally, imposing greater stresses on the heels, which then are prone to collapse.[61,64] Toe grabs and heel calks alter the foot axis and concentrate stress.[64] Thoroughbred horses may have a genetic predisposition for a broken back foot-pastern axis.

At rest, elevation of the heel causes the distal interphalangeal and pastern joints to flex and the metacarpophalangeal joint to extend.[70] The effect is greatest at the distal interphalangeal joint and least at the metacarpophalangeal joint.[70] In vitro, elevation of the heels with a wedge decreases the strain in the DDFT, the extensor branches of the SL, and medial hoof wall[71] and decreases the moment about the center of rotation of the distal interphalangeal joint.[72] The horizontal distance between the center of rotation of the distal interphalangeal joint and the toe decreases with heel elevation. In addition, the horizontal distance between the toe and a imaginary vertical line bisecting McIII dropped to the ground is decreased. This decreases stresses on the heels. Decreasing the hoof wall angle increases the stress on the heels.[2]

Lowering the heel or raising/lengthening the toe to create an acute hoof angle or long toe increases the likelihood of toe-first landing.[19,21] Elevating the heels increases the likelihood of heel-first landing.[19] The overall impulse (force × time) on the foot is least when the foot-pastern axis is straight, indicating that a straight foot-pastern axis is least injurious to the foot.[19] At a walk, elevating the heels decreases the strain in the DDFT and its AL[31,72] with little effect on the SDFT and SL. The decreased strain in the DDFT is reflected in decreased pressure on the navicular bone.[41]

Elevating the toe results in a marked increase in strain in the ALDDFT and a lesser increase in strain in the DDFT at the end of the stride as a result of increased extension of the distal interphalangeal joint.[31] Strains in the SDFT and SL are either reduced or unchanged.[31,72] Horses with small hoof wall angulation have a prolonged breakover, but the length of stride,

duration of the stance, and swing phases are unchanged.[19,21] When hind feet are trimmed with more acute hoof wall angulation, breakover is delayed but the timing of impact is unchanged as normal coordination is restored during the swing phase of the stride. There is an increase in over-reach distance, the distance between the print of the front foot and the landing point of the hind foot.[73] Heel wedges delay the dorsal shift in the GRF and decrease the maximum torque about the distal interphalangeal joint during the second half of the stride. Toe wedges have an opposite effect.[31,68] However, neither toe nor heel wedges alter the dorsopalmar position of the point of force during mid-stance of the stride, indicating that the heels are not unloaded. Both toe and heel wedges cause medial displacement of the point of force.[68] Increasing the length of the toe prolongs breakover but does not alter stride length; however, it increases maximal flexion of the metacarpophalangeal joint during the swing phase.[74,75]

The position of breakover in the sagittal plane appears to influence the angle of the dorsal hoof wall and the distal phalanx. Moving the point of breakover palmarly from the most dorsal margin of the hoof wall increases the angle of the dorsal hoof wall and the ground and increases the alignment between the middle and distal phalanges.[63,76] Whether this effect is related to the biomechanical properties of the dorsal hoof wall or relief of pain within the foot is undetermined. The effect of increased hoof angle on hoof wall strain is inconsistent. In an in vitro model, hoof wall strain did not change with increased hoof wall angle.[71] In contrast, in an in vivo experiment, increased hoof wall angle increased hoof wall strain more at the lateral quarter than at the toe and not at all at the medial quarter.[77]

The effect of pastern length and the angle of the foot-pastern axis are less well established. The angle of the hoof-pastern axis to the ground is a feature of a horse's conformation. It cannot be changed experimentally, but comparing horses with different conformations shows that the point of force in horses with a small hoof-pastern axis angle is more palmarly positioned than in horses with a larger axis angle.[27]

Prolonged dorsopalmar imbalance also has delayed effects because of the nature and growth of the hoof capsule. In barefoot horses, leaving the toe long causes the toe to grow faster than a short, trimmed toe. The hoof walls and frog become narrower, and the shape of the ground surface tends to skew away from a circular shape.[78] The length of the frog does not change. Clinically, the same appears to occur in shod horses. In horses with extremely long toes the foot becomes "hoof bound." The heels of long-toed horses are predisposed to become underrun because the heels bend dorsally. Both of these phenomena are seen in Tennessee Walking Horses or American Saddlebreds intentionally shod with long hooves.

Other Forms of Imbalance

Many horses with imbalanced feet have a combination of mediolateral and dorsopalmar imbalance. For example, a foot with a sheared heel has one heel longer than the other, which is frequently associated with a flared toe quarter on the opposite side of the foot.[79] Diagonal imbalance has been described dynamically. The hoof lands on one corner of the hoof capsule and then loads the diagonal corner, with consequent distortion of normal hoof capsule shape and alignment with the rest of the distal limb.[80] Other local deformations of the hoof wall occur, either uniaxially or symmetrically, that do not fit the classical description.

IMBALANCE AND POOR CONFORMATION AS A CAUSE OF LAMENESS

Poor conformation and imbalance of the distal limb are common, as is lameness that can be isolated to the distal limb. However, demonstrating the correlation is not always straightforward. In some horses an obvious disease process and obvious imbalance co-exist, and when the imbalance is treated the lameness improves. In other lame horses, imbalance is evident with no other clinical, radiographic, or scintigraphic evidence of disease, and treating the imbalance also improves the lameness. In yet other lame horses, there is evidence of imbalance, with or without other evidence of disease, but treating the imbalance does not improve the lameness. To my knowledge, only one study thoroughly investigated the effects of hoof balance on injury. The odds of catastrophic musculoskeletal injury and suspensory apparatus failure were lower when the lateral sole area was greater than the medial sole area.[60] Suspensory apparatus failure was more likely the greater the difference between the angles of the dorsal hoof wall and the heels. McIII condylar fractures were less likely with a steeper toe angle.

Mediolateral imbalance is associated with a shift in the point of force of the GRF, distortion of the hoof capsule, induced asymmetry of the articulations of the distal limb, and rotation of the distal interphalangeal joint. With increased compressive stresses the following problems are clinically presumed to follow imbalance: sub-solar bruising, hemorrhage in the white line from laminar tearing, pain from shearing heels, quarter or heel cracks, thrush in narrow frogs, pedal osteitis, fractures of the palmar process of the distal phalanx, sidebone, synovitis, and osteoarthritis, and more proximal fractures[1,54,61,64]

The effects of dorsopalmar imbalance should be separated into the effects of broken-forward and broken-back foot-pastern axes. A broken-back foot-pastern axis increases the load on the palmar aspect of the foot during weight bearing and increases the stresses in the toe at breakover. It causes hyperextension of the distal interphalangeal joint and increases the tension in DDFT and pressure on the navicular bone. Therefore it can be expected to be associated with heel bruising, lamellar tearing at the toe, pedal osteitis of the palmar processes, navicular disease, tenopathy at the insertion of the DDFT, and more proximal injuries of the tendons or suspensory apparatus.[3,54,61,64] In the hindlimb a broken-back foot-pastern axis appears to be associated with tarsal and back pain.

A broken-forward foot-pastern axis (upright) appears to be less pernicious.[3,54] It increases the load on the dorsal half of the foot and decreases tension in the DDFT. The principal findings are sub-solar bruising distal to the dorsal distal margin of the distal phalanx and subsequent osteitis of the distal phalanx.[54]

An upright foot-pastern axis has traditionally been considered to predispose toward concussive injuries of the weight-bearing structures in the limb, whereas a foot-pastern axis with an acute angle to the ground predisposes toward strains and sprains of the flexor apparatus. Similarly, a long pastern has been considered to predispose toward strains and sprains.

Any angular deformity more proximal in the limb that increases the mediolateral symmetrical loading of the foot can be expected to have effects similar to mediolateral imbalance. Rotational deformities do not seem to be a frequent cause of problems. Toe-out conformation is more likely to cause interfererence but anecdotally, toe-in conformation is considered more likely to cause lameness.

Undoubtedly, technological advances in diagnostic imaging and pain localization will lead to identification of other combinations of disease and structure. Epidemiological studies will confirm the relationships between the different features of poor conformation and imbalance on disease.

CLINICAL IDENTIFICATION OF HOOF IMBALANCE

The horse must be observed while it is standing squarely to assess each limb in relation to the whole and in motion on a flat, level surface. Each foot must be examined on and off the ground.

Mediolateral Imbalance

Visual inspection should note the position of the entire limbs to identify angular or rotational deviations more proximally in the limb that may have repercussions for the foot (Fig. 27-4). Visual inspection of the foot on the ground should note rotational deformities of the metacarpophalangeal joint and placement of the foot in relation to the sagittal axis of the limb. The hoof capsule should be inspected closely for asymmetry of the coronary band. This is frequently a strictly visual inspection, but graphing the height of corresponding medial and lateral points on the coronary band provides objectivity that can highlight an imbalance and provide a record for future comparison.[52,81] The medial and lateral walls should be inspected for flares and evidence of underrun heels, lipping at the coronary band, and even spacing between the growth rings.

The ground surface of the foot reflects changes elsewhere in the hoof capsule. The foot should be approximately symmetrical about the center of the frog. In mediolateral imbalance, the sole may appear wider on the side with a flare in the wall and narrower on the side with an underrun wall. Dorsal displacement of the ground surface of one heel in relation to the other accompanies proximal displacement of the coronary band at the heel commonly associated with sheared heels. Wear of the shoe or wall at the toe indicates the point of breakover. Alternatively, the breakover point may be identified by lifting the antebrachium cranially, allowing the metacarpal region and pastern to hang passively, and then lowering the foot; the point of breakover is the first part of the foot to touch the ground.[54] Asymmetrical breakover may indicate mediolateral imbalance, but it also may follow angular or rotational deformities of the limb. Asymmetrical bruising adjacent to the wall at either quarter may signify excessive concussion caused by mediolateral imbalance or laminar tearing in a wall with a flare.

Examination of the distal limb for rotational or angular conformation by viewing the limb on the ground from the dorsal aspect may be misleading because it is influenced by weight bearing. The ground surface of the foot automatically aligns with the surface of the ground regardless of the relative lengths of the medial and lateral hoof wall. This causes secondary rotation within the phalangeal axis. To circumvent this rotation the limb can be examined off the ground by lifting the foot in the same way described to find the point of breakover; the angulation or rotation within the distal limb is observed by sighting down the metacarpal region, pastern, and hoof.[54]

The traditional way to assess mediolateral balance is to sight across the ground surface of the foot with the leg off the ground, holding the proximal metacarpal region and allowing the digit to hang downward in the sagittal plane with the metacarpophalangeal and interphalangeal joints in passive extension. A line drawn across any two corresponding points on the circumference of the ground surface of the wall should be perpendicular to the axis of the limb as judged by the metacarpal region. If the limb is perfectly symmetrical about the axis of the limb, without angular or rotational deformities, and the observer is directly above the limb, this technique is probably satisfactory within the limits of the observer. I question the accuracy of this technique because most distal limbs are not symmetrical but have at least some element of rotation about the metacarpophalangeal joint. A smaller number of horses have true angular deformities at the metacarpophalangeal joint. For reliability, there must be consistency in the

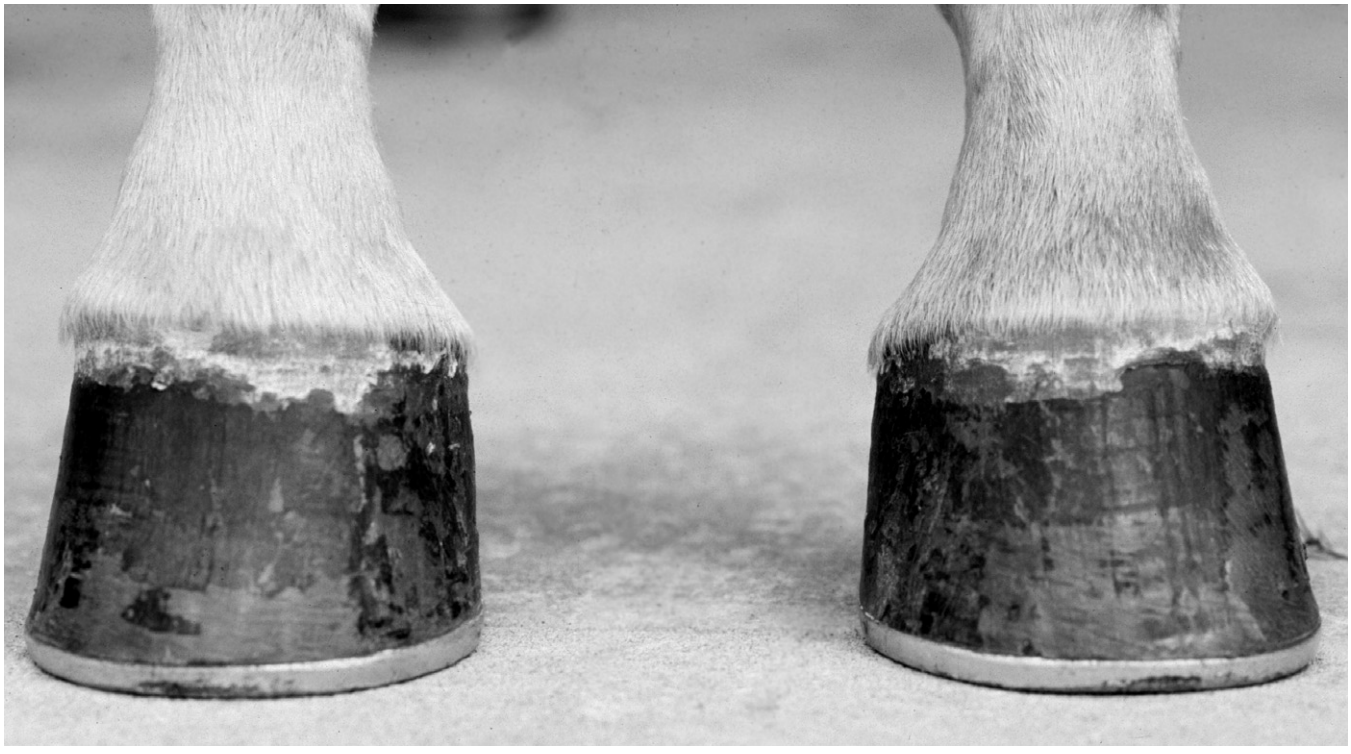

Fig. 27-4 Feet of a yearling with bilateral mediolateral static imbalance. The lateral wall of both front feet reaches higher than the medial wall so that the lateral coronary band is higher than the medial coronary band; consequently, the dorsal coronary band is sloping distally and medially. The growth rings are also tilted in the same direction as the coronary band. The imbalance creates the impression that the pastern is no longer centered in the foot but is displaced laterally.

extension of the metacarpophalangeal joint and in the position of the observer. T-squares have been used to improve the reliability of this observation,[4,54] but misalignment of the T-square with the axis of the metacarpal region decreases accuracy.

Dynamic mediolateral balance is assessed by observing the horse from in front and from behind at a walk and at a trot. Because the degree to which symmetry of landing can be detected is a function of the frequency of observation and speed of the horse, only more severe imbalances can be detected at a trot compared with a walk, and more subtle differences in timing remain undetected unless video recorders or more sophisticated measuring equipment is used. During the flight phase of the stride, movement of the foot, phalangeal axis, and more proximal limb is observed in relation to the plane of travel to correlate with previously noted rotational and angular deformities.

A dorsopalmar radiograph is the only means to assess the relationship between the hoof capsule and the phalanges (Fig. 27-5). Overt imbalance can be detected on routine dorsopalmar radiographs, but detection of more subtle changes requires strict technique because apparent radiographic imbalance can be readily induced artificially. Both feet must be weighted equally, because unilateral weight bearing induces mediolateral asymmetry and rotation of the interphalangeal joints.[40] The foot must be allowed to assume its natural orientation to the rest of the limb, only reliably possible if it is placed on a swiveling block. Deformation of the hoof capsule may be induced by rotation within the limb, which may change the angulation of the distal phalanx with the ground.[82] The metacarpal region must be within 10° of vertical in the frontal plane. The x-ray beam must be horizontal and centered on the mid-sagittal plane so that a wire marker centered on the dorsal hoof wall bisects the central sulcus of the frog on the radiographs. Neither toe-in nor toe-out conformation alters the radiographic measurements of mediolateral balance if assessed in this manner.[83,84]

Although mediolateral imbalance can unquestionably cause lameness, the hoof capsule is not the site of pain that is associated with lameness. Rather the pain is associated with the effects of imbalance, that is, stress on the deeper structures of the hoof and the musculoskeletal structures of the distal limb. Therefore it is not surprising that the lameness may improve with perineural or intra-articular analgesia of the distal limb in a similar manner to osteoarthritis of the distal interphalangeal joint or navicular disease.

Dorsopalmar Imbalance

The limb is examined for angulation at the carpus and metacarpophalangeal joint. The foot-pastern axis is visually inspected to determine whether the axis is straight or broken forward or back. This method provides only a rough guide. Visual examination can be improved by using a gauge, one limb of which is aligned with the pastern and one with the dorsal hoof wall. However, deciding exactly what landmarks to use on the pastern for alignment introduces irregularity. In addition, the axis changes as the horse shifts its weight or posture. Similarly, concavity of the dorsal hoof wall in the sagittal plane raises the issue of what to align the pastern with. If the dorsal hoof wall is concave, usually the top third of the hoof wall is the most closely aligned to the dorsal surface of the distal phalanx.

The length of the toe from the proximal coronary band to the ground surface in the mid-sagittal plane of the hoof is readily measured with a tape and compared against reference values or previous measurements for the same horse. This information is greatly under-used. The length and angle of the heel also are evaluated. Only the wall at the heel distal to the bulb should be evaluated, because inclusion of the heel bulb causes the angle to be underestimated. Heels that are angled more acutely to the ground than the dorsal hoof wall are longer than heels that are parallel to the dorsal hoof wall.

A long toe is associated with elongation and narrowing of the ground surface of the foot. The frog width decreases in a comparable manner with the width of the foot. The length of the frog should remain almost constant, so that an increase in the length of the ground surface of the foot is reflected in an

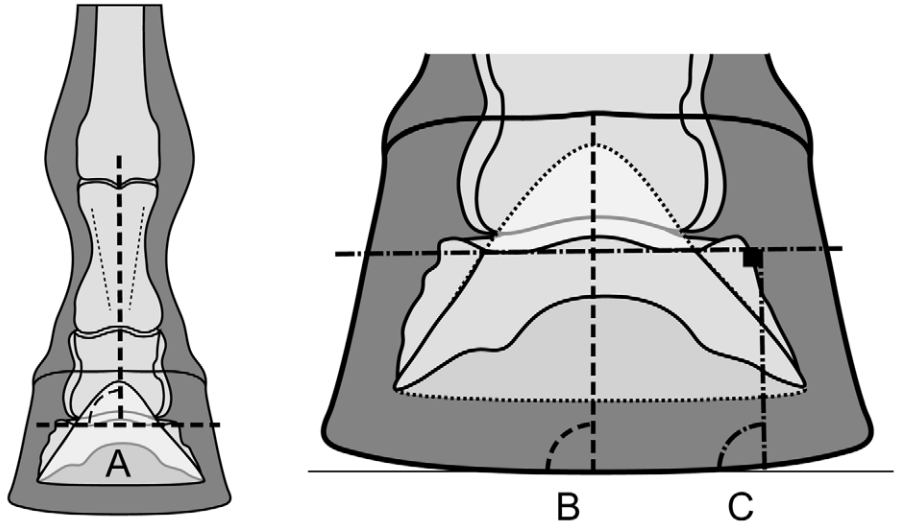

Fig. 27-5 Radiographic assessment of balance has been described by Caudron et al.[84] **A,** Rotation between the phalanges is indicated when a line bisecting the distal phalanx is not perpendicular to the articular surface of the distal phalanx. **B,** A tilt in the axis of the hoof capsule is determined radiographically when a mid-sagittally placed wire marker is not perpendicular to the ground surface of the foot. **C,** Tilting of the distal phalanx is evident when a line drawn perpendicular to a line drawn across the articular surface of the distal phalanx is not perpendicular to the ground. All measurements presuppose that the radiographic beam bisects the foot (the dorsal wire marker bisects the apical process of the distal phalanx and the central sulcus).

increase in the distance between the apex of the frog and the most dorsal aspect of the toe or breakover point. The ground surface of the heels should be adjacent to the base of the frog. If the ground surface of the heels projects dorsally to the base of the frog, the heels are either too long, angled too acutely, or both. Hemorrhage in the white line at the toe caused by lamellar tearing may be a secondary indicator that the toe is too long.

In a horse with a broken-forward foot axis, a flexural deformity of the distal interphalangeal joint must be distinguished from heel contraction secondary to pain. In a foot with a flexural deformity the heels and frog are wide and the ground surface of the foot is triangular, resembling a hind foot. In contrast, a contracted foot has a narrow heel and frog.

The relationships between the individual phalanges and between the phalangeal axis and the hoof are determined radiographically. The phalanges are closest to alignment when the foot-pastern axis is straight. However, the proximal interphalangeal joint usually appears slightly extended, even with a straight foot-pastern axis.[70] The dorsal hoof wall should be parallel to the distal phalanx. The angle of the solar surface of the distal phalanx with the ground and the thickness of the sole, or the distance from the solar surface from the dorsal distal margin of the distal phalanx, should be measured. The center of rotation of the distal interphalangeal joint should be equidistant from the weight-bearing surface of the toe and the heels.

In hind feet, dorsopalmar imbalance associated with hyperextension of the distal interphalangeal joint appears to take a slightly different form compared with the fore feet. The dorsal hoof wall is not necessarily long when viewed from the side. The toe frequently has been dubbed back, and it acquires a marked convexity. Viewed from the ground surface the concavity of the sole is exaggerated and if the foot is shod, the frog lies between the branches of the shoe. This may be from descent of the frog, or proximal displacement of the heels. In my experience, these horses almost invariably are shod.

Evaluation of dynamic dorsopalmar balance suffers from the same limitations as the evaluation of dynamic mediolateral balance. However, gross imbalances can be observed at a walk or trot. Normally, when viewed from the side the foot is expected to land flat or slightly heel first.

TREATMENT OF IMBALANCE AND POOR CONFORMATION

Treatment of imbalance provides at least two challenges. The first is to have a clear objective of what ideal balance is for any given horse because, although there are some hard and fast rules, other areas are unclear. For example, a straight foot-pastern axis is beneficial, but the exact length of toe that is optimal for the same foot is not clear.

The second challenge is interpreting the measured indicators of balance. For example, the measures of mediolateral imbalance, symmetry of the coronary band, sighting along the axis of the metacarpal region and across the ground surface of the foot, footfall, and dorsopalmar radiographs may not agree on whether the foot is imbalanced, the direction of the imbalance, or provide varying degrees of imbalance. Radiography appears to be the most accurate and is substantiated scientifically.[84]

Difficult balance problems may not have easy solutions, and judicious experimentation that can be both time consuming and expensive may be necessary. Balance may be corrected, but long-standing problems may not be correctable. Poor conformation can be compensated for but can never be corrected with trimming or shoeing in an adult horse. The ability to compensate for poor conformation is inversely proportional to the height in the limb from which the defect originates.[5] Major imbalances should be corrected gradually over time, or lameness may result from the correction.[4,54,61]

Rebalancing the foot can be performed by visual assessment of the hoof and foot-pastern axis before and after trimming, repeating until a satisfactory result is obtained. Rebalancing may be facilitated by radiographic assessment of balance. Radiographs, with markers to indicate the angle of the dorsal hoof wall, the location of the apex of the frog, and the position of the coronary band, obtained before the trim has been performed are used as a baseline on which to base the trim. Deformation of the hoof capsule in the pre-treatment radiographs may result in underestimation of the imbalance,[83] and radiographs obtained after trimming may be helpful to assess the accuracy of treatment.

Treatment of imbalance is complicated by the necessity to consider both the immediate and delayed effects. The reversal of the immediate effects of imbalance is the most straightforward, because it requires nothing more than restoring the normal length of the wall around the circumference and ground surface of the foot. The effects of viscoelastic deformation of the hoof wall caused by uneven pressure should correct spontaneously if the force is removed and sufficient time is allowed before the horse is reshod. Other deformation of the hoof wall caused by contraction or aberrant hoof growth may have to wait for new hoof wall to grow from the coronary band to the ground surface after the biomechanical forces have been optimized by trimming and shoeing.

Mediolateral and dorsopalmar imbalance may respond to trimming and allowing the horse to go barefoot.[64] Application of poultices to the foot has been advocated to encourage hoof realignment.[81] This allows the effects of prolonged imbalance from the viscoelasticity of the hoof and misdirected hoof growth to adjust without the influence of the shoe. However, the quality of a wall that is usually shod may deteriorate over several weeks, before it improves, and flat-footed horses may become more lame.

In foals, trimming, shoeing, or surgery corrects poor conformation associated with abnormal angulation in either the sagittal or frontal planes. Conformation in adult horses is fixed, but changing foot balance or shoeing sometimes can compensate for poor conformation. However, when the stresses are realigned to benefit a musculoskeletal structure, they become less than optimally aligned for the hoof capsule and predispose to a different problem. For example, in a horse with a long pastern at a normal angle or with a sloping pastern of normal length, the foot is further dorsal than is ideal. This creates a greater moment about the metacarpophalangeal joint. This can be compensated for in part by extending the heels of a shoe further past the heels of the foot than normal. This technique moves the most palmar ground support palmarly, but it also moves the point of application of the GRF further palmarly, increasing both the stresses on the heels and the propensity to deform. Treatment of poor conformation is almost always a compromise.

Mediolateral Imbalance

Uncomplicated mediolateral imbalance is corrected by decreasing the length of the wall on the side of the foot in which it is longest, or lengthening the wall of the side that is shortest. If the horse has sufficient foot, it is always preferable to correct the imbalance by trimming the longer wall. However, when the hoof is insufficient to permit trimming the longer wall sufficiently to restore the equilibrium, then the shorter wall must be lengthened. A wall can be lengthened in several ways. However, care must be taken because alteration of the balance may actually cause lameness. The simplest method is to add a pad, either a shim or a full wedge

pad. The shim pad is riveted to the branch of the shoe so that it is interposed between the foot and the shoe, or a full wedge pad is positioned so that the thickest portion of the pad is on the shortest side of the foot. Alternatively, the shorter wall can be lengthened by addition of acrylic. This can be allowed to set and a shoe nailed to the hoof and composite, or the shoe can be attached to the foot with the acrylic so that the position of the shoe in relation to the hoof capsule is adjusted as the acrylic dries.[85] A shoe with branches of different thickness can be used. This makes the thinner branch lighter. The width of the web can be increased to compensate for the thinner branch so that the asymmetrical effect of uneven weight distribution is minimized.

Flares and under-run walls that accompany mediolateral imbalance must be addressed by seeing where the hoof wall should be at the ground surface. The longitudinal axis of the frog is a good guide to the location of the center of the ground surface of the foot in the sagittal plane. The shoe should be positioned symmetrically about the frog so that the flare that extends abaxially to the shoe is dressed back and the shoe extends abaxially past the underrun wall.[1] The flare and underrun wall then should correct as new hoof wall grows distally.

The most controversial issue in balancing a foot is restoring mediolateral imbalance in the following circumstances: (1) when the coronary band is asymmetrical with one heel/quarter higher, usually the medial quarter/heel, which is obvious with the horse standing; (2) when the ground surface of the shorter wall appears farthest from the metacarpophalangeal joint when the axis of the limb is sighted with the foot off the ground; and (3) when the shorter wall contacts the ground first at a walk. This is typical with a sheared heel. Should the long wall be shortened, potentially increasing the discrepancy in footfall, or should the long wall be lengthened, exacerbating an already imbalanced appearance? I cannot see any benefit in lengthening an already long wall or determine how this might improve the wall. Radiographic evidence of joint asymmetry may resolve the dichotomy. If this fails, the horse can be shod with a shoe positioned perpendicular to the axis of the limb with the long heel floated and extra support provided by a heart bar shoe. Alternatively, a groove can be cut through the stratum medium immediately distal to the displaced coronary band to encourage it to move distally; this process may need to be repeated.

Compensation for angular or rotational deformities in the limb is seldom attempted because they are not the cause of pain and attempts to compensate for them may increase the asymmetry of stresses imposed on the hoof capsule. However, if an angular limb deformity causes a secondary mediolateral imbalance, this may need correction whenever the horse is trimmed.[61] If a rotational deformity causes one heel to land long before the other, aligning the shoe branches perpendicular to the axis of the metacarpal region rather than the pastern may be beneficial.[54] A medial or lateral extension may be used to extend the ground surface of the foot/shoe under the axis of the limb to improve the distribution of forces in the more proximal limb.[3,54] However, these corrections improve function in one part of the limb at the expense of another. When angular or rotational deformities cause limb interference, it may be necessary to change shoeing to change the flight of the limb to reduce the likelihood of injury.

Dorsopalmar Imbalance

An imbalanced foot usually is related to a toe that is too long or heels that are underrun, less frequently to heels that are too long, and seldom to heels or a toe that is too short. The toe or heels are trimmed until the horse has a straight foot-pastern axis. Since dorsopalmar imbalance starts to develop immediately after shoeing because of disparate growth of the toe and heel, deliberately trimming the foot 1° to 2° broken forward may be useful.

Correction of a broken-back foot-pastern is more complicated after secondary changes have occurred. If excessive toe length has caused the sole to change shape so that it is longer and narrower than normal, the breakover should be brought back to a more natural position. The angle of the dorsal hoof wall should be re-evaluated to determine whether it has improved before other measures are taken. In a barefooted horse the breakover is set by the trim, and in a shod horse by both the position and roll of the toe of the shoe. The foot width should increase naturally in time, and the shoe should be set full at the quarters to accommodate the abaxial movement of the quarters. An extended period may be required to regain the optimal ratio of the length to width of the ground surface of the foot. If underrun heels are a result of a long-toe conformation, the weight-bearing surface of the heels can be moved palmarly (backed up), but the angle of the heels often is too acute to allow alignment of the foot-pastern axis regardless of toe length. This can be corrected immediately by raising the heels. The heels can be raised with a wedge pad, either a full pad or shim, a shoe with wedged heels, or building up the heels with acrylic.[3,86] However, this process increases the pressure on the coronary band at the heels, and may decrease the rate of heel growth. Alternatively, a bar shoe may be used to limit the heels from digging into the ground.[4] Allowing the horse to go barefoot may result in the fastest improvement in conformation, but this precludes the horse working, and flat-footed horses may become more lame. Contraction of the width of foot often accompanies dorsopalmar imbalance, and grooving or thinning the heels has been recommended.[1,62] For long-term soundness, I believe it is preferable to realign the foot-pastern axis as well as possible and then allow wider, straighter heels to grow from the coronary band. This may require time out of work. Raising the heels is frequently the fastest way to return a horse to work; thus the manner of treatment may depend on the immediate athletic demands placed on the horse.

A broken-back hoof-pastern axis in the hind limbs is treated by trimming the toe, using radiography to assess the improvement. Frequently the solar margin of the distal phalanx is tilted by several degrees with the dorsal margin farther from the ground than the palmar processes. In my experience, it may not be initially possible to improve the angle of the solar margin of the distal phalanx beyond parallel to the ground. It frequently is ideal to allow the hind feet to go bare. When the foot must be shod, in contrast to the fore foot, I recommend setting the toe of a rim shoe in line with where the wall at the toe should meet the ground and provide no more coverage of the heels than is necessary.

Treatment of a broken-forward foot-pastern axis caused secondarily from pain or a flexural deformity, by decreasing the dorsal hoof wall angle may exacerbate the lameness. A broken-forward axis from pain must be addressed by treating the primary problem. Correction of a broken-forward foot-pastern axis associated with a flexural deformity is not always necessary, but when it is, it should be accompanied by desmotomy of the ALDDFT.

Compensation for a long pastern or a low foot-pastern axis when the weight-bearing surface of the foot is too far dorsal in relation to the limb may be achieved by extending the heel of the shoe.[81] This correction is directed at reducing the moment about the metacarpophalangeal joint by moving the palmar aspect of the foot/shoe farther palmarly, usually by using an egg bar shoe, but any heel extension that is not likely to result in the shoe being pulled by the hind foot will work. Compensation for an upright conformation is not readily feasible and fortunately seldom necessary.

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HORSESHOES AND SHOEING

• Andrew Parks

An equine veterinarian must be able to assess the way a horse is shod in relation to the current use of the horse and make recommendations for shoeing to improve the function of the distal limb. This necessitates an understanding of the basic construction and fit of a shoe and its use. Determining how a shoe affects the horse's performance requires an understanding of how the elements of a shoe function. To understand how to change the function of the distal limb, it is necessary to know which modifications to form and fit of a shoe can achieve the desired result. It is more important to consider how a basic shoe may be modified to achieve a given result than list a variety of different shoes for specific conditions. Farriery developed as an art guided by individual and collective experience and has recently been supplemented by scientific studies. Some guidelines are derived empirically and others have a scientific basis, but the skill of the individual is a vital third element.

HORSESHOES

Foot Preparation

Shoeing cannot be considered in isolation from foot trimming. The foot is trimmed to remove the excess length of wall that accrues because the distal wall is protected from wear by the shoe. After the foot has been cleaned, the exfoliating sole and ragged margins of the frog are debrided with a hoof knife. The wall is trimmed with nippers and then leveled with a rasp.

In addition to maintaining optimal balance in a sound horse by trimming the foot to restore the length of the foot, several other modifications to the hoof capsule may be performed, usually with the intent of improving hoof capsule shape, and often in conjunction with specific shoeing practices. *Floating the hoof wall* refers to trimming part of the hoof wall short so that the hoof will not touch the shoe at that point. The shoe supports the wall on either side of the floated area dorsally and palmarly unless the heel is floated. Floating the hoof wall is used to relieve that section of the wall from weight bearing. This encourages faster growth at the coronary band, permits proximally displaced wall to descend, and relieves stress in the wall so that new wall growth may redirect itself. The heels are floated to treat under-run heels. This is most effective when the palmar aspect of the foot is given additional support (e.g., with a heart bar shoe).

Grooving of the hoof capsule is designed to mechanically dissociate the capsule on one side of the groove from the other. The groove is made through the full thickness of the stratum medium. The grooves may be created parallel or perpendicular to the horn tubules. The use of several grooves parallel to the horn tubules between the quarters and heel of the hoof capsule has been advocated to encourage expansion of the heels by increasing the flexibility of the wall but is of dubious value. Grooves perpendicular to the horn tubules usually are made around part of the circumference of the hoof capsule immediately distal to the coronary groove, typically at the toe or heels. These grooves relieve the pressure on the coronary band from the stresses in the weight-bearing wall. This increases the speed of new wall growth proximal to the groove and allows the new wall to grow independent of distracting forces in the distal wall.

Resection of the hoof capsule involves removal of the stratum medium, usually starting at the weight-bearing surface and extending a variable distance proximally, typically involving no more than 20% to 40% of the circumference of the hoof capsule. Because this creates instability of the hoof capsule, it is used only for treatment of laminitis, white line disease, and hoof wall avulsion injuries. Thinning the hoof wall at the heels so that partial thickness of the stratum medium is removed has been recommended to increase the flexibility of the wall and encourage expansion of the hoof capsule, but it is seldom performed.

The Horseshoe Form

In its most basic configuration a horseshoe is a curved steel bar, rectangular in cross section, that is shaped to conform to the contour of the ground surface of the hoof wall and wide enough to cover the ground surface of the hoof wall and the immediately adjacent sole. The shoe has four surfaces: the foot and ground surfaces and the inner and outer edges, called *rims*. The parts of the shoe are named after the corresponding section of the hoof, that is, the *toe*, *quarter*, and *heel*. Each shoe has two branches, medial and lateral, that extend from the center of the toe to the medial and lateral heels, respectively. The substance of the branches is called the *web*, which has a width and thickness. The shoe is punched with nail holes, three or four in each branch, three of which are usually used. This shoe is described as *flat* because the ground surface of the shoe is level, *stamped (punched)* because it has nail holes, and *open* because the bar of metal forming the shoe does not form a continuous loop between the heels.

The shoe should fit flush with the outer margin of the dorsal half of the hoof wall from one quarter to the other. From the quarter palmarly/plantarly the shoe should incrementally extend further abaxial to the wall until it extends approximately 0.3 cm abaxial to the wall at the heel to allow for expansion and contraction of the hoof capsule as the foot alternates between weight bearing and non–weight bearing. The shoe branches extend marginally palmar/plantar to the heels to allow for growth of the hoof capsule during the shoeing cycle. This basic shoe pattern and fit may suffice for many horses but frequently is altered to suit the exercise performed by a horse, the ground surface on which the horse is worked, and for therapeutic purposes.

Materials and Size

Horseshoes may be made of metal, synthetic polymers, or a composite of the two materials. Altering the material of a shoe alters the weight of a shoe for a given size, the durability of the shoe against wear and other damage, shock absorption, workability, and cost. Most horseshoes are made of steel for reasons of effectiveness, cost, wear, and workability. Aluminum also is used frequently because it is lighter and easier than steel to cold forge, but it wears faster than steel and is not as stiff. Shoes recently have been made from various synthetic polymers and composites of more than one material and are used in specialty situations, but shoe status is in constant flux as new products appear on the market and others disappear.

Metal shoes may be hand forged from bar stock or manufactured. Hand-forged shoes offer the advantage of being customized to the individual foot. However, manufactured shoes are used far more frequently than hand-forged shoes because they save time and a wide range of shoes is suitable for most feet. Bar stock is usually rectangular in cross section and is available in different sizes varying from 0.6 to 1.2 cm thick and 1.2 to 3.1 cm wide, although the most commonly used size is 0.8 × 1.9 cm. Concave stock is frequently used in Europe. Manufactured shoes, also called *keg shoes*, are sized. However, although sizing within and usually between product lines is consistent for a given manufacturer, there is no universal standard for sizing horseshoes. Manufactured shoes may be generically shaped to the general shape of a horse's foot or specifically designed for a fore or hind foot. The former requires more shaping but are cheaper and require less stock on hand. The dimensions of the stock of either manufactured or hand-forged shoes affect the weight of the shoe, stiffness, coverage of the

ground surface of the foot, height the shoe elevates the foot off the ground, and rate at which the shoe wears out.

Shoe weight influences the biomechanics of movement. The heavier the shoe, the more energy is expended accelerating and decelerating the limb at the beginning and end of each stride. Therefore the lightest shoe is used that is compatible with protecting the wall and adjacent sole and providing the stiffness and wear required. Shoes made from concave stock are lighter than shoes made from regular bar stock. The width and thickness of the shoe usually is uniform around the circumference of the shoe so that biomechanical influence of shoe weight and the stresses imposed are usually balanced about the axis of the limb. Occasionally a shoe may be unevenly weighted to alter the animation or balance of a gait. The increased weight may be at the toe, in one branch, or at one or both heels. Increasing the weight at the heels is done to increase animation but has not been scientifically tested. Increasing the weight of the toe has been used to encourage the horse to reach farther at the beginning of the stride. Toe weights do not increase stride length but do increase flexion in the limb of horses with poor limb flexion during protraction of the limb. They have no impact on horses that already have good limb flexion.[1]

Although the width of the web of a shoe is related to the thickness of the hoof and, at least in part, the size of the foot, it is common to increase the width of the web of the shoe to provide increased protection to the margins of the sole. The thickness of the shoe is related to the rate at which it is expected to wear and to a lesser extent the rigidity needed to prevent the shoe from bending out of shape. The height the shoe raises the foot off the ground also is influenced by the thickness of the shoe, but this is usually a secondary consideration to wear and rigidity.

Cross-Sectional Profile of Shoe Stock

The cross-sectional profile of the shoe may be modified by altering the ground surface, the solar surface, either rim of the shoe, or a combination, and may affect the whole shoe or part of the shoe, such as the toe, the branch, or a heel (Fig. 27-6).

Several common modifications are made to the ground surface of the whole shoe. Softening the 90° angles at the junction of the ground surface of the shoe and the inner and outer rims by beveling or rounding, called *rolling,* increases the ease of breakover. A shoe with a rounded outside rim is called a *roller motion shoe,* which improves the ease of breakover in any direction. A similar effect is achieved with a *half round shoe,* so called because it is made from half round stock that resembles a semi-circle in cross section. The toes of flat shoes frequently are rolled or beveled only at the toe to improve the ease of breakover (Fig. 27-7). A similar effect is achieved by rockering the toe, that is, bending the full thickness of the shoe proximally. Less commonly, a flat shoe may be asymmetrically beveled or rounded to improve the ease of breakover toward one side or other of the shoe to encourage a horse to breakover toward that side and direct the flight of the foot. Rounding the margin of a shoe to prevent interference is called *safing.*

There is little traction between smooth steel and the ground. Grooves in the ground surface of the shoe, called *fullering* or *creases,* increase traction. The fullering fills with dirt; the friction between dirt in the fullering and the dirt on the ground is greater than steel on dirt. In addition, the crease provides two additional edges that may bite into the ground. The full circumference of the shoe may be fullered, or it may be limited to the branches of the shoe and less frequently just the toe. Fullering a single branch of the shoe enhances traction uniaxially and delays breakover on that side of the foot. When the branches of the shoe are fullered, the nail holes are centered in the groove, which is formed to conform with the inside and outside of the nail heads.

Fig. 27-6 The cross-sectional profile of the shoe branch can be modified to vary traction and breakover. **A,** Half round; **B,** flat shoe incompletely creased or fullered; **C,** concave stock; **D,** fully creased rim shoe; **E,** incompletely creased rim shoe (Eventer, St. Croix Forge, Forest Lake, MN).

Fullering and modifying the shoe edges frequently are performed in conjunction with each other. Rim shoes are fully fullered, and the ground surface of the rims is beveled toward the fullering of the shoe. More specialized rim shoes have a higher inside or outside rim, hence the names *inside* or *outside rim shoes* (Fig. 27-8). A polo plate is a specialized form of inside rim shoe. Because the higher rim is on the inside, the horse is less likely to cause severe injury to another horse in competition yet still benefits from the additional traction of the rim. A barrel racing shoe is a form of outside rim shoe that provides greater traction than an inside rim shoe, although ease of breakover is sacrificed. A similarly modified profile of racing or training plates is termed *swedging.* A classic example of a shoe that uses all these techniques is a half round, half-swedge shoe worn on the hind feet by harness horses. The inner branch is half round in cross-sectional profile and the lateral branch of the shoe is swedged. Thus the medial branch, enhanced by the half round, breaks over rapidly, whereas the breakover of the lateral branch is delayed by the swedge.

The only common modification to the solar surface of the shoe is gentle beveling of the inner half of the web toward the inside, called *seating out* or *concaved inner surface.* Horses with flat soles are shod with seated-out or concaved shoes to decrease pressure on the sole adjacent to the wall. Less frequently the heels of the shoe are beveled to the outside margin of the shoe to encourage heel expansion, that is, abaxial movement of the wall during weight bearing. This practice is of questionable benefit, and if the shoe is beveled severely, it causes undue stress in the white line. Beveling the part of the heel of the shoe that extends abaxial to the quarters and heels, called *boxing,* decreases the likelihood of the shoe catching on another object or being trod on by another foot and pulled off.

Extensions

An *extension* is any projection of the shoe that extends outward from the normal outline of the shoe in the horizontal plane (i.e., away from the center of the sole). Extensions may be

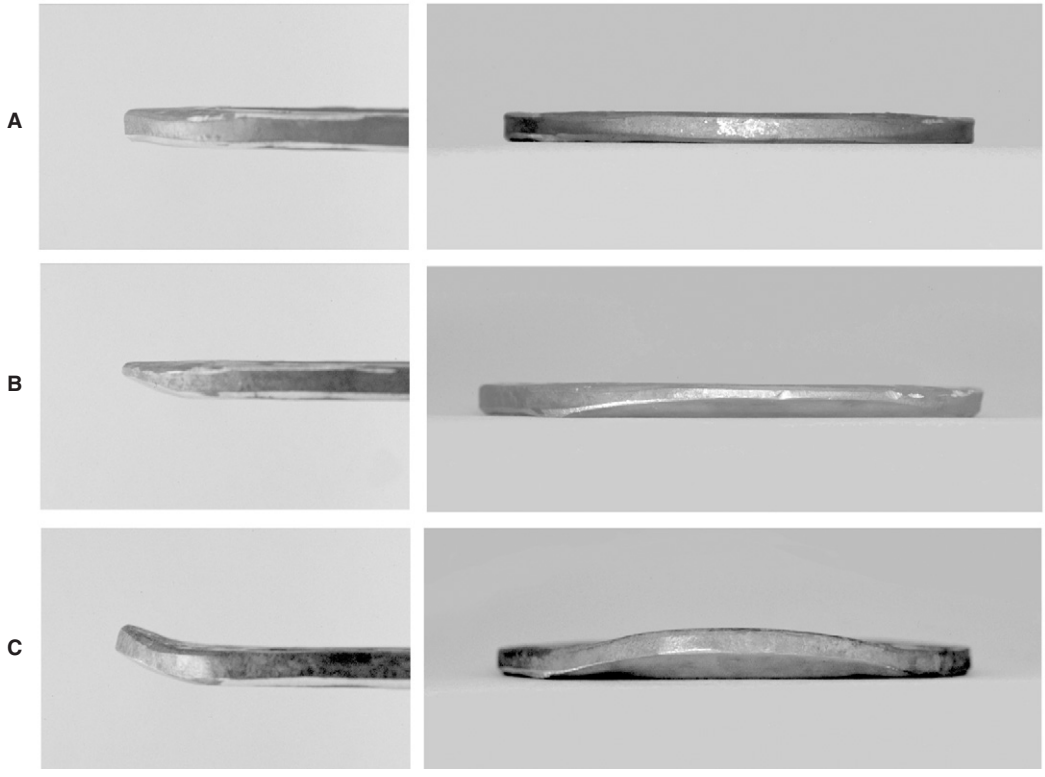

Fig. 27-7 The toe of a shoe frequently is modified to move the point of breakover palmarly compared with a flat shoe. **A,** Flat shoe; **B,** rolled-toe shoe; **C,** rocker toe shoe.

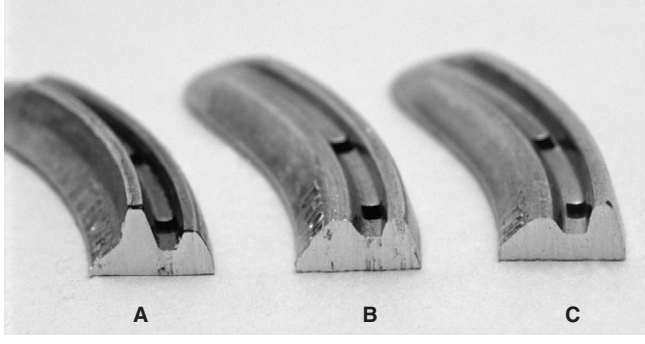

Fig. 27-8 Cross-section of the branch of three shoes. **A,** A high inside rim; **B,** rims of equal height; **C,** a high outside rim.

positioned anywhere around the circumference of the foot. An extension can be forged with the shoe at the time of manufacture or welded onto the outside rim of the shoe. A similar effect is obtained by using an oversized shoe positioned either forward or backward or by setting one branch of the shoe wide. Every extension has the potential to cause the shoe to act as a lever, and this lever action may be either static, when the animal is stationary and the foot bearing weight, or dynamic, particularly during the landing and breakover phases of the stride. When force is exerted on the extension by the ground, the stresses are increased in the adjacent wall and decreased in the opposite wall as the point of action of the GRF shifts. Because the digit is approximately symmetrical in the sagittal plane, the effect of medial or lateral extensions is similar. However, the absence of dorsopalmar symmetry means that dorsal and palmar/plantar extensions function differently. In addition to acting as levers, extensions increase the surface area available for ground contact, which decreases the amount the shoe descends into soft footing at that point in the circumference of the foot. If the surface area of one part of the shoe is altered, it causes the foot to be supported by a soft ground surface more than the opposite side so the interaction between shoe and ground becomes more complex because the foot is tilted.

Abaxial extensions may be used to force either the opposite wall of the hoof capsule to the ground or support the wall adjacent to the extension. In doing so, the lever decreases the compression in the opposite wall and increases the compression in the ipsiaxial wall, which imposes a moment in the frontal plane. This effect is used in foals with angular deformities. The extension is placed on the opposite side of the limb to the side of the deviation to increase the load in the side of the limb with the extension, slow growth, and apply an angular force across the mid-sagittal plane of the limb.

Toe extensions usually are applied to foals with a flexural deformity of the distal interphalangeal joint. The toe extension increases the moment about the distal interphalangeal joint at breakover, as the point of action of the GRF moves dorsally, and during weight bearing if the heels are off the ground. This increases tension in the DDFT and muscle. Therefore the toe extension acts to stretch the musculotendonous unit of the DDF. Success depends on the severity of the flexural deformity. In more severely affected animals the pain generated by the increased tension in the DDFT, with or without increased compression at the toe, becomes counterproductive. The change in location of the GRF places the wall at the toe under greater compression and it is prone to deform.

Heel extensions frequently are used uniaxially or biaxially. In performance horses, extensions frequently take the form of short continuations of the heel that are called *trailers*. Trailers are used almost exclusively on the lateral branch of hind

shoes. Front shoes with trailers are likely to be removed by interference from a hind foot. Egg bar shoes project palmar/plantar to the heels of the hoof capsule and act as heel extensions. They are most commonly used on the front feet, where they are less likely to be removed by interference than shoes with trailers. Force on a biaxial heel extension decreases the moment about the distal interphalangeal joint and the tension in the DDFT. Therefore horses convalescing from a DDFT injury benefit from heel extensions, frequently used in conjunction with heel elevation. Horses with navicular disease appear to benefit from egg bar shoes, theoretically because the navicular bone is under less compression from the DDFT,[2] but this has not been substantiated.[3] The consequence of this benefit is that the heels are under greater compressive stress.

Heel extensions alter the way the foot strikes the ground. If, as happens frequently, the heels are closer to the ground at impact, the extension contacts the ground first. A lateral uniaxial extension, either in line with or diverging up to 45° from the mid-sagittal plane of the foot, is used to force the foot to pivot toward the side of the trailer as the foot lands. The toe of the foot is directed laterally after impact and breakover is redirected.

Bars

A *bar* is any part of a shoe that extends from one branch of a shoe toward the other. A *complete bar* extends from one branch to the other; a *partial bar* extends part of the way across the shoe. Most bars extend from one heel to the other to form a closed shoe. A bar may extend from one quarter to the other, or even diagonally across the shoe. There are several patterns of complete bar shoe in common usage including the straight bar, egg bar, heart bar, and heart bar–egg bar (or full-support shoe). Bar shoes offer several benefits: increased stability and ground contact surface area, local protection, and recruitment of additional weight-bearing area of the foot.

Closing the shoe is considered to make the shoe more stable by decreasing movement between the branches of the shoe and is frequently used, often in conjunction with other shoeing techniques, for instability within either the hoof capsule or distal phalanx. Bars that extend palmar/plantar to the normal position of the heel of the shoe act as a palmar/plantar extension. Bars that set under the ground surface of the foot can be adjusted to protect that part of the foot from ground contact, apply pressure to that part of the foot, or recruit that part of the foot for weight bearing. A straight bar shoe may decrease pressure on the palmar/plantar third of the frog and protect the underlying navicular bone. The heart bar shoe is used to recruit or increase the role of the frog in weight bearing, particularly in the treatment of laminitis. However, heart bar shoes also may be used to support the palmar/plantar aspect of the foot to reduce the stress in the adjacent wall and permit floating of the heels.

An incomplete bar extends part of the way across the ground surface of the foot, most commonly from one heel onto the frog, which supports or reduces weight bearing on a single heel. Alternatively, a full bar across the full width of the foot may be used in conjunction with an incomplete shoe so that the bar covers both heels, but the shoe is incomplete between one quarter and heel. The bar of a Z bar shoe is shaped with two 90° bends that are incorporated into a three-quarter shoe, so that one leg of the Z is attached to the heel of one branch and the other is attached to the quarter of the opposite branch. This shoe also relieves one heel from weight bearing.

Calks, Grabs, and Other Devices Added to the Ground Surface of the Shoe

Various devices are added to the ground surface of a shoe to increase traction. They also influence the speed and direction of landing and breakover. Calks are projections of almost any size and shape, although most are round, square, or rectangular, on the ground surface of a shoe (Fig. 27-9). The terminology to describe the different types is confusing and at times inconsistent. Different types are called *blocks, stickers,* and *studs.* They are made of steel or steel with a tungsten carbide core. Toe grabs and bars welded to the shoe are, in essence, greatly elongated calks. Borium, tungsten carbide crystals in a flux, is welded onto the surface of a shoe in an incremental manner so that any shape or sized projection can be formed. Some calks are permanent, whereas others are temporary. Permanent calks are forged with the shoe, molded in the case of aluminum shoes, at the time of manufacture, or welded or brazed onto the shoe at the time of fitting. Drive calks are semi-permanent and are driven into a hole drilled into the shoe. Temporary calks, also called *screw-in calks* or *studs,* are screwed into tapped holes so they can be attached and removed as needed. Cotton wool is used to plug the hole when not in use. The size and shape of the studs may be changed with the ground conditions.

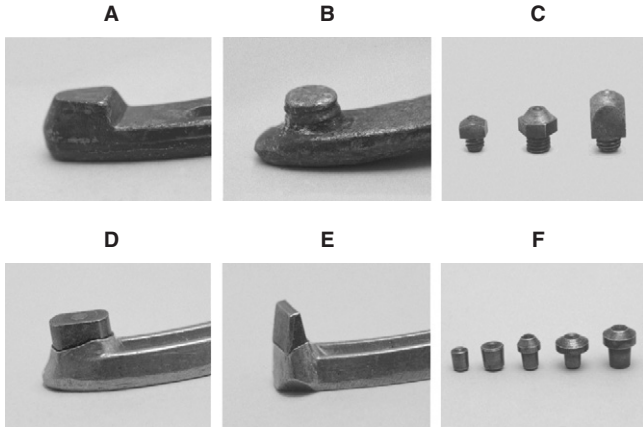

Fig. 27-9 Calks may be forged with the shoe at the time of manufacture or added later. **A,** Calk of a "heeled" shoe formed at the time of manufacture; **B,** calk formed by addition of borium; **C,** various sizes of screw in calks; **D,** a block inserted into an aluminum shoe at the time of manufacture; **E,** a sticker inserted into an aluminum shoe at the time of manufacture; **F,** drive-in calks.

Calks may be positioned at any point around the circumference of the shoe. The choice of whether to use calks, what type of calks to use, and where to position the calks follows no dogmatic guidelines, but usually is based on the preference of the farrier. However, although square and round calks probably offer equal resistance to both mediolateral and dorsopalmar motion, rectangular calks offer greater resistance to motion against movement perpendicular to the long axis. Bilateral heel calks typically are used on jumpers and event horses. Racehorse shoes may be equipped with toe grabs, with or without one or two heel calks (blocks or stickers). Draft animals usually have shoes with biaxial heel calks and, less frequently, a large toe calk.

Projections from the surface of the shoe, in addition to providing traction, inevitably alter the balance of the foot by altering the way the foot contacts the ground. The harder the ground surface, the greater the effect. The taller the calk, the greater the effect. A single calk at either the heel or toe alters mediolateral and dorsopalmar balance. Two heel calks of equal height alter dorsopalmar balance. The addition of a toe calk of equal height restores dorsopalmar balance. The addition of calks to the shoe concentrates stress in the wall immediately proximal to the calk. Therefore the lowest broadest calk compatible with adequate traction is recommended.

The effect of calks on breakover and landing is a secondary consideration, because other methods usually are applied to

achieve the same objective. A single heel calk acts in much the same way as an extension, causing the foot to turn toward the side with the calk as the foot lands. Symmetrical placement of two pairs of calks, one pair on either side of the toe and one pair at the heels, encourages the foot to break over in the center of the toe.

Pads

A pad is a layer of material inserted between the hoof capsule and the shoe (Fig. 27-10). Pads may provide protection, diminish concussion, and alter the effective angle, length, or both of the foot and shoe. Traditionally they have been divided by form into full and rim pads, and by composition into leather and synthetic. However, in the last two decades an imaginative range of products has become available, a range too great for comprehensive discussion. Pads may be riveted to the shoe, which is then nailed to the foot. However, shoes are available that are manufactured with a rim pad bonded to the shoe. Full pads are fitted between the foot and the shoe and cover the entire ground surface of the foot. The cavity between a full pad and the sole usually is filled with pine tar and oakum, or a synthetic equivalent such as silicone, to prevent the cavity filling with dirt. In contrast to full pads, rim pads are fitted to the contour of the shoe so that the sole is not covered.

Pads that cover the sole protect the ground surface from direct trauma. However, efficacy has been questioned because the pad and packing effectively lower the contact point with the ground. Pressure on the pad can then be transmitted to the sole, although in a more diffuse pattern. Any pad interposed between the shoe and the wall has the potential to diminish concussion on the wall. However, products vary greatly.[4] Wedge pads, either full pads or bar wedges, change the angulation of the hoof capsule to the ground. Wedge pads most commonly are used to raise the heels. Wedge pads also are used to raise either the medial or lateral wall to improve the mediolateral balance when this is not feasible by trimming. This can be achieved with a full pad, but it is more common to use a uniaxial wedged shim.

More novel pad designs include extension of the ground surface of the pad between the branches of the shoe to fill part or all of the cavity between the ground and the sole to form a flexible heart bar, or to recruit more weight-bearing surface area and support the ground surface of the foot. Similarly, silicone putty and pour-in polyurethane can be applied to form a pad in situ after the horse has been shod. Once set, these materials can be trimmed to selectively apply or relieve pressure.

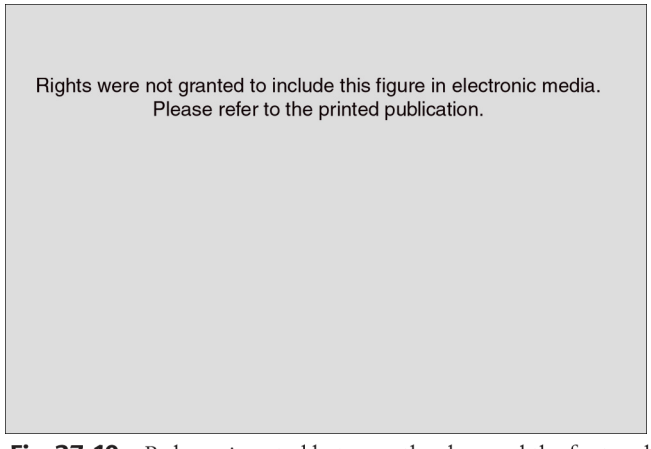

Fig. 27-10 Pads are inserted between the shoe and the foot and take various forms. **A,** Full leather pad; **B,** full plastic pad; **C,** oval plastic rim pad; **D,** full wedge pad; **E,** a bar wedge pad; **F,** a full plastic pad formed with a molded heart bar (Cushion Frog Pad; Castle Plastics, Leominster, MA).

Pads have several disadvantages. All pads compress, which causes the nails to loosen sooner than they otherwise would. Full pads trap moisture against the sole and frog. The underlying sole and frog are softer, and the horse is predisposed to developing thrush.

Miscellaneous Changes in Shoe Form or Fit

A shoe shape is not limited to covering the entire ground surface of the wall. Partial shoes cover part of the circumference of the hoof wall. Tips that cover just the toe are used to prevent wear at the toe in young horses. A three-quarter shoe extends from one heel to the opposite quarter and limits weight bearing in the uncovered heel. A shoe may be reversed so that it is open at the toe but closed at the heels. This functions as an egg bar shoe palmarly with a shortened breakover at the toe.

Hot Versus Cold Shoeing

In cold shoeing a previously manufactured shoe is shaped cold and applied to the trimmed foot. In hot shoeing a newly forged shoe or a manufactured shoe is heated in the forge and applied to the ground surface of the foot while the shoe is still hot. The heat sears and levels the trimmed surface of the foot. It should cause no discomfort to the horse. Cold shoeing is quicker and requires less equipment and less skill. Hot shoes are easier to shape than cold shoes because the steel is softer. Hot shoeing produces a better fit, because the searing levels any minor irregularities in the trim and highlights any overtly unleveled areas. This is no substitute for correct trimming of the foot beforehand. The searing also aids seating of clips. It is also considered to seal the ends of the horn tubules.

Attachment of the Shoe to the Hoof

The large majority of horseshoes are attached to the hoof capsule with nails. Horseshoe nails are available in a variety of styles and sizes, but all share a similar pattern. Each has a head, a shank (blade), and a point beveled at the tip. There are several types of head that vary in size and the angle of bevel. Heads with less bevel, such as European head nails, are suited for thicker webbed shoes. Other nails with thinner or narrower shanks, such as race and slim blade nails, minimize damage to the wall. The size and style of nail is chosen based on experience to provided secure attachment of the shoe to the hoof capsule, without unnecessarily damaging the hoof capsule. The shape of the crease and nail hole should be stamped to fit the shape of the nail head, which provides the greatest support to the nail. The heel nail is located at the bend in the quarter, the toe nail at the junction of the toe and quarter, and the quarter nail(s) evenly spaced in between. In North America, three nails are usually used in each branch of the shoe. In the United Kingdom, three nails are usually used in the medial branch and four nails in the lateral branch.

The nail should be driven into the ground surface of the foot at the outside of the white line and exit the wall approximately 1.9 cm proximally. The bevel at the point of the nail is always directed toward the sole to limit the likelihood of the nail entering the underlying sensitive structures. Once the nail is driven, only the flat head of the nail should extend distal to the shoe. Because there is some variability in thickness of hoof wall for a given sized foot, the position of the nail hole in relation to the thickness of the web should be adjusted so that it is immediately distal to the white line. When the nail holes are positioned more toward the inner rim than normal, the shoe is coarse punched; when they are toward the outer rim, the shoe is fine punched. Because the slope and thickness of the wall changes between the toe and quarter, the angle at which the nail is driven decreases from toe to heel. This should be reflected in the stamping of the nail holes. These adjustments to the position, shape, and angle of nail holes are easier to accommodate on hand-forged shoes, although modern manu-

factured shoes are available in sufficient variety and made to stringent specifications that most situations can be addressed satisfactorily. The number of nails, size of the nails, the congruency of the nail holes to the shoe, and the skill with which they are applied determine the security of attachment of the shoe to the hoof. Small nails may not adequately attach the shoe to the foot. Large nails may split the hoof capsule.

Recently the use of various adhesives to attach shoes has become more popular. The shoe can be affixed with an adhesive by either tabs or cuffs that are attached to the circumference of the shoe and extend proximally. The cuffs are made of semi-rigid plastic or synthetic cloth. Alternatively, the adhesive may be directly interposed between an aluminum shoe and the ground surface of the wall and immediately adjacent to the sole. Attachment of the shoe with adhesives offers several advantages over nailing in the following circumstances: (1) when nailing is too painful; (2) when there is insufficient wall for nailing the shoe; and (3) depending on the specific application, it permits greater expansion of the foot. However, there are disadvantages. The glue-on shoes or the adhesive are more expensive, and application of some adhesives to the side of the wall decreases the quality of the underlying wall over time. There is a also perception that glued-on shoes do not stay on as well as nailed-on shoes, but this impression is in part the result of poor case selection or poor application.

Clips are triangular-shaped projections that extend proximally from the periphery of the solar surface of the shoe. Clips may be forged from the shoe at the time of fitting, or shoes with preformed clips are purchased. When a shoe is fitted, the outer surface of the clip is congruent with the surface of the hoof wall. They reduce movement between the shoe and the hoof capsule, which decreases the shear stress on the hoof nails. A single clip usually is used in the center of the toe of fore shoes, and two clips are placed near the toe quarter junction of hind shoes. A toe clip is not used on hind feet in case it should injure the fore foot, but there is no reason why side clips should not be used on fore feet. For additional stability of the hoof capsule, clips may be positioned elsewhere around the periphery of the shoe to constrain expansion of the hoof capsule. Optimally, two or more clips should be located at 180° to each other around the circumference of the shoe so that the flat surface of the clips are aligned perpendicular to the direction of expansion.

Horseshoe Functions

The principal objective of shoeing a horse is to provide protection to the hoof wall from excessive wear. More far-reaching goals include improving balance, providing traction, modifying breakover, increasing animation, providing support or stabilization, and limiting interference.

Balance

Mediolateral and dorsopalmar balance are discussed elsewhere (see pages 252 to 253).

Protection

Shoes and accessories can provide protection from excessive wear, trauma, and excessive concussion. Protection from excessive wear is primarily required for the ground surface of the wall and is provided by any shoe that is interposed between the foot and the ground around the full circumference of the hoof wall and the adjacent sole. Protection of the rest of the ground surface of the foot, namely, the sole and the frog, may be achieved by interposing a layer between the ground and the ground surface of the foot. If a specific area needs protection, frequently this can be provided by modifying the shoe. For example, a seated-out wide web shoe protects more of the sole adjacent to the wall than a shoe with a narrower web. A bar added to the shoe protects the underlying sole or frog. For more extensive protection to the ground surface of the foot or alleviation of concussion, a pad may be inserted between the shoe and the foot (see page 266). Some shoes are manufactured with a composite of a metal backbone to provide rigidity and synthetic polymers to provide protection against concussion.[4]

Traction

Traction is required to facilitate optimal, confident movement of a horse over the ground as the limb decelerates at the beginning of the stride, as the horse propels itself forward and around turns, and as the foot breaks over. There is an indirect inverse relationship between traction and ease of breakover. Many modifications to a shoe that increase traction delay breakover and vice versa. The amount of traction required depends on the horse, type of exercise, and the ground surface.

Increased traction is provided by decreasing the ground surface area of the shoe, creasing or fullering the shoe, adding ridges to the cross-sectional profile of the shoe, or using calks, grabs, and nail heads that project below the surface of the shoe. Increasing the ground surface area of the shoe or rounding the outer rim or both rims of the shoe reduces traction.

Breakover Modification

The speed of breakover is related to the moment created about the distal interphalangeal joint by the dorsal hoof wall at the end of the stride. This is determined by the anatomical relationship between the center of rotation of the distal interphalangeal joint and the dorsal-most point of ground contact, termed the *breakover point*, and the shoe resistance to being elevated off the ground. The moment about the distal interphalangeal joint is dictated by the direct distance between the center of rotation of the joint and the breakover point and the angle this line forms with the ground. These are in turn influenced by the length and angle of the dorsal hoof wall. Breakover can be enhanced by shortening the toe, moving the breakover point palmarly and increasing the angle of the dorsal hoof wall. Breakover is delayed by lengthening the toe[5] and decreasing the angle of the dorsal hoof wall.[6] Shortening the toe is accomplished by trimming the foot and is limited by the underlying sensitive structures. Toe length can be increased by letting it grow out or addition of pads or a thicker webbed shoe. The breakover point can be moved palmarly by trimming a barefooted horse or rolling or rockering the toe of the shoe, squaring the toe of the shoe, or setting the shoe back. However, the use of rolled or rockered toes did not influence the timing of breakover.[7,8] The angle of the dorsal hoof wall may be increased by elevating the heels with wedge pads or using shoes with wedged heels. These practices are both preferable to leaving the heels long. The degree to which the shoe adheres to the ground is indirectly related to modifications made to the shoe to enhance traction.

The direction and the point on the circumference of the hoof at which breakover occurs are related to the conformation of the limb, namely, the presence of angular or rotational deformities. To change the breakover point, either the orientation of the shoe on the ground before breakover or the ease of breakover between the two branches can be changed. The direction the foot is oriented on the ground is influenced by the way it lands, and the way it lands is influenced by the path of the foot during the flight phase of the stride, which is in turn influenced by the way the foot breaks over to complete the full phase cycle. Uniaxial trailers and heel extensions turn the foot ipsilaterally at landing, which directs breakover toward the opposite side of the toe at the end of the stride.

Breakover may be redirected by changing the relative ease with which the branches leave the ground, changing the relative length of the lever arm, adherence of the two branches of the shoe to the ground, or a combination of these factors. Adherence is increased by use of traction devices or decreased by using a non-fullered branch. The length of the lever arm of a shoe branch may be increased by setting it more dorsal to create a small extension between the center of the toe and the

toe/quarter junction or decreased by setting it back. In essence, this is what happens when the toe is squared. Increasing the ease with which one branch lifts off the ground, while decreasing the ease the other branch lifts off, shifts the breakover point toward the side with slower breakover. Any maneuver to change the direction of breakover is accompanied by increased torsional stress in the limb.

Animation

Gait animation indicates increased range of joint movement with exaggerated temporal characteristics. Animation is primarily a cosmetic change created because such characteristics are considered esthetically desirable in certain equestrian disciplines. It may be improved by shoeing.[9] Increased animation of gait is achieved by increasing the weight of the foot and shoe and increasing the length of the foot, shoe, or both.[5] These manipulations delay breakover, which is then followed by an exaggerated response both in distance moved and joint angulation. Animation obtained by increasing the length of the foot creates imbalance and increases stresses associated with moments about the distal interphalangeal joint. Animation obtained through increased weight of the foot is accompanied by greater energy expenditure by the horse, fatigue, and a greater chance of injury. Interestingly, egg bar shoes decrease animation compared with flat shoes.[3]

Support

Support is a term widely used, seldom defined, and often ambiguous. Support usually means to hold a structure up or prevent it from collapsing. This can be interpreted in at least two ways. First, it can refer to the relationship between the foot and the ground. Second, it can refer to supporting a structure within the foot. On occasion, support may fit both of these circumstances. Specifically, supporting the foot in relation to the ground implies keeping the whole or part of the foot from descending into a ground surface that gives, or keeping the foot level. Increasing the ground surface area of the shoe at any juncture around the periphery of the foot will support that area. Typically, the palmar aspect of the foot is most likely to be supported by the use of a straight bar or an egg bar shoe.

Support as a concept applied to structures within the foot is not always straightforward. Collapse of the hoof wall under excessive compressive strain is a simple example. However, in horses with laminitis or rupture of the DDFT, the distal phalanx displaces from its normal position; however, the concepts behind providing support are quite different. In a horse with a ruptured DDFT the relationship between the distal phalanx and the hoof capsule is intact, whereas in a horse with laminitis, it is disrupted.

An injured tissue may require stress relief. The clinician should determine whether the tissue is stressed under tension (tendons, ligaments, lamellae) or compression (bone, hoof wall), at what point in the stride the stress is greatest, and whether the stress is associated with weight bearing, moments about the distal interphalangeal joint, or a combination of these two factors. The stresses that are greatest during the weight-bearing phase of the stride are redirected by altering the balance of the limb by trimming the foot, applying wedges, using shoe extensions as levers, or recruiting additional parts of the ground surface of the foot to bear weight. For example, in a horse with laminitis with uniaxial damage to the lamellae, the distal phalanx displaces distally on one side of the foot only and the distal interphalangeal joint becomes correspondingly asymmetrical. Support of the distal interphalangeal joint requires an extension to the side of the displacement, but this would further increase the strain in the damaged lamellae and cause further displacement. To protect the lamellae, contralateral extension, used with other measures to support a laminitic foot, would load the contra-axial lamellae and protect the damaged lamellae by reducing the load. An ipsilateral extension would be appropriate to treat a strain of the collateral ligament. However, redistributing the stress from one structure inevitably increases the stress on others.

Decreasing the moment about the joint reduces stresses that are greatest during extension of the distal interphalangeal joint. For example, in a horse with acute laminitis, the already damaged lamellae have a greater propensity to separate with the stress associated with breakover. To reduce this stress, shortening the toe decreases the length of the lever arm and elevating the heels decreases the tension in the DDFT.

A strain of the insertion of the DDFT is under stress during both weight bearing and breakover. A heel extension with elevation decreases the stress during weight bearing by moving the point of action of the GRF palmarly. Moving the breakover point palmarly decreases the tension in the DDFT at breakover by decreasing the torque about the distal interphalangeal joint. If the DDFT were severely strained, a heel elevation and extension would prevent the toe of the foot from lifting off the ground and subluxation of the distal interphalangeal joint.

Importantly, the practitioner needs to understand which tissue to support, how to achieve it, and anticipate the side effects. In addition to the immediate benefits of the support provided, the clinician must contend with the changes within the hoof capsule and changes in the relationship between the hoof capsule and the distal phalanx from movement between the epidermal and dermal lamellae, the viscoleasticity of the hoof capsule, and the altered pattern of growth of the hoof.

Prevention of Interference Injury

Interference occurs when the foot of one limb contacts another limb during the stride cycle, which frequently results in injury. Interference can be divided into two types. *Brushing* occurs when a forelimb or hindlimb interferes with the contralateral limb between the coronary band and the carpus or hock. When a limb interferes with the ipsilateral limb, the hindlimb strikes the forelimb (forging, overreaching) or, less frequently, the forelimb strikes the hindlimb (scalping, speedy cutting). *Cross firing* occurs when a forelimb and contralateral hindlimb interfere. The causes of interference include poor balance, poor conformation, fatigue, and lameness. When poor balance, or lameness, is identified as the cause, correction of these problems should resolve the interference. Increasing the fitness of the horse or using a lighter shoe may reduce interference from fatigue in unfit horses. However, the conformation of a horse cannot be changed. For example, a horse that is toed out will wing in during the flight phase of the stride, increasing the likelihood of interference with a contralateral limb. A horse with a short back in relation to the length of its limbs has an increased tendency for interference between a forelimb and a hindlimb. Treatment of interference that cannot be eliminated by removing the cause is directed at preventing interference by influencing the flight pattern of the feet and reducing the severity of injury when interference does occur.

Contact between ipsilateral limbs occurs as the forelimb is breaking over and the hindlimb is landing. Traditionally, prevention has been aimed at encouraging the forelimb to break over faster so that it moves out of the way of the hindlimb and delaying breakover of the hindlimb so that it lands later. Forelimb breakover has been discussed previously. Delaying breakover in the hindlimbs has been accomplished by lowering the heels of the hoof or thinning the branches of the shoe at the heel. However, although this does delay breakover, limb coordination is restored during the swing phase of the stride.[10] It also creates a dorsoplantar imbalance, which may cause other problems.

Contact between contralateral forelimbs occurs because the path of the limb in the swing phase is too close to the

position of the weight-bearing limb, which is most likely to occur in base-narrow, toed-out horses. Prevention aims to redirect breakover laterally away from the medial toe so that the phalanges do not diverge as far medially from the mid-sagittal plane of the limb as the metacarpophalangeal joint flexes.

Contact between a hind foot and the contralateral forelimb in pacers occurs between strides as the horse is suspended. Prevention of cross firing is aimed at encouraging the hindlimb to stay further lateral so that it does not contact the forelimb. This is accomplished by encouraging the foot to move laterally as it lands, adding a trailer to the lateral branch of the shoe, and encouraging the foot to break over toward the outside of the foot by using a half round–half swedge shoe.

To decrease the severity of injury when impact between the limbs is unavoidable, the clinician should reduce the likelihood of the shoe itself, particularly sharp edges, from contact of the limb likely to be injured and directly protect the injured limb. To limit injury caused by sharp edges at the periphery of the shoe, the margin of the shoe can be safed in the area of contact, or a shoe with a curved contour, such as a half round shoe, can be used. To limit contact between the shoe and the injured limb, the shoe can be fitted so that it is set back from the margin of the hoof wall by moving the toe of the shoe back or by moving the medial branch axially. Alternatively, the shoe is forged so that that part of the shoe making contact is set back from the wall. Boots can be worn during exercise to directly protect the part of the limb likely to be injured.

THE PRACTICE OF SHOEING

Effects of Shoeing on Foot Function

A horseshoe is not simply an extension of the hoof capsule. By interposing a shoe that has markedly different physical characteristics from the hoof capsule between the foot and the ground, a single interface is replaced by two interfaces, that between the foot and the shoe and that between the shoe and the ground. This inevitably has consequences for foot function.

A flat shoe nailed onto the hoof decreases movement of the hoof during the impact phase of the stride,[11] though the heels are still able to expand.[12] It increases the maximum deceleration of the foot,[4] increases the frequency of vibrations within the foot as it strikes the ground,[13] and maximizes vertical GRF.[14] This influence of shoeing on the shock caused by foot impact decreases proximally to the point where the influence is minimal at the metacarpophalangeal joint.[14] Shoeing does not change the principal compressive forces within the hoof wall, although it does cause slight reorientation and decreases the variability of the stresses within the wall.[15] In addition, shoeing increases the pressure on the navicular bone by the DDFT[3] and accentuates the decrease in tissue pressure within the digital cushion that occurs soon after impact.[13] Shoeing increases stride duration, but not stride length, in young horses shod for the first time.[9] Shoeing and the addition of weight to a shod foot increase the animation of the gait.[5,9] Shoeing has minimal effect on the point of force during the stride.[16] However a horse shod with flat steel shoes is likely to slide farther after impact on a firm, flat surface, such as macadam or concrete, than would a barefooted horse.[17] It has been suggested that the rigidity of the shoe alters the way the hoof capsule accommodates to irregularities on the ground surface.[17] A rigid shoe causes the whole foot to tilt or twist, whereas the viscoelastic hoof capsule might permit local distortion of the hoof capsule with less displacement of the whole foot. Finally, shod horses do not wear the feet naturally. Although virtually no wear of the distal hoof wall occurs between the toe and the middle of the quarter, because there is no movement of the hoof wall against the shoe, the heels do wear against the shoe as they expand and contract.[17] Therefore during the course of a shoeing cycle, the angle of the dorsal hoof wall decreases.

It is my impression that shoeing over a prolonged period results in a thinner hoof wall. The quality of the wall adjacent to the shoe is not as good as that of the same horse barefoot. It is also my impression that the distal phalanx may change shape as the result of certain shoeing practices and imbalance. Mediolateral imbalance may change the shape of the distal phalanx.[18] Of greater importance is the effect of shoeing on the development of the feet of young horses. The feet finish growing when a horse is between 4 and 6 years of age. If abaxial movement of the hoof is restricted by shoes before that age, it may affect the growth of the hoof and potentially the shape and size of the distal phalanx. I prefer to allow young horses to go barefoot until they are mature if the intended use of the horse permits.

The deleterious effects of shoeing have led to a resurgence of interest in maintaining horses barefoot with routine trimming as necessary. Although there are unquestionable benefits to keeping a horse barefoot, maintaining a horse under modern management conditions so that its feet are in a condition to withstand the demands of work without becoming lame can be challenging.

Shoeing Sound Horses for Performance

Routine shoeing is directed at maintaining optimal balance and foot function and then addressing any specific needs that are related to the nature of work the horse performs. Balance is discussed elsewhere (see pages 252 and 253). The specific needs of a horse for a given type of work often have an established tradition. Observation of how other horses competing in the same discipline are trimmed and shod is a good starting point. Specialized farriers who shoe horses competing at high levels are a valuable resource. Fads are as much a part of the present history of shoeing as they are of many other disciplines and should be viewed with circumspection. However, although most fads disappear, a few become part of the standard armamentarium. In addition to traditional guidelines, there are rules regulating certain trimming and shoeing practices set by the governing bodies of different branches of equine sports and competitions. These frequently regulate the type of traction devices, weight of shoe, or length of toe that may be used. These rules generally are aimed at limiting excessive injury to the horses. For example, the use of toe grabs is prohibited in racehorses in certain states in the United States because of the established association between use and severe musculoskeletal injuries. Polo ponies are allowed to compete with inside rim shoes on the fore feet but not outside rim shoes.

Corrective Shoeing

Modification of a shoe in the pursuit of one objective almost inevitably has other consequences, which may be untoward. For example, to alter the mediolateral imbalance of a foot by decreasing the thickness of the web of one branch, either the width of the web can be maintained with loss of weight or the weight of the web can be maintained but the width must be increased. Changing the weight of the web alters the inertia about the distal limb. Changing the width of the web potentially changes the traction between the ground and the shoe and the depth the shoe sinks into the ground surface.

Corrective shoeing is directed at preventing lameness in a horse with poor conformation or balance (see pages 253 to 259) or treating a horse that has become lame. Therapeutic shoeing of lame horses requires knowledge of the cause of the problem and how to treat it. Lameness usually is related to pain that is often the response to inflammation after

low-grade repetitive injury, insult from stresses within tissues, or at the interface between tissues. These stresses may be compressive, tensile, bending, torsional, shearing, or a combination of these. For example, compressive strains within the sole result in bruising and in some horses, osteitis of the distal phalanx. Bending or shearing stresses in the wall may result in hoof cracks. Long-toe, low-heel conformation increases tension in the DDFT, compression of the navicular bone, and tension within the dorsal lamellae.

Accuracy of the diagnosis may be the first impediment. Sometimes pain causing lameness can be isolated to a specific tissue with a combination of physical examination, local analgesia, radiography, and scintigraphy, but frequently the pain can only be isolated to part of the foot, and sometimes simply to the foot. The precipitating factor may not be within the tissue or structure exhibiting pain but may be a conformational change elsewhere. The more accurate the diagnosis, the more closely localized the source of pain and the more knowledge of any underlying stresses, the more appropriate treatment is likely to be and the greater the chance of success.

The less specific the diagnosis, the more symptomatic the therapy. This is most likely to be directed at the balance of the foot and conformation of the limb, not necessarily an easy task. However, if lameness and pain persist after optimal balance is restored, the pain is most likely to be related, directly or indirectly, to the concussive forces associated with impact and weight bearing or flexion and extension of the distal limb. The ground surface of the foot can be protected from direct impact with the ground, and the maximal deceleration and frequency of vibrations within the structures between the ground surface of the foot and the metacarpophalangeal joint can be decreased. Extension of the distal interphalangeal joint can be decreased by reducing the extent that the heels sink into a soft ground surface and the moment about the distal interphalangeal joint at breakover. Torsional stresses within the distal limb may be reduced by encouraging the foot to break over in the most natural position.

Lameness Associated with Shoeing

Although the incidence of lameness associated with shoeing is unknown, poor trimming, shoe selection, and shoe attachment are well-recognized causes of lameness. Trimming that leaves a hoof too long or imbalanced, either mediolateral or dorsopalmar, predisposes the distal limb to abnormal stresses and lameness. Similarly, trimming the wall and adjacent sole too short causes undue pressure on the sole, bruising, and lameness.

Shoe selection includes size, weight, and traction devices. If the shoe is too small or short for the foot, the heels are unlikely to have adequate coverage. Pressure is concentrated in the wall and adjacent sole, which leads to bruising, hoof cracks, and underrun heels. Shoes that are too long or wide are at greater risk of being pulled off. If the ends of long branches are redirected axially to prevent the shoe from being pulled off, the angle of the sole may bruise. If the web of the shoe is narrow, the adjacent sole is unprotected. Conversely, a wide web shoe may impinge on a dropped sole.[19] Shoe size influences its weight. A heavy shoe may cause fatigue, decreased agility, and predispose to interference.[19] The size of a shoe also affects the effective surface of the foot. Too small a ground surface area concentrates the stresses of weight bearing.

Inappropriate use of traction devices predisposes to injury. The use of calks and toe grabs alters the balance of the limb and concentrates stress wherever they are applied. Too much traction may cause shearing within the limb as the horse places the limb on the ground because the limb decelerates too rapidly. Traction devices that anchor the limb in the ground once the foot is planted may cause fractures of McIII or the proximal and middle phalanges as a horse pivots on the limb. An inside rim shoe provides a good compromise among traction, ability to pivot, and ease of breakover. Too little traction at least decreases a horse's confidence as it works and at worst may cause a horse to slip and injure itself.

If a shoe is unintentionally set eccentrically, so that one branch extends further abaxial than the other, greater stresses occurs in the wall proximal to that branch. If the shoe is unintentionally rotated, the coverage of the heels is uneven and the heel with the least coverage responds as if the foot is shod short on that branch. If the shoe is to be rotated intentionally, a larger sized shoe should be used.

Inappropriate attachment of the shoe to the foot may cause direct injury to the underlying sensitive tissues, damage the hoof capsule, and impair expansion. The use of larger nails than needed displaces more tissue as they are driven and therefore is more likely to fracture the hoof wall or impinge on the underlying sensitive structures. Nails that directly injure the sensitive tissues when driven, called *nail prick*, cause an instantly recognizable problem that is immediately corrected. More insidiously, a nail that is close to, but not within the sensitive structures, called *nail bind*, applies pressure to the lamellae or may cause the inner hoof wall to fracture toward the lamellae. This creates a tract through which infection may become established. Nail prick and nail bind may occur because the nail was driven too high or was started too far axially. The angle at which the nail holes are punched and the location in the web of the shoe may contribute to the problem. Nails set behind the middle of the quarter adversely affect the expansion of the foot and should be avoided.

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NATURAL BALANCE TRIMMING AND SHOEING

• Gene Ovnicek

The feet of wild or domestic horses have the same anatomy, function similarly, and require stimulation to develop and perform optimally. The equine foot is a product of its environment, and the foot modifies and changes so that the basic requirements for health, function, and soundness can be met. This works well when the horse is roaming free in an area that is large enough for optimal foot maintenance, or if used aggressively barefooted. The sole, bars, and frog become callused and durable, with the wall chipping and wearing so that the wall at the toe is worn to the same level as at the sole. Feet managed in this self-maintaining manner are of the highest quality and generally are trouble free and healthy.

APPEARANCE OF A SELF-MAINTAINED FOOT

The wall of a self-maintained foot is worn down to the level of the sole callus in the toe region. The quarters are broken away at the widest part of the foot in horses that live in soft and sandy areas. This allows dirt to compact only in the palmar region of the foot, in the area of the bars. The heels grow beyond the height of the frog, which helps to form a trap for the dirt. The wall is worn to the level of the sole dorsal to the frog apex. The frog apex is generally in contact with the ground when the foot is loaded and is often enlarged and callused as a result of its use. Radiography shows that this area is in the center of the distal phalanx. Support for the distal phalanx is provided by the dorsal wall and sole callus, frog apex and frog buttress, bars, heels, and dirt compaction.

Fig. 27-11 This imprinted feral foot shows the wall is worn to the same level as the sole at the toe. This is typical of self-maintained feet of both feral and domestic horses.

Horses that are ridden or live in more abrasive areas, such as pavement or dry gravel, frequently wear the heels shorter than the level of the frog. The wall is always worn equal in height to the sole in the front part of the foot (Fig. 27-11). The thickness of the live sole callus (recognized as the functional epidermal sole tissue that extends beyond the dorsal distal border of the distal phalanx) at the toe, and the thickness of the live sole at the heel are a consistent distance from the distal border of the distal phalanx.[1] Therefore the sole is a reliable reference for mediolateral balance. The wall can be trimmed or rasped to the same distance from the live sole on each side of the foot so that the distal aspect of the distal phalanx is level, parallel with the sole.

The quality of the frog and digital cushion play a major role in dorsopalmar balance of the foot. The natural, healthy frog is designed to hold dirt between the bars and frog, and dirt seldom exceeds the level of the frog. The frog, along with the dirt compaction, is partially responsible for the alignment of the distal phalanx and the pastern. The frog, bars, and sole are all important in weight bearing.

The hoof wall breaks away while the sole and frog become callused and durable for the horse to walk on. With shod feet there is little control of the hoof wall trimming by wear and chipping. If the wall of a shod hoof is not prepared to an equal depth to the live sole plane medially and laterally, the coronary band distorts and quarter cracks and toe cracks may develop. Horses that break and crush the heels and those that develop toe cracks and quarter cracks all experience a natural but crude, deliberate way of trimming the foot so that the distal phalanx maintains a parallel medial/lateral orientation to the ground. Breaking and cracking of the wall allows the frog, bars, and sole to contact the ground to fulfill the natural function.

To maintain a horse barefooted without availability of a large, free roaming area, owners should try to keep the horse's living area and exercising area the same. If a horse is kept in a soft, sandy pasture or large, sandy paddock, then it should be ridden daily in that same type of sandy terrain. The same is true for horses kept in dry, rocky pastures. In addition to a consistent environment, regular activity also is required with exercise for 5 to 25 miles daily.

Most horses are housed in small, confined stalls or paddocks that are often soft, wet, and non-abrasive. The feet do not wear at all and adapt to that environment. These horses are reluctant to work barefoot on a more abrasive surface. Some horses have poor-quality, sub-standard feet that need some type of protection for any form of work.

NATURAL BALANCE TRIMMING FOR A BAREFOOTED HORSE

Natural balance trimming is maintaining the feet of domestic horses consistent with self-maintained feet. These instructions are for *domestic horses that are left barefoot*.

The majority of horses' feet can be separated into four types: (1) normal feet, (2) feet with underrun heels and long toes, (3) clubbed or upright feet, and (4) unusually flat feet. In all types the sole callus, the apex of the frog, and the callused portion of the frog buttress are the support structures of the foot (Fig. 27-12).

Fig. 27-12 Important support structures of the equine digit. *A*, Sole callus; *B*, apex of the frog; *C*, Callused portion of the frog buttress.

Normal Feet

The following trimming sequence is used for horses that have normal feet and are left barefooted without suitable activity to wear the feet to the natural hoof shape. After removal of dirt, the clinician should identify the sole callus in the toe area. This is the functional epidermal tissue that extends beyond the dorsal, distal border of the distal phalanx and is seen as the raised area just inside the hoof wall. The sole callus maintains its relationship with the distal phalanx at the 10-o'clock and 2-o'clock positions and adapts quickly to changes in the environment to best offer protection and support to the distal phalanx. The *live sole* is the functional epidermal sole tissue that extends beyond the distal border of the distal phalanx and has a waxy surface appearance. The clinician should remove only enough of the loose, flaky chalky sole material so that the live sole and the sole callus are clearly seen. Most horses left barefooted have little or no sole that needs to be exfoliated or removed.

If an imaginary line is drawn across the toe at the back edge of the sole callus, this is the place where natural breakover occurs in the self-maintained foot. With a normal foot the wall in this region is firmly attached to the sole callus at ground level. The sole callus on most normal bare feet is narrow and well defined, but not in flat or clubbed feet, because in these horses the toe of the distal phalanx is closer to the ground. The hoof wall should be conservatively rasped or nipped to the back edge of the sole callus. The rocker or roll should not exceed 10° to 15° from the flat plane of the sole (i.e., what is normally found on a well-worn shoe). Next, the wall is rasped or nipped down to the level of the sole callus on each side of the toe, behind the rockered portion. The hoof wall should be trimmed so the height of the sole callus and wall are equal. The length of that flattened area depends on the size and type of the foot and sole callus (2.5 to 3.5 cm).

The wall behind the toe callus is trimmed to the level of the live sole through the quarters. The heel that remains is flattened so that the heels are equal in height to each other and at the same level as the frog buttress or slightly shorter. This generally means that the quarters at the widest part of the foot are floated.

Only the cleft of the central sulcus of the frog is routinely trimmed to lessen the chance of bacterial colonization in less active horses. The rest of the frog should not be trimmed at all, unless parts are hanging by a small attachment from the live frog structure. The bars are trimmed only if they start to turn, roll over and become flat to the sole, or if cracked or diseased. If flares exist on the outer hoof wall, the clinician should find the most prominent growth ring near the middle of the dorsal hoof wall and remove only the amount necessary to make the wall straight from top to bottom. Rasping should not extend beyond the white line. Finally, the outer rim of the hoof wall that is closest to the ground is rounded (the rim is chamfered).

Flat Feet

Horses with flat, sensitive feet that are used for trail riding and multi-terrain activities often are unsuitable to leave barefoot. Many horses with flat feet have a thin sole that separates from the wall at ground level, causing laminar tearing at the distal aspect of the distal phalanx. With the sole callus used for weight bearing, the outer wall is removed in a dubbed, vertical manner to lessen the pull on the wall. The wall is brought back very close to the edge of the sole on the ground side so that no dirt will pack under the wall next to the sole. The sole of the foot is never touched with the knife or rasp. Feet of this type never need exfoliating. They need more sole thickness below the distal phalanx, which may develop if the wall is reattached closer to the ground surface of the sole. When the distal 3 to 4 cm of the wall is left to full thickness, the distal phalanx is supported proximally by stable wall. The sole callus may become more durable and develop dense protective tissue once the laminae are not torn by the wall pulling away from the sole.

The heels are rasped back to a solid horn structure and the frog is left untouched. The clinician should not rocker the toe until the wall attaches more normally to the sole at ground level. With repeated trimming the gap between the sole and wall disappears and eventually the callus at the toe quarters bonds tightly with the wall, and the solar surface may become concave.

Clubfeet

An upright or clubfoot often is smaller. The practitioner should not try to change it but treat it separately, using the same guidelines as for a normal foot. The sole callus at the toe is located to determine the point for breakover. The live sole in the heel region is used as a guide for trimming the heels, leaving the sole full thickness to protect the distal border of the distal phalanx. In my opinion, the most fragile part of the horse's foot is the distal border of the distal phalanx; in an upright foot, it may be more susceptible to trauma. However, if the heel is lowered excessively while the toe is left to

improve the digital alignment, damage to the distal border of the distal phalanx is more likely.

A normal foot has an even curve to the outer hoof wall at the heel buttress and an even arch to the bars with the heel buttress terminating slightly ahead of the back of the frog. The heel buttress (end of heel) of an upright foot has an abrupt curve with bars that are quite straight. The heel ends close to the back of the frog. Excessive removal of the heel of a clubfoot does not allow the horse to land heel first and increases the chances of distal phalanx trauma from landing toe first.

The sole callus on a clubfoot is slightly different from that of normal feet. There is usually a broad, raised formation to the sole, seen just ahead of the frog apex. The callus on each side of the frog apex is more prominent and extends well behind the tip of the frog. The natural place for breakover is closer to the frog apex because of the position of the sole callus. Therefore the wall is rockered ahead of the sole callus just as in a normal foot. The live sole in the back part of the foot is deeper. The hoof wall at the heel should not be trimmed equal to the live sole. The sole callus continues palmarly to the widest part of the foot and beyond, giving the appearance of a flat, thick sole from heel to heel. If the dorsal hoof wall is severely flared and resembles a foot with chronic laminitis, the flare should be removed.

Feet with Long Toes and Underrun Heels

Underrun heels grow forward under the foot with a sharp curve in the heel. The underrun heel ends ahead of the frog buttress with bars that are curved similarly. The frog apex is often elongated. This heel conformation is abnormal and often is painful. Natural balance trimming helps to restore the foot to a near natural shape, with alleviation of pain. The sole callus is broad and looks more like a small mound around the sole ahead of the frog apex. If the horse can be kept in a dry, soft area for 1 to 2 weeks, the toe can be aggressively rockered ahead of the callus, leaving the sole callus and medial and lateral walls to walk on. The heels need to be trimmed back below the level of the frog if the bars and heels are severely curled and appear to end in front of the back of the frog. However, the heel should never be trimmed down past the live sole at the heel or any other part of the foot. The wall is finished normally. This aggressive trimming rapidly starts to repair the deformed feet and can be done quickly and successfully as long as the bottom of the foot is hardened and protected with hoof and sole hardeners. Alternatively, more wall can be left at the medial and lateral sole callus to not overload the sole callus. The foot will respond well with each trimming.

NATURAL BALANCE SHOEING

When preparing the sole surface for a shoe, the frog, bars, and sole are prepared conservatively, similar to feet that are left bare. Feet that are shod do not exfoliate effectively, and sole material that is showing cracks in the sole and is chalky and crumbles when it is cut with a knife should be removed. When the sole and frog material changes from a chalky, crumbly state to a waxy-appearing surface, the live or functional sole and frog has been reached, and absolutely no more cutting should be done. The live, functional sole at the toe quarters (where the sole callus is) and the live sole at the heel quarters (behind the widest part of the foot) are references to the distal border of the distal phalanx. Trimming the wall to these live, functional structures offers the best guide to attain accurate medial/lateral and dorsopalmar balance.

The bottom surface of the foot is finished flat when the foot is prepared for shoeing (not relieved in the quarters as when trimmed to be left barefoot) and is hot seated if possible. Vigorous hot seating helps to dehydrate and strengthen the sole callus. It also pulls the sole proximally from the ground level to eliminate sole pressure.

Fig. 27-13 The aluminum Natural Balance Shoe is designed to be applied 0.3 to 0.9 cm (depending on foot size) from the frog apex to the inside edge of the shoe at the toe. This placement closely meets breakover requirements with respect to the sole callus. The seated-out reverse arch on the inside border of the shoe at the toe helps to protect the distal border of the distal phalanx from sole pressure.

Shoe Placement and Application

Shoe selection is important, and wide-web rim type shoes work best for easy modification. The outer rim is normally tapered-in to the nail groove, which is helpful and some what mimics the way the bare foot naturally wears. That same feature is equally helpful at the toe when the shoe is squared somewhat and positioned on the foot so the breakover point of the shoe fits directly over the back edge of the sole callus at the center of the toe. The heel of the shoe should extend to the full length of the frog. A good reference for that position is the back of the crease in the central sulcus. Radiographs can be used to determine the natural position for breakover.

When pre-made aluminum (Thoro'Bred Race Plate Company, Inc.; Anahaeim, CA) or steel (Malaysian Horseshoe Company; Malaysia) Natural Balance Shoes are used, the same criteria of shoe placement for breakover and heel length should be followed. A wide-web rim shoe is broadened at the toe and tapered from the inner rim to the toe between the toe quarters. The shoe is placed on the foot so that the inner rim (part of breakover) is over the inside edge of the sole callus. The Natural Balance Shoe is positioned a variable distance from the frog apex to the inside edge of the shoe for placement (Fig. 27-13). That distance is regulated with the heel position. If a line is drawn across the widest part of the foot where the bars end, one third of the foot mass is ahead of this line to the point of breakover.

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HOOF RECONSTRUCTION MATERIALS AND GLUE-ON SHOES

• Robert Sigafoos

Advancements in adhesive technology have substantially improved the dependability of adhesive-bonded shoes. The ability to control plasticizer migration in adhesives has allowed the use of plasticizers in industrial adhesives, increasing adhesive flexibility and impact resistance without inhibiting adhesive strength at the bond line interface.

ADHESIVE TYPES

Most adhesives used to bond hoof wear to hooves evolved from use in hoof reconstruction.[1] Important features include impact resistance, the type of adhesive joint loads, speed of polymerization, heat production, surface sensitivity, and environmental compatibility.

Impact resistance is particularly important because of factors such as horse's weight, speed, and high level of cyclic loading of the feet. Adhesives that remain flexible once polymerization is complete (elastomeric adhesives) perform better than rigid adhesives with similar tensile strength characteristics. When the bonding is applied directly to the hoof wall (as opposed to bonding individual components of a shoe together), at least one surface (the hoof) is always flexible. The primary load will be in peel, for which elastomeric adhesives are preferable.

Speed of polymerization is critical because the horse needs to bear weight and ambulate immediately after shoeing. The cure profile of an adhesive involves a "green" phase when the adhesive has solidified, but not reached full strength. Challenging the bond in the early portion of the green phase can cause irreversible bond line failure. However, very fast polymerization may be at the expense of reduced impact strength.

The polymerization reaction produces heat that increases with the speed of the reaction. It is surface area dependent for a given volume of polymer. In concentrated volumes that are not allowed sufficient surface area to dissipate heat, temperatures can exceed 120° C in volumes typically used for hoof reconstruction or bonded shoes. Spreading the adhesive to provide increased surface area relative to volume allows dissipation of heat. Some adhesive manufacturers add fillers to very fast systems to dissipate heat, resulting in a temperature reduction as much as 25%. Submural temperatures underlying reacting exothermic resins vary depending on the hoof wall thickness but generally do not achieve dangerous levels. However, dermal layers underlying thin hoof walls (such as those found at the quarters, or in foals) may be at risk to substantial thermal trauma.

Surface sensitivity affects the ability of an adhesive to bond to a contaminated surface. Adhesives that solvate surface contaminants into the bond line are preferable. The adhesive must be resistant to moisture and microbial degradation. The adhesives commonly used for hoof reconstruction and bonded shoes are polymethyl/cyclohexyl methacrylate, cyanoacrylate, polyurethane, and, to a far lesser extent, epoxy resins. Each has distinct advantages.

Polymethyl/cyclohexyl methacrylate (PM/CHMA) systems are commonly referred to as *acrylic* adhesives. The newest elastomeric acrylic adhesives offer good impact resistance, rapid cure, minimal surface sensitivity, and excellent wetting characteristics for hoof wall and other substrates commonly used with hoof care applications. However, they have an intense odor, relatively high exothermic temperatures, and a high vapor pressure that limits shelf life in opened containers. As with all elastomeric adhesives subjected to high peel loads, the acrylic adhesives should be used with a thick bond line.

Polyurethane adhesives produce low-modulus adhesive joints. These joints exhibit excellent impact strength and perform remarkably well under high cyclic loads. Polyurethane has the best abrasion resistance and shock attenuation of all the adhesives commonly used. However, these adhesives require extensive substrate cleaning and preparation to ensure good bonds, which is often beyond the scope of practical field applications. These adhesives also do not form effective bonds with many of the plastics commonly used with hoof care, including acrylonitrile butadrene styrene (ABS), polyvinyl chloride (PVC), and acrylic/PVC copolymers.

Thermoplastic adhesives are principally dependent on mechanical bonding for adhesion, limiting structural use in horses.

Cyanoacrylate adhesives are thermoplastic, single-component systems that are cure inhibited through acid stabilization. Ambient surface moisture increases constituent pH and allows the polymerization process to begin. The principal advantage of these adhesives is the ability to form bonds with substrates that are difficult to bond for most adhesives. Cyanoacrylates are the only adhesive for many types of glue-on shoes that have polyurethane as a structural bonding substrate. They have excellent strength when loaded in sheer but poor peel and impact strength. Cyanoacrylate adhesives also are highly susceptible to post-cure, moisture degradation. Because cyanoacrylates become rigid after curing, the bond line rapidly develops a "mosaic" fracture pattern when exposed to impact. This allows capillary intrusion of water into the bond line, further subjecting the bond to environmental degradation. They also have limited gap-filling characteristics. The ideal surface for successful cyanoacrylate bonding is a virtually polished surface. The bond line must be very thin.

Epoxy resins offer excellent environmental resistance, very good sheer characteristics, and are the adhesive of choice when assembling shoe components that involve engineered fabric lay-ups and when an extended or elevated temperature are curing is acceptable. These systems are not useful when bonding directly to the hoof wall, because the hardeners that are commonly used markedly increase the rigidity of the cured polymer.

Polyester resins have some value in cosmetic repair of hoof wall, but they lack tensile strength and environmental resistance and have limited use.

Shoe Types

Currently four principal types of glue-on shoes are available. These include the "direct glue" method using PM/CHMA adhesive, the molded polyurethane "tab type" shoes that use a cyanoacrylate adhesive, the flocked-lined plastic cuff that used an epoxy adhesive, and the fabric cuff that uses a PM/CHMA adhesive.

Two techniques have been used for direct-glue shoes. The first method involves the use of a PM/CHMA adhesive without fillers, with the bond line between the distal aspect of the hoof wall and the shoe.[2] The hoof side of the shoe is cleaned and sanded. The hoof is prepared by cleaning the dirt and loose debris from the wall and sole. The adhesive then is applied directly to the shoe and hoof, and the shoe is positioned on the hoof so that the bond line is continuous from heel to heel and incorporates an increasing percentage of the sole toward the heels. The hoof must be held non–weight bearing through the green phase of the adhesive cure cycle, approximately 3 to 5 minutes depending on ambient temperature. Given the extended cure profile of this type of adhesive, the bond should be challenged as little as possible for 12 hours. A modification of this system has been developed using

staple fiberglass fibers as a filler for the adhesive.[3] The primary disadvantages are the need to keep the hoof non–weight bearing until the adhesive has green cycled and the need to bond the heels securely to the shoe.

Polyurethane "tab type" shoes use a cyanoacrylate adhesive to bond a component of the shoe (the polyurethane tab) to the hoof wall. These shoes require careful substrate preparation, including solvent cleaning and finish sanding with extremely fine sand paper. Because cyanoacrylates have limited gap-filling properties, the prepared hoof wall must match the profile of the tab precisely. Since these adhesives have limited moisture and impact resistance, shoe retention for competitive animals may prove difficult.

Plastic flock-line cuffs (Dalric Glue on Shoes, Advance Equine, Versailles, KY) are not actually shoes, but they act as a conjoining device to attach shoes to the dorsal aspect of the hoof wall. These devices use an epoxy (or a PM/CHMA adhesive) to attach the shoe to the hoof wall through a mechanical lock of the adhesive to the flock lining and rivets to attach the shoe to the cuff. This system is considerably more robust than the polyurethane tab system described previously. However, fitting the cuff to oddly configured hooves can be difficult.

The fabric cuff (Sigafoos Series Adhesive Bonded Shoes, Sound Horse Technologies; Unionville, PA) system uses a PM/CHMA adhesive to bond a braided fabric cuff that is an integral part of the shoe to the dorsal aspect of the hoof wall. This system comes as a fully assembled shoe (Series One) or a modular system (Series Two) that allows the farrier to assemble any type of pattern configuration desired. The primary disadvantage of this system is the limited choice of types of shoes currently available in the Series One system.

Glue-on shoes offer distinct advantages over mechanically attached shoes because of the non-invasive and non-destructive nature of the attachment. They are expensive, but this cost usually is recouped if the actual cost of lost shoes and the resultant hoof loss is considered. If the widespread acceptance of adhesives in other industries is any reflection on the potential for its use in the farrier industry, adhesives will become the dominant method of attachment of shoes for horses in the foreseeable future.

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CHAPTER • 28

Trauma to the Sole and Wall

Robin M. Dabareiner, William Moyer, and G. Kent Carter

PROBLEMS ASSOCIATED WITH HORSESHOE NAILS

Nail Bind

History and Clinical Signs A *close nail* or *nail bind* refers to placement of a horseshoe nail not necessarily in the sensitive structures of the hoof but close enough such that the nail exerts pressure on these structures to cause discomfort. The horseshoe nail is designed to be driven obliquely through the hoof wall. When the nail is driven, the tip is placed at the inner edge of the white line with the bevel of the nail tip facing inward. When driven, the bevel contacts the hard hoof wall and curves outward and exits 1 to 2 cm above the level of the shoe. The tip of the nail is removed and the remainder is bent over to form a clinch to hold the shoe firmly to the hoof. Correct nail placement is important because if the nail is placed too shallow (superficial), the hoof wall will weaken and possibly split; if is it placed in too far, the sensitive structures of the hoof may be entered (pricked) (Fig. 28-1). Overzealous clinching of the nails causes inward bending of the nail that can result in pressure on sensitive tissues, which may result in immediate or delayed pain and lameness. Slight displacement of a shoe also can result in nail pressure on sensitive tissues.

Diagnosis Diagnosis of nail bind is difficult and often determined by eliminating other causes of foot pain. Lameness varies from subtle to severe. Sometimes a horse exhibits a change or lack in performance. The problem may not arise until several days after shoeing. Sometimes a horse is sound when trotted in a straight line, but it shows lameness when pulled in a tight circle or when making a turn. It is important to realize that nail bind is not always associated with poor farriery. A good nail can become a problem nail days or weeks after shoeing if the shoe shifts, causing abnormal nail pressure, or if the horse had a poor quality hoof wall and hoof wall loss occurs. This is a common problem in Thoroughbred (TB) racehorses and usually involves

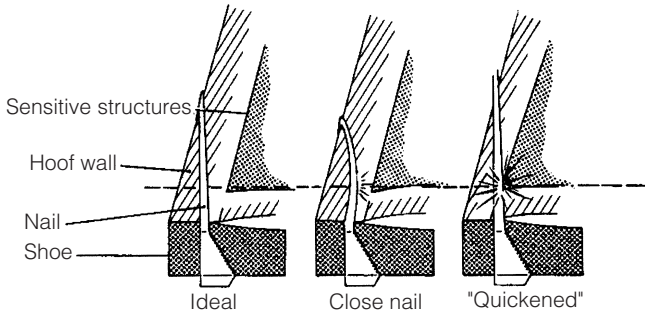

Fig. 28-1 Schematic of ideal, close, and pricked (quickened) horseshoe nail placement.

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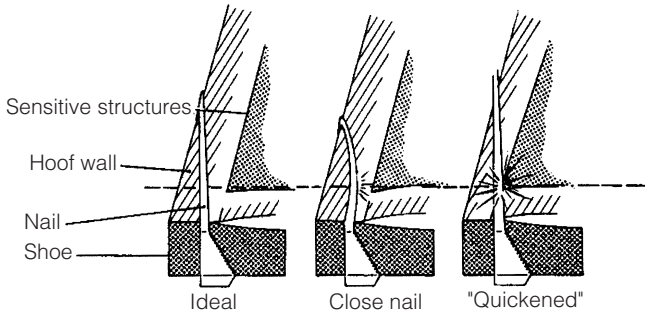

Fig. 28-1 Schematic of ideal, close, and pricked (quickened) horseshoe nail placement.

the inside heel nail (the inside quarter and heel wall usually is the thinnest aspect of the hoof wall). Hoof tester evaluation using both pressure and percussion may cause a painful response over the offending nail. Heat and increased digital pulse amplitudes may not be present, depending on the duration of the close nail. Often diagnosis is determined by pulling single nails one by one from the shoe and evaluating the lameness after each nail is removed. Paradoxically, lameness is sometimes transiently accentuated after removal of the offending nail.

Treatment and Prognosis The treatment of nail bind is removal of the offending nail; usually lameness resolves within a few days. No additional treatment is usually needed. Prognosis is good once the nail is removed provided infection does not ensue.

Nail Prick

History and Clinical Signs *Nail prick* or *quicking* refers to penetration of the sensitive hoof structures, usually the sensitive laminae, by a driven horseshoe nail (see Fig. 28-1). The horse usually reacts as the farrier drives or clinches the nail by jerking the foot from the farrier.[1] Sometimes blood appears on the nail or leaks from the nail hole. Nail pricks occur for many reasons and are not always caused by a misdirected nail. Poorly made shoes, misdirected nails, selection of a nail that is too large, poorly placed nail holes, and faulty nails can result in a nail prick. Horses with poor hoof quality, thin hoof walls, or flaring hoof walls can be very difficult to nail and thus are at greatest risk. Fractious horses and young horses that have not been previously shod may lean on the farrier or repeatedly pull the foot from the farrier, making driving a nail difficult. A rushed farrier predisposes to nail pricking, but it can also happen to the best of farriers. Damage from an improperly driven nail can vary from minimal to serious infection.

Diagnosis Diagnosis of a misdirected nail warrants great diplomacy from the veterinarian, because many owners become unjustifiably upset with the farrier. Some horses repeatedly stomp the affected foot or paw the ground immediately after shoeing. Others point the affected limb after shoeing. Lameness may not be apparent immediately but may occur days after shoeing when the nail hole becomes infected and the trapped pus begins to exert pressure. The horse usually becomes acutely lame and the lameness worsens over time unless it is treated. An infection may migrate up the lamellae (white line) and create an abscess or soft spot at the hairline of the coronary band. The abscess is directly aligned with the hoof wall tubules leading to the infected nail hole, which is an important diagnostic aid. Hoof tester examination, using both pressure and percussion over each nail, is essential to locate the offending nail. Increased digital pulse amplitudes and heat may be present.

Treatment Pricks from nails can be potentially serious and require immediate treatment. If the nail prick is discovered by the farrier at the time of shoeing, the nail is removed. The nail should be examined for moisture or blood. The nail hole can be irrigated with dimethyl sulfoxide, povidone-iodine solution, or hydrogen peroxide. The nail hole is packed with iodine-soaked cotton and left open. Often the nail is redirected and no further treatment is needed. Tetanus prophylaxis is essential for an unvaccinated horse.

If the offending nail cannot be localized or the nail hole is infected, the shoe is removed. Hoof testers then are used to localize the painful nail hole. Many times the pressure from the hoof testers cause black, malodorous liquid pus to exit from the hole. This may not be obvious immediately, but if the foot is replaced to the ground and the horse walks a few steps, pus may become obvious. The basis of treatment is to establish drainage. The infected nail hole often requires enlargement with a loop hoof knife or curette. Ideally a cone-shaped hole is made with the larger opening at the bottom of the hoof. The hole is irrigated or the entire foot is soaked in an Epsom salt and povidone-iodine foot bath for 20 to 30 minutes twice daily until the infection is resolved. It is important to protect the foot from the environment (mud, dirt) by keeping the foot bandaged between foot soaks. Alternatively, a poultice can be applied to the foot for several days. Additional medications usually are not necessary unless infection is widespread. Anti-inflammatory medication may be beneficial to decrease pain. Once the infection has cleared, the shoe is replaced. The affected nail hole can be packed with iodine-soaked cotton and the horse re-shod with a plastic pad covering the sole. Alternately, a hole can be drilled into the shoe over the affected nail hole and the shoe can be replaced, leaving access to the infected area for daily irrigation and povidone-iodine packing.

Prognosis Prognosis after nail prick usually is good, provided that minimal damage occurs to vital structures of the foot. Establishing drainage for infection is important to avoid potential complications, such as infectious osteitis of the distal phalanx or infection of the distal interphalangeal joint.

Solar Bruising

History and Etiology A *bruise* is a contusion or impact injury that causes focal or generalized damage with subsequent hemorrhage of the solar corium. Sole bruising occurs commonly in all types and breeds of horses, especially in racing TBs and Standardbreds (STBs).[2] The degree and severity of lameness varies from acute, severe lameness to chronic mild or intermittent pain, depending on location and degree of damage. It is important to determine the cause of the bruise, because this dictates proper treatment and prevention. The general cause is abnormal focal weight bearing on the solar surface of the foot. The location of the bruise is helpful in determining the cause of the injury. The most common location is the junction between the bars and the walls of the heel, termed a *corn*.[3] Corns occur most frequently on the medial side of the front feet. Heel bruising may be the result of improper shoeing or trimming. Some farriers bend the medial branch of the shoe toward the frog to prevent the horse from stepping on and pulling the shoe. This shoe position causes direct pressure to the sole at the heel angle, instead of the heel wall, resulting in continued concussion and bruising. A shoe that is too small or does not extend far enough back under the heels can lead to heel bruising.[4] The ends of the shoe should extend to the widest aspect of the frog for proper heel protection. Horses with long-toe–low-heel hoof conformation are susceptible to heel bruising. Toe bruising can be caused by excessive impact or weight bearing on the toe region secondary to another cause such as heel pain. An improperly positioned horseshoe that rests on the sole instead of the hoof wall also causes toe bruising. Horses with long toes and those shod with toe grabs concentrate impact at the toe region.[4] Sole bruising occurs often in horses with flat feet because the sole repeatedly strikes the ground surface. A flat foot can be congenital or created by trimming the hoof wall too short or excessive quarter wall breakage. Thin-soled horses or excessive trimming of the sole reduces the sole protection and predisposes to sole bruising. Loose shoes can shift position, and improperly balanced feet cause excessive impact forces to specific regions of the foot and cause bruising.[4] Riding on hard and rocky ground can result in stone bruises. A shod foot that has overgrown to the point where the shoe is riding on the sole is at risk of bruising.[4]

Clinical Signs The degree of lameness from sole bruising can change daily and lameness varies between horses. Removal of the shoe usually increases the degree of lameness. The bruising can be acute or chronic depending on the cause. Digital pulse amplitudes are increased after exercise, and careful hoof tester evaluation often reveals a focal painful response.

Discoloration is common, but if the bruise is chronic, deep, or if the horse's sole is pigmented, it may be difficult to identify. Bruising often affects several feet. In most horses, lameness will improve after perineural analgesia of the palmar digital nerves. Radiographic changes are rare but may reveal a serum pocket (fluid line) between the distal phalanx and external sole. Persistent, chronic bruising may lead to osteolytic lesions or solar margin fractures of the distal phalanx.

Treatment Initial treatment with phenylbutazone (2.2 mg/kg bid) and soaking the feet in Epsom salts help decrease inflammation. Corrective or proper shoeing is imperative to shift the weight-bearing forces away from the damaged area of the foot. Sole paint consisting of a combination of equal parts of phenol, iodine, and formalin can be applied to toughen the solar surface. Hoof balance problems and shoeing causes, such as heel calks, toe grabs or tucked heels, should be eliminated. One of many different shoeing techniques then is used to decrease impact and protect the bruised area. One method is use of a rim pad that is cut out over the bruised area so that the affected heel or quarter is floated and thus receives minimal weight-bearing forces. Another method is application of a bar shoe with a deeply concaved solar surface to stabilize the foot and alleviate any sole pressure from the shoe itself. A wide web shoe may provide relief by covering and providing protection over a larger surface. It is important that this shoe be properly positioned on the hoof wall so that it does not increase sole pressure. Application of full pads packed with silicone or oakum may provide temporary relief by distributing weight-bearing forces over a wider area but often leads to a weakened and softer sole, causing recurrent problems.[5]

Prognosis The prognosis is good if the inciting cause of the bruising can be corrected. Corrections may be difficult in horses with flat feet or long-toe–low-heel conformation, and such horses often have recurrent solar bruising.

Thrush

Thrush is a bacterial infection characterized by an accumulation of black, malodorous, necrotic material, usually originating within the central or collateral sulci of the frog of the hoof. This degenerative condition may spread to involve deeper structures of the foot, such as the digital cushion, hoof wall, and heel bulb region, causing inflammation and breakdown of these structures.[6] Many keratolytic organisms may be present, but *Fusobacterium necrophorum* is often isolated. Thrush is most often caused by poor environmental conditions; horses standing in soiled stalls, deep mud, swampy land, or wet pastures are at risk, especially if the feet are not cleaned daily.[7] Poor hoof conformation also predisposes to thrush. Saddlebreds, Tennessee Walkers and other gaited horses, and some Warmblood breeds have long feet with naturally deep frog sulci and are at risk of thrush.[7] Horses with sheared heels or acquired frog deformity also are predisposed. Horses shod with full pads may develop thrush secondary to moisture and dirt collection under the pad. Other well-kept, clean horses can develop thrush for no apparent reason.

Clinical Signs and Diagnosis Lameness often is not apparent, but if present, the severity can vary. With severe thrush lameness can be obvious, but in most horses thrush is an additional finding and the primary source of pain is elsewhere. Diagnosis is based on the presence of black, malodorous discharge located most commonly within the frog sulci. The central frog sulcus often is malformed and very deep. A painful response may occur when the affected sulci are cleaned, because the degenerative process may extend to sensitive structures of the foot. If structural damage has occurred, the heels may move independently of each other, causing pain on manipulation.

Treatment The predisposing cause should be identified and, if possible, removed. The horse should be moved to a clean, dry environment and the feet should be cleaned daily. Any necrotic debris and undermined tissue are carefully debrided and cleaned using a hoof knife. Foot bandages may be necessary if the debridement is extensive. Systemic antibiotics may be necessary if deep or more proximal tissues are affected, but infection is usually managed by topical medication. Several caustic materials have been recommended, including a combination of phenol, tincture of iodine, and 10% formalin, Kopertox solution (Fort Dodge, Fort Dodge, IA), or methylene blue. Others have recommended foot soaks in chlorine bleach (30 ml of bleach in 5 L of water). Corrective trimming and farriery may be necessary. If heel instability is present, a bar shoe may be necessary to stabilize the palmar aspect of the foot. Exercise is important to strengthen the palmar aspect of the foot and will naturally clean the feet.[7] The best treatment for thrush involves prevention by educating the client on proper hoof hygiene.

Prognosis The prognosis for horses with thrush is favorable if the cause can be identified and eliminated and if the condition is treated before extensive hoof damage has occurred.

Sheared Heels

History and Clinical Signs *Sheared heels* refers to instability between the medial and lateral bulbs of the heel. Mediolateral foot imbalance may be a predisposing cause. It is frequently but not invariably associated with distortion of the hoof capsule. The medial bulb of heel often is displaced proximally, with a steep medial wall and flaring of the lateral wall (see Fig. 6-3). However, instability between the bulbs of the heel can also occur in a more normally conformed foot, and distortion of the hoof capsule as previously described is not synonymous with sheared heels. It is also important to recognize that sheared heels can be present without causing lameness, although sheared heels may be a cause of lameness.

Sheared heels may be present in one or multiple feet and may be associated with mild to moderate lameness. Lameness is usually worst on firm footing. There may be distortion of the coronary band, which usually is higher medially. There may be a deep cleft dissecting between the medial and lateral bulbs of the heel. Sheared heels may predispose to thrush.

Diagnosis Instability of the bulbs of the heel is detected by grasping each bulb of the heel with the left and right hands and twisting each bulb in opposite directions in a shearing motion. In a normal horse the bulbs of the heel cannot be moved independently. Considerable independent motion can be associated with pain causing lameness. However, if the lameness is severe, another co-existing cause of lameness should be considered. Lameness associated with sheared heels is removed by perineural analgesia of the palmar digital nerves.

Treatment and Prognosis Any mediolateral or dorsopalmar foot imbalance should be corrected. The affected foot should be shod with a bar shoe to provide stability to the heel region. This may need to be continued for many months, and occasionally indefinitely, until some physical attachment between the heel bulbs has become established. If the hoof capsule is distorted in shape as previously described, the medial branch of the shoe should be set slightly wide to encourage the medial wall to grow down to it and prevent it from collapsing laterally. Any excess flare on the lateral wall should be removed. The prognosis is generally good.

Hoof Wall Separation (White Line Disease, Seedy Toe)

History and Clinical Signs The white line, visible at the sole, is created by the junction of the insensitive laminae of the hoof wall and the horn of the sole. *White line disease* has historically been a term to describe the separation of the hoof wall from its laminar attachments. A crack or opening occurs

within the white line, allowing a bacterial or fungal infection to invade the stratum medium, with proximity to the laminae causing cavities to develop between the laminae and outer hoof wall.[8] Environmental conditions of either too much moisture (continuous wet pastures) or drought conditions producing excessively dry feet predispose to development of a crack or opening in the white line. Horses with poor-quality hoof walls that split or crack or those with chronic laminitis and a thickened or stretched white line in the toe region may develop white line disease. The term *seedy toe* has been used differently in North America and Europe. In North America it is used to describe thickened or stretched white line at the toe in horses with chronic laminitis, whereas in Europe it is used to describe separation at the white line, filled with crumbly material, that is not associated with laminitis. The hoof wall separation usually is a chronic condition beginning weeks or months before veterinary advice is sought, since there usually is no associated lameness. Hard ground may exacerbate any lameness seen.

Diagnosis The degree of lameness varies, but if severe, white line disease can cause clinical signs of pain. However, the clinician should resist the temptation to incriminate this disease as the primary source of lameness until a thorough lameness examination is completed. Because lameness is abolished after palmar digital analgesia or a dorsally directed ring block, this disease can easily be confused with the many other conditions of the foot. Visual examination of the white line, assisted by a probing instrument, reveals a cavity with separation of outer hoof wall from the laminae. Radiographic evaluation determines the full extent of hoof wall separation. Often the cavity is either dry or filled with necrotic debris, which may involve a bacterial or fungal infection. The cavity is usually not painful to probing.

Treatment The separated outer hoof wall is removed using hoof nippers, hoof knife, and motorized tools. The aim is to remove cracks or crevices that could harbor bacteria. A Dremel tool burr (Dremel, Racine, WI) is useful to smooth any cracks in the insensitive laminae that are exposed after hoof wall removal. Large defects in the hoof wall require protection. A heart bar shoe redistributes weight-bearing forces to the frog and palmar region of the foot and away from damaged and weakened areas. The hoof wall defects prevent normal nailing procedures; therefore clips can help secure the shoe to the hoof. After hoof wall removal the exposed laminae may still have an active infectious component. The horse should be kept in a clean, dry stall and the exposed laminae are treated topically with iodine or merthiolate daily for 10 days or until they are dry. The horse may then be a candidate for prosthetic hoof wall repair using a product such as Equilox (Equilox International, Pine Island, MN). The plastic acrylic is trimmed and shaped to the horse's natural hoof wall at the next shoeing. The hoof to which the acrylic was applied should be kept dry to avoid losing the acrylic patch. The horse may return to normal activity once the prosthetic patch is in place.

Prognosis Prognosis depends on response to treatment and cause of the original problem. Horses with poor hoof quality or primary laminitis often have recurrence. If the horse responds to original treatment and environmental conditions improve, prognosis is good.

Poor Hoof Wall Quality

Poor hoof quality plagues many horses. Many factors determine hoof quality, including the environment, farrier management, hoof conformation, and use of the horse. Drought conditions may result in dry, brittle hooves that are prone to hoof wall splitting and cracking. Excessive moisture creates a weakened hoof wall that may flatten or collapse under normal weight-bearing forces. Improper trimming and shoeing methods can cause substantial hoof wall damage. Horseshoe nails that are placed too far outward in the hoof wall or exit too low in the hoof wall weaken the hoof wall and cause splitting and cracking. Many TB horses with long-toe–low-heel hoof conformation often have collapsing heels and thin, weak hoof walls that make proper nail placement and farrier management difficult. The most common cause of weak, poor-quality feet is lack of exercise and stall housing.

Diagnosis Diagnosis is somewhat subjective as poor hoof quality is usually in the eye of the beholder. There are no objective criteria for determining hoof quality. Diagnosis is usually made by visual examination of the foot. Communication with the farrier is imperative for the diagnosis and future treatment plan. Horses with poor hoof wall quality often have palmar heel pain or are prone to sole and heel bruising. They are prone to losing shoes, which can accentuate the problem.

Treatment If possible, the cause of the poor hoof wall quality should be corrected if identified. In drought conditions, painting the entire hoof and coronary band daily with a lanolin-based hoof dressing is sometimes beneficial. In an excessively wet environment, confining the horse to a stall and avoiding standing water or wet pastures may improve hoof wall quality. Poor farriery can be addressed, but in many horses poor hoof conformation and poor environment are more difficult to manage. Long-toe–low-heel hoof conformation should be balanced as much as possible with proper trimming of the heels back to the widest portion of the frog. The toe of the foot is shortened as much as possible and a rockered, round, or squared-toed shoe is used to further decrease toe length and ease breakover. Egg bar shoes are used if more palmar heel support is needed. Access to the outdoors and being able to move about are helpful but often difficult to achieve.

Hoof quality also is a function of proper diet and exercise. The horse's nutrition should be evaluated, paying particular attention to protein quantity. Biotin supplements may be beneficial.

Prognosis Poor hoof quality usually is not corrected but managed. Prognosis is generally unfavorable unless a specific cause can be identified and eliminated.

PENETRATING INJURIES OF THE SOLE

Sub-Solar Abscess

History and Clinical Signs Sub-solar abscess (gravel) is one of the most common causes of acute lameness in all horses. Sub-solar abscesses may originate from a penetrating wound in the white line, nail hole, or deep sub-solar bruise. A cause may not be identified. Lameness is usually acute and severe (grade 3 to 4 of 5) and may worsen over time until drainage is established. Lameness that develops during work or when a horse is turned out may falsely lead to the suspicion of a traumatic injury. The horse often points and may not bear full weight on the affected limb. Distal limb swelling often accompanies a sub-solar abscess that has not drained, leading the owner to suspect tendon injury. Systemic signs of infection (fever, lethargy) may be present if deeper structures are involved. The infected tract may migrate and open at the coronary band. Before breaking open, a soft, painful area can be located by digital palpation of the coronary band.

Diagnosis Digital pulse amplitudes are usually increased and the hoof capsule may have heat. A focal painful area can usually, but not always, be located with careful hoof tester examination. Careful paring of the sole and frog may be helpful in locating the abscess, but the clinician must be careful *not* to damage good, healthy tissue while looking for the infection site. Unnecessary, aggressive paring may lead to

large painful areas that take months to heal. Foot poultices and hot water foot baths with Epsom salts help to eventually localize the affected area, especially in horses with hard horn. Grey or black, malodorous liquid leaks from the infected tract. Firm digital palpation of the surrounding area can help to determine the extent to which adjacent tissues are underrun. Similar clinical signs can also be seen in weak-footed TB horses, especially in the palmar aspect of the foot, associated with frank sub-solar hemorrhage. Radiography sometimes is useful to identify a gas or fluid pocket.

Treatment Treatment is aimed at establishing adequate drainage. If the tract is open at the sole surface, it should be enlarged just enough for good irrigation and drainage. This may require sedation or perineural analgesia of the foot. If pink tissue or blood is encountered, debridement should be discontinued. Large holes should not be used to avoid solar corium protrusion, which can be a painful sequela to overzealous hoof paring. If drainage occurs at the level of the coronary band and solar surface, through-and-through lavage is beneficial. Debridement at the coronary band level should be minimal to prevent iatrogenic distal interphalangeal joint contamination. Once drainage is established, the foot is protected from the environment and re-contamination with a foot bandage or poultice. Continued foot soaks in warm water povidone-iodine and Epsom salt foot baths should be continued until infection and inflammation are eliminated. The shoe is replaced when the affected area is dry and cornified. Large areas may require a plastic pad under the shoe for solar protection. Antibiotics and non-steroidal anti-inflammatory drugs (NSAIDs) are rarely needed, unless infection is severe or deeper structures have been penetrated. Many practitioners consider antibiotics contraindicated because administration may prolong clinical signs. However, if swelling and infection of the coronary band and subcutaneous tissues of the pastern region occur, antibiotic therapy is indicated. Because lameness can be severe in horses with this type of disseminated infection, involvement of the distal interphalangeal joint is often suspected but usually not present. Tetanus prophylaxis is mandatory.

Prognosis Prognosis in horses with simple sub-solar abscess is excellent but decreases if complications develop in which deeper structures of the foot are involved.

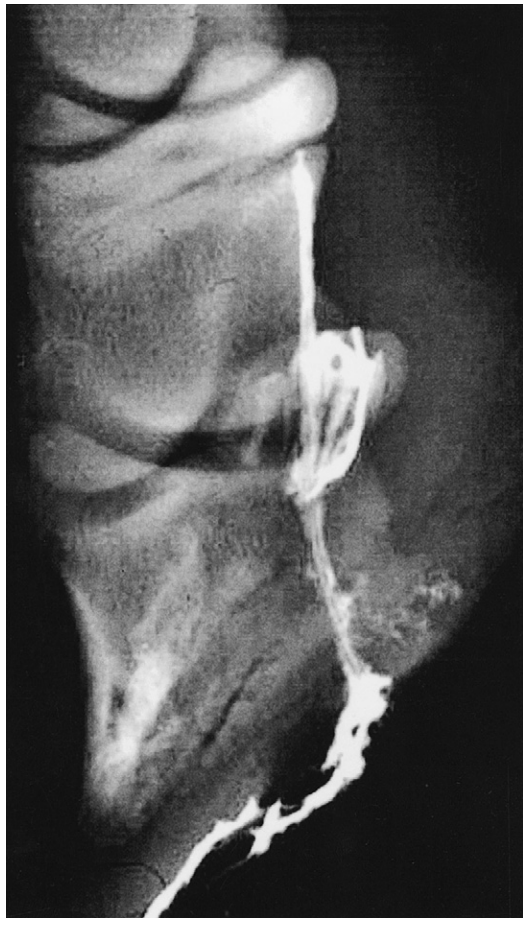

Fig. 28-2 Oblique radiographic view of a foot; radiopaque contrast material has been injected into a hole through the sole, the result of a penetrating injury. The contrast medium extends proximally. A lateromedial view is also required to define better which structures may be involved.

Deep Penetrating Injuries to the Sole

History and Clinical Signs A horse's environment is filled with sharp objects that can penetrate the sole, causing severe damage to structures deep within the hoof capsule. All puncture wounds should be considered potentially serious, but those in the solar white line or palmar frog area require special attention because of the potential for navicular bursa, digital flexor tendon sheath (DFTS), deep digital flexor tendon (DDFT), or distal phalanx involvement.

The clinical signs vary with anatomical structure involved and chronicity of the injury. Lameness may be mild at the time of injury but moderate to severe once inflammation and infection occur. Penetrating wounds of the navicular bursa or DDFT result in severe lameness and a reluctance to bear weight on the heel.

Diagnosis If a foreign body is found in the bottom of the foot, the owner should usually be instructed to leave the object in the foot, unless there is danger of further penetration. Radiography is performed immediately to determine depth of penetration and orientation. In many horses, little superficial evidence of penetrating injury is present. Digital pulse amplitudes are increased and the foot is usually warm to touch. Hoof testers are useful to determine a focal point of pain, but often the entire surface of the foot is reactive. If necessary, the horse should be sedated and the foot desensitized to facilitate further examination. Light paring of the sole and frog areas with a hoof knife may reveal a black spot indicating the penetration site. Often, however, the entry is not discovered because the elastic nature of the hoof structures collapse the site of penetration. If an entry wound is discovered, the foot is scrubbed thoroughly before inserting a sterile, flexible probe. Care must be used so that inadvertent force and horse movement do not cause the probe to penetrate previously unaffected structures. A less invasive and preferred method is to place a sterile teat cannula into the hole and inject sterile radiopaque material to determine the affected structures (Fig. 28-2). It is important to obtain a true lateromedial radiographic view to determine the dorsal spread of the contrast medium.

If infection of the navicular bursa, distal interphalangeal joint, or DFTS is suspected, paracentesis, synovial fluid cytological studies, and culture and microbial sensitivity tests should be performed. Comprehensive radiographic examination should be performed to assess the distal phalanx and the navicular bone. Initial radiographs may appear normal, but lysis or new bone formation may become apparent within 10 to 14 days.

Treatment If penetration of deep hoof structures is suspected, broad-spectrum systemic antibiotics, NSAIDs, and tetanus prophylaxis should be administered. Antibiotic therapy usually should be continued for 2 weeks after resolution of clinical signs of infection. Establishing drainage, copious lavage with sterile ionic fluid, and debridement of all necrotic tissue is indicated. Management of infection of the navicular bursa is discussed in Chapter 24.

If the DDFT is involved (infectious tendonitis), debridement and removal of frayed and infected tendon fibers may be performed in a standing, sedated horse using a tourniquet and perineural analgesia, or with the horse under general anesthesia depending on horse temperament and owner financial constraints. After debridement, use of a 4- to 8-degree wedge shoe decreases forces on the DDFT and provides some pain relief. The wedge shoe angle is gradually decreased over several months as the DDF tendon begins to heal and strengthen. The bottom of the foot requires protection with a bandage or hospital plate until the surgical site granulates in and cornifies.

Infectious Osteitis of the Distal Phalanx

Deep penetrating wounds to the sole, especially the solar–white line junction, can result in infectious osteitis of the distal phalanx. Usually a chronic, recurrent draining tract is located at the coronary band or solar surface of the foot, associated with variable lameness. Infection of the distal phalanx also can result from undetected soft tissue infection, dissection of sub-solar abscesses, or as a sequela to laminitis secondary to recurrent abscessation and ischemia at the toe. As the bone infection progresses, blood supply to the area is compromised and the area of avascular bone separates from the parent bone forming a sequestrum.[9] Radiographic abnormalities may not be detectable for weeks after a penetrating injury.[9] Radiography reveals a radiolucent area in the margin of the distal phalanx, with or without sequestrum formation (Fig. 28-3). Debridement and curettage of all soft and necrotic bone often can be performed in a standing, sedated horse, but general anesthesia may be required. The foot is desensitized using perineural analgesia, cleaned with a hoof knife and steel brush, and prepared for aseptic surgery. Hemostasis is achieved by wrapping a roll of elastic bandage firmly around the fetlock joint to compress and occlude the palmar digital arteries. The infected bone is accessed by removal of sequential layers of the sole using either a motorized Dremel tool or a Galt trephine (Miltex, Bethlage, NY) with a retractable pilot bit.[10] The infected bone usually is discolored and soft and should be curetted to healthy bone margins. Culture of the infected bone and microbial sensitivity testing should be performed. A post-operative radiograph should be obtained to ensure complete debridement. After surgery the surgical site is packed with sterile gauze sponges soaked in antiseptic or antimicrobial solutions and then the foot is bandaged. Disposable diapers and duct tape are inexpensive materials used to make a waterproof foot bandage. The bandage is changed at 1- to 2-day intervals for the initial few weeks. A bar shoe and hospital plate provide solar protection. A plastic pad secured with duct tape also works well. The bolts and metal plate are removed from the hospital plate and the surgery site can be cleaned and treated; the plate then is bolted back in place. After surgery the horse is confined to a small area until the hole granulates and cornifies, which usually requires 4 to 6 weeks. If granulation tissue becomes excessive at the surgery site, application of 2% tincture of iodine speeds healing. If severe infection is present, the surgery site can be packed lightly with antibiotic-impregnated beads for continued antibiotic release at the infected site. Regional limb perfusion with antibiotics may also be beneficial.

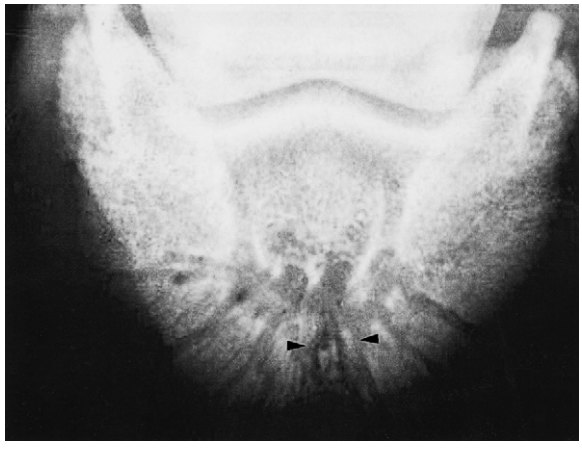

Fig. 28-3 Dorsoproximal-palmarodistal oblique radiographic view of the distal phalanx demonstrating infectious osteitis with a sequestrum *(arrowheads)*.

Prognosis Prognosis depends on whether the infection is severe and chronic. Horses with acute penetrating wounds that receive immediate and aggressive treatment have a good chance of returning to athletic use. Horses with penetration injuries that have an established infection involving the navicular bursa,[11] DFTS, or distal interphalangeal joint have a poorer prognosis. Prognosis for horses with infectious osteitis of the distal phalanx is good if the cause of infection is not laminitis. In one study, up to 24% of the distal phalanx was removed with successful results.[12]

Hoof Wall Cracks

History and Clinical Signs Hoof wall cracks occur from improper foot balance; coronary band defects; excessive hoof growth; and thin, dry, or wet hoof walls. The horny hoof wall often fails internally before being visible on the external hoof surface.[13] Central toe cracks usually are associated with distal phalanx rotation and club feet. Hoof cracks are characterized by location (toe, quarter, heel or bar), length (complete or incomplete), depth (superficial or deep), site of origin (ground surface or coronary band), and whether hemorrhage or infection is present.[13] Hoof cracks are usually obvious by visual examination of the foot, except those originating at the hairline, which may be only 1 to 2 cm long and difficult to see. Lameness may be present, depending on whether the hoof crack involves the sensitive laminae and if infection is present.

Diagnosis Hoof wall cracks are diagnosed by visual assessment of the hoof capsule. The depth of the hoof crack, pain, and associated instability surrounding the crack are determined by careful hoof tester examination. Pain associated with a hoof crack is usually determined by digital pressure and hoof tester manipulation over the crack. Purulent material may exude during hoof tester pressure. Radiography is useful to evaluate rotation of the distal phalanx in horses with central toe cracks. Bleeding from a hoof crack after exercise indicates that the sensitive laminae are involved.

Treatment Treatment varies with hoof crack location, depth, horse use, and presence of exposed sensitive laminae and infection. Superficial hoof cracks do not extend into laminar tissue and therefore are not painful. These may be the result of improper foot balance or basic neglect regarding trimming. Treatment involves balancing the foot and providing stability by application of a full bar shoe. If the horse is shod correctly and restricted from strenuous activity, most hoof cracks will resolve.[14]

Treatment of deeper hoof cracks varies somewhat with location of the crack. If lameness exists, diagnostic analgesia should be performed to confirm that the hoof crack is the source of pain. Careful observation of the affected foot as the horse walks slowly often shows that the defect is unstable and actually closes and pinches the underlying sensitive laminae as the foot strikes the ground, causing pain. The hoof crack is explored and debrided with a hoof knife or motorized burr (Dremel tool) to remove all necrotic and infected tissue. Any undermined hoof wall is also removed. The area is treated for 24 to 48 hours with an antiseptic such as merthiolate

(Thermisol; Eli Lilly, Chicago, IL) or tincture of iodine, until the crack is dry and free of infection. The hoof wall must be stabilized so that it can regrow. Previous recommendations have suggested grooving or burring the proximal extent of the crack, but this is rarely successful.[13] Many techniques for hoof wall stabilization use a combination of frog support with a heart bar shoe and clips combined with either a fiberglass patch,[14] drill and lace,[13] or metal plate technique. The heart bar shoe is essential to oppose the forces causing collapse of the crack during weight bearing. The toe must be trimmed short and squared before application of the shoe. The drill and lace technique is used if the deep crack does not extend to the coronary band, and the metal plate technique is used if it does. Using the metal plate technique, two drill holes are placed 1 to 2 cm on either side of the trough directly opposite each other. Care is needed to ensure that drilling too deep does not affect deeper hoof structures. The foot should *not* be desensitized to avoid iatrogenic penetration of deeper tissues with the screws. One or two plates are cut that are longer than the exposed hoof crack and about 0.6 cm wide. With the foot held in a non–weight-bearing position, the metal plate is drilled and bolted in place to stabilize the crack. The hoof wall adjacent to the toe crack is trimmed shorter than the remaining hoof wall to minimize weight-bearing forces on the damaged area and decrease potential bending forces on the plate. The crack is treated for several days with Thermisol or tincture of iodine until the sensitive structures begin to cornify and infection has resolved. The hoof crack can then be filled with an acrylic material (e.g., Equilox; Equilox International, Pine Island, MN). Adhesion of the acrylic to the hoof wall is enhanced by sanding the hoof wall, applying acetone to dry the area, and using a hair dryer at the external hoof surface before acrylic application.

Quarter and heel hoof cracks often are incomplete and may be predisposed by low-heel–long-toe conformation with underslung heels.[13] After the crack is debrided and infection eliminated, the foot is balanced and a heart bar shoe applied. Two holes are drilled using a 0.24-cm drill bit approximately 1 to 2 cm apart on either side of and parallel to the crack. The holes begin at the ground surface and extend up the hoof wall. A shoelace or synthetic multifiber suture is laced in a far-near–near-far suture pattern to stabilize the crack.[13] After the crack is dry and free of sensitive tissue and infection, it is filled with acrylic material. Readers are also referred to Chapter 27.

Prognosis The prognosis for horses with both superficial and deep hoof cracks is good and lameness usually resolves as soon as the hoof is stabilized, but recurrence is common. Success is improved if the mechanical cause of the hoof crack can be identified and eliminated.

Coronary Band and Hoof Wall Lacerations

History and Clinical Signs The hoof wall is thicker and stronger at the toe region and becomes thinner through the quarters and heel, where the younger hoof has a greater moisture content.[14] The quarter and heel regions of the foot are susceptible to traumatic injuries. Coronary band and hoof wall lacerations usually occur from the horse catching a segment of hoof on an object as it steps down or kicking or stepping on a sharp object. Hoof avulsion injuries also can occur when the foot is entrapped between fence boards or in a cattle guard. Continued hoof imbalance, improper shoe removal, and repetitive trauma to the coronary band region result in a chronic hoof avulsion or spur—a fibrous bed of scar tissue beneath a displaced segment of hoof wall. Horses that overreach are predisposed to coronary band spurring or heel avulsion injuries. Steeplechase horses and horses racing on grass often slip and lacerate the heel region of a front foot.

Hoof avulsions are described as acute (lacerations) or chronic (repetitive trauma and spur formation) injuries. Avulsions can be complete, with total tissue loss, or incomplete in that a border of hoof remains intact. Hoof wall, coronary band, sole, distal phalanx, laminae, and the distal interphalangeal joint may be involved in deep lacerations. The degree of lameness varies with duration, depth, and location of the injury. Horses with acute, superficial injuries may show mild lameness, and horses with deeper structure involvement may be non–weight bearing. If degree of lameness does not seem appropriate for severity of the laceration, the integrity of the palmar digital vein, artery, and nerve should be investigated.

Diagnosis Diagnosis is straightforward, but involvement of deeper structures can be difficult to identify. Careful manipulation of the foot causes a painful reaction if deep structures are involved and provides valuable information regarding the integrity of the supporting hoof structures. Instability of the distal interphalangeal joint may indicate collateral ligament damage. If manipulation produces a sucking noise, the distal interphalangeal joint or DFTS may be involved. Before further evaluation the coronary band hair should be clipped, the outer hoof wall rasped, and sole trimmed to eliminate any superficial contamination. The area should be scrubbed with antiseptic solution and lavage performed. The wound then is digitally explored by the veterinarian using sterile gloves. Radiography is recommended for all deep lacerations, because fractures of the distal or middle phalanges may be present. Contrast radiographic studies may be necessary to identify openings in synovial structures. Ultrasonographic examination may help to determine the extent of soft tissue damage.

Treatment The equine foot heals primarily by epithelialization and reformation of the corium.[15] Decreasing motion at the affected site and hoof stabilization are essential for a successful outcome. Treatment varies with duration, severity, and type of injury. Incomplete, superficial hoof wall lacerations without coronary band involvement are treated by excision of the separated hoof wall and bar shoe application until healing occurs. The goal of shoeing is to eliminate weight-bearing forces at the damaged site and provide hoof stability.

Incomplete, clean, acute hoof avulsions involving the coronary band can be treated by cleaning and debriding the displaced flap of tissue and suturing it back in place. This generally requires general anesthesia. Interrupted vertical mattress sutures with No. 1 or No. 2 monofilament suture are recommended.[14,15] Any undermined or contaminated hoof wall is removed. Immobilization is essential and is provided by applying a foot cast. The cast is usually left in place for 2 to 3 weeks. When applying the foot cast, the clinician should ensure that the proximal extent of the cast is located at midpastern level and does not impinge on the fetlock joint during movement. Administration of systemic antibiotics and NSAIDs may be necessary if contamination is suspected. If deeper structures are involved, cast application should be delayed until infection is eliminated. Open synovial structures can be lavaged daily, and the foot is protected with a sterile foot bandage. Antibiotic-impregnated beads and regional limb perfusion with antibiotics may be necessary with severe contamination.

Horses with compete avulsion injuries that appear stable during movement are treated by daily cleaning and bandaging until healing occurs. Bar shoe application is required if the hoof is unstable and contaminated. A foot cast can be used if infection is not a problem.

Repair of a chronic avulsion injury, or spur, usually requires surgical excision with the horse under general anesthesia. The hoof is rasped and prepared for aseptic surgery. The hoof wall distal to the avulsed segment should be thinned with a rasp to allow placement of the sutures through the hoof wall. The excessive cornified tissue growing from the displaced coronary band is trimmed to a level just distal to the coronary band. The fibrous tissue under the avulsed segment, which lies in

the bed of the defect created by the hoof avulsion, is resected. This provides a vascularized area and room for replacement of the avulsed segment back to its original location. The displaced coronary band and hoof wall are replaced and sutured in the correct anatomical position using No. 2 monofilament suture in a simple interrupted or vertical mattress suture pattern. The surgery sites are covered with a light bandage and the foot immobilized in a foot cast for 2 weeks.

Prognosis Treatment is usually prolonged and often takes 3 to 5 months for complete healing, which can be costly to the owner.[15] Incomplete superficial avulsion injuries or injuries that can be sutured usually heal by first intention, with a good functional, and sometimes cosmetic, end result. A roughened or thickened hoof wall distal to the defect often occurs at the site of avulsion, but it usually does not create a clinical problem. Prognosis decreases if deeper structures or synovial structures are involved. Complications such as infectious arthritis, fractures, and potential osteoarthritis can occur often with a guarded prognosis.

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CHAPTER • 29

Functional Anatomy of the Palmar Aspect of the Foot

Robert Bowker

The palmar portion of the foot consists of several important structures functioning to support the foot and the limb of the horse, as well as being an integral part of the energy dissipation mechanisms present within each limb. These palmar foot structures include the cartilages of the foot (also called the *lateral collateral* or *ungual* cartilages), the digital cushion, and an extensive vascular network. Although each of these structures is present in every foot, morphological features and tissue composition vary widely among horses, which may be responsible for differing efficiencies in ability to dissipate energy. Furthermore, such differences may in part account for differences between the feet of a sound horse compared with feet of a horse with chronic lameness associated with the foot. Awareness of how these tissues interact and relate to each other during foot impact is important for understanding how the foot dissipates energy and how potential problems may arise to produce lameness, when energy dissipation is not efficient and the concussive forces are transmitted to the bones and other connective tissues. The domestic horse spends considerable time standing, so the structure of the palmar aspect of the foot is important for support to minimize the weight of the horse being shifted dorsally toward the connective tissues at the toe of the foot.

The medial and lateral cartilages of the foot extend from the palmar surface of the distal phalanx to the bulbs of the heel as large vertical sheets, whereas the digital cushion lies between the medial and lateral cartilage and extends dorsally toward the solar surface of the distal phalanx ventral to the deep digital flexor tendon (DDFT). Associated with each cartilage of the foot is a venous network that connects with the venous vessels under the distal phalanx and the vessels associated with the dermis of the hoof wall. This venous microvasculature forms a hydraulic system that is hypothesized to form the mechanism for how the ground impact energies are dissipated, before these forces are transmitted to and damage the bone and other connective tissues within the foot. Those horses with good to excellent hydraulic systems should be more efficient in dissipating the impact energy compared with horses with feet with less well-developed hydraulic networks.

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CARTILAGES OF THE FOOT

The cartilages of the foot lie beneath the skin and dermis and the coronary venous plexus and have previously been described as rhomboid-shaped, with a convex surface abaxially and a concave surface axially. Several ligaments secure the cartilages of the foot to the digital bones.[1-4] However, these descriptions are over-simplistic and appear to have been obtained from horses with underdeveloped structures in the palmar aspect of the foot. The morphological features of the cartilages of the foot vary greatly with a range of shapes and thickness, the presence of an axial projection from its distal edge, and the extent of vascularity.[5] The structure of the cartilages of the foot is best determined by viewing the foot in frontal and transverse sections (Figs. 29-1 and 29-2). In frontal sections cut perpendicular to the ground, beginning at the bulbs of the heels, the cartilages of the foot have a C- to L-shaped configuration. Both the upright and the base parts of the L-shaped cartilage vary in thickness among horses. The mean thickness of the upright part at the level of the navicular bone ranges from 0.5 to 2.0 cm in an adult horse (450 to 550 kg body weight). The base part or axial projection of the L-shaped cartilage varies in its thickness and the distance that it extends toward the midline of the foot overlying the bars and the frog. The cartilages of the foot are thinnest in the heel region (0.45 to 1.3 cm) but become thicker closer to the distal phalanx (0.6 to 1.5cm) and thin slightly as they attach onto the distal phalanx (0.5 to 1.0 cm). The cartilages of the foot are thicker in forelimbs than hindlimbs.

Overlying the abaxial surface of the cartilages of the foot are a venous plexus, the laminar dermis, and the hoof. Loose connective tissue extends from each cartilage to the DDFT. On the axial surface the axial projection of the cartilage extends toward the midline of the foot. In most feet, this axial projection extends from the dorsal half of the cartilage; the palmar half of the cartilage has virtually no, or very small, extension into the substance of the digital cushion. However, in some feet it extends the entire dorsal-palmar extent of the cartilages of the foot. This axial projection extends to overlie the epidermal ridges of the bars, with many finger-like projections extending into the substance of the digital cushion, and may extend across the midline of the palmar foot under the digital cushion to fuse with that of the opposite side of the foot. These white bundles of fibrous and fibrocartilaginous tissues are easily discerned from the surrounding yellow elastic, adipose, and collagen fibers of the digital cushion. The relative thickness of the axial projection in a distal-proximal orientation varies. In horses younger than 4 to 5 years of age the axial projection usually is not fully developed along the entire length of the cartilage, and in young foals there is only a thin sheet of fibrous tissue.

The cartilages of the foot contain primarily hyaline cartilage, but in many horses from 4 to 5 years of age the medial border of the cartilage develops fibrocartilage. A fibrocartilaginous ligament of variable thickness develops between the cartilage and the DDFT; this is consistently larger in forelimbs than hindlimbs. In some horses the cartilages of the foot can ossify. This may be genetically controlled, but stress on the foot, such as that produced with certain shoe types improving traction, may promote ossification.

Several ligamentous attachments connect the cartilages of the foot to the distal and middle phalanges, as well as to the navicular bone.[3,4] The chondroungular ligaments attach the cartilage to the distal phalanx along the palmar process, whereas the medial and lateral chondrocoronal ligaments attach the cartilage to the distal end of the middle phalanx. The medial and lateral ligaments of the cartilages of the foot (collateral chondroungular ligaments) attach the cartilage to the angle of the distal phalanx. The paired chondrosesamoidean ligaments attach the axial surface of the cartilage to the navicular bone. A pair of elastic ligaments extends between the proximal phalanx and the proximal surface of the cartilages of the foot and are most prominent in larger horses, such as draft breeds. Cruciate ligaments of the cartilages of the foot (cruciate chondroungular ligaments) connect the axial surface of the cartilage to the palmar process on the opposite side of the foot. Within the digital cushion are fiber tracts radiating from the connective tissue ventral to the attachment of the DDFT (digital torus), through the digital cushion, to the axial surface of the cartilages of the foot.

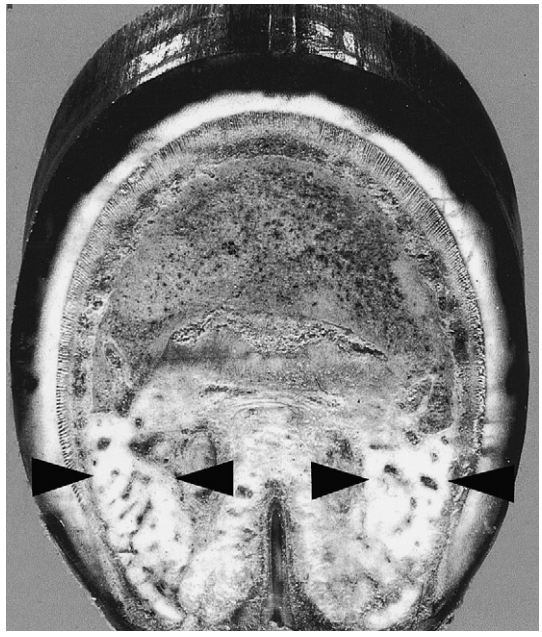

Fig. 29-1 Transverse section through the distal foot of a Quarter Horse 25 years of age, with no history of foot problems. *Arrows* show the thick cartilages of the foot. An axial projection composed of fibrocartilage extends between each cartilage of the foot.

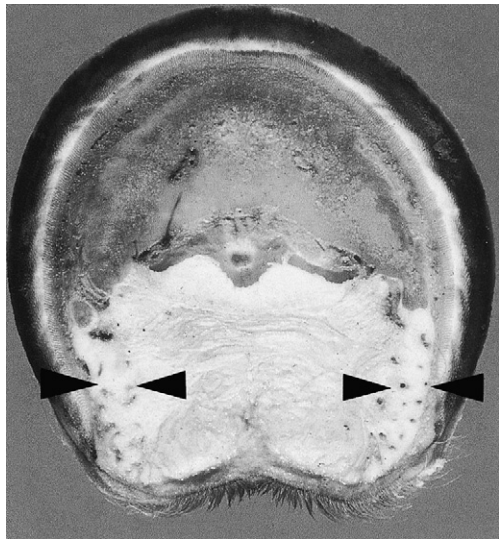

Fig. 29-2 Transverse section of the foot of a horse with chronic foot pain with thin cartilages of the foot *(arrows)*. The digital cushion is primarily composed of fat and elastic tissue, with little fibrocartilage.

Each hoof cartilage is perforated by numerous vascular foramina, the number of which varies depending on the thickness of the cartilages of the foot. Within the vascular channels is a large central vein, with a rich network of microvessels termed *veno-venoanastomoses*.[5-7] These microvessels exit the large central vein and, after a variable course within the vascular channels, re-enter the same vein. More of the vascular channels are present at the distal level of the cartilages of the foot, but in feet with relatively thicker collateral cartilages, there are more vascular channels proximally compared with feet with much thinner cartilages. The veins coalesce proximal to the cartilage into a venous plexus before uniting to form the medial and lateral palmar digital veins.[7,8]

DIGITAL CUSHION

The digital cushion consists of a meshwork of collagen and elastic fiber bundles, with small areas of adipose tissue, and lies between the cartilages of the foot, extending dorsally as a wedge-shaped tissue attached to the DDFT and the solar surface of the distal phalanx, near the distal attachment of the DDFT.[1-4] It overlies the frog and its dermis and the axial projection from the cartilages of the foot. Proximally and dorsally the digital cushion fuses with the distal digital annular ligament and bulges into the bulbs of the heel, that are separated superficially by a central shallow groove. Some horses have areas of fibrocartilage in the digital cushion and if present, these extend between the cartilages of the foot and the DDFT. The digital cushion has two arteries that pass through the digital cushion to the area distal to the digital cushion, but proximal to the axial projection of the cartilages of the foot, and then branch extensively to supply the frog.[2-6] Only a few vessels ramify through the digital cushion from these two arteries.

ENERGY DISSIPATION

The function of the digital cushion is controversial. The structural organization of the cartilages of the foot, the digital cushion, and the vasculature suggests a role in energy dissipation.[5] The pressure theory suggests that at ground contact the frog stay is pushed upward into the digital cushion, forcing the cartilages of the foot outward. The depression theory emphasizes a downward movement of the pastern into the digital cushion during ground impact, forcing the cartilages of the foot to move outward. However, neither theory is consistent with measurement of negative pressure within the digital cushion during stance and locomotion.[9] The hemodynamic hypothesis provides a hydraulic mechanism during ground contact, so that impact energy is transmitted to the fluid within the blood vessels.[5] During ground impact the outward expansion of the cartilages of the foot probably occurs through the bars contacting the axial projections and the downward movement of the bony column within the hoof capsule. This creates a negative pressure within the digital cushion. At this brief moment of impact the venous blood within the vessels of the palmar aspect of the foot is forced into the microvenous vasculature within the vascular channels of the cartilages of the foot. Hydraulic resistance to flow through the microvasculature dissipates the high-frequency energy waves, which are potentially deleterious to bone and other tissues. Negative pressure in the foot enables refilling of the vasculature before the next footfall. In feet with thick cartilages of the foot, enclosing more microvessels within the vascular channels, more energy is dissipated on ground contact compared with feet with thin cartilages of the foot. The fibrocartilage content of the digital cushion also is crucial to energy dissipation, because the fibrocartilage has its own energy-absorbing mechanisms. The elastic tissue acts only like a spring and absorbs little energy on ground contact, serving to return the foot to its original position as the foot leaves the ground.

The shape of the foot appears to influence the development of the cartilages of the foot and the structure of the digital cushion. In well-balanced feet, with the frog on the ground along with the bars, cartilages of the foot tend to be thick with fibrocartilage in the digital cushion.[5] In feet with a low heel and long toe, the site of ground contact of the hoof wall at the angle of the wall and the bars usually is beneath the bony part of the distal phalanx, rather than underneath the cartilages of the foot. Therefore more of the energy of impact is transferred to the bone and hoof wall laminae, as the cartilages of the foot and the digital cushion are in essence by-passed during ground impact.

NAVICULAR SUSPENSORY APPARATUS

The navicular suspensory apparatus consists of several ligaments functioning to suspend the distal sesamoid bone or navicular bone on the palmar surface of the distal interphalangeal joint.[1-4] Proximally, paired collateral suspensory ligaments of the navicular bone (or the collateral sesamoidean ligament) arise from the distal surface of the proximal phalanx and pass in a distopalmar direction, attaching along the abaxial surface of the middle phalanx,[10] to insert on the extremities of the navicular bone. In addition, small branches attach to the axial surface of the cartilages of the foot and the distal phalanx. The attachment along the middle phalanx is important biomechanically during forward movement of the limb, because high loads are created on the joint surfaces between the navicular bone and the middle phalanx and between the navicular bone and the distal phalanx.[11] These ligaments are composed of collagen fibers with an abundance of elastic tissue fibers. Distally the distal sesamoidean impar ligament extends from the distal border of the navicular bone to the entire flexor surface of the distal phalanx adjacent to the insertion of the DDFT.[1-4] At its insertion the distal sesamoidean impar ligament contains an extensive network of microvessels containing arteriovenous complexes and nerves fibers within loose connective tissue septae.[10] The arteriovenous complexes are innervated by many peptidergic nerve fibers, including substance P, neurokinin A, and calcitonin gene-related peptide, which are present in the many sensory fibers innervating the foot.[12] The peptide nerves substance P and neurokinin A also have pharmacological receptors, located on the small isolated microvessels and the arteriovenous complexes within the distal sesamoidean impar ligament, to control blood flow through this intricate vascular network. When these peptides are released from the sensory nerve fibers in the foot, they promote an active vasodilatation of these small vessels, presumably through a nitric oxide pathway. The locations of these arteriovenous complexes suggest that they may have two possible functions, including a protective mechanism for detection of high-pressure differences within the region during movement, and maintaining the hydration status of the distal sesamoidean impar ligament and other nearby connective tissues for optimal function.

The distal border of the navicular bone has a narrow, elongated facet for articulation with the distal phalanx. Between this and the attachment of the distal sesamoidean ligament is a fossa containing foramina for blood vessels. The proximal border also has several small foramina. The dorsal articular surface of the navicular bone and the articulation between the navicular bone and the distal phalanx create a substantial palmar extension of the articular surface of the distal interphalangeal joint. During extension of the distal interpha-

langeal joint, with fixation of the foot on the ground and the movement of the body over the distal limb, the middle phalanx contacts the dorsal articular surface of the navicular bone, and the navicular bone becomes a weight-bearing structure. Loads transmitted through the navicular bone are supported by the proximal and distal suspensory ligaments. The role of the DDFT is discussed further in Chapter 33.

DISTAL INTERPHALANGEAL JOINT

The distal articular surface of the middle phalanx, the articular surface of the distal phalanx and the articular surfaces of the navicular bone form the distal interphalangeal joint. The relatively short medial and lateral collateral ligaments of the distal interphalangeal joint arise from the distal ends of the middle phalanx to insert on the distal phalanx and the dorsal part of the cartilages of the foot. The joint capsule has a small dorsal pouch and an extensive palmar pouch, and blend with the collateral ligaments and common digital extensor tendon. The palmar pouch of the distal interphalangeal joint is greatly expandable (25 to 30 ml in volume) and is subdivided into a proximal palmar pouch and a small distal palmar pouch extending between the navicular bone and the distal phalanx. In the midline the palmar pouch extends proximally beyond the two small secondary tendons of the DDFT, which attach to the distal end of the middle phalanx. The proximal palmar pouch almost surrounds the collateral sesamoidean ligaments.[13,14] The distal interphalangeal joint cavity also has several small abaxial, dorsal projecting outpouchings that are in close proximity to the sensory nerves of the medial and lateral palmar digital nerves. The distal interphalangeal joint capsule therefore has a large surface area through which local anesthetic solution may diffuse. The comma-shaped navicular bursa is much smaller (approximately 3 ml in volume). Proximally it can extend over the proximal border of the navicular bone to protrude dorsally.

INNERVATION

The innervation of the equine distal forelimb is via the medial and lateral palmar nerves and the medial and lateral palmar metacarpal nerves.[1-4] The distal continuation of the palmar nerves course parallel to the accompanying artery and then obliquely across the abaxial surface of the ligament of the ergot to supply most tissues of the palmar half to third of the foot (see Chapter 10). The Editors point out that given their clinical experience and current research results, diagnostic analgesia of the digital nerves result in analgesia of most of the foot, including the solar surface. Several small nerves branch from the medial palmar digital nerve, to course with the artery of the digital cushion, to supply the palmar foot, including the dermis of the overlying skin and frog, parts of the digital cushion, laminae of the bulbs of the heel, cartilages of the foot, and portions of the quarters. A small branch of the lateral palmar nerve may supply the ligament of the ergot. The dorsal branches of the palmar nerve continue with the palmar digital vein to innervate the dorsal aspects of the foot, including the distal interphalangeal joint, laminar and solar dermis, and the dorsal part of the cartilages of the foot. An intermediate branch from this nerve occurs in approximately a third of horses. In some horses a branch of the medial palmar metacarpal nerve supplies the coronary band. No communication occurs between the palmar metacarpal nerves and the dorsal branches of the palmar digital nerves. Variable branches occur, including one from the lateral palmar nerve in the proximal metacarpal region extending obliquely to the coronary band, and one from the medial palmar digital nerve to the navicular bursa. In the hindlimbs an additional nerve supplies the coronary and laminar dermis of the dorsal foot, provided by the medial and lateral dorsal metatarsal nerves (terminal branches of the deep fibular nerve). Rarely branches from the plantar metatarsal nerves course under the distal ends of the second and fourth metatarsal bones to supply the dermis of the periople and coronet. The widespread distribution of these nerves provides a broad sensory and sympathetic autonomic innervation pattern to the tissues and vasculature of the foot.

The sensory nerves have many diverse functions in addition to conveying consciously perceived sensations, including touch, proprioception, and pain. The palmar digital nerves are composed of both small, unmyelinated nerves and larger myelinated nerves in a ratio of nearly 4:1. Approximately 25% of the unmyelinated fibers are sympathetic nerves and 75% are afferent fibers. Much of the sensory information from the foot and the activity of the sympathetic autonomic nerves is conveyed to the spinal cord through the unmyelinated fibers, which are the more slowly conducting nerve fibers. Many neurochemicals are present within the nerves including noradrenaline and adrenaline, substance P, neurokinin A, calcitonin gene-related peptide, neuropeptide Y, peptide histidine isoleucine, vasoactive intestinal peptide, and the enkephalins. These are released locally from the peripheral processes of the sensory nerves, and the effect on surrounding tissues depends on which neurochemical(s) is emitted.

Most of these peptides have been identified in the foot, usually in close association with blood vessels and other microvasculature. Within the dorsal hoof wall the sympathetic fibers containing noradrenaline and neuropeptide Y are present along the small arterioles within the dermal laminae and form dense plexuses around the arteriovenous anastomoses.[15] The same neurotransmitters are present in the navicular region, including the arteriovenous complexes.[13] Noradrenaline and neuropeptide Y promote vasoconstriction, whereas vasoactive intestinal peptide is a prominent dilator of the smooth muscle of the microvessels.[16] The other peptides, such as substance P, calcitonin gene-related peptide, and peptide histidine isoleucine, are present in the sensory nerve fibers of the dorsal hoof wall and the distal aspect of the distal sesamoidean impar ligament[10,12] and also promote vasodilation by means of an endothelial-dependent mechanism and the activation of a nitric oxide pathway.[17,18] Activation of these sensory nerves either directly, or indirectly through pain mechanisms, produces a measurable increase in the concentration of these peptides in joints and tissues as they interact with tissue elements, such as inflammatory cells and macrophages, and controls edema formation.[19]

Other sensations, such as touch and proprioception, are mediated to the spinal cord through the larger myelinated fibers. The receptors of these nerve fibers are present in the bulbs of the heel and in association with the collateral ligaments of the navicular bone.[20-22] The locations of these lamellated receptors appear to be critical for the perception of proprioceptive stimuli by the horse during movement when heel-first landing occurs. Activation of these receptors at ground impact and the rapid conduction to the spinal cord through these thickly myelinated fibers enable this sensory information to become incorporated into the spinal cord reflex mechanisms controlling locomotion. During toe-first or flat-footed landings, activation of these sensory receptors is presumably less. If a horse is shod with a pad, it may shorten its stride because the pacinian receptors are not adequately stimulated. If a little, finger-sized piece of rubber is attached temporarily to the pad, within one or two steps the stride extends forward maximally, as the horse "realizes" the importance of this area of the foot, by activation of the sensory receptors. If the rubber is removed after several days, the gait does not revert. Together the unmyelinated and the myelinated nerves

enable the horse to smoothly negotiate the varying surfaces of the terrain during locomotion and provide a means for monitoring changes in the peripheral tissues and controlling the physiological and pathological environment within the foot.

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CHAPTER • 30

Navicular Disease and Other Soft Tissue Causes of Palmar Foot Pain

Sue J. Dyson

PATHOPHYSIOLOGY OF NAVICULAR DISEASE

Navicular disease is a chronic forelimb lameness associated with pain arising from the distal sesamoid or navicular bone and closely related structures, including the collateral ligaments of the navicular bone, the distal sesamoidean impar ligament, the navicular bursa, and the deep digital flexor tendon (DDFT), but does not include primary DDF tendonitis. Although historically considered to be a single disease, given its variety of clinical manifestations it is likely that there are probably a number of different clinical conditions, of different origins, that give rise to pain. It is difficult to conceive of a single disease that can result in an insidious-onset, slowly progressive bilateral forelimb lameness, or an acute-onset, relatively severe unilateral forelimb lameness, each with a variety of different radiological manifestations and with some horses never developing radiological changes. It is curious that sometimes clinical signs become apparent in young horses just

enable the horse to smoothly negotiate the varying surfaces of the terrain during locomotion and provide a means for monitoring changes in the peripheral tissues and controlling the physiological and pathological environment within the foot.

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commencing work, whereas more typically lameness is seen in mature riding horses. It is also seen in horses with vastly different distal limb conformation. It is a common condition in Quarter Horses, which tend to have narrow, upright, boxy feet, small relative to the body size, as well as in European Warmblood horses, many of which have relatively tall narrow feet. It is also common in Thoroughbred horses, which frequently have rather flat feet, with low collapsed heels, often associated with dorsopalmar foot imbalance. Recent evidence has suggested a heritable tendency toward the development of navicular disease in the Dutch Warmblood.[1,2]

Navicular disease has not been reproduced experimentally; therefore, all proposed causes remain speculative. Earlier theories suggesting a vascular etiology with arteriosclerosis,[3] or thrombosis, resulting in ischemia within the navicular bone,[4] have largely been rejected because of failure to identify ischemic bone or thrombosis, failure to reproduce clinical signs or pathological changes by occluding blood supply to the bone, and increasing evidence demonstrating increased bone modeling.[5-8] Post-mortem studies to date have focused principally on horses with long-term, chronic lameness, generally with advanced radiographic abnormalities, reflecting the end stage of a disease complex. However, studies of aging changes in the navicular bone of normal horses and comparison with those with navicular disease have given valuable evidence that there is a degenerative aging process that may be accelerated in horses with navicular disease.[7-9] Non-physiological biomechanical factors may promote this degenerative disease.[7,10] The explanation for pain and lameness in horses with no detectable radiological change has been poorly investigated by post-mortem studies.

The navicular apparatus comprises the navicular bone, the collateral ligaments of the navicular bone, the distal sesamoidean impar ligament (DSIL), the navicular bursa, the DDFT, and the distal digital annular ligament. The navicular bone or distal sesamoid bone, which articulates with the middle and distal phalanges, provides a constant angle of insertion, and maintains the mechanical advantage of the DDFT, which exerts major compressive forces on the distal one third of the bone. Contact studies between the phalanges in isolated limbs have demonstrated that the greatest forces are applied in the propulsion phase of the stride, during extension of the distal interphalangeal joint, with increased tension of the DDFT on the palmar aspect of the navicular bone, increased contact between the navicular bone and the middle phalanx, and increased tension in the collateral ligaments.[11,12] Tension in the DDFT and the distal digital annular ligament promotes stability of the distal interphalangeal joint. Forces may be altered by foot conformation: in a horse with weak heels, there is less flexion of the distal interphalangeal joint compared with a horse with strong heels, which results in increased pressure concentrated on the distal aspect of the navicular bone.[13]

Compressive forces and stress on the navicular bone have been compared in clinically sound horses and horses with navicular disease.[10] Although the mean peak force and stress were similar, the force and stress in the horses with navicular disease were approximately double early in the stance phase of the stride. This early peak stress resulted in a much higher loading rate in the group with navicular disease. The difference in loading patterns was associated with an increased force in the DDFT in the early and mid-stance phases, probably as a result of increased contraction of the DDF muscle. This contraction of the DDF muscle may result in toe-first ground contact seen in some horses with navicular disease. It is suggested that pain associated with the navicular bone may result in a positive feedback by increasing the force in the DDFT and hence increasing the compressive force on the navicular bone. This hypothesis is supported by reduction in peak forces on the navicular bone throughout the stance phase in horses with navicular disease after perineural analgesia of the palmar digital nerves.[14]

The shape of the navicular bone may be determined at birth, and this may influence the biomechanical forces subsequently applied to the bone and hence influence the risk of development of navicular disease[1,15] (Fig. 30-1). Finnhorses and Friesian horses tend to have a straight or convex contour of the proximal articular border of the navicular bone and rarely develop navicular disease. The incidence of navicular disease is much higher in the Dutch Warmblood, and horses in which the proximal articular margin is concave or undulating appear to be at highest risk of development of the disease.[2,14]

Aging results in degenerative change in the fibrocartilage on the palmar aspect of the navicular bone.[7,8] Physiological forces result in adaptive remodeling of the subchondral bone with cortical thickening. Non-physiological forces result in subchondral sclerosis associated with thickening of the trabeculae with focal areas of lysis (Fig. 30-2). There is edema, congestion, and fibrosis of the marrow stroma within the

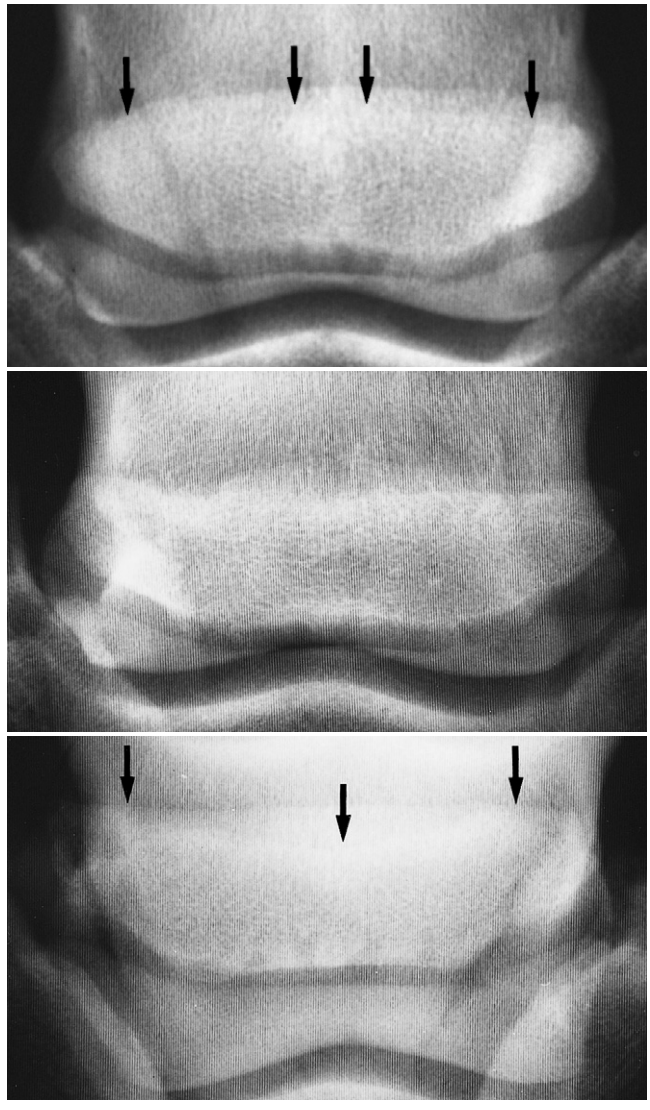

Fig. 30-1 Dorsoproximal-palmarodistal oblique radiographic views of the navicular bone to show differences in shape of the proximal articular margin *(arrows)*. **A,** Convex; **B,** undulating; **C,** concave views.. There are variably shaped lucent zones along the distal border of the bones.

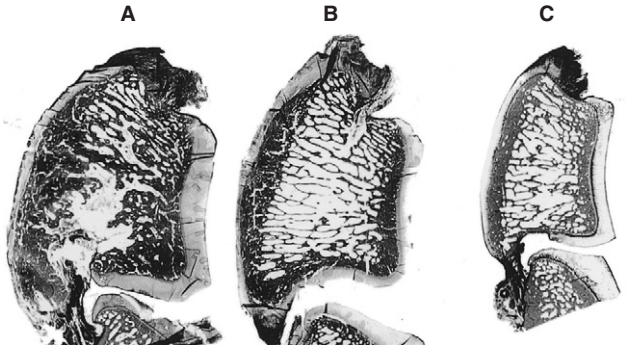

Fig. 30-2 Sagittal sections of the navicular bones of **(A)** a horse with navicular disease, **(B)** an age-matched control, and **(C)** an immature control horse. The subchondral bone has an increased area and porosity in **A** compared with **B** and **C;** the trabecular area is decreased, but the trabeculae are thickened. (From Wright IM, Kidd L, Thorp BH: Gross, histological and histomorphometric features of the navicular bone and related structures in the horse, *Equine Vet J* 30:220, 1998.)

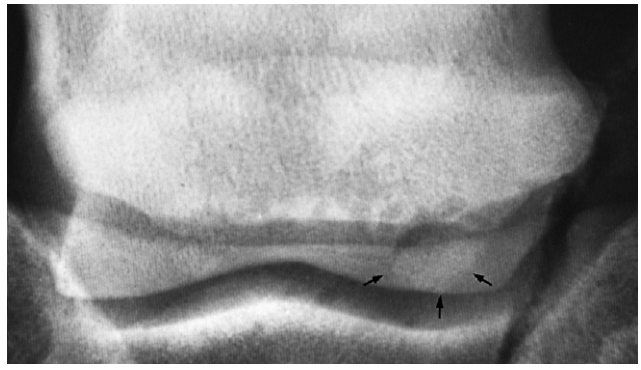

Fig. 30-3 Dorsoproximal-palmarodistal oblique radiographic view of a navicular bone showing a large discrete mineralized fragment on the distal lateral aspect of the bone *(arrows)*, an abnormal concave contour of the adjacent distal border of the bone, and the lucent zones along the distal border of the bone. The horse had bilateral forelimb lameness associated with distal border fragments in both front feet.

medullary bone, which may result in cyst-like formation. Enlarged synovial invaginations are the result of recruitment and activation of osteoclasts following the course of the nutrient vessels into the spongiosa. Concurrently there may be fibrillation of the dorsal surface of the DDFT, which may predispose to adhesion formation between the DDFT and regions of partially or fully eroded fibrocartilage on the palmar aspect of the navicular bone. Whether lesions in the DDFT are primary or secondary to pre-existing damage of the fibrocartilage currently remains open to debate.

Aging changes also have been recognized in the articular cartilage of the navicular bone and the opposing face of the distal phalanx.[12] There is loss of proteoglycan and tidemark advancement, which is thought to reflect excessive shear stress in the zone between the mineralized and non-calcified articular cartilage. A greater number of tidemarks were seen in horses with clinical signs of navicular disease than normal horses of similar age.

To date, active lesions of the collateral ligaments have not been well recognized, whereas the presence of entheseous new bone on the proximal border of the navicular bone, reflecting previous insertional desmopathy, is well documented in both normal horses and horses with navicular disease.[7,8,16] Its significance remains uncertain, although more extensive new bone tends to be associated with other signs of navicular disease.[16] Mineralized and osseous fragments in the distal sesamoidean impar ligament also have been recognized in both normal horses and in horses with navicular disease, and the clinical significance remains difficult to determine. Fragments associated with a defect in the distal margin of the navicular bone were more common in horses with navicular disease than in age-matched controls[8] (Fig. 30-3). Although other gross and histological abnormalities of the distal sesamoidean impar ligament have not been well documented, inflammation has recently been recognized histologically at the intersection of the distal sesamoidean impar ligament and DDFT in horses with clinical signs of navicular syndrome.[17] This region is rich in sensory nerve endings, with many arteriovenous complexes that are damaged in horses with navicular disease.[18] Aging changes have been seen in the region of insertion of the distal sesamoidean impar ligament and the DDFT, with a change in fibroblast shape and an increase in proteoglycans.[12] The functional significance of this is not yet known.

The incidence and cause of primary bursitis of the navicular bursa is not known, nor is its relationship to the development of navicular disease. Villous hypertrophy, hyperplasia of synovial lining cells, and venous congestion have been described in association with navicular disease, whereas the synovial membrane appeared uniform in six normal horses of undetermined age.[19] However, in anther study comparing immature horses, horses with navicular disease, and age-matched controls, 3 of 25 age-matched controls had evidence of asymptomatic chronic synovitis. In both the navicular disease group and the age-matched controls, mild hyperplasia and hypertrophy was seen compared with immature horses up to 3 years of age.[8]

Pain associated with navicular disease may be due to venous distention following venous hypertension. Dilated venules and sinusoids entrapped in fibrous marrow have been identified only in horses with navicular disease.[7] Raised intraosseous pressure has been measured in horses with navicular disease.[20,21] The contribution of other causes or sources of pain remains open to speculation, although many sensory nerve endings also have been identified in the collateral ligaments of the navicular bone and the distal sesamoidean impar ligament.[22]

DIAGNOSTIC CONSIDERATIONS

History

Most horses present with a history of an insidious onset of loss of performance, shortening of stride, or intermittent shifting bilateral forelimb lameness that usually is worst on firm ground. The complaint from the owner may be loss of action, stiffness, unwillingness to jump, especially drop fences, and inability to lengthen stride. Less commonly a horse may have acute-onset, moderate to severe, usually unilateral but sometimes bilateral, forelimb lameness. The condition is rare in ponies compared with horses and, although hindlimb lameness associated with navicular navicular disease is unusual, it does occasionally occur in both ponies and horses.[23,24]

Clinical signs often are first apparent when the horse is approximately 7 to 9 years of age, although the disease can occur in young horses of 3 to 4 years of age, which may have fairly advanced radiographic abnormalities. Lameness may first become apparent after a period of enforced rest because of some other unrelated problem or after a change in management. Development of lameness soon after change of ownership, associated with a change in trimming and shoeing,

different work patterns, and altered periods of turn-out is not uncommon.

The incidence of navicular disease varies among breeds. The Quarter Horse,[25] Warmblood horses,[26] and Thoroughbred cross horses[27] have a relatively high incidence, whereas the incidence, recognition, or both in some breeds such as the Finnhorse, the Arab,[28] and the Frisian is relatively low. The incidence in higher level competition horses appears to be lower than in horses subject to lesser athletic demands.[8,24]

Clinical Signs

Pain when the horse is standing at rest may be a feature of navicular disease; however, this is a variable finding and some clinically normal horses habitually point one or both front feet. A horse that has resting pain associated with navicular disease may stand pointing one front foot, sometimes alternating between feet. Alternatively, an affected horse may pack bedding under its heels or have a tendency to sit on a manger to relieve pressure on the palmar aspect of the foot. Navicular disease is recognized in horses with a wide variety of foot shapes: narrow, boxy upright feet of the Quarter Horse and low, collapsed heels typical of many Thoroughbreds. Navicular disease often is seen in association with poor mediolateral or dorsopalmar foot balance. If lameness is consistently worse on one forelimb, the feet may become asymmetrical in shape, with the lamer foot being narrower with a taller heel.

The digital vessels sometimes are palpably enlarged, but this is an inconsistent, non-specific finding. The response to hoof testers applied to the frog region is often negative,[5,24,29] although other authors[30] have described a positive response as a fairly consistent feature of navicular disease. Pain may be elicited at the toe if the horse has been repeatedly overloading the toe, resulting in sub-solar bruising. Differences in clinical signs observed by different clinicians may reflect genuine differences in horse populations in different geographical locations. Distention of the distal interphalangeal joint capsule is sometimes seen in association with navicular disease, but not invariably so. Raised intra-articular pressure (>40 mm Hg) is said to be associated with navicular disease,[31] although in my experience there is considerable variability in the degree of distal interphalangeal joint distention and pressure in both normal and lame horses.

Lameness is sometimes apparent when the horse is moving on a hard surface in straight lines. It may fluctuate in degree within an examination period or between examinations performed on different days. The horse may show a tendency to stumble associated with an altered foot placement. Overt unilateral lameness may be evident, but in some horses there is only marginal shortening of stride and reduced lift to the stride, which may be difficult to detect if the horse was not previously known to the observer. The horse may move better on a soft surface, even in circles. No clinical signs are detectable in some horses when examined moving in hand on a hard surface. Lameness generally is accentuated if the horse moves in circles on a hard surface, especially with the lame limb on the inside of the circle. In some horses, lameness is apparent only under these circumstances. Less commonly lameness is accentuated when the lame limb is on the outside of a circle. Sometimes lameness cannot be detected unless the horse is ridden, when it may move in a slightly stiff, "flat," and restricted fashion. Recognition of this requires prior knowledge of the horse, knowledge of the expected quality of movement of a horse of that type, or respecting the opinion of the rider that the horse has "lost some action." A vast difference may be detected by desensitizing both front feet. This may be easier to appreciate when riding the horse than watching it.

The response to distal limb flexion is extremely variable. Many horses with navicular disease show a transient, mild increase in lameness.[5,24,29] Resistance to flexion or marked accentuation of lameness after flexion is unlikely to reflect navicular bone pain. Elevation of the toe of the foot on a wedge or wooden board, with the contralateral limb picked up, resulting in extension of the distal interphalangeal joint, may increase lameness, but this response is neither consistent nor pathognomonic for navicular disease.

If the horse's foot conformation is poor and the feet are not trimmed and shod optimally, it is worthwhile improving the trimming and shoeing and re-assessing the lameness after several weeks. If the lameness has markedly improved, it is unlikely to reflect navicular disease.

Response to Local Analgesic Techniques

Perineural analgesia of the palmar digital nerves, using 1.5 to 2 ml of mepivacaine hydrochloride (2%) per site, performed immediately axial to the cartilages of the foot usually results in improvement in lameness. However, lameness often is not alleviated fully.[29,32] Occasionally there is no response. Lameness is generally alleviated completely after perineural analgesia of the palmar nerves, performed at the base of the proximal sesamoid bones, unless there is another concurrent source of pain. Horses should always be re-evaluated in both straight lines and circles to determine if lameness becomes apparent or is accentuated on the contralateral limb.

Intra-articular analgesia of the distal interphalangeal joint using 6 ml of mepivacaine can alleviate or improve pain associated with the navicular bone within 5 minutes of injection.[28,32,33] A negative response does not preclude navicular pain, because approximately 20% of horses have a negative response to intra-articular analgesia of the distal interphalangeal joint and a positive response to intra-thecal analgesia of the navicular bursa[32]; this has correlated with both radiographic and post-mortem abnormalities of the navicular bone. A positive response to intra-articular analgesia of the distal interphalangeal joint is a non-specific result (Figs. 30-4 and 30-5), since this technique can alleviate solar pain[34] and pain associated with the palmar processes of the distal phalanx, the distal sesamoidean impar ligament, the DDFT, and the joint itself.[32,33,35] Misleading positive results are more likely to occur with larger volumes of local anesthetic solution and evaluation of the response more than 10 minutes after injection.[36]

A positive response to analgesia of the navicular bursa (3 to 4 ml of mepivacaine) usually reflects primary navicular bone pain or primary bursal pain. For accurate injection into the navicular bursa the needle should be sited in the middle of the flexor surface of the navicular bone. Placement of a needle into the navicular bursa is best performed under radiographic control or should be followed by injection of a radiographic contrast agent to check its position. If the needle is positioned too far proximally, there is danger of injection into the proximal palmar outpouching of the distal interphalangeal joint or the digital flexor tendon sheath. If the needle is too far distal, it may enter the distal palmar outpouching of the distal interphalangeal joint. If synovial fluid appears spontaneously in the needle hub, it is unlikely that the needle is positioned correctly. Improvement in lameness is usually appreciated within 5 minutes of injection of local anesthetic solution. A negative response to analgesia of both the distal interphalangeal joint and the navicular bursa makes it unlikely that the horse has navicular disease.

Radiographic Examination

Radiographic examination of the navicular bone should be performed after removal of the shoes and appropriate preparation of the feet, and should include lateromedial (LM), dorsoproximal-palmarodistal oblique (DPr-PaDiO), and palmaroproximal–palmarodistal oblique (PaPr-PaDiO) views. Flexed oblique views of the distal interphalangeal joint

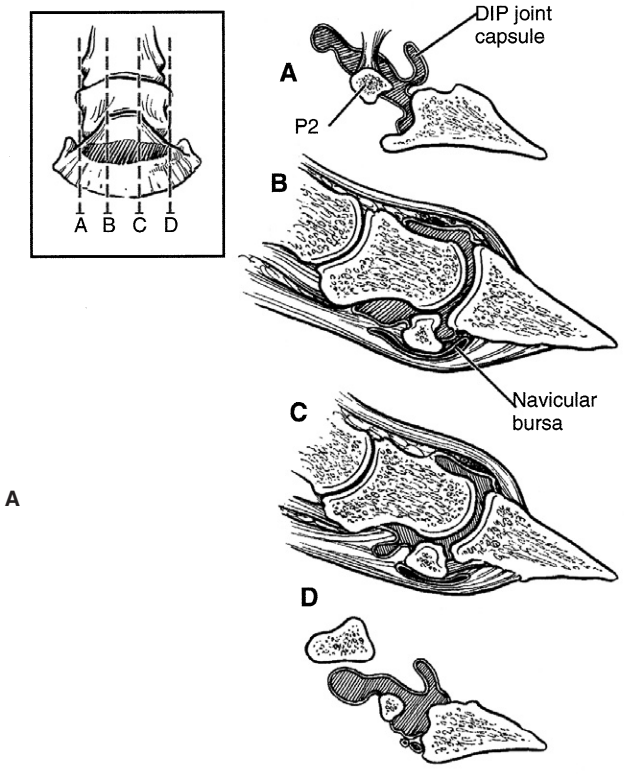

Fig. 30-4 **A,** Diagram of the relationship between the distal interphalangeal joint *(DIP)* capsule, the collateral ligaments of the navicular bone, and the distal sesamoidean impar ligament. The four sagittal sections through the foot are denoted with lines *A, B, C,* and *D.* **B,** Lateromedial radiographic view of a foot. Three milliliters of radiopaque contrast medium has been injected into the navicular bursa to show its proximodistal extent. *P2,* Middle phalanx.

also are useful for evaluation of its articular margins. Appropriate positioning of the feet is critical for high-quality images. Correct angulation of the x-ray beam for PaPr-PaDiO views is crucial for diagnostic images. Artifacts can be created unless the x-ray beam is tangential to the palmar surface of the navicular bone.[37] The correct angle can vary between 35° and 50° depending on the shape of the foot.

The proximal border of the navicular bone has two margins: the flexor surface, which is always convex in a DPr-PaDiO view, and the articular margin, which is variable in shape. It can be categorized as concave, undulating, straight, or convex. In the Dutch Warmblood an enhanced risk of the development of navicular disease has been suggested if the articular margin is concave or undulating, rather than convex.[2]

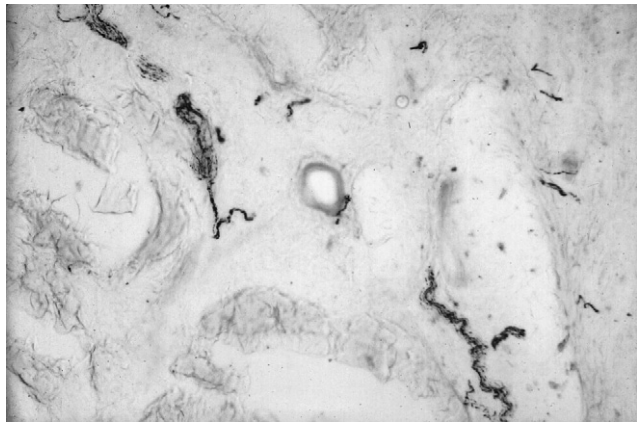

Fig. 30-5 Histological sample stained to show substance P, a small peptide, within thinly myelinated sensory nerves in the collateral ligaments of the navicular bone, appearing to innervate microvessels and pass toward the navicular bone. (Courtesy Dr. R. Bowker.)

The following features should be evaluated in all views[37] (Figs. 30-6 through 30-11): the presence, number, shape, size, and location of radiolucent zones along the distal borders of the navicular bone; the presence of lucent zones along the proximal border of the navicular bone; lucent zones within the medulla of the bone; trabecular pattern within the medulla; entheseophyte formation on the proximal or distal aspects of the bone; presence of mineralization within a collateral ligament of the navicular bone; presence of articular osteophytes; thickness of the flexor cortex; regularity of outline of the flexor cortex; lucent areas in the flexor cortex; new bone formation on the flexor surface; corticomedullary definition; and presence of mineralized fragments distal to the navicular bone.

A grading system for evaluation of the navicular bone in LM and DPr-PaDiO views was devised by Dik.[38] An adapted version is presented in Box 30-1. The degree of lameness and the degree of radiological abnormality often are poorly correlated. Some horses with navicular bone pain have no detectable radiological change. Some horses, especially young horses, have relatively advanced radiographic changes when lameness is first recognized. In many horses with suspected navicular disease the radiological abnormalities are equivocal. The ability to document the progression of radiological change over time (years) is relatively unusual. The appearance of the navicular bone varies in normal horses,[39,40] which may in part be related to breed and conformation and thus biomechanical forces placed on the navicular apparatus. Horses with very upright conformation tend to have a thinner flexor cortex compared with horses with lower heels.[24]

Some changes are more likely to be seen in older athletes than in immature horses and reflect the biomechanical stresses placed on the bone and its supporting structures. Older horses are more likely to have entheseophyte formation on the proximal border of the bone, especially laterally, compared with immature athletes. The presence of new bone on the proximal border is not always associated with lameness.[41] The flexor cortex generally increases in thickness with age as an adaptive remodeling process in athletic horses. The number, shape, and size of radiolucent zones along the distal border of the navicular bone may vary between breeds. Mineralized fragments on the distal border of the navicular bone may be seen as an incidental radiological abnormality, although there was a higher incidence (39%) in horses with navicular disease at postmortem examination compared with age-matched controls

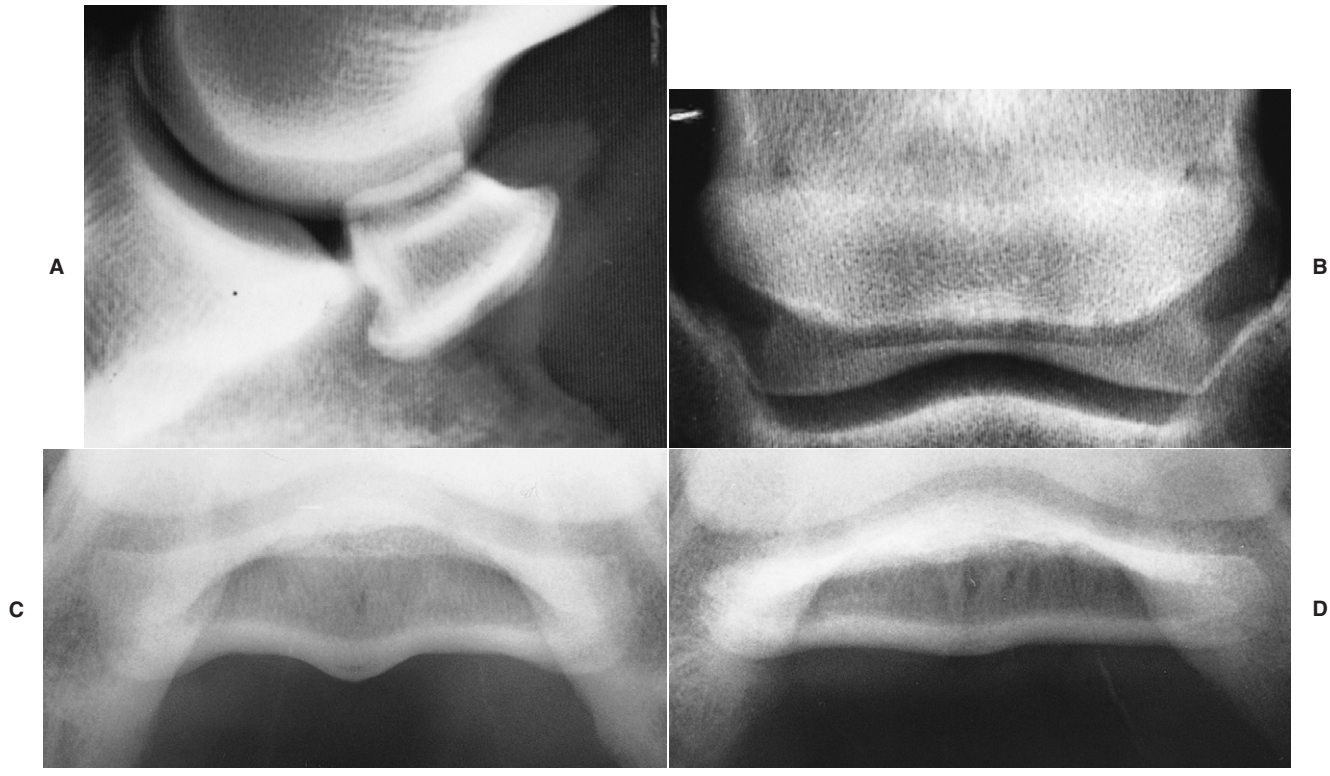

Fig. 30-6 **A,** Lateromedial view of a normal navicular bone. **B,** Dorsoproximal-palmarodistal oblique view of a normal navicular bone. **C,** Palmar 45° proximal-palmarodistal oblique view of a normal navicular bone with a well-defined, crescent-shaped lucent zone in the central eminence of the flexor cortex. **D,** Palmar 50° proximal-palmarodistal oblique view of a normal navicular bone in a horse with upright foot conformation. The flexor cortex is flatter compared with **C.**

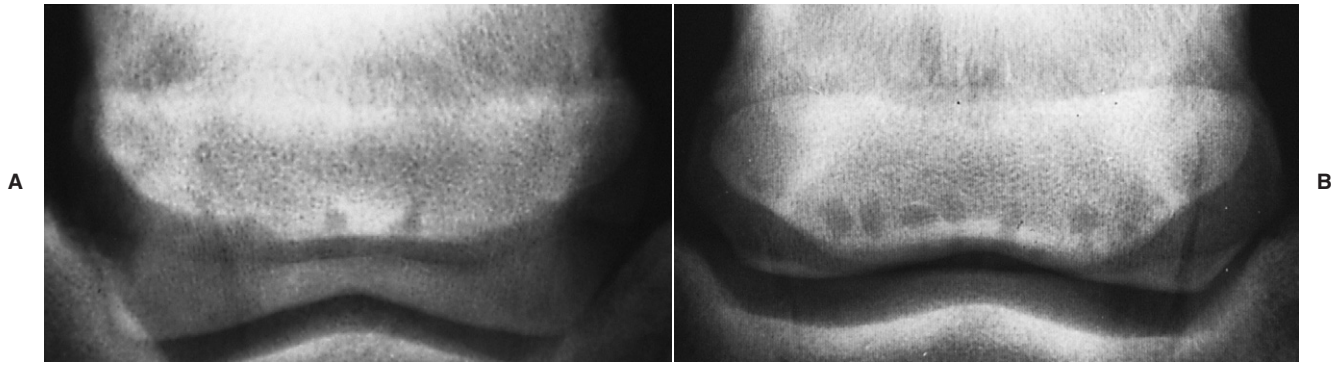

Fig. 30-7 **A,** Dorsoproximal-palmarodistal oblique view of a navicular bone of a normal horse. There are several small lucent zones along the distal border of the bone. **B,** Dorsoproximal-palmarodistal oblique view of a navicular bone of a horse with clinical signs of navicular disease. Note the variable shape, size, and position of the lucent zones along the distal borders of the bone.

(4%).[8] In some of the horses with clinical evidence of navicular disease, mineralized fragments occurred in the absence of other pathological changes. Mineralized fragments were found in 3% of 1111 sound horses examined radiographically, whereas a 28% incidence was seen radiographically in 118 horses with navicular disease.[16]

A high-quality PaPr-PaDiO view is the most sensitive projection for detection of subtle, but significant radiological changes, including medullary sclerosis (see Fig. 30-11), reduced corticomedullary demarcation, change in contour of the flexor cortex resulting from either erosions (see Fig. 30-10, *A* to C) or new bone formation (Fig. 30-10, *D*), and lucent zones in the flexor cortex. Radiographic abnormalities may not be detectable in other views. Small, crescent-shaped lucent zones within the flexor cortex at the sagittal ridge are probably an aging change reflecting adaptive remodeling of the subchondral bone.[42]

Generally the larger number of radiological changes that are present in all radiographic projections, the more likely it is that the horse has clinical navicular disease. The most significant radiological changes likely to reflect navicular disease include cyst-like lesions within the medulla (see

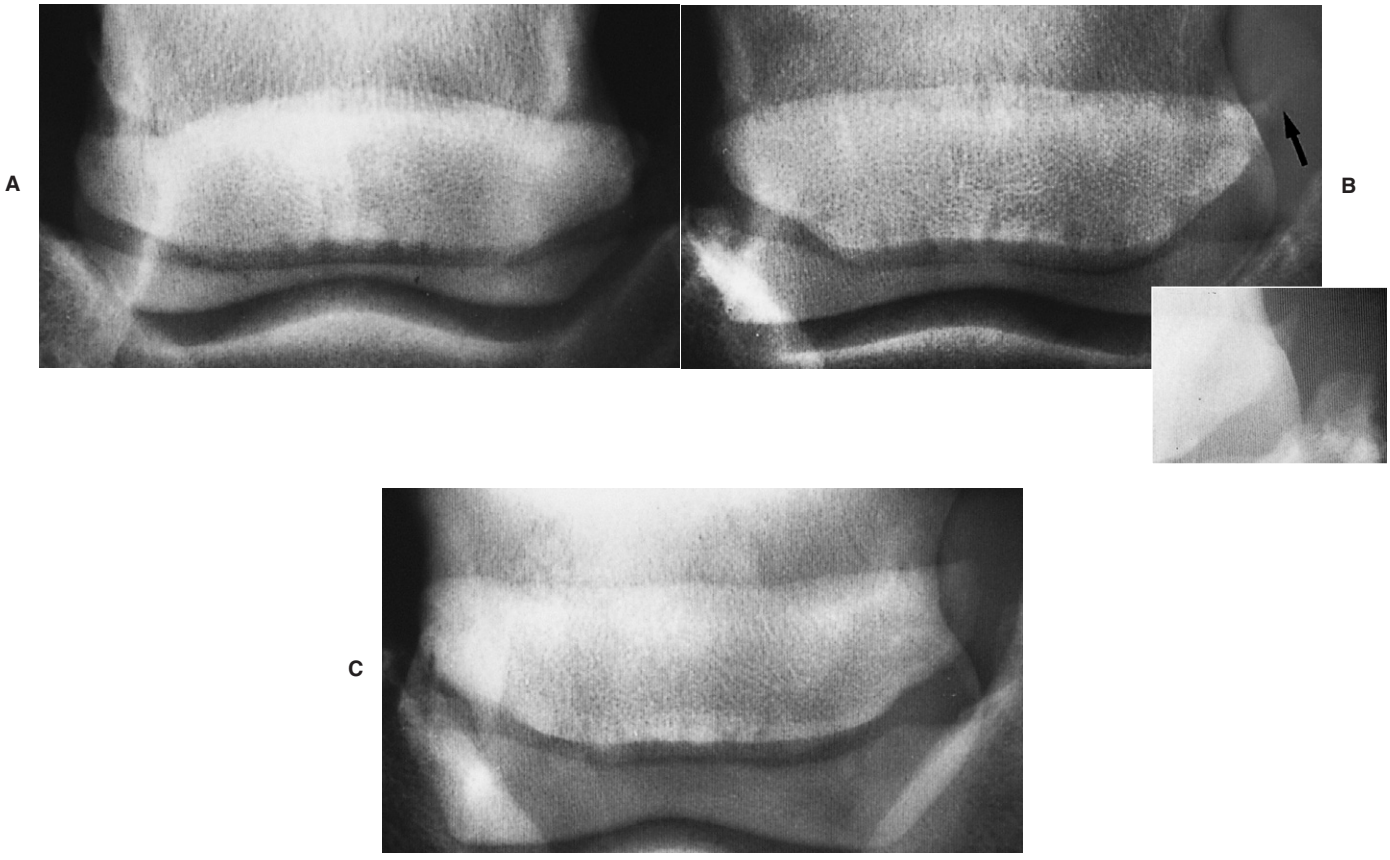

Fig. 30-8 **A,** Dorsoproximal-palmarodistal oblique view of a navicular bone of a normal horse. The asymmetry of the medial and lateral aspects of the bone is the result of mild entheseophyte formation. **B,** Dorsoproximal-palmarodistal oblique view of a navicular bone of a horse with clinical signs of navicular disease. There is a prominent entheseophyte on the proximolateral aspect of the bone *(arrow)*. Note also the lucent zones along the distal aspect of the bone. The *inset* shows detail of the entheseophyate. **C,** Dorsoproximal-palmarodistal oblique view of the navicular bone of a horse with clinical signs of navicular disease. Lateral is to the right. The modeling of the proximal border and lateral margin of the bone is the result of entheseophyte formation.

Fig. 30-9, *A*) that are discrete from distal border lucencies, medullary sclerosis, reduced corticomedullary demarcation, new bone on the flexor surface, and erosions of the flexor cortex of the bone (see Fig. 30-9, *B* and *C*).

In some horses, concurrent degenerative changes of the distal interphalangeal joint are also seen, with modeling* of the proximal dorsal articular margin of the navicular bone, the dorsal and palmar articular margins of the middle phalanx, and the extensor process of the distal phalanx.

In some horses the development of advanced radiological changes precedes the recognition of clinical signs. However, the absence of radiological abnormalities of the navicular bone does not preclude the presence of pain associated with the navicular bone and scintigraphic evidence of increased bone modeling.

*There is confusion between the histological and radiographic usage of the terms *remodeling* and *modeling.* Histologically, *remodeling* refers to resorption and formation of bone that is coupled and occurs in basic multicellular units. This regulates the microstructure of bone without altering its shape and is a continuous process, replacing damaged bone with new bone. Thus it cannot be appreciated on radiographs. The term has been used with regard to radiographs to describe the reshaping of bone to match form and function (e.g., after fracture repair), but strictly speaking, the term *modeling* should be used.

Histologically, *modeling* refers to resorption and formation of bone that is not coupled and occurs at anatomically different sites (bone drift). It is a continuous process that regulates the macroscopic appearance of bone according to Wolff's law. In terms of radiography, *modeling* has been used to describe the formation of bone relative to the cartilage model that is being replaced (i.e., the normal formation of bone). Thus the two definitions do not agree; to avoid confusion, strictly speaking the term *modeling* should be applied to describe the change in shape of a bone as it adapts to the stresses applied to it.

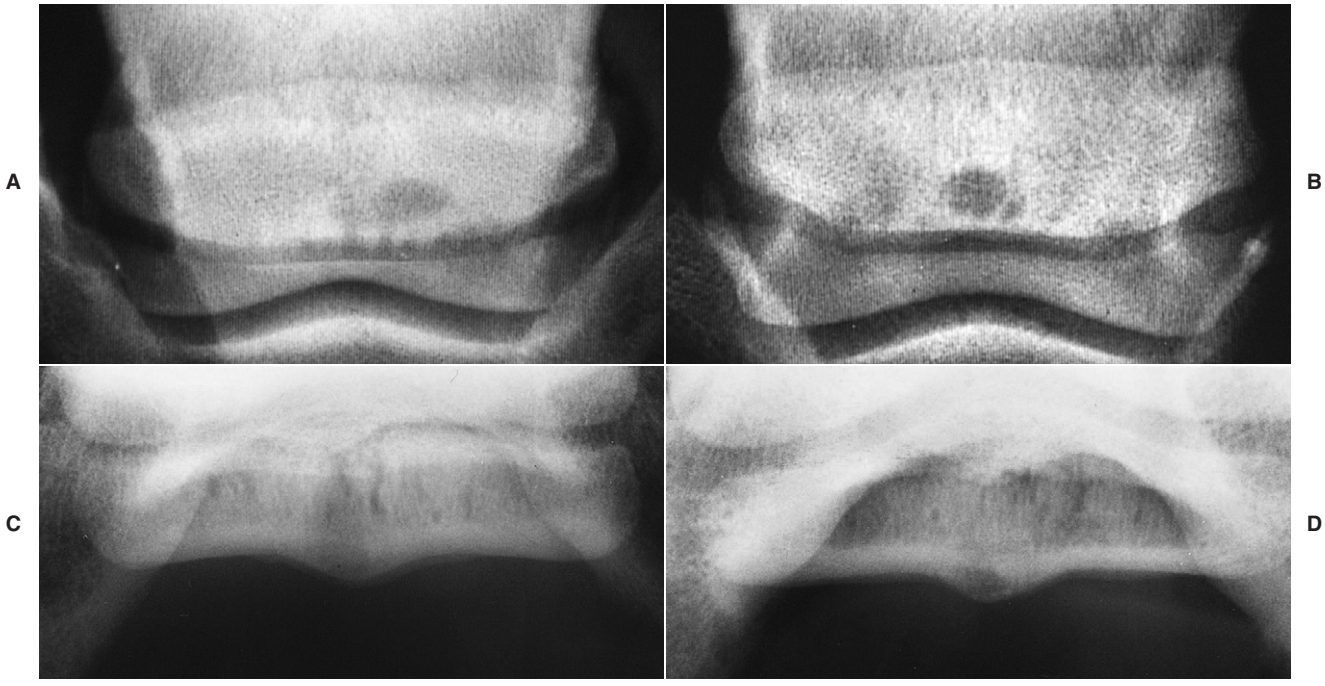

Fig. 30-9 **A,** Dorsoproximal-palmarodistal oblique view of a navicular bone with a large cyst-like lesion within the medulla of the navicular bone. This lesion did not penetrate the flexor cortex of the bone. The horse had a bilateral forelimb lameness, which was improved by analgesia of the distal interphalangeal joint or navicular bursa. **B,** Dorsoproximal-palmarodistal oblique view of a navicular bone with a large cyst-like lesion within the medulla of the bone, which did penetrate the flexor cortex of the bone (see **C**).The horse had a bilateral forelimb lameness that was worse on soft ground with the lamer limb on the outside of a circle. Lameness was improved by palmar (abaxial sesamoid) nerve blocks. **C,** Palmaroproximal-palmarodistal oblique view of the same navicular bone as in **B,** with a large lucent area penetrating the flexor cortex of the bone. **D,** Palmaroproximal-palmarodistal oblique view of a navicular bone of a 7-year-old event horse that had been rested because of a tendon injury and developed left forelimb lameness soon after resuming work. Lameness was alleviated by analgesia of the navicular bursa. There is a well-defined lucent zone in the flexor cortex in the medial aspect of the sagittal ridge of the navicular bone (compare with Fig. 30-6, C). Radiographic abnormalities were detectable only in this projection.

Box • 30-1

Radiographic Findings of the Navicular Bone in Normal and Diseased Horses

Grade	*Condition*	*Radiographic Findings*
0	Excellent	Good corticomedullary demarcation; fine trabecular pattern. Flexor cortex of uniform thickness and opacity. No lucent zones along the distal border of the bone, or several (fewer than six) narrow conical lucent zones along the horizontal distal border. Right and left navicular bones symmetrical in shape.
1	Good	As above, but lucent zones on the distal border of the navicular bone are more variable in shape.
2	Fair	Slightly poor definition between the palmar cortex and the medulla as a result of subcortical sclerosis. Crescent-shaped lucent zone in the central eminence of the flexor cortex of the bone. Several (fewer than eight) lucent zones of variable shape along the distal horizontal border of the navicular bone. Mild entheseophyte formation on the proximal border of the navicular bone. Navicular bones asymmetrical in shape.
3	Poor	Poor corticomedullary definition as a result of medullary sclerosis. Thickening of the dorsal and flexor cortices. Irregular opacity of the flexor cortex of the bone. Many (more than seven) radiolucent zones along the distal horizontal or sloping borders of the navicular bone. Lucent zones along the proximal border of the bone. Large entheseophyte formation on the proximal border of the bone. Discrete mineralization within a collateral ligament of the navicular bone. Radiopaque fragment on the distal border of the navicular bone.
4	Bad	Large cyst-like lesion within the medulla of the navicular bone. Lucent region in the flexor cortex of the navicular bone. New bone on the flexor cortex of the navicular bone.

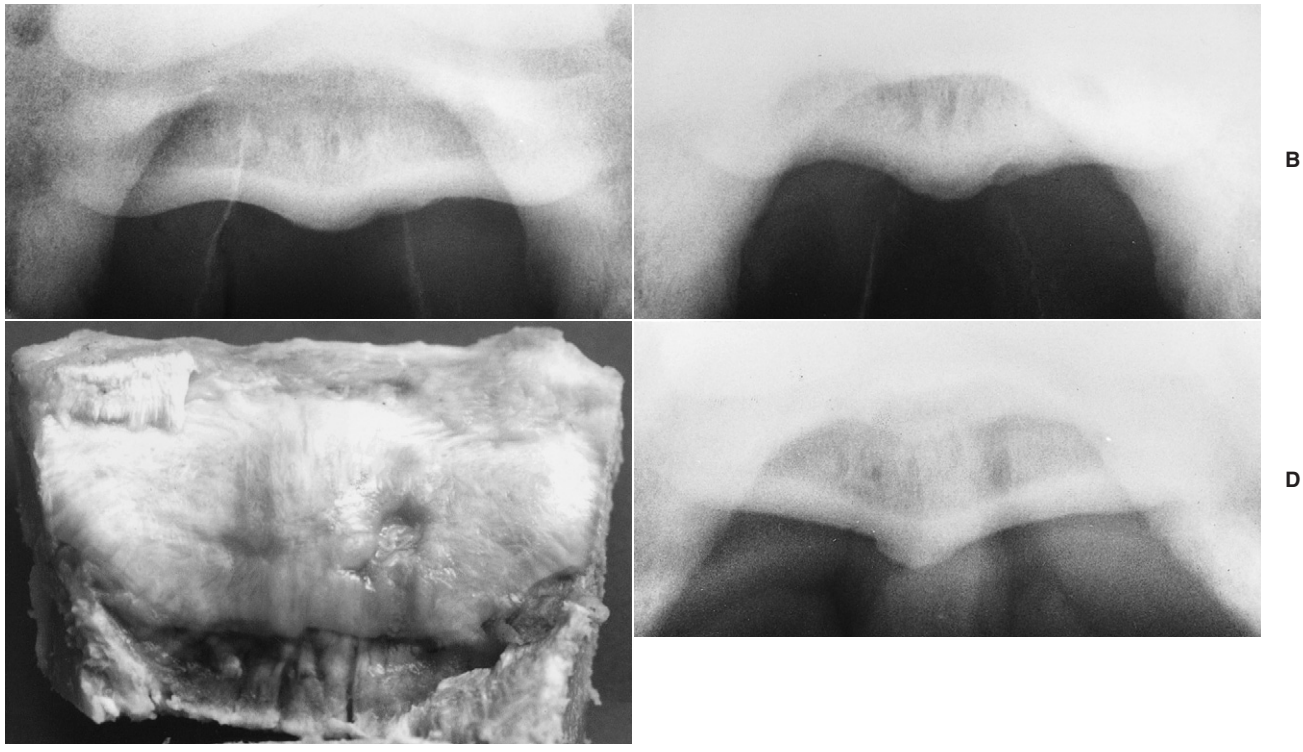

Fig. 30-10 **A,** Palmar 45° proximal-palmarodistal oblique view of a navicular bone with an irregular contour of the lateral aspect of the flexor cortex. Lateral is to the right. The horse had a bilateral forelimb lameness, which was improved by intra-articular analgesia of the distal interphalangeal joint or analgesia of the navicular bursa. **B,** Palmar 35° proximal-palmarodistal oblique view of the same navicular bone as in **A.** The irregularity of the flexor cortex is more obvious. Apparent reduction in corticomedullary demarcation is an artifact, the result of the angle of projection. Lateral is to the right. **C,** The same horse as in **A.** The gross pathological specimen has a defect in the flexor surface, lateral to the sagittal ridge. Lateral is to the right. **D,** Palmaroproximal-palmarodistal oblique view of a navicular bone of a horse with unilateral forelimb lameness improved by analgesia of the navicular bursa. There is new bone on the flexor cortex of the navicular bone. Radiological abnormalities were detectable only in this projection.

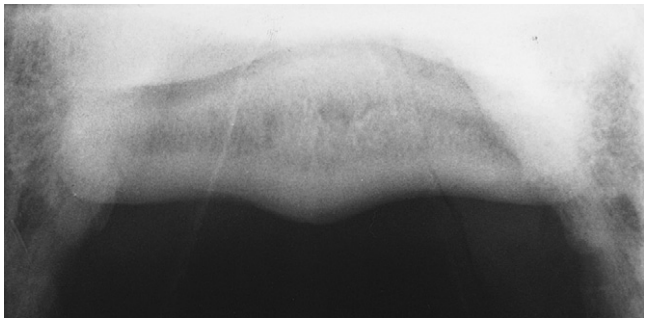

Fig. 30-11 Palmaroproximal-palmarodistal oblique view of a navicular bone of a 6-year-old riding horse with acute-onset right forelimb lameness that was improved by perineural analgesia of the palmar digital nerves or intra-articular analgesia of the distal interphalangeal joint. There is increased cortical thickness, medullary sclerosis, and reduced corticomedullary demarcation (compare with Figs. 30-6, C and *D*). The left front navicular bone appeared normal. The lameness persisted.

Contrast Radiography of the Navicular Bursa

Contrast radiography of the navicular bursa may reveal a number of abnormalities not detectable on plain radiographs, including thinning or erosion of the flexor fibrocartilage of the navicular bone, loss of the dye column thought to be caused by adhesion formation between the DDFT and the navicular fibrocartilage, and filling defects on the palmar aspect of the bursa perhaps associated with surface fibrillation of the DDFT.[24,43] The clinical significance of these findings is currently questionable, because it has been well recognized that thinning of the flexor fibrocartilage is a normal aging change, and the significance of surface fibrillation of the DDFT is still open to debate.

Nuclear Scintigraphy

Tetracycline labeling of bone has revealed increased bone turnover in the navicular bone in navicular disease.[5,6] Nuclear scintigraphy offers a sensitive method of detecting increased bone turnover; however, this is not necessarily synonymous with either pathological change or pain.

Lateral pool- (soft tissue) and lateral and palmar (solar) bone–phase images of the front feet are required for accurate diagnosis of navicular disease.[44-46] In solar views of normal horses, uptake of the radiopharmaceutical in the navicular bone and the distal phalanx is approximately similar (Fig. 30-12, *A*). However, uneven uptake in various regions of the distal phalanx can complicate interpretation. Care must be taken when interpreting lateral views not to confuse uptake in the cartilages of the foot with uptake in the navicular bone. In solar views, uptake associated with the proximal interphalangeal joint can potentially be superimposed over the navi-

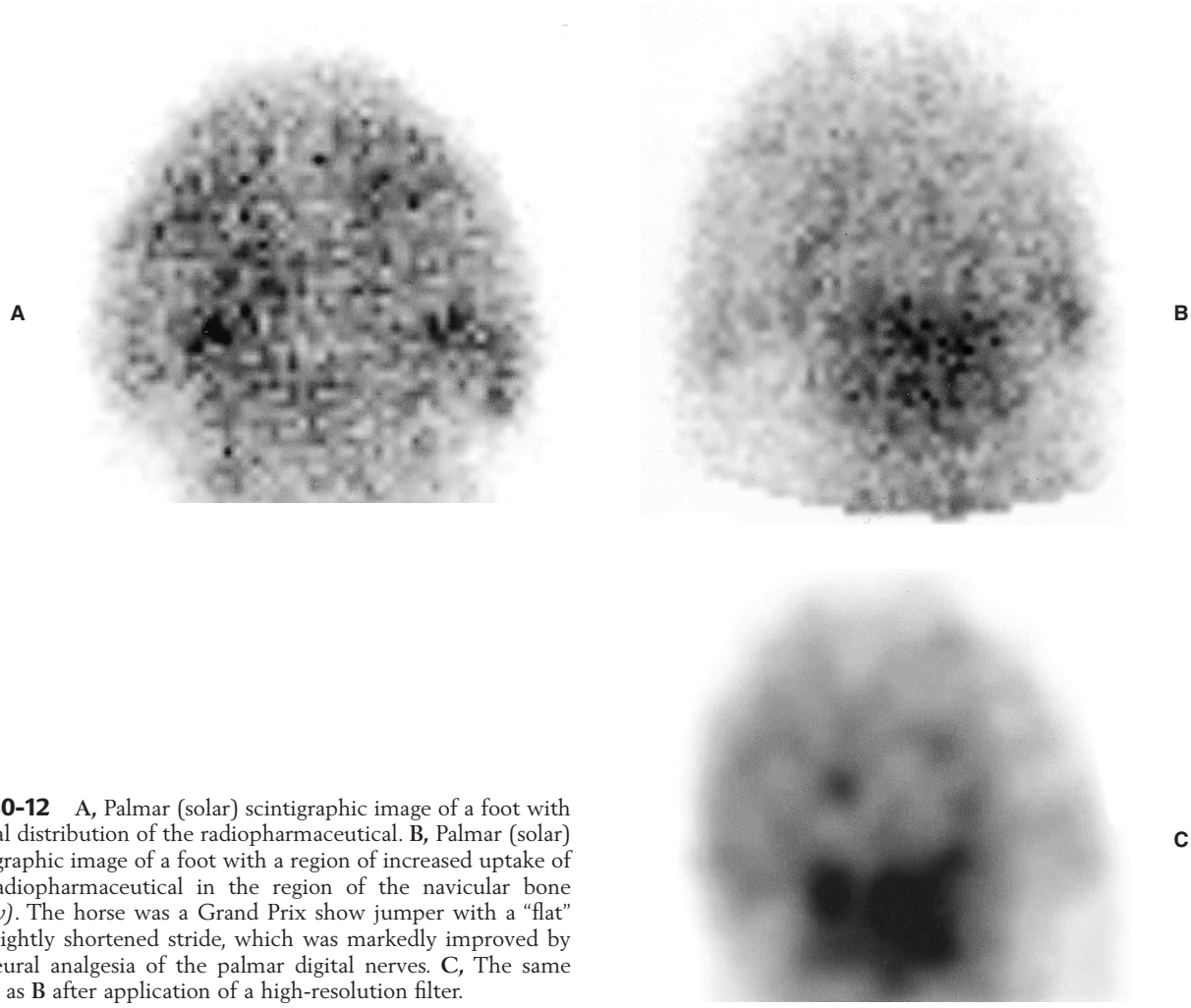

Fig. 30-12 **A,** Palmar (solar) scintigraphic image of a foot with normal distribution of the radiopharmaceutical. **B,** Palmar (solar) scintigraphic image of a foot with a region of increased uptake of the radiopharmaceutical in the region of the navicular bone *(arrow)*. The horse was a Grand Prix show jumper with a "flat" and slightly shortened stride, which was markedly improved by perineural analgesia of the palmar digital nerves. **C,** The same image as **B** after application of a high-resolution filter.

cular bone, unless the foot is well extended during image acquisition and the pastern region is masked with a lead shield. Interpretation can also be difficult, because some horses with unilateral lameness have a region of increased uptake of the radiopharmaceutical in both the left and right front navicular bones while apparently experiencing pain in only one limb.

Various patterns of uptake of the radiopharmaceutical have been identified in association with horses with palmar foot pain: focal or generalized increased radiopharmaceutical uptake (IRU) in the navicular bone (Fig. 30-12, *B*),[44-47] IRU in the navicular bone and the distal phalanx in the region of insertion of the DDFT,[24,48] IRU in both the navicular bone and the palmar processes of the distal phalanx,[44] IRU in one or both palmar processes of the distal phalanx,[24,44] and IRU in the distal phalanx at the sites of insertion of the DDFT.[48] The close proximity of the regions of insertion of the DDFT and the distal sesamoidean impar ligament makes accurate differentiation of insertional injuries difficult. It also may be difficult to differentiate IRU in the subchondral bone of the distal interphalangeal joint.

IRU in the navicular bone, with or without IRU at the region of insertion of the DDFT, has correlated well with a positive response to intra-thecal analgesia of the navicular bursa.[48] False-positive results can be found in clinically normal horses, although a relatively high percentage have subsequently developed lameness associated with palmar foot pain.[48,49]

There is a relatively high incidence of horses with clinical signs compatible with navicular disease and a positive response to intra-articular analgesia of the distal interphalangeal joint or intra-thecal analgesia of the navicular bursa, which have no detectable radiological abnormalities of the navicular bone but have IRU of radiopharmaceutical associated with the navicular bone.[48] Thus nuclear scintigraphy offers a sensitive method of diagnosis of navicular disease, although specificity is probably less good.

Computed Tomography and Magnetic Resonance Imaging

Computed tomography (CT; see Chapter 20) and magnetic resonance imaging (MRI; see Chapter 21) are potentially more sensitive than radiography in determining structural lesions of the navicular bone[50-53] (Fig. 30-13) and identifying degenerative changes in the articular cartilage of the distal interphalangeal joint and primary lesions in the DDFT, the distal sesamoidean impar ligament, and the collateral ligaments of the navicular bone. An abnormal amount of fluid within the distal interphalangeal joint or the navicular bursa and synovial proliferation may be identified by MRI (Figs. 30-14 and 30-15). MRI permits detection of pathophysiological changes within the navicular bone, such as bone edema. However, both CT and MRI require the horse to be under general anesthesia. These techniques may permit clinicians to develop further understanding of the pathological processes earlier in the course of navicular disease.

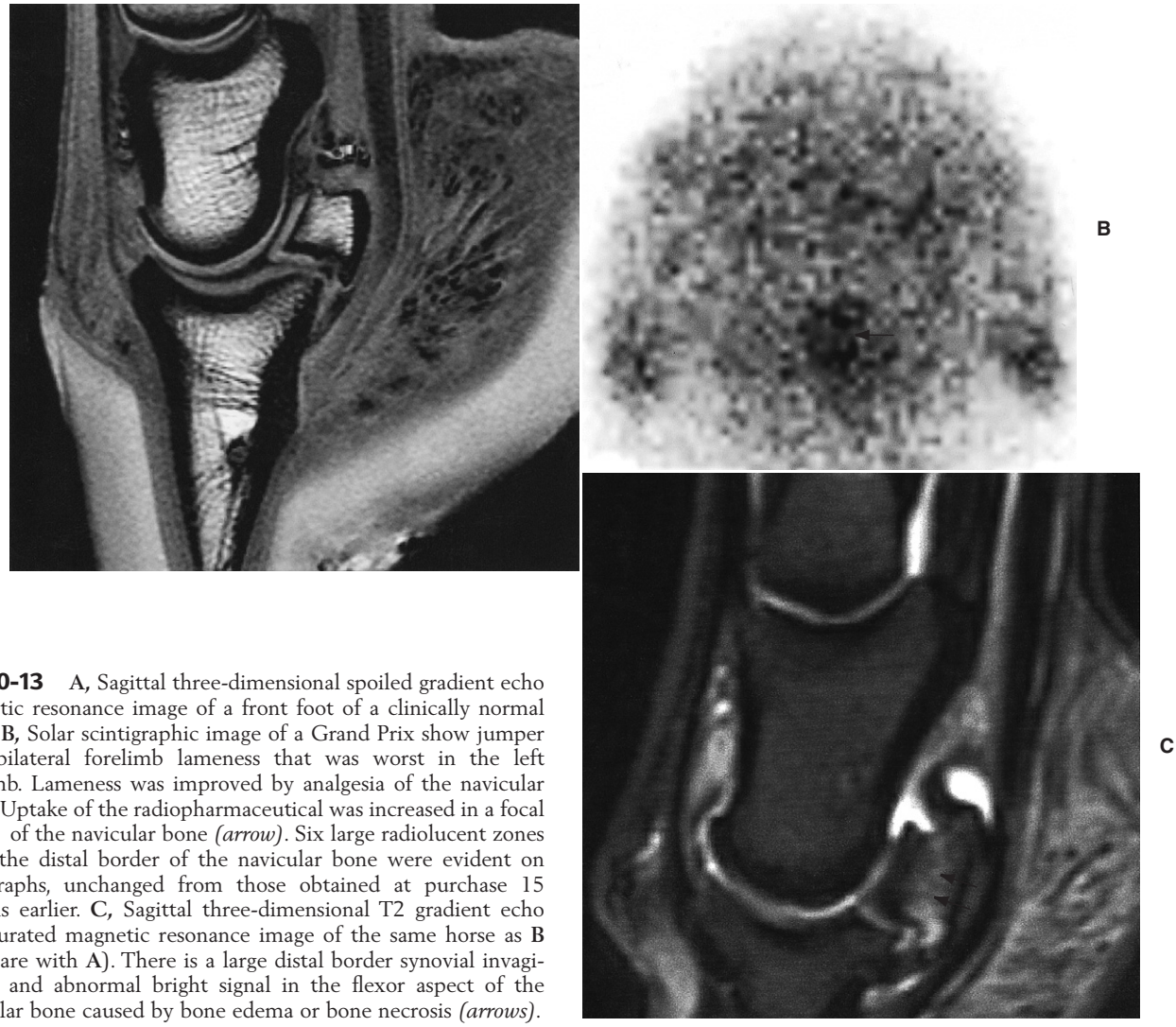

Fig. 30-13 **A,** Sagittal three-dimensional spoiled gradient echo magnetic resonance image of a front foot of a clinically normal horse. **B,** Solar scintigraphic image of a Grand Prix show jumper with bilateral forelimb lameness that was worst in the left forelimb. Lameness was improved by analgesia of the navicular bursa. Uptake of the radiopharmaceutical was increased in a focal region of the navicular bone *(arrow)*. Six large radiolucent zones along the distal border of the navicular bone were evident on radiographs, unchanged from those obtained at purchase 15 months earlier. **C,** Sagittal three-dimensional T2 gradient echo fat-saturated magnetic resonance image of the same horse as **B** (compare with **A**). There is a large distal border synovial invagination and abnormal bright signal in the flexor aspect of the navicular bone caused by bone edema or bone necrosis *(arrows)*.

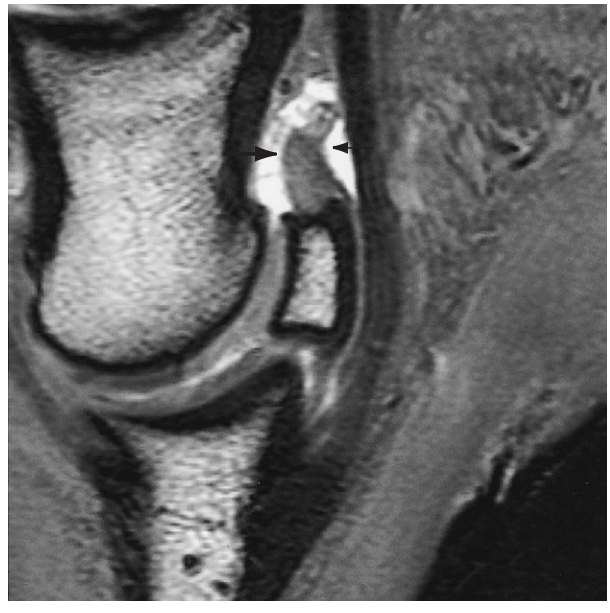

Fig. 30-14 Sagittal three-dimensional T2* gradient echo magnetic resonance image of a Grand Prix show jumper with lameness improved by analgesia of the navicular bursa. There is an increased amount of fluid in the navicular bursa *(small arrow)* reflecting synovitis (compare with Fig. 30-13, *A)* and an abnormal amount of fluid within the distal interphalangeal joint *(large arrow)*.

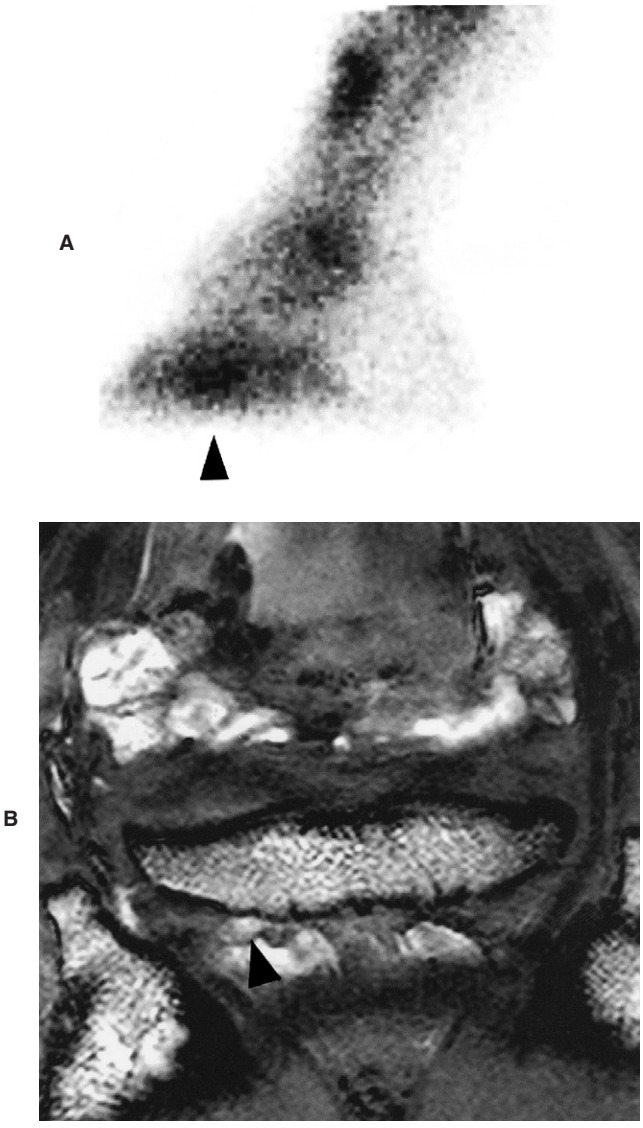

Fig. 30-15 **A,** Lateral scintigraphic image of the left front foot of a 9-year-old event horse with unilateral lameness that was improved by analgesia of the distal interphalangeal joint or navicular bursa. Uptake of the radiopharmaceutical is increased on the solar aspect of the distal phalanx in the region of insertion of the deep digital flexor tendon and distal sesamoidean impar ligament (DSIL). No significant abnormality was detectable radiographically. **B,** Dorsal three-dimensional T2* gradient echo magnetic resonance image of the same horse as in **A** shows an osseous fragment in the DSIL *(arrow)* and an abnormal signal in the DSIL lateral to it. Lateral is to the right.

Endoscopic Evaluation of the Navicular Bursa

Endoscopic examination of the navicular bursa (see Chapter 24) permits evaluation of the fibrocartilage on the flexor surface of the navicular bone, the navicular bursa itself, the overlying dorsal surface of the DDFT, and a limited view of the distal sesamoidean impar ligament.[24,54] Thus it is possible to definitively identify adhesions between the DDFT and the palmar aspect of the navicular bone, thinning or full-thickness erosion of the flexor fibrocartilage of the bone, fibrillation of the dorsal aspect of the DDFT, and synovitis of the bursa. Tears in the distal sesamoidean impar ligament have also been identified,[24] but these are thought to be of traumatic origin rather than part of the navicular disease complex. It is important to differentiate between age-related changes and pathological abnormalities.

OTHER SOFT TISSUE CAUSES OF PALMAR HEEL PAIN

Our understanding of soft tissue pain associated with the palmar aspect of the foot has been limited until recently to identification of pain seen in association with poor foot balance, poor shoeing, or both factors that responded to corrective trimming and shoeing (see Chapter 27). Horses that respond satisfactorily to trimming and shoeing alone are unlikely to have chronic navicular disease and although navicular bone pain may be present, the precise source of pain cannot be determined. A hemodynamic function for the cartilages of the foot and associated vascular system may contribute to force dissipation.[55] Peptidergic sensory nerve endings also have been identified in the vascular channels of the cartilages of the foot, close to pacinian-like corpuscles, indicating that this part of the foot may respond to sensory and proprioceptive stimuli. In horses with long toes and underrun heels the cartilages of the foot and associated vasculature are less well developed than in a better conformed foot. This may result in less effective force distribution, predisposing to the development of palmar foot pain. In addition, tissue morphological differences have been identified in the digital cushions between "healthy" and "weak" feet, so the digital cushion also may be involved in force dissipation.

Relatively recently other soft tissue causes of palmar heel pain have been confirmed. These horses usually have no detectable radiological abnormalities. Primary lesions of the DDFT are discussed in detail in Chapter 33. Other injuries that should be considered include the following:

- Desmitis of the distal sesamoidean impar ligament
- Desmitis of the collateral (suspensory) ligaments of the navicular bone
- Navicular bursitis
- Synovitis of the palmar pouch of the distal interphalangeal joint
- Combination injuries

Definitive diagnosis of most of these conditions requires MRI, and to date, only small numbers of horses have undergone imaging.[56] Therefore clinical manifestations must be restricted to case examples that may prove unrepresentative in the future. Desmitis of the lateral aspect of the distal sesamoidean impar ligament has been seen in conjunction with a focal tear in the articular cartilage of the distal interphalangeal joint on the ipsilateral side and an abnormal amount of fluid within the navicular bursa. Lameness had been acute in onset and had persisted despite rest. Lameness was alleviated by perineural analgesia of the palmar digital nerves and was improved by intra-articular analgesia of the distal interphalangeal joint. Scintigraphic examination revealed IRU in both pool- and bone-phase lateral images in the palmar aspect of the distal phalanx in the region of insertion of the DDFT and distal sesamoidean impar ligament.

A focal tear in the distal sesamoidean impar ligament also has been identified by endoscopic examination of the navicular bursa in an event horse with acute-onset severe lameness that improved after perineural analgesia of the palmar digital nerves or intra-articular analgesia of the distal interphalangeal joint[57] (Fig. 30-16).

Ectopic mineralization within a collateral ligament of the navicular bone has been seen in conjunction with other radiological abnormalities of the navicular bone compatible with navicular disease. Primary desmitis has not yet been recognized.

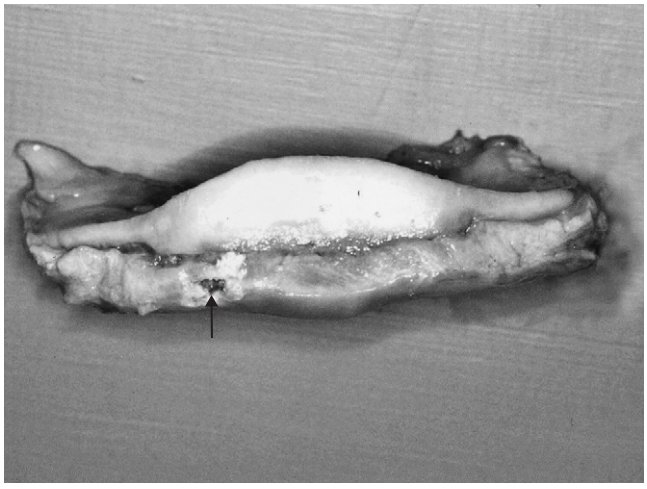

Fig. 30-16 Gross post-mortem specimen of the distal aspect of the navicular bone and the and distal sesamoidean impar ligament (DSIL). There is a focal tear in the DSIL *(arrow)*, which had been identified by arthroscopic evaluation of the navicular bursa. Surface fibrillation of the deep digital flexor tendon had also been seen.

Lameness alleviated by analgesia of the navicular bursa, but unassociated with any radiological sign of navicular disease, has previously been attributed to navicular bursitis, but further evidence for primary bursitis has been lacking. These horses frequently have scintigraphic evidence of abnormal modeling of the navicular bone[58] and respond only transiently to medication of the navicular bursa with either corticosteroids or hyaluronan. It therefore seems unlikely that these horses have a primary bursitis. MRI has demonstrated distention of the navicular bursa and the distal interphalangeal joint capsule in a horse with bilateral forelimb lameness that was substantially improved by analgesia of the navicular bursa. There was scintigraphic evidence of increased modeling of the navicular bones, but other than enlarged synovial invaginations on the distal border of the bones, no structural abnormality could be identified using MRI. The response to treatment of the bursae was only transient. MRI also has demonstrated synovial proliferation and distention of the bursa in association with desmitis of the distal sesamoidean impar ligament.

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CHAPTER • 31

Treatment and Prognosis of Horses with Navicular Disease

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NAVICULAR DISEASE

For the purposes of this chapter, *navicular disease* is defined as pain believed to originate from the navicular bone based on clinical signs, response to local analgesic techniques, and presence of substantial radiological abnormalities of one or both navicular bones (see Box 30-1) or scintigraphic evidence of increased modeling of the navicular bone(s). If the clinical signs are not suggestive of navicular disease, especially in horses with unilateral lameness, the possibility of a soft tissue injury should be considered. In these horses rest may be indicated.

In navicular disease the problem is managed because there currently is no cure for the condition. Rest is not a useful

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CHAPTER • 31

Treatment and Prognosis of Horses with Navicular Disease

John B. Madison and Sue J. Dyson

NAVICULAR DISEASE

For the purposes of this chapter, *navicular disease* is defined as pain believed to originate from the navicular bone based on clinical signs, response to local analgesic techniques, and presence of substantial radiological abnormalities of one or both navicular bones (see Box 30-1) or scintigraphic evidence of increased modeling of the navicular bone(s). If the clinical signs are not suggestive of navicular disease, especially in horses with unilateral lameness, the possibility of a soft tissue injury should be considered. In these horses rest may be indicated.

In navicular disease the problem is managed because there currently is no cure for the condition. Rest is not a useful

strategy in the management of most horses with pain in the navicular region, because although lameness improves somewhat in most horses after a period of rest, it often returns shortly after the horse resumes exercise. A period of rest for some other cause often precedes the onset of clinical signs of navicular disease.

The recent work of Wilson et al.[1] suggests that relief of pain is vitally important to alleviate a potentially vicious circle. The horse should be encouraged to land normally, rather than toe first, to avoid increased forces on the navicular bone from the deep digital flexor tendon (DDFT).

In horses with early navicular disease without major radiological abnormalities, medical management using non-steroidal anti-inflammatory drugs (NSAIDs), isoxsuprine, and careful trimming and shoeing may be effective, but obviously these drugs cannot be used if the horse will compete. The aim of medical management is to attempt to return the horse to regular work as soon as possible, starting initially with work predominantly in straight lines. Horses should be exercised as much as possible daily, combining ridden exercise with either turnout or walking on a horse walker. In some horses, use of corrective trimming and shoeing combined with isoxsuprine is sufficient, whereas in others additional analgesia is required using a NSAID. In horses with marked medullary sclerosis, lesions involving the flexor surface of the navicular bone, or central osseous cyst-like lesions, the response to medical management often is unsatisfactory and surgical treatment may be indicated.

MANAGEMENT STRATEGIES

Trimming and Shoeing

Successful management depends on a good relationship with an accomplished, reliable farrier. There is an art and science to farriery, and finding the right combination of the method of trimming and the selection of an appropriate shoe for a given horse often requires some trial and error. Each foot and each horse must be examined individually with regard to the distal limb and foot conformation, limb flight, foot placement, and the intended use of the horse. Both the farrier and the veterinarian must agree, and both must also agree to alter the initial approach if it does not work. Some degree of compromise is always involved. However, some principles apply for all horses with navicular disease. The degree to which lameness can be improved by trimming and shoeing alone is dependent on the horse's hoof-pastern angle and the previous suitability of trimming and shoeing. If the horse already had a well-conformed foot, little will be achieved in most horses with navicular disease.

Care must be taken either to correct or preserve dorsopalmar and lateromedial foot balance whenever possible. This depends to some extent on the natural shape of the horse's foot and its distal limb conformation. Ideally, the hoof-pastern axis should be straight, but whether this can be achieved depends on the horse's conformation. Radical changes in foot trimming may temporarily result in increased lameness; therefore it may be necessary to achieve correct foot balance in stages. The foot should be trimmed to maintain heel mass and shorten the toe to facilitate breakover. Sufficient shortening of the toes usually requires trimming the horn from the solar and dorsal aspects of the foot. The use of the so-called four-point or natural balance trim has recently been favored in some quarters, but the same principles of breakover can probably be achieved with more traditional trimming, provided that the toe is shortened sufficiently (see Chapter 27). Although the four-point trim results in clinical improvement in some horses, in others lameness has been increased, possibly caused by excessive trimming, resulting in altered hoof wall strain,[2] or by excessive sole pressure.

Elevation of the heel may relieve pressure from the DDFT on the palmar aspect of the navicular bone, with subsequent pain relief. In a study of normal Dutch Warmblood horses, elevation of the heel using a 6° wedge reduced the maximal force on the navicular bone by 24% compared with flat shoes.[3] However, not all horses with heel pain need to have the heel elevated and in many, the response is only temporary. One of us (J.B.M.) strives for a hoof angle between 50° and 55° and establishment of a straight foot-pastern axis. However, many horses with low, collapsed heels have a natural angle of no more than 35° and it is impractical to achieve an angle of 50° to 55°. Lateromedial radiographic views may be helpful to demonstrate to a less skilled farrier whether a horse needs more or less heel. If the horse's lameness is worsened after elevating the toe using the wedge test, some degree of heel elevation may be beneficial. Elevation of the heel can be achieved by using a wedge heel shoe or a rim or complete wedge pad.

A variety of shoes have been used successfully in the management of navicular disease, including egg bar, egg bar–heart bar, straight bar shoes, and the so-called Tennessee shoe, which moves the point of breakover to just in front of the apex of the frog by placing rails on the palmar aspect of the branches of the shoe. Success has also been achieved in some horses using the natural balance shoe.

The strategy of using shoes that move the weight-bearing axis in a palmar direction in horses with low, collapsed, and underrun heels is quite successful. However, a recent study has shown that using egg bar shoes in clinically normal Dutch Warmblood horses with well-conformed feet did not reduce force on the navicular bone compared with flat shoes.[3] The forelimb gait was also described as less animated with egg bar shoes compared with flat shoes. However, these findings cannot necessarily be translated to lame horses with less than ideal foot conformation. Many top-level show jumpers and dressage horses have been shod with egg bar shoes with no apparent detriment to the quality of the gaits. Egg bar shoes provide a greater surface area through which forces are transmitted and clinically seem to help reduce pain associated with either navicular bone pain or distal interphalangeal joint pain. Of 55 horses with clinically diagnosed navicular disease, 53% had permanent relief of lameness after application of egg bar shoes in a follow-up period of 12 to 40 months.[4] In some horses, clinical improvement took many weeks. Resolution of lameness was seen despite the persistence of low, collapsed heels, which is also our experience. In another study, treated horses showed histomorphometric evidence of altered navicular bone modeling compared with untreated controls.[5] The shoes should be made to measure and of sufficient size and length to provide adequate support to the heel region (Fig. 31-1).

Fitting an egg bar shoe is not necessarily easy and depends on the shape of the horse's foot. If the quarters are very wide it can be difficult to "pick up" the heels. In such horses a traditional open shoe fitted long and wide at the heels or a straight bar shoe may be more appropriate. The correctly fitted egg bar shoe should project beyond the ground-bearing surface of the foot. It is more likely to be pulled off than a standard shoe, and the use of over-reach boots should always be considered, at least when the horse is worked or turned out. These shoes are not suitable for a horse that is going to work in deep mud, since they are readily sucked off.

In summary, the following principles may aid in the selection of a shoe: correction and then maintenance of dorsopalmar and lateromedial balance, ease of breakover achieved by rolling the toe of the shoe, maintenance of foot (and especially heel) mass, and protection of the palmar aspect of the foot from concussion.

Screw-in studs are used in many competition horses to enhance traction. Some riders just use studs in the lateral

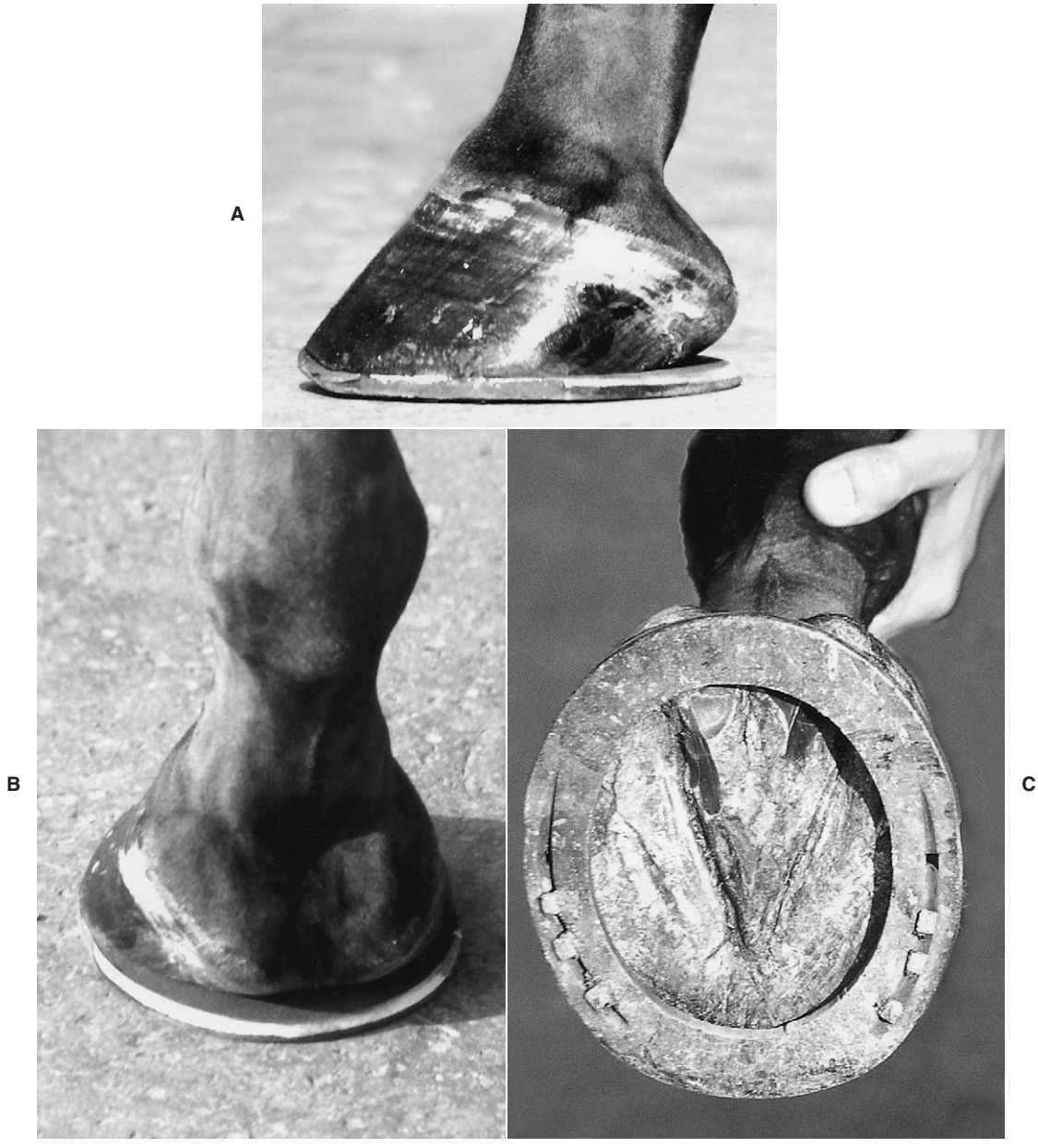

Fig. 31-1 **A,** Lateral; **B,** palmar, and **C,** solar views of a correctly fitted egg bar shoe. The shoe projects beyond the ground bearing surface of the foot to cover the bulbs of the heel.

branch of the shoe to avoid the risks of tread injuries created by a medial stud in the contralateral limb. This inevitably creates mediolateral imbalance, and therefore care should be taken either to use studs in both branches or to avoid the use of studs. The studs should be positioned as far palmarly as possible to avoid shortening the effective length of the shoe. When the ground is hard, small, pointed studs are preferable to large, square-shaped studs.

Non-Steroidal Anti-Inflammatory Medication

A wide variety of NSAIDs are used in horses. In general, these drugs should be administered at the lowest dose necessary to achieve the desired effect. There are anecdotal reports that certain NSAIDs are better for horses with foot pain (e.g., meclofenamic acid [Arquel]), but there are no controlled studies to support these claims. Phenylbutazone is widely used, and many, but not all, horses with navicular pain respond to this drug. To determine the lowest effective dose of phenylbutazone, the horse is given a dose of 4 mg/kg twice daily for 3 days and the dose is then gradually decreased until the lameness returns. Depending on lameness severity, NSAID treatment may only be necessary when the horse is being used. It should be borne in mind that it has been demonstrated that phenylbutazone may influence bone turnover.[6] After bone biopsy of the tibia, mineral apposition rate was significantly decreased in horses treated with phenylbutazone (4.4. mg/kg BID) compared with untreated, control horses. The clinical significance of this finding in the management of navicular disease is unknown. For horses that are intolerant of phenylbutazone, carprofen ([Rimadyl] 1.0 to 1.5 mg/kg SID) has been a useful alternative in one author's (J.B.M.) practice.

Isoxsuprine

Isoxsuprine is a β-agonist that causes peripheral vasodilation in people. Its mode of action in the treatment of navicular disease is unknown. It has been suggested that there are no

measurable cardiovascular effects of isoxsuprine given orally in the horse.[7,8] However, orally administered slow-release isoxsuprine resin was absorbed and resulted in thermographic evidence of increased skin temperature of the distal forelimbs for about 8 hours after administration.[9] Isoxsuprine also binds strongly to α-adrenoreceptors[10] and therefore may be active despite insignificant measurable levels in plasma.[9] The drug also may have some anti-inflammatory or hemorheologic properties that could explain why some horses with navicular disease appear to respond to isoxsuprine therapy. A double-blind clinical trial evaluating the response to isoxsuprine in the treatment of navicular disease demonstrated a decrease in lameness in the treated horses.[11]

If the horse responds to a trial dose of 0.6 mg/kg twice a day for 30 days, then continued treatment at the same dose given once or twice a day indefinitely may be indicated. In one author's (S.J.D.) experience, some horses have been treated for 12 weeks and responded satisfactorily and have then shown remission of clinical signs for up to 9 months without treatment. If no response is seen within 30 days, it is unlikely that further treatment will be beneficial. However, some horses that did not respond a dose of 0.6 mg/kg twice daily did respond satisfactorily to 0.9 mg/kg twice daily. If lameness is moderate to severe, then treatment should be combined with a NSAID for the first 3 to 4 weeks of treatment. The response to treatment in horses with major radiological abnormalities is generally very poor.

Intra-Articular Medication

Although lameness associated with navicular disease may be improved by intra-articular analgesia of the distal interphalangeal joint, the intra-articular administration of hyaluronan with or without corticosteroids (e.g., triamcinolone, 10 mg) does not usually result in a similar degree of improvement, unless there is concurrent synovitis of the distal interphalangeal joint. Intra-thecal injection of corticosteroids into the navicular bursa may provide transient relief of clinical signs for up to 2 to 3 months.[12] In one author's (S.J.D.) experience, this treatment is effective only in horses with low-grade radiographic abnormalities but can result in very substantial although temporary improvement.

Other Drugs

Warfarin previously was used to treat horses with navicular disease, and in one series an 80% success rate was claimed.[13] However, these were predominantly pleasure horses and low-level competition horses. Experience in higher level competition horses produced disappointing results.[14] Warfarin therapy requires careful adjustment of the dose guided by monitoring of prothrombin time at least at monthly intervals. This, combined with the drug's limited efficacy, has largely resulted in the abandonment of this treatment.

Pentoxyfylline and propentofylline are hemorheologic drugs that alter the deformability of the red blood cell membrane and inhibit platelet aggregation.[8,15] These drugs are advocated by some clinicians for the treatment of navicular disease, although clinical efficacy to date is largely unknown. No measurable effect on digital and laminar blood flow was seen in healthy horses after oral administration of pentoxifylline.[8] A small clinical study using propentofylline resulted in improvement, but not alleviation of lameness associated with navicular disease, during a 6-week treatment period.[15] A long-term clinical trial using metrenperone found similar efficacy to isoxsuprine; only 27% of horses had long-term resolution of lameness.[16]

The pathological similarities between navicular disease and osteoarthritis have prompted the use of drugs used for the management of osteoarthritis. Systemic administration of a polysulfated glycosaminoglycans (PSGAG [Adequan], 500 mg IM every 4 days for 7 treatments] resulted in clinical improvement in lameness in treated horses compared with controls during the treatment period.[17] In one author's (S.J.D.) experience, PSGAG treatment has not had any long-lasting benefit on lameness associated with navicular disease.

Chemical "Neurectomy": Cryoneurectomy

Temporary resolution of palmar foot pain can be achieved by chemical ablation of sensory fibers in the palmar digital nerves. Several products are capable of causing a temporary loss of sensation when injected over the nerves. These include Sarapin (a plant alkaloid that is thought to alter transmission in type C fibers), P block, and cobra venom. The product usually is injected over, or directly into, the nerve with a corticosteroid. Injection of absolute alcohol into the perineurium may also be used. Any of these products may result in localized perineural fibrosis, which makes subsequent surgical neurectomy more complicated. Alternatively, the digital nerves may be frozen percutaneously using liquid nitrogen. Both methods result in 2 to 3 months of clinical improvement when successfully applied. In our experience, these methods work well on occasion but are unreliable. Return of sensation in most horses is accompanied by a return of the lameness.

Desmotomy of the Collateral (Suspensory) Ligaments of the Navicular Bone

Navicular suspensory desmotomy has been described as a treatment for navicular disease.[18,19] In an initial report from the United Kingdom, 13 of 16 horses treated with this technique were able to work without lameness,[18] and in a subsequent report, 76% of 118 horses were sound at 6 months, but only 43% remained sound 3 years later.[20] Diehl[19] from Germany reported clinical improvement in 50% of 57 horses. In a study from New Zealand, 12 of 17 horses were sound at least 6 months post-operatively.[21] In our experience, navicular suspensory desmotomy often results in short-term (6 to 12 months) improvement in lameness. One author (J.B.M.) has never seen complete resolution of lameness, whereas the other (S.J.D.) has. The difference in response may relate to post-operative management. One author (S.J.D.) starts walking exercise from the first post-operative day, with a rapidly ascending exercise program after suture removal, whereas the other (J.B.M.) starts light exercise 3 weeks post-operatively with a return to full work after 3 months. It was originally suggested that this surgical procedure resulted in alteration of the biomechanical forces on the navicular bone, but clinical improvement may also be the result of sectioning the sensory nerve fibers that run in the suspensory ligaments. The original surgical technique described transecting the ligaments near the origin through oblique skin incisions, but the proximal interphalangeal joint capsule can be penetrated by this approach. Better results have been achieved via vertical skin incisions further distally, centered over the collateral ligaments of the navicular bone.

Desmotomy of the Accessory Ligament of the Deep Digital Flexor Tendon

Desmotomy of the accessory ligament of the deep digital flexor tendon has been described as a treatment for navicular disease in horses with a markedly upright foot conformation.[22] Long-term follow-up results are not available.

Periarterial "Sympathectomy"

Litzke et al.[23] described a technique for cutting the sympathetic nerve supply to the lateral and medial digital arteries, either alone or with palmar digital neurectomy. This technique is still practiced in Germany, but the results have not been well documented.

Palmar Digital Neurectomy

If lameness associated with navicular disease is completely eliminated by perineural analgesia of the palmar digital nerves, then palmar digital neurectomy may be considered as a treatment of last resort.[24] The technique has been associated with a significant number of complications, including failure to alleviate lameness, recurrence of lameness, complete or partial rupture of the DDFT, and subluxation or luxation of the distal interphalangeal joint, neuroma formation, and failure to recognize either sub-solar infection or penetrating injuries of the palmar aspect of the foot. Case selection is very important. Horses with pre-existing pathology of the DDFT are probably at highest risk of subsequent rupture of the DDFT. If there is radiological evidence of a substantial defect in the flexor surface of the navicular bone, DDFT lesions are likely and these horses should not be treated by neurectomy.

A variety of techniques of palmar digital neurectomy have been described. Recent work suggests that the simple guillotine technique results in the longest period of desensitization and less neuroma formation compared with epineural capping and carbon dioxide laser division.[25] Surgery should be delayed for at least 7 days after perineural analgesia. We both prefer to use two long (3- to 4-cm) incisions just dorsal to the medial and lateral edges of the superficial digital flexor tendon starting just below the base of the sesamoid. The nerve is gently stretched and transected using a scalpel blade as far distally and proximally as possible. Approximately 50% of horses have one or more (up to five) accessory branches that must be divided to achieve complete desensitization of the heel region. In most instances the accessory branches are located abaxial to the palmar digital nerve and run in a palmar direction and parallel to the ligament of the ergot. If an accessory branch is located at one surgical site, there are invariably accessory branches associated with the remaining palmar digital nerves. The incision is closed in a single layer, and the horse is confined to a box stall with hand walking only for a period of 30 days. Phenylbutazone (2.2 mg/kg BID) is administered for a period of 10 days. This limited exercise, combined with minimal handling of the nerve(s) intraoperatively, seems to reduce the risk of neuroma formation.

The horse is shod in the manner that was most successful before surgery. This procedure usually provides relief of clinical symptoms in approximately 65% to 70% of horses for approximately 12 to 18 months post-operatively.[26,27] Individual horses may have recurrence of lameness sooner.

With careful handling of the nerve intraoperatively, followed by conservative postoperative management, neuroma formation is uncommon. A painful neuroma, which may occur several months after surgery, may be successfully managed with perineural injections of Sarapin and triamcinolone. Occasionally a second neurectomy proximal to the neuroma is required.

If lameness occurs because of re-innervation, surgery may be repeated, assuming that perineural analgesia of the palmar digital nerves still results in relief of pain. However, re-innervation may be due to axons sprouting from the proximal nerve stump and anastomosing with other local nerves. Repeated surgery may therefore be less successful.

Surgical Drilling

A technique for drilling cyst-like lesions in the navicular bone through an arthroscopic approach has recently been described, but long-term follow-up results are not available.[28]

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CHAPTER • 32

Fracture of the Navicular Bone

Sue J. Dyson

Fractures of the navicular bone occur in a variety of configurations. The most common is a slightly oblique sagittal fracture, medial or lateral to the midline. Y-shaped fractures and other comminuted fractures are less common. Distal border fragments are discussed elsewhere (see Chapter 30). Fractures occur more commonly in forelimbs than hindlimbs. In a series of 40 navicular bone fractures, 28 (70%) occurred in a forelimb.[1] Fractures usually are traumatic in origin, although it is not always possible to identify the cause. Fractures in hindlimbs have been the result of kicking a wall. Bipartite and tripartite navicular bones also have been described and should be differentiated from a fracture.[2] It has been suggested that many fractures are pathological secondary to severe navicular disease,[3] but this confusion probably arises because lucent zones adjacent to the fracture line and along the distal border of the navicular bone develop rapidly, within months of fracture occurrence.[4] Palmar (plantar) displacement of fracture fragment(s) may result in laceration of the deep digital flexor tendon.[5] Other fractures of the middle or distal phalanx occasionally occur concurrently.

HISTORY

Lameness is usually acute in onset and severe. However, sometimes a horse may develop less severe lameness and radiographic examination reveals evidence of an old fracture, which presumably healed by fibrous union but has recently become unstable.

CLINICAL EXAMINATION AND DIAGNOSIS

The horse may be reluctant to bear full weight on the limb. Digital pulse amplitudes may be increased, and in some horses, pain can be elicited by application of hoof testers to the heel or frog regions. Percussion of the frog may be resented. Lameness usually is severe with an acute fracture and may be accentuated as a horse turns. Lameness is usually substantially improved by perineural analgesia of the palmar digital nerves, although it sometimes is unaffected. Diagnosis is dependent on radiographic identification of the fracture(s). Most fractures are best identified in a dorsoproximal-palmarodistal oblique view (or an "upright pedal" plantarodorsal view in a hindlimb) (Fig. 32-1). It is important that frog cleft artifacts not be confused as a fracture. The clinician should examine any lucent line carefully to see whether it extends beyond the bone margins, or if it changes position relative to the medial and lateral margins of the bone if the x-ray beam is reoriented 5° medially or laterally. The frog clefts should be well packed with a moldable compound (e.g., PlayDoh; Hasbro, Pawtucket, RI) and if necessary re-packed. If doubt persists, a weight-bearing dorsopalmar view and a palmaroproximal-palmarodistal oblique view of the navicular bone should determine whether a fracture is present. The palmaro-

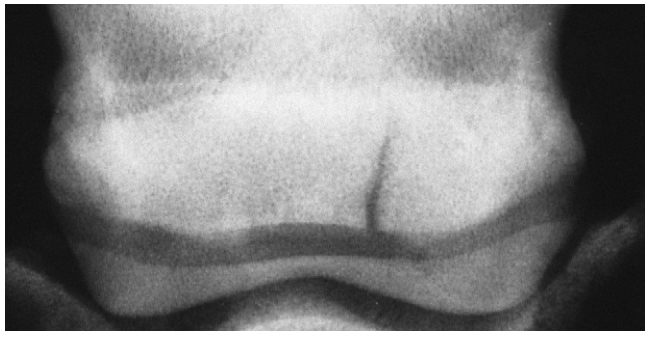

Fig. 32-1 Dorsoproximal-palmarodistal oblique radiographic view of a navicular bone. There is an acute parasagittal fracture lateral to the midline. Lateral is to the right.

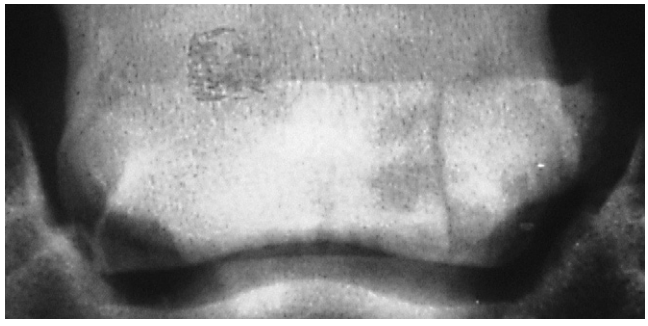

Fig. 32-2 Dorsoproximal-palmarodistal oblique view of a navicular bone with a chronic fracture involving the lateral aspect. There are large lucent areas adjacent to the fracture line and modeling of the lateral proximal margin of the navicular bone.

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Fracture of the Navicular Bone

Sue J. Dyson

Fractures of the navicular bone occur in a variety of configurations. The most common is a slightly oblique sagittal fracture, medial or lateral to the midline. Y-shaped fractures and other comminuted fractures are less common. Distal border fragments are discussed elsewhere (see Chapter 30). Fractures occur more commonly in forelimbs than hindlimbs. In a series of 40 navicular bone fractures, 28 (70%) occurred in a forelimb.[1] Fractures usually are traumatic in origin, although it is not always possible to identify the cause. Fractures in hindlimbs have been the result of kicking a wall. Bipartite and tripartite navicular bones also have been described and should be differentiated from a fracture.[2] It has been suggested that many fractures are pathological secondary to severe navicular disease,[3] but this confusion probably arises because lucent zones adjacent to the fracture line and along the distal border of the navicular bone develop rapidly, within months of fracture occurrence.[4] Palmar (plantar) displacement of fracture fragment(s) may result in laceration of the deep digital flexor tendon.[5] Other fractures of the middle or distal phalanx occasionally occur concurrently.

HISTORY

Lameness is usually acute in onset and severe. However, sometimes a horse may develop less severe lameness and radiographic examination reveals evidence of an old fracture, which presumably healed by fibrous union but has recently become unstable.

CLINICAL EXAMINATION AND DIAGNOSIS

The horse may be reluctant to bear full weight on the limb. Digital pulse amplitudes may be increased, and in some horses, pain can be elicited by application of hoof testers to the heel or frog regions. Percussion of the frog may be resented. Lameness usually is severe with an acute fracture and may be accentuated as a horse turns. Lameness is usually substantially improved by perineural analgesia of the palmar digital nerves, although it sometimes is unaffected. Diagnosis is dependent on radiographic identification of the fracture(s). Most fractures are best identified in a dorsoproximal-palmarodistal oblique view (or an "upright pedal" plantarodorsal view in a hindlimb) (Fig. 32-1). It is important that frog cleft artifacts not be confused as a fracture. The clinician should examine any lucent line carefully to see whether it extends beyond the bone margins, or if it changes position relative to the medial and lateral margins of the bone if the x-ray beam is reoriented 5° medially or laterally. The frog clefts should be well packed with a moldable compound (e.g., PlayDoh; Hasbro, Pawtucket, RI) and if necessary re-packed. If doubt persists, a weight-bearing dorsopalmar view and a palmaroproximal-palmarodistal oblique view of the navicular bone should determine whether a fracture is present. The palmaro-

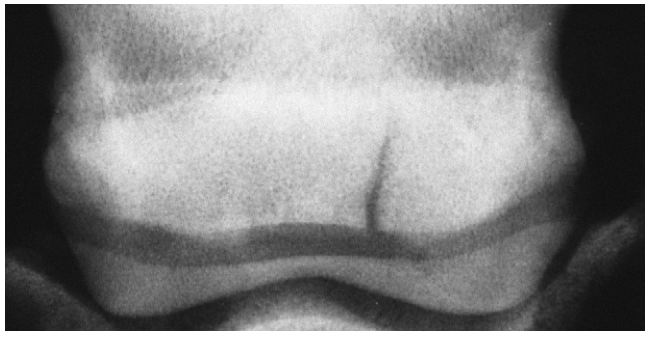

Fig. 32-1 Dorsoproximal-palmarodistal oblique radiographic view of a navicular bone. There is an acute parasagittal fracture lateral to the midline. Lateral is to the right.

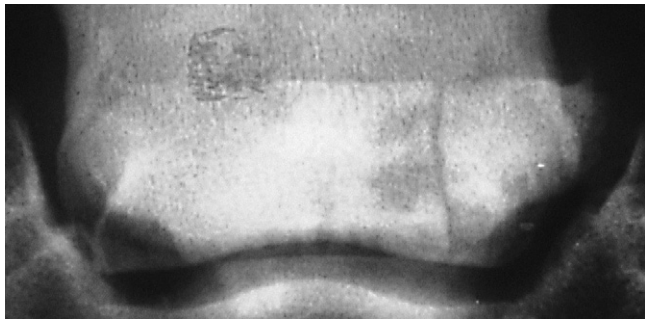

Fig. 32-2 Dorsoproximal-palmarodistal oblique view of a navicular bone with a chronic fracture involving the lateral aspect. There are large lucent areas adjacent to the fracture line and modeling of the lateral proximal margin of the navicular bone.

proximal-palmarodistal oblique view is also useful for determining the presence of comminution or displacement. The presence of lucent zones along the fracture line indicates that it is not of recent origin but is likely to have been present for at least several months (Fig. 32-2).

Treatment

Lameness associated with a simple sagittal fracture usually improves progressively with rest, but the prognosis for complete resolution of lameness or return to full athletic function is guarded. Some horses do become sound enough for light hacking, but lameness may be recurrent.[3,6] Better results have been described in a limited number of horses by trimming the foot to establish a normal hoof-pastern axis followed by application of four 3° wedge pads and a flat shoe to reduce the weight-bearing function of the navicular bone.[7] The horse is confined to box rest for 60 days and then starts walking exercise for an additional 2 months. The shoe is refitted every 4 weeks with 3° less elevation at each shoeing. However, healing probably is only by fibrous union. Treatment by palmar digital neurectomy may provide symptomatic relief in some horses, but a substantial proportion develop osteoarthritis of the distal or proximal interphalangeal joints and associated lameness.[6] Internal fixation using a lag screw technique with a specially designed limb jig and radiographic control offers the best prognosis for simple, oblique sagittal fractures.[1,8] Twenty-three (68%) of 40 horses resumed work. The prognosis was significantly better for fractures of less than 4 weeks' duration than for older fractures. Potential complications included splitting the fragment, inability to reduce the fracture resulting in a step deformity, and poor stabilization of the fracture. The prognosis for more complicated fractures is guarded.

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CHAPTER • 33

Primary Lesions of the Deep Digital Flexor Tendon within the Hoof Capsule

Sue J. Dyson

ANATOMY

Within the hoof capsule the deep digital flexor tendon (DDFT) is molded to the palmar (plantar) surface of the navicular bone and separated from it by the navicular bursa. The distal recess of the navicular bursa separates the DDFT and the distal sesamoidean impar ligament. The DDFT has a terminal fan-like expansion containing cartilage that occupies the entire space between the medial and lateral palmar processes of the distal phalanx. It inserts on the facies flexoria and semilunar crest of the distal phalanx. The dorsal portion of the DDFT joins with the distal sesamoidean impar ligament immediately before insertion on the facies flexoria of the distal phalanx. There are parallel fibers of dense connective tissue, separated by loose connective tissue, within which are many sensory nerves and numerous blood vessels.[1] A prominent line (tidemark) indicates the transition from nonmineralized tendon and ligament to mineralized regions of the DDFT and distal sesamoidean impar ligament before attachment to the distal phalanx.[2]

Within the digit the DDFT induces axial compression of the articular surfaces of the proximal and distal interphalangeal joints.[2,3] It has an important role in stabilizing the distal interphalangeal joint. The anatomical arrangement of the collateral ligaments of the navicular bone facilitates compression of the articular surfaces of the navicular bone into those of the middle and distal phalanges.[2] The DDFT has a dorsal fibrocartilaginous pad that supports pressure of the tuberositas flexoria, the transverse prominence on the proximopalmar aspect of the middle phalanx.[3]

The relationship of the DDFT to the navicular bone varies with the phase of the stride. During the full weight-bearing stance of the stride the DDFT is only in contact with the distal aspect of the bone, whereas in the propulsion phase the DDFT bends over the distal scutum (the fibrocartilaginous insertion of the straight sesamoidean ligament on the middle phalanx) and comes into full contact with the navicular bone. Tension in the DDFT is maximal, and active muscle contraction and the elasticity in the tendon and in its accessory ligament result in extension of the distal interphalangeal joint.[3] At the beginning of the swing phase of the stride the tension in the DDFT contributes passively to induce flexion of the interphalangeal joints. During extension of the distal interphalangeal joint, which is maximum at the propulsion phase of the stride, pull on the DDFT creates a shear force between the DDFT and the distal sesamoidean impar ligament.[2]

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PATHOPHYSIOLOGY

Primary lesions of the DDFT have only recently been recognized clinically, and only limited information concerning pathophysiology is available. Currently it is not known whether degenerative aging changes that are common to many other tendons and ligaments may predispose to injury. A progressive increase in proteoglycans with age has been seen in the distal aspects of the DDFT and distal sesamoidean impar ligament, which may be an adaptation to stress.[2] It was suggested that horses with low, weak heels may be more susceptible to these changes. Similar but more extensive changes were seen in two horses with navicular disease. Lesions within the DDFT within the foot were identified in less than 10% of horses with unknown history in a post-mortem study of SDFT and DDFT lesions in the metacarpal region and digit.[4]

In a post-mortem study of 38 horses with suspected navicular disease, 1 horse had a core lesion of the DDFT with no associated pathological condition of the navicular bone[5]; 4 additional horses had histological evidence of focal regions of necrosis within the tendon concurrent with other pathological conditions of the navicular bone. Surface fibrillation was a common finding in horses with navicular disease but not in age-matched controls. No lesions of the DDFT were described in the age-matched controls. Pool et al.[6] considered lesions in the DDFT to be secondary to navicular disease. Inflammation at the intersection of the DDFT and distal sesamoidean impar ligament was recognized in the Quarter Horse in association with other signs compatible with navicular disease.[7] It was considered that these lesions may be involved with the pathogenesis of navicular disease.

Ectopic mineralization within the DDFT has been identified radiographically, at the level of the navicular bone and immediately proximal to it, but its clinical significance has been speculative.[8] Entheseous new bone at the site of insertion of the DDFT on the facies flexoria and semilunar crest of the distal phalanx has been described associated with lameness, unassociated with navicular disease.[9] Nuclear scintigraphic examination of horses with palmar foot pain has revealed horses with focal regions of increased uptake of the radiopharmaceutical in the region of insertion of the DDFT,[10] either alone or in association with a region of increased uptake in the navicular bone. Many of these horses had no detectable radiographic abnormality.

Primary DDF tendonitis may be the result of repetitive overstress or an acute-onset traumatic tear. Lesions identified in association with navicular disease have been identified in the region where the tendon passes over the palmar aspect of the navicular bone, whereas primary tendonitis has been identified not only in this region but also further proximally and distally, with some lesions confined to the actual insertion into the distal phalanx.[11]

Rupture of the DDFT secondary to previous neurectomy of the palmar digital nerves is considered a separate condition; however, it is likely that a pre-existing, unrecognized pathological condition of the DDFT predisposes to rupture.

HISTORY AND CLINICAL SIGNS

Lameness associated with primary DDF tendonitis within the hoof capsule has been identified most commonly in horses that jump,[1-13] but it has also been seen in general-purpose riding horses. Lameness usually is unilateral, acute in onset, and moderate to severe in intensity. In horses with severe lameness the horse may point the affected limb at rest. In mild to moderate injuries, lameness may resolve with rest but recur with work and progressively worsen.

There are generally no substantial palpable abnormalities of the limb, unless the lesion extends proximally into the pastern region. There is also no response to pressure applied to the foot with hoof testers. Lameness often is worse on a soft surface, especially on a circle, and in some horses is apparent only under these circumstances. The response to distal limb flexion is variable. Extension of the distal interphalangeal joint using the board or wedge test may accentuate the lameness.

LOCAL ANALGESIA

Perineural analgesia of the palmar digital nerves immediately proximal to the cartilages of the foot usually but not invariably improves lameness but rarely is lameness fully alleviated. Perineural analgesia of the palmar nerves at the level of the base of the proximal sesamoid bones usually relieves the lameness. Intra-articular analgesia of the distal interphalangeal joint results in rapid improvement in lameness in some horses, but in others lameness persists unchanged. There has been no correlation between the analgesic response and the proximodistal site of the lesion.[11] Analgesia of the navicular bursa often results in improvement in lameness but rarely alleviates it fully.

DIAGNOSTIC IMAGING

Radiography

In some lateromedial radiographic projections of the foot a faint outline of the DDFT can be seen; the significance of this finding is unknown. Occasionally a focus of mineralization is seen within the DDFT, at the level of or proximal to the navicular bone, but to date this has not been correlated with primary tendonitis of the DDFT. In a well-positioned, high-detail lateromedial projection, entheseous new bone or a radiolucent focus may be identified on the facies flexoria of the distal phalanx. This finding has been seen more commonly in heavier Warmblood breeds[9] than in Thoroughbred and cross-bred horses[11] and has been correlated with increased bone activity identified by nuclear scintigraphy.

Ultrasonography

Diagnostic ultrasonographic imaging of the DDFT within the hoof capsule is not easy because of the horny hoof capsule and the difficulties in orientating the ultrasound transducer perpendicular to the line of the tendon fibers. Thus it is easy to create artifacts. The proximal part of the DDFT may be imaged using a 6.5-MHz transducer positioned between the bulbs of the heel, unless the heel is very contracted. The region of the DDFT overlying the navicular bone and close to its insertion is potentially seen by a solar approach, placing the transducer (7.5-MHz) on the frog. The frog must be sufficiently soft; this generally is achieved by soaking the foot by bandaging a water-soaked sponge on the foot for several hours. If the horn is exceptionally hard, soaking may have to be continued for several days. The horn must also be pared to provide a relatively flat site for placement of the ultrasound transducer. This is difficult to achieve if the horse has a very narrow foot with a deep frog cleft and sulci. Only the region of the DDFT in the mid-sagittal plane can be successfully imaged. Orientation is achieved most easily in longitudinal images.

A normal DDFT has linear, parallel echoes and its margins are well defined.[14] An anechoic region representing fluid in the navicular bursa is interposed between the DDFT and the echoic line of the palmar aspect of the navicular bone. There are limited reports of correlation of postulated lesions within the DDFT and either post-mortem studies or

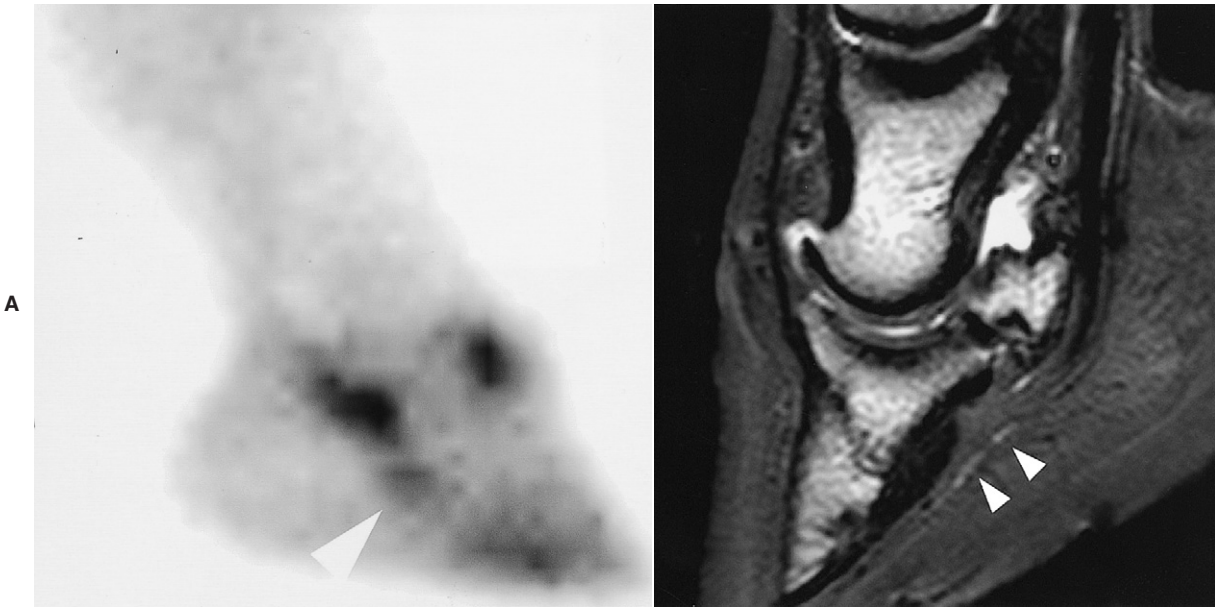

Fig. 33-1 **A,** Lateral pool-phase scintigraphic image of the right front foot of a 9-year-old event horse with a history of acute-onset severe lameness after competing. Lameness had persisted despite rest. Lameness was improved by perineural analgesia of the palmar digital nerves or intra-articular analgesia of the distal interphalangeal joint, but it was not affected by analgesia of the navicular bursa. There is a region of increased uptake of the radiopharmaceutical *(arrow)*. Superimposition of a lateromedial radiograph demonstrated that this was at the site of insertion of the deep digital flexor tendon (DDFT) on the distal phalanx. Magnetic resonance imaging demonstrated an abnormal signal at the insertion of the DDFT. **B,** Sagittal three-dimensional T2-weighted gradient echo magnetic resonance image of the same foot in **A.** There is an unusual bright signal immediately distal to the DDFT *(arrows)*, the significance of which is currently unknown. The increased signal in the DDFT distally is due to its structural composition and the magic angle effect. There is also a large amount of fluid in the distal interphalangeal joint.

confirmation of the presence of a lesion using alternative means[15]; thus the sensitivity and specificity of the technique are currently unknown.

Nuclear scintigraphy

Pool- and bone-phase nuclear scintigraphic lateral and solar images of the foot have been used to identify a region of increased uptake of ^{99m}Tc either at the site of insertion of the DDFT on the facies flexoria of the distal phalanx or further proximally within the DDFT[9,16] (Figs. 33-1 and 33-2). Superimposition of a lateromedial radiographic image and a lateral scintigraphic image helps to accurately locate the site of increased uptake in the lateral view.[17] Pool-phase images have been more sensitive than bone-phase images in identifying lesions of the DDFT proximal to its insertion. Although the bone-phase solar (palmar) view may be more sensitive for identification of abnormal modelling associated with insertional stress, there is a relatively high incidence of false-positive results.[16]

The results from pool-phase lateral images have correlated well with findings using magnetic resonance imaging, arthroscopic evaluation of the DDFT through the navicular bursa, and post mortem studies.[17] However, the close proximity of the insertion of the distal sesamoidean impar ligament to that of the DDFT makes differentiation of insertional injuries potentially difficult.

Computed tomography

Computed tomography (CT) can only be performed with the horse under general anesthesia, but has the potential to give information about the size, shape, and internal architecture of the DDFT within the hoof capsule. It is therefore an accurate method of diagnosis of primary DDFT lesions. Lesions have been identified in one or both of the two lobes of the DDFT and were seen as enlargement of the DDFT with an increase or decrease in density[13,18] (Fig. 33-3). Thirteen of 78 horses

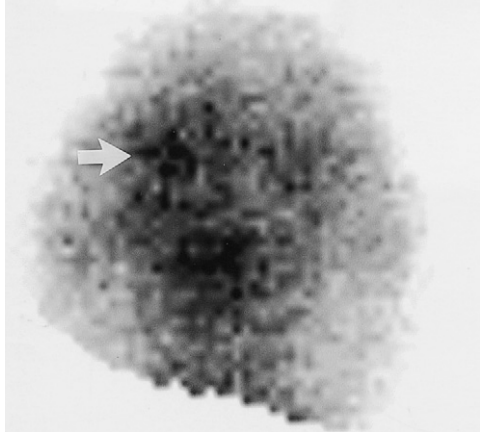

Fig. 33-2 Solar (palmar) bone-phase scintigraphic image of the left front foot of a 7-year-old show jumper with acute-onset, chronic left forelimb lameness that was alleviated by perineural analgesia of the palmar digital nerves. There is a focal region of increased uptake of the radiopharmaceutical *(arrow)*. Post-mortem examination revealed focal bone necrosis and reactive bone at this site associated with the insertion of the deep digital flexor tendon.

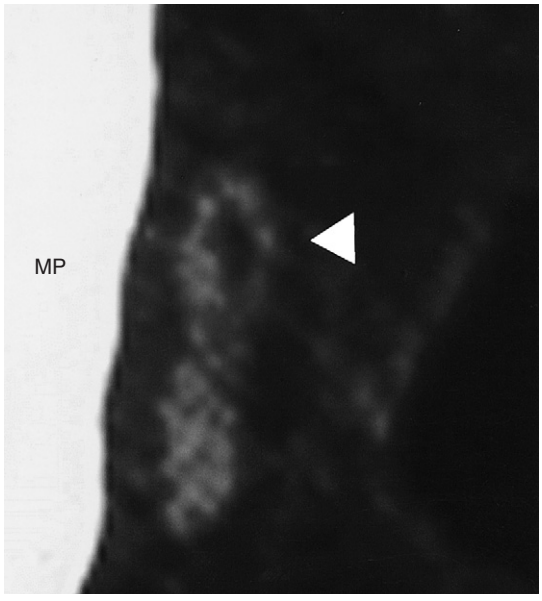

Fig. 33-3 Transverse computed tomographic image at the level of the middle phalanx (MP). Dorsal is to the left. The medial lobe of the deep digital flexor tendon has a focal decrease in signal density *(arrow)*.

examined using CT had abnormalities of the DDFT within the hoof.[13] To date, the results of clinical cases have not been correlated with post mortem examination and thus the clinical significance of the lesions remains speculative. However, correlation of an abnormal CT scan result with abnormalities verified at post mortem examination has been described without clinical details.[18]

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) can only be performed with the horse under general anesthesia, but it potentially offers the optimal method of diagnosis of primary DDFT lesions[12] (Fig. 33-4). Experience is still required to determine the clinical significance of relatively subtle changes in signal intensity. Three-dimensional T2*-weighted gradient echo sequences, with slice thickness of 1 to 1.5 mm, provide the best image quality.[19] Lesions are seen best in sagittal and transverse planes. Fat-suppressed two-dimensional fast spin echo or three-dimensional gradient echo sequences also are useful for detection of bone pathology at the insertion.

Most primary DDFT lesions have been identified proximal to the navicular bone, extending from the middle phalanx a variable distance distally. A small number of lesions have been identified at the insertion of the DDFT. Lesions usually are restricted to either the medial or the lateral lobe, but occasionally they occur in both lobes. Core lesions or dorsal fiber disruption are most common, sometimes with herniation of fibers and granulation tissue into the navicular bursa. Focal, full thickness, sagittal plane splits have also been seen at the level of the navicular bone, in association with abnormal signal in the palmar cortex of the bone. Seven of 18 horses with palmar foot pain of previously undetermined cause had primary DDFT injuries diagnosed using MRI.[20]

Correlation of the results of MRI examination of cadaver specimens from horses with clinically suspected DDFT lesions and post mortem examination has been good[17,21] (Fig. 33-5). Collagen necrosis and chondroid metaplasia within the DDFT have been seen in regions of increased signal intensity.[17,21] Care must be taken in the interpretation of sagittal plane images in the region of insertion of the DDFT,

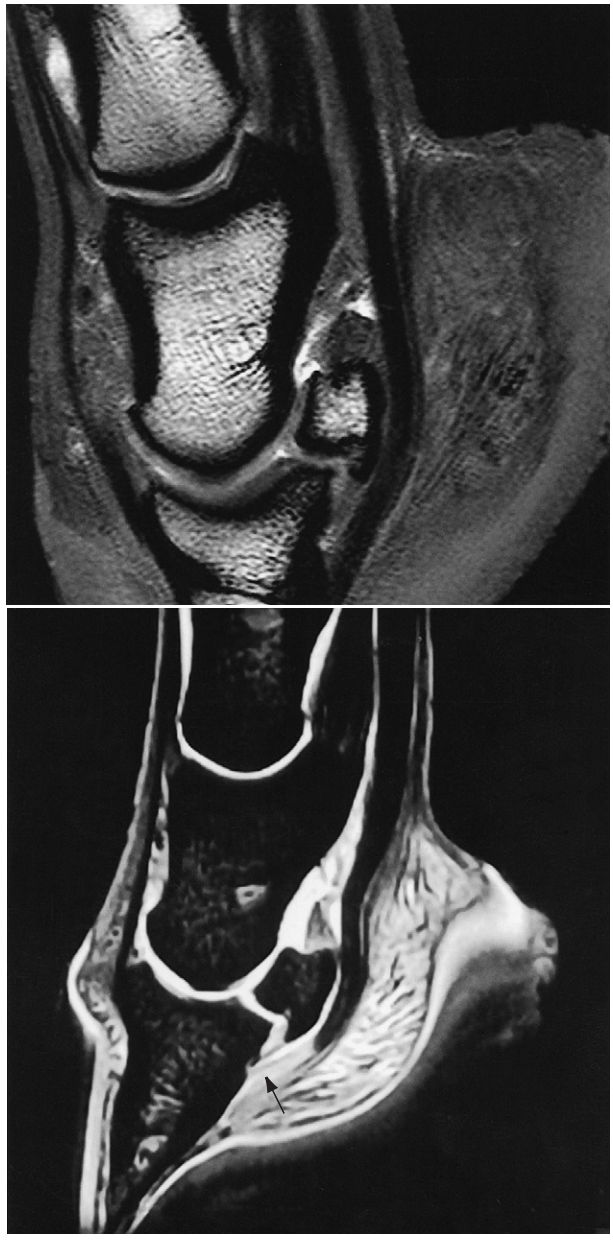

Fig. 33-4 **A,** Sagittal magnetic resonance image of a normal foot obtained using a three dimensional T2-weighted gradient echo sequence. There is a uniform signal throughout the deep digital flexor tendon (DDFT). **B,** Sagittal three-dimensional T2-weighted, fat-saturated, gradient echo magnetic resonance image of a normal foot. There is increased signal in the distal aspect of the DDFT *(arrow)*.

because there is an increased signal intensity in normal horses (see Figs. 33-1, *B,* and 33-4, *B*). This is related in part to the composition of the tendon, but also to the magic angle effect. This is due to orientation of fibers at approximately 55° to the main magnetic field.

SURGICAL EXPLORATION

Endoscopic examination of the digital flexor tendon sheath and the navicular bursa allows visual evaluation of the surface of the DDFT and assessment of its integrity by probing (see Chapter 24). Core lesions of the DDFT frequently have been

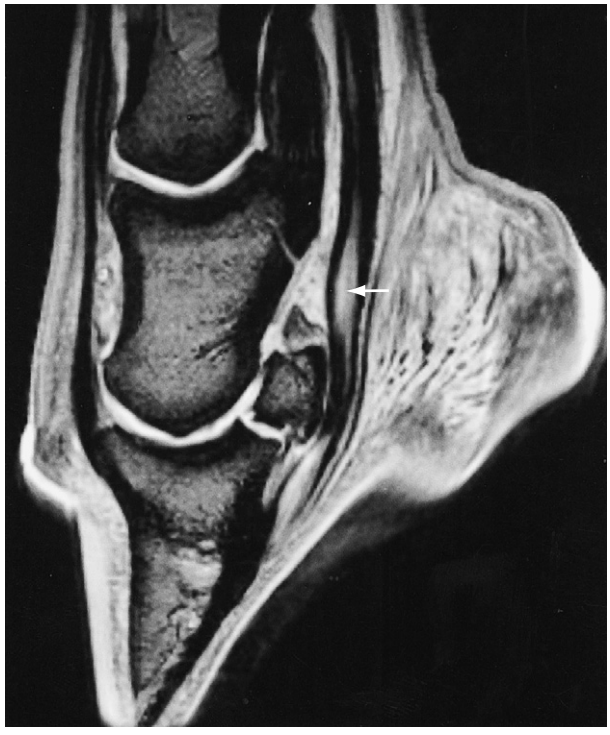

Fig. 33-5 Sagittal magnetic resonance image of the right front foot of an advanced event horse with acute-onset severe lameness that was alleviated by perineural analgesia of the palmar digital nerves and improved by intra-articular analgesia of the distal interphalangeal joint. The deep digital flexor tendon is enlarged and has increased signal intensity proximal to the navicular bone *(arrow)*. This correlated with an extensive core lesion at postmortem examination.

associated with extensive surface fibrillation of the tendon and inflammation of the synovial lining of the navicular bursa with villous proliferation.[11] However, this technique is both invasive and provides only limited information about the internal architecture of the tendon.

TREATMENT

Although Ueltschi[10] has reported a favorable outcome for horses with scintigraphic, radiographic, or both types of evidence of insertional lesions of the DDFT following prolonged rest,[10] in my experience with both these lesions, or those located further proximally, lameness frequently has persisted over the long term. These lesions have been confirmed at postmortem examination. Lesions have been identified within the DDFT immediately proximal and distal to the navicular bone, or localized to the insertion of the DDFT, with focal bone necrosis at that site.[17]

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CHAPTER • 34

The Distal Phalanx and Distal Interphalangeal Joint

PRIMARY PAIN ASSOCIATED WITH THE DISTAL INTERPHALANGEAL JOINT

• Sue J. Dyson

FUNCTIONAL ANATOMY

The distal interphalangeal joint is a complex structure comprising not only the articulation between the middle and distal phalanges, with supporting collateral ligaments, but also the articulation with the navicular bone. It therefore forms a close relationship with the distal sesamoidean impar ligament, the collateral ligaments of the navicular bone, and the navicular bursa.

The distal interphalangeal joint can move in three planes with flexion and extension movements in the sagittal plane, lateromedial movements in the frontal plane, and rotation and sliding in the transverse plane.[1,2] During normal load bearing and propulsion on a flat, level surface, distal interphalangeal joint movement is principally flexion and extension. On an uneven surface and a circle, or if the foot is unbalanced, passive movements result in the distal phalanx sliding and twisting relative to the middle phalanx. Movement is restricted by the collateral ligaments of the distal interphalangeal joint, the deep digital flexor tendon (DDFT), the distal digital annular ligament, and the distal sesamoidean impar ligament and collateral ligaments of the navicular bone. The degree of sliding and axial rotation within the distal interphalangeal joint[3] may predispose the horse to distal interphalangeal joint injury and may explain why lameness associated with the distal interphalangeal joint frequently is accentuated on a circle.

HISTORY

Lameness associated with the distal interphalangeal joint may be acute or insidious in onset and unilateral or bilateral. It is more common in forelimbs than in hindlimbs but does occur in both. Lameness in horses with unilateral disease tends to be sudden in onset, but those with bilateral lameness may be evaluated because of poor performance (e.g., shortened stride, unwillingness to jump drop fences, or unwillingness to land with one forelimb leading).

CLINICAL SIGNS

The distal interphalangeal joint capsule often is distended unilaterally or bilaterally. Distention of the joint capsule also can occur in clinically sound horses; therefore this finding is not pathognomonic for distal interphalangeal joint pain. However, chronic distention does reflect synovitis and treatment may reduce the risk of future problems. The dorsal proximal outpouching of the joint capsule can be palpated on the distal dorsal aspect of the pastern. Distention may result in obvious swelling, but it may be difficult to appreciate unless the horse has a fine hair coat. When distention of the joint capsule is present, ballottement of fluid from medial to lateral of the dorsal midline should be possible. The degree of distention may vary according to the recent work history.

Pain may be present on flexion or rotation of the distal limb joints. However, a marked reaction to distal limb flexion more likely reflects metacarpophalangeal joint pain. Mediolateral, dorsopalmar, or both types of foot imbalance are frequent findings and are considered important predisposing factors for the development of distal interphalangeal joint pain. The degree of lameness varies depending on the nature of the underlying pathological change, recent work history, and whether lameness is unilateral or bilateral. With bilateral distal interphalangeal joint pain the horse may move just a bit "flat" with a slightly shorter than normal stride. Severe lameness may reflect trauma to one of the supporting soft tissue structures of the joint. Lameness may be accentuated by distal limb flexion or rotation of the distal limb joints, but the response is variable. Lameness often is worse on a circle, especially on a hard surface, either with the lamest limb on the inside or outside of the circle.

DIAGNOSIS

Local Analgesia

Pain associated with the distal interphalangeal joint often improves after perineural analgesia of the palmar digital nerves and sometimes is alleviated fully. However, in some horses perineural analgesia of the palmar nerves at the level of the proximal sesamoid bones is required to completely eliminate lameness.

Intra-articular analgesia of the distal interphalangeal joint is not specific for pain that affects the joint itself. The potential exists for relief of pain from the navicular bone, insertions of the distal sesamoidean impar ligament and the DDFT on the distal phalanx, the palmar processes of the distal phalanx[4] and the sole, even at the toe.[5,6] A standard approach is suggested to aid in interpretation of the response. A maximum volume of 6 ml of local anaesthetic solution should be used. After injection through a dorsal midline approach the horse should stand still until re-assessment 5 minutes after injection. The clinician should ascertain whether sensation remains around the coronary band and whether any response to hoof testers has been eliminated to determine the specificity of the block. Lameness caused by primary distal interphalangeal joint pain usually improves rapidly and substantially after intra-articular analgesia. If the lameness persists, the veterinarian should re-assess the horse after an additional 5 minutes. If lameness is still apparent, the distal interphalangeal joint is not a likely primary source of pain. However, after this time the block still could result in further improvement in lameness as the local anesthetic solution diffuses to adjacent structures, thereby potentially confounding the response to any other block performed at this stage.[4,7] Intra-articular analgesia of the distal interphalangeal joint can substantially improve lameness associated with navicular disease within 5 minutes of injection,[8,9] although 20 % of horses with navicular bone

pain had a negative response to intra-articular analgesia of the distal interphalangeal joint.[8] Therefore the result of intra-articular analgesia of the distal interphalangeal joint is best interpreted in comparison with the response to analgesia of the navicular bursa.[7,8]

A slight or negative response to intra-articular analgesia of the distal interphalangeal joint does not eliminate completely a response to treatment of the joint, especially if the joint capsule is distended. For example, medication of the joint with hyaluronan and triamcinolone (10 mg) sometimes may resolve lameness that was not altered by intra-articular analgesia.

Retrieval of synovial fluid from the distal interphalangeal joint depends on the synovial fluid pressure within the joint and position of the needle. Using a dorsal approach to the joint and a 20-gauge needle, synovial fluid usually appears spontaneously in the needle hub and, if the joint capsule is distended, may flow out under pressure. Relief of this pressure may help to resolve lameness. However, not all horses with considerable pressure within the distal interphalangeal joint respond to intra-articular analgesia or medication of the joint. The pressure within the distal interphalangeal joint also increases if the contralateral limb is picked up; therefore if the contralateral limb is picked up to aid restraint of the horse for injection of the distal interphalangeal joint of the ipsilateral limb, a tendency for backflow through the needle puncture site may occur until the contralateral limb is placed on the ground.

Imaging Techniques

Radiography

Comprehensive radiographic examination of the distal interphalangeal joint should include weight-bearing dorsopalmar, lateromedial, dorsoproximal-palmarodistal oblique, and flexed oblique views of the interphalangeal joints.[10] Because intra-articular analgesia may influence pain associated with the palmar processes of the distal phalanx and the navicular bone, these structures also should be evaluated carefully.

The shape of the extensor process of the distal phalanx varies considerably among horses on lateromedial views (Fig. 34-1), but the shape usually is bilaterally symmetrical.[10] Modeling changes of the extensor process can be present without associated lameness. Care should be taken in interpretation of the bony prominences on the distal medial and lateral aspects of the middle phalanx, which always appear larger in bigger-boned horses. The distal interphalangeal joint should be inspected carefully for recognition of small osteophytes on the distal palmar aspect of the middle phalanx and the dorsoproximal aspect of the navicular bone. Joint space congruity and the shape of the proximal articular surface of the distal phalanx should be assessed carefully. A smoothly outlined depression sometimes is seen in the middle of the proximal articular surface of the distal phalanx in clinically normal horses.

The flexed oblique views—dorsal 60° lateral-palmaromedial oblique and dorsal 60° medial-palmarolateral oblique—enhance detection of periarticular new bone (Fig. 34-2) and modeling of the distal aspect of the middle phalanx. Care is

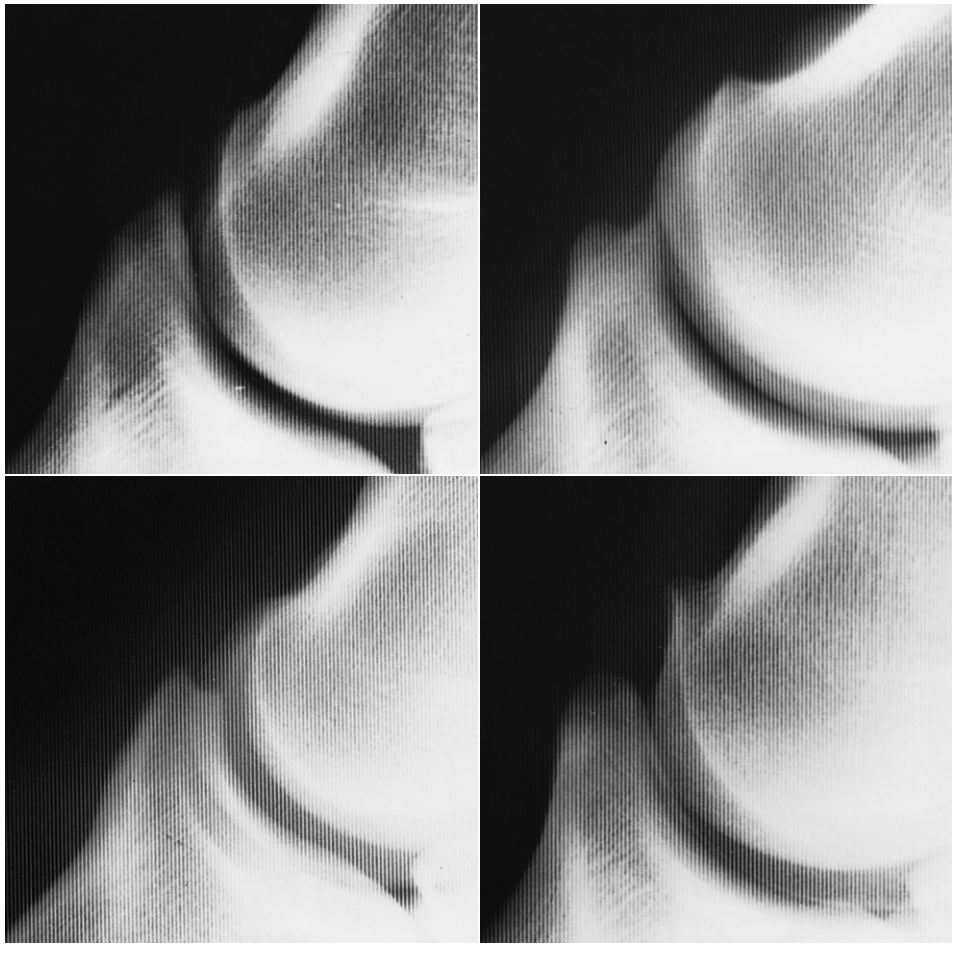

Fig. 34-1 Lateromedial radiographic views of the extensor process of normal distal phalanges. Note the variability in shape.

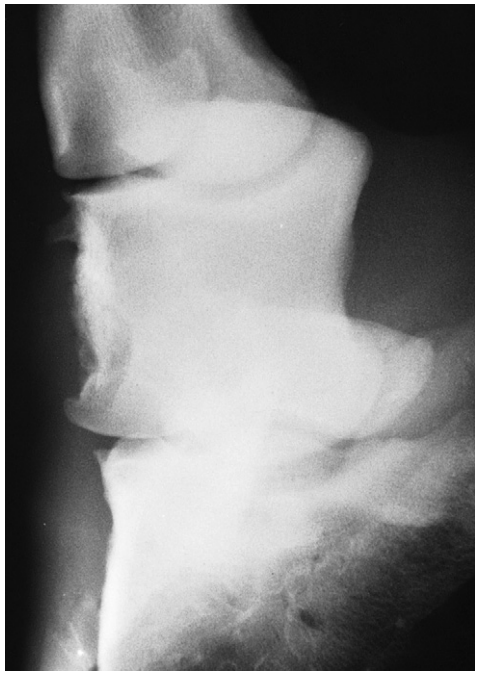

Fig. 34-2 Flexed dorsal 60° lateral-palmaromedial oblique view of the distal interphalangeal joint. There are modeling changes of the articular margins of both the the distal interphalangeal joint and proximal interphalangeal joints.

needed to differentiate between periarticular osteophytes and entheseous new bone at the insertion of the digital extensor tendon. Evaluation of the integrity and thickness of the subchondral bone plate of the middle and distal phalanges is important. Discontinuity of the subchondral bone plate may be the first radiological sign of the development of an osseous cyst–like lesion. Increased thickness of the subchondral bone plate may occur with osteoarthritis.

New bone also may develop on the dorsal aspect of the diaphysis of the middle phalanx. Smoothly outlined new bone usually is subsynovial and insignificant, whereas active-appearing, pallisading new bone usually is associated with lameness. Palisading is one of the earliest radiographic signs but often is missed. The dorsal cortex of the middle phalanx may become sclerotic (thicker), and early proliferative new bone may be seen.

Small, well-rounded mineralized opacities on the dorsoproximal aspect of the distal phalanx are not uncommon and may be present unassociated with clinical signs. Large mobile pieces are more likely to be associated with lameness.

The distal border of the navicular bone also should be evaluated carefully, because the lucent zones along the distal border represent synovial invaginations from the distal interphalangeal joint. An increase in size and number of these lucent zones has been observed with chronic synovitis of the distal interphalangeal joint.

Ultrasonography

Diagnostic ultrasonography with a 7.5-MHz transducer and a stand off is invaluable for assessment of the dorsal pouch of the distal interphalangeal joint, the amount of fluid within the joint, and the presence of synovial proliferation. Assessment of the palmar pouch is much more difficult, and evaluation of the articular cartilage is extremely limited. The structure of the collateral ligaments of the distal interphalangeal joint proximal to the hoof capsule can be assessed[11,12] and the chondrocompedal, chondrocoronal, and distal digital annular ligaments.[13]

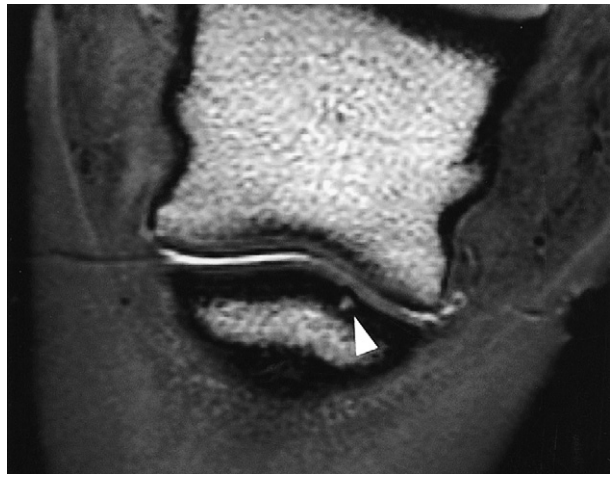

Fig. 34-3 Dorsal three-dimensional T2* gradient echo magnetic resonance imaging of the left hind foot of a riding horse with lameness of 3 months' duration. The lameness was completely resolved by intra-articular analgesia. Radiographic examination was negative; scintigraphy revealed focal increased radiopharmaceutical uptake in the proximal aspect of the distal phalanx. A focal lesion is present in the subchondral bone plate of the distal phalanx *(arrow)*.

Nuclear Scintigraphy

Nuclear scintigraphy has been useful in the identification of horses with distal interphalangeal joint capsule and subchondral bone trauma. It appears to be rather insensitive to the identification of osteoarthritis unless the disease is advanced.[14] Abnormal uptake of the radiopharmaceutical is best detected in lateral images.

Diagnostic Arthroscopy

The dorsal and palmar pouches of the distal interphalangeal joint may be inspected arthroscopically; however, the view of joint surfaces is very limited and complete assessment of the integrity of the articular cartilage is not possible. Access may be enhanced after joint trauma with resultant instability of the joint. Affected horses usually develop long-term lameness problems. Lavage of the joint may be beneficial therapeutically in some horses with chronic distal interphalangeal joint pain without joint instability. A limited view of the distal sesamoidean impar ligament can be seen through the navicular bursa.

Magnetic Resonance Imaging

Sagittal, frontal, and transverse magnetic resonance images of the distal interphalangeal joint permit excellent evaluation of the articular cartilage and subchondral bone of the joint and the dorsal and palmar pouches of the distal interphalangeal joint capsule (Fig. 34-3). The distal sesamoidean impar ligament, DDFT, collateral ligaments of the navicular bone, and the navicular bone and bursa also may be assessed. Magnetic resonance imaging (MRI) is the imaging modality of choice for horses with chronic distal interphalangeal joint pain that does not respond adequately to medical treatment.

DIFFERENTIAL DIAGNOSIS OF PRIMARY DISTAL INTERPHALANGEAL JOINT PAIN

Synovitis

The most common cause of distal interphalangeal joint pain is synovitis, which may occur unilaterally or bilaterally. Lameness is mild to moderate in degree and palpable distention of the distal interphalangeal joint capsule usually is present. Intra-articular analgesia generally resolves the lame-

ness. Treatment should be directed to identification of any predisposing causes. Corrective trimming to restore correct foot balance and appropriate shoeing is essential for successful management. Horses with collapsed heels usually benefit substantially from properly fitted egg bar shoes (see Fig. 31-1). The timing of trimming and shoeing can be crucial: if the feet are allowed to get too long, soreness may return.

In my experience, systemic administration of hyaluronan is generally of limited benefit in the initial treatment of horses with acute or chronic lameness, but it may have a role in longer-term management. Intra-articular medication using hyaluronan, with or without short-acting corticosteroids (e.g., triamcinolone) or polysulfated glycosaminoglycans (PSGAG) is the most effective treatment method. In horses with acute synovitis with only mild lameness a single treatment with hyaluronan alone may be sufficient, but if the lameness is more severe or chronic, better results may be achieved with a combination of triamcinolone and hyaluronan. Improvement usually is evident within 5 days of treatment. If lameness persists, better results may be achieved by two additional injections using hyaluronan alone at weekly intervals. Intra-articular treatment with PSGAG is contraindicated if acute inflammation is present, but in horses with more chronic lameness, good results have been achieved using serial (up to five) weekly treatments.[15,16] PSGAG used systemically may be useful for longer-term management. Treatment is followed by walking for 7 days and then a progressive resumption of work after the final treatment. In some horses, intra-articular therapy results in long-term resolution of the problem. Others require repeated treatments at intervals as needed.

Care is necessary during injection of the distal interphalangeal joint to avoid puncturing the large vessels on the distodorsal aspect of the pastern. Puncture tends to cause localized fibrosis and future injections are more difficult. With excellent technique the distal interphalangeal joint will tolerate well many injections, and the prognosis for future soundness is good.

Osteoarthritis

Osteoarthritis without Radiographic Abnormalities

Scintigraphy may be useful in the diagnosis of early subchondral lesions associated with osteoarthritis. The definitive diagnosis of osteoarthritis without radiographic abnormalities is possible pre-mortem only by MRI (Fig. 34-4). Horses may have signs similar to those of primary synovitis, but the degree of lameness may be more severe, especially if the horse is exerted maximally, and the response to intra-articular medication tends to be shorter and less complete. MRI may show a generalized loss of signal within the articular cartilage of the distal interphalangeal joint, in addition to irregularity in the cartilage surface, with or without concurrent abnormalities in the subchondral bone. The prognosis for sustained future soundness is guarded.

Osteoarthritis with Radiographic Abnormalities

Correlation is lacking between modeling in the region of the extensor process of the distal phalanx and lameness associated with the distal interphalangeal joint. Entheseophytes at the site of insertion of the common digital extensor tendon should be differentiated from osteophytes. Entheseophytes often are not associated with current lameness. However, the presence of periarticular osteophytes on the distodorsal and palmar aspects of the middle phalanx and the proximal articular surface of the navicular bone is more likely to be associated with lameness (Figs. 34-5 and 34-6). Radiographic evidence of osteoarthritis of the distal interphalangeal joint can be seen with other causes of lameness, such as navicular disease. Horses with primary osteoarthritis of the distal interphalangeal joint may respond better to serial treatments with PSGAG than treatment with hyaluronan and corticosteroids.[15,16] However, if concurrent severe synovitis is present, primary treatment with triamcinolone and hyaluronan, followed by intra-articular PSGAG, may yield the best results. In my experience, better results are achieved in horses that are sound after intra-articular analgesia compared with horses that show partial improvement in lameness. Prognosis usually is inversely related to the severity of the radiographic abnormalities.[7]

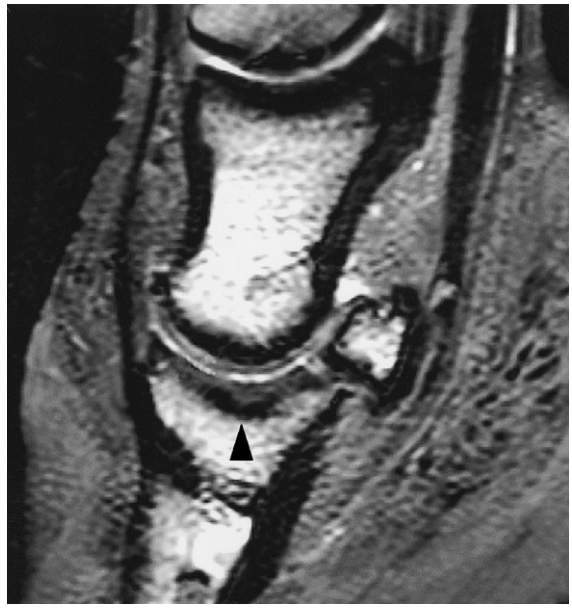

Fig. 34-4 Sagittal three-dimensional T2* gradient echo magnetic resonance image of the foot of a Grand Prix show jumper. There is loss of the normal homogenous signal in the cartilage of the distal interphalangeal joint and irregularities in the subchondral bone in the center of the distal phalanx *(arrow)*. Mild periarticular osteophyte formation was evident on radiographs. Scintigraphic examination was unremarkable.

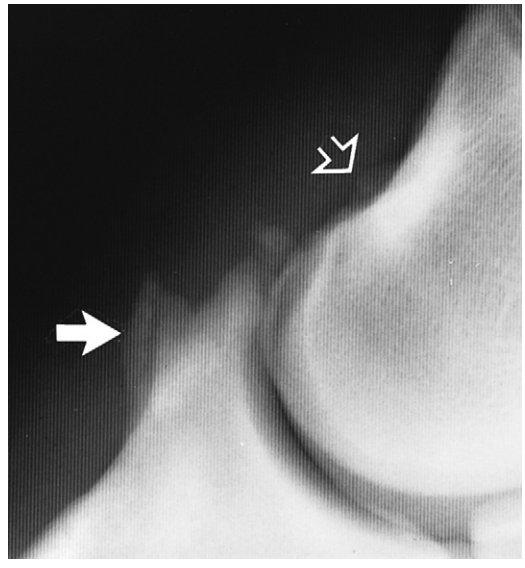

Fig. 34-5 Lateromedial radiographic view of the distal interphalangeal joint of a 7-year-old pleasure horse. Marked entheseophyte formation is present on the dorsoproximal aspect of the distal phalanx *(arrow)*. In addition, modeling of the extensor process of the distal phalanx and slight new bone formation on the distal dorsal aspect of the middle phalanx are seen *(open arrow)*.

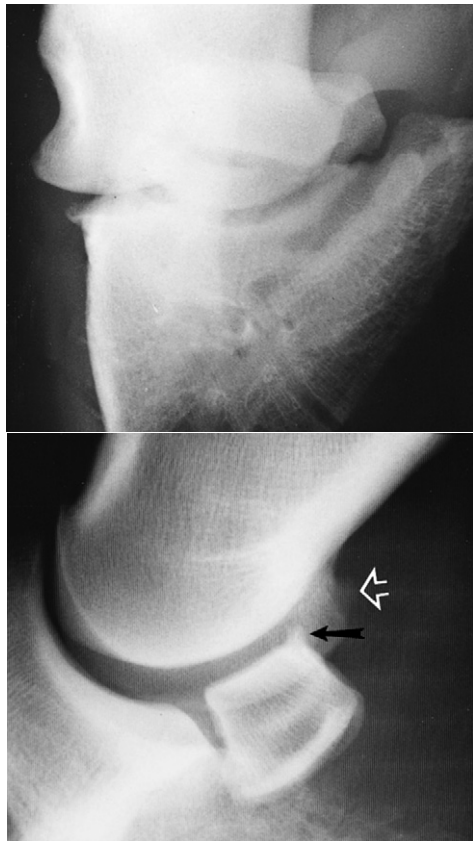

Fig. 34-6 **A,** Dorsolateral-palmaromedial oblique view of a flexed distal interphalangeal joint of an 8-year-old show jumper with bilateral forelimb lameness alleviated by intra-articular analgesia of the distal interphalangeal joints. Modeling of the proximal articular margin of the distal phalanx is seen, radiographic evidence of osteoarthritis (compare with Fig. 34-2). No radiographic abnormalities were seen in lateromedial or dorsopalmar views. **B,** Lateromedial radiographic view of the left front foot of a riding horse with lameness improved by intra-articular analgesia of the distal interphalangeal joint. An articular osteophyte is seen on the dorsoproximal aspect of the navicular bone *(arrow)*, and osteophyte formation is visible on the distal palmar aspect of the middle phalanx *(open arrow)*.

Traumatic Damage to Articular Cartilage

Sudden onset of unilateral lameness may be related to traumatic damage to the articular cartilage of the distal interphalangeal joint, with or without other concurrent soft tissue damage. Definitive diagnosis is possible only with MRI. Intra-articular medication may provide temporary relief of clinical signs but the long term prognosis is guarded.

Joint Capsule Trauma

Traumatic damage to the joint capsule, with or without subchondral bone trauma, usually results in sudden-onset, severe lameness that persists despite rest. Lameness may be accentuated markedly when the horse turns. In the acute stage, no abnormalities are detected on radiographic examination. However, periarticular new bone may develop after several weeks (Fig. 34-7). Nuclear scintigraphic examination may show generalized increased uptake of the radiopharmaceutical in the region of the distal interphalangeal joint (Fig. 34-8). Arthroscopic evaluation in these horses has been unrewarding. The response to intra-articular medication has been poor,

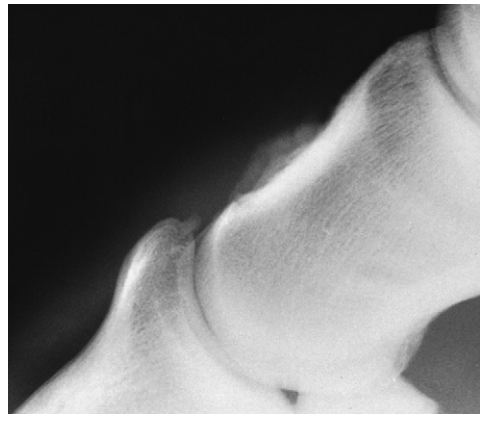

Fig. 34-7 Lateromedial radiographic view of the left front foot of a 3-year-old Thoroughbred filly with severe lameness that improved substantially after intra-articular analgesia of the distal interphalangeal joint. Entheseous new bone is seen on the dorsal aspect of the middle phalanx, in addition to the small fragment at the extensor process of the distal phalanx. Nuclear scintigraphic examination had revealed a similar pattern of uptake to that seen in Fig. 34-8.

and the prognosis for return to athletic function despite prolonged rest is guarded.

Subchondral Bone Trauma

Subchondral bone trauma may be focal or more generalized and usually is associated with unilateral lameness, which responds poorly to intra-articular medication and short periods of rest. No detectable radiological abnormalities may be apparent in the acute stage, although Ross[14] described subtle proliferative changes on the distal aspect of the middle phalanx and the proximal aspect of the distal phalanx and a variable degree of subchondral lucency in the proximal aspect of the distal phalanx. Nuclear scintigraphy is required for definitive diagnosis.[14] MRI has demonstrated a focal lesion in the proximal subchondral bone plate of the distal phalanx (see Fig. 34-3). Whether this finding may be a precursor to the development of an osseous cyst-like lesion is unknown.

Osseous Cyst-Like Lesions

Osseous cyst-like lesions occur most often in the center of the proximal aspect of the distal phalanx.[17,18] They vary in size and the presence of visible communication with the distal interphalangeal joint. Not all are detectable radiographically; some have been identified only at postmortem examination after a poor response to medical treatment of the distal interphalangeal joint.[16] Horses with large osseous cyst-like lesions may be asymptomatic but be lame at a later date. Lameness usually is unilateral, sudden in onset, and moderate to severe. Osseous cyst-like lesions may occur in young immature horses and mature athletes. Not all osseous cyst-like lesions that cause pain have active bone turnover; therefore nuclear scintigraphy may not be helpful in determining whether a long-standing osseous cyst-like lesion is the current cause of lameness. However, currently or recently developing osseous cyst-like lesions usually are associated with marked increased bone activity and may be evident on nuclear scintigraphic scans before radiographic evidence is evident. Conservative treatment may result in spontaneous resolution of the lameness, with or without resolution of the cyst, but in some horses lameness persists, with or without enlargement of the cyst. Arthroscopic access to a cyst usually

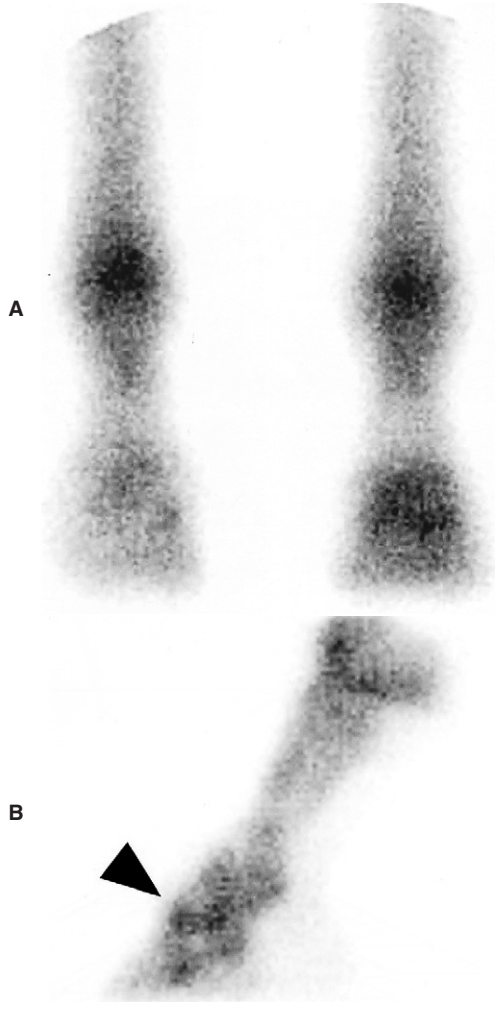

Fig. 34-8 **A,** Dorsal nuclear scintigraphic image of the front feet of a 6-year-old show jumper with sudden-onset, severe lameness markedly improved by intra-articular analgesia of the distal interphalangeal joint. The left forelimb is on the right. Radiopharmaceutical uptake is increased in the subchondral bone of the distal interphalangeal joint. **B,** Lateral scintigraphic image of the left front foot shows increased uptake of the radiopharmaceutical centered on the distal interphalangeal joint *(arrow)*. Slight periarticular new bone formation was evident radiographically. Arthroscopic evaluation revealed synovial proliferation. The horse remained lame.

is poor if it is in the central portion of the distal phalanx. Therefore surgical debridement of a well-defined osseous cyst-like lesion usually is performed through the hoof wall. Cysts located more dorsally can be debrided arthroscopically, however. Prognosis depends in part on the integrity of the overlying articular cartilage and, in some horses, lameness persists.[19,20]

Palisading New Bone on Dorsal Aspect of Middle Phalanx

Pain associated with the distal interphalangeal joint occasionally is associated with new bone formation on the dorsal aspect of the middle phalanx. The origin of this new bone is unknown. Arthroscopic evaluation of the joint may reveal crumbly bone, which is easily debrided, with resolution of lameness in some horses.[21]

Desmitis of Collateral Ligaments of Distal Interphalangeal Joint

Desmitis of the medial or lateral collateral ligament of the distal interphalangeal joint results in sudden-onset, severe unilateral lameness.[4,11,22] The desmitis is believed to be due to abnormal sliding and rotation of the joint (e.g., lateral sliding of the distal phalanx and medial rotation, which causes the medial collateral ligament to undergo great strain). Avulsion fractures also may accompany this injury. Damage to the body of the ligament results in obvious periligamentous soft tissue swelling and pain, but no palpable abnormality may exist with insertional injuries. Detection of joint instability may be possible with severe injury.[22]

Desmitis of the body of the medial or lateral collateral ligament is confirmed ultrasonographically[11,12] and is characterized by enlargement in cross-sectional area of the ligament and a diffuse reduction in echogenicity or a hypoechogenic core lesion. Modeling at the insertion on the middle phalanx may also be seen. In a few horses a well-defined radiolucent area has been identified in the distal phalanx at the site of insertion of a collateral ligament.[4,22]

Treatment is box rest for a minimum of 2 months followed by at least 4 months of walking exercise. When the medial collateral ligament is damaged, a shoe with a wide medial branch and narrow lateral branch is recommended. For lateral collateral desmitis a shoe with a wide lateral branch with a lateral extension and a narrow medial branch is preferred.[2]

The prognosis for return to athletic function is fair for horses with injuries that affect primarily the body of the ligament.[11] The prognosis for horses with avulsion injuries at the insertion of a collateral ligament on the distal phalanx, with joint instability and radiographic abnormalities, is much more guarded.[4,22]

Osseous Fragments on Dorsal Aspect of Distal Interphalangeal Joint

Small osseous fragments on the dorsoproximal aspect of the extensor process of the distal phalanx may be seen in clinically normal horses. Some are pointed proximally with a flat base and look like the tip of the extensor process, whereas others are well rounded. These fragments may represent separate centers of ossification or be a manifestation of osteochondrosis. If the fragments are seen in association with distal interphalangeal joint pain, the significance should be interpreted with care. Arthroscopic removal should be considered only if medical therapy of the joint fails.

Fracture of Extensor Process of Distal Phalanx

Fractures of the extensor process of the distal phalanx are discussed on page 316.

Articular Chip Fracture of Middle Phalanx

Articular chip fractures of the medial or lateral condyle of the middle phalanx occasionally cause acute-onset lameness.[16] Such fractures can be detected only by flexed oblique radiographic views of the distal interphalangeal joint. The prognosis after surgical removal is favorable provided joint stability has not been compromised. Surgical repair with arthroscopic guidance also is possible.

Damaged Distal Sesamoidean Impar Ligament

Focal tears in the distal sesamoidean impar ligament have been seen in association with sudden-onset, severe lameness that improves after intra-articular analgesia of the distal interphalangeal joint. In horses with acute lameness the synovial fluid may be hemorrhagic. Usually no radiological abnormalities are present. Definitive diagnosis is possible with MRI or arthroscopic evaluation of the navicular bursa.[4] The prognosis for athletic function is guarded.

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FRACTURES AND FRAGMENTATION OF THE EXTENSOR PROCESS OF THE DISTAL PHALANX

• Gayle Trotter

HISTORY

Osteochondral fragments of the extensor process of the distal phalanx are classified as type IV fractures and exist as two clinical entities.[1-3] Small fragments variably are considered as traumatically induced or unusual manifestations of osteochondrosis. Large fragments are considered as traumatically induced or separate centers of ossification[2] or a variation of the traumatically induced, non-union fracture osseous bodies often observed in other locations on the distal phalanx in foals.[4]

Both types of fragments involve predominantly the front feet, and lameness may be present. Fragments can be seen as incidental radiological abnormalities. The fragments almost always are intra-articular, but they usually are non-displaced to minimally displaced.

CLINICAL AND IMAGING FINDINGS

Low-grade lameness may be present in horses with small fragments. Lameness alleviated by intra-articular analgesia of the distal interphalangeal joint was present in 16 of 21 horses with osteochondral fragments.[3] Lameness may not develop until the horse is in full work when the presence of the fragment induces synovitis. The fragment is seen readily on a lateromedial radiographic view. Many fragments are removed prophylactically.[3]

Lameness associated with large fragments may be delayed in onset; radiological abnormalities often appear chronic when lameness is first recognized. A strong fibrous union may maintain stability of the fragment until trauma causes minor fragment loosening. Lameness apparently is unrelated to fragment size.[1] I have observed large bilateral fragments in a horse with unilateral lameness, and surgery was performed to remove the smaller but clinically relevant fragment. Seven years later lameness related to the fragment developed in the opposite limb that also was corrected surgically. In some horses the hoof shape becomes triangular or pyramidal; this clinical appearance has been described as *pyramidal disease* or *buttress foot*.[2]

TREATMENT AND PROGNOSIS

Removal of small fragments under arthroscopic guidance is a well-accepted treatment with a highly favorable prognosis.[3] Lameness resolved in 14 or 16 horses with lameness associated with a small fragment, and 5 of 5 horses remained sound after the fragment was removed prophylactically.[3] Horses can return to work within 2 weeks of fragment removal if surgery is prophylactic and at 10 weeks later if preoperative lameness was present.

Guidelines for treatment of horses with large fragments are less clear. Full recovery after internal fixation of these fragments was reported for two horses in four reports (six horses).[1,3-5] At least one horse had an acute fracture.[5] Fragment removal by dorsal midline arthrotomy has been reported in 14 horses with a successful outcome in 8 (57%).[1] Of the 6 horses with unfavorable results, preoperative duration of lameness exceeded 2 years in 3 horses, suggesting more favorable results might be expected if preoperative lameness duration were shorter. Horses usually return to full work by 4 months after surgery.

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OSSEOUS CYST-LIKE LESIONS IN THE DISTAL PHALANX

• Gayle Trotter and Sue J. Dyson

HISTORY

Osseous cyst-like lesions in the distal phalanx usually are unilateral, solitary, and range in location from the extensor process to deep within the weight-bearing surface. Debate continues on whether the lesions have a traumatic or developmental origin. When large bilateral lesions are present in a young horse, a developmental component is strongly suggested.[1-3]

CLINICAL AND IMAGING FINDINGS

Affected horses often are young but mature, and acute-onset, moderate to severe lameness frequently is present. Less commonly the history includes a low-grade, intermittent lameness. Occasionally osseous cyst-like lesions are identified as incidental radiological findings. Osseous cyst-like lesions sometimes are seen in older horses with pain localized to the foot. Such lesions may be clinically significant. Lameness may be exacerbated by distal limb flexion, but results of hoof tester examination usually are negative. Lesions are seen more commonly in the forelimbs, although the hindlimb was affected in 3 of 15 horses in one report.[1] Effusion of the distal interphalangeal joint occasionally is present, and large bilateral lesions sometimes are associated with a buttress foot appearance.

Lameness often is unaltered by perineural analgesia of the palmar digital nerves, but it is resolved by palmar (abaxial sesamoid) nerve blocks. Intra-articular analgesia of the distal interphalangeal joint improves lameness associated with osseous cyst-like lesions that communicate with the joint. Small articular lesions may not be detectable radiographically, but large osseous cyst-like lesions usually are readily identifiable on dorsoproximal-palmarodistal (upright pedal) views. Lesions involving the extensor process are seen readily on lateromedial projections. Smaller osseous cyst-like lesions located in a very medial or lateral location within the more weight-bearing aspect of the joint are best identified in weight-bearing dorsopalmar views. Nuclear scintigraphic examination may identify small osseous cyst-like lesions that are not identifiable radiographically. Osseous cyst-like lesions may be associated with increased uptake of radiopharmaceutical, depending on the stage of lesion development and activity of the surrounding bone. However, an osseous cyst-like lesion that is scintigraphically silent still may be a cause of lameness.

TREATMENT AND PROGNOSIS

Reports documenting results after either conservative or surgical treatment are limited. In one study, conservative treatment resulted in clinical improvement in approximately 30% of horses, but the prognosis for full recovery was guarded.[1] One report documented favorable results in a horse with bilateral extensor process fragmentation through what were presumed to be pre-existing cystic lesions in the extensor processes.[4] Anecdotally arthroscopic debridement of smaller extensor process lesions can be associated with full return to function.[5] Many lesions are not accessible by arthroscopy or arthrotomy through a dorsal approach. Debridement of distal phalanx lesions through the hoof wall has been described,[6] but this approach has not gained universal acceptance. However, good results have been achieved in a limited number of horses.[7] Intra-articular medication can be useful in the ongoing management of selected horses; occasionally conservative treatment results in a horse suitable for a limited amount of riding activity.

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KERATOMAS, NEOPLASTIC, AND NON-NEOPLASTIC SPACE-OCCUPYING LESIONS IN THE HOOF

• Sue J. Dyson

Keratomas are classified as aberrant, possibly hyperplastic, keratin masses originating from epidermal horn producing cells of the coronary band.[1] They also have been defined as benign neoplasms originating from the coronary dermis.[2] As the abnormal horn tubules grow distally toward the toe, hoof wall deformation may occur with disruption of the white line, which may permit entry of infection. Expansion of the mass results in pressure necrosis of the adjacent distal phalanx. Keratomas occur most often in the dorsal half of the foot.[3] A keratoma is the most common space-occupying lesion in the hoof, but other neoplastic conditions occur occasionally and result in similar clinical signs, although they usually can be differentiated radiographically.[4-8] Other non-neoplastic space-occupying lesions, such as epidermal inclusion cysts, have been identified that can be differentiated from a keratoma only by histological examination.[9]

HISTORY

Often mild intermittent forelimb or hindlimb lameness exists, or more severe episodes of lameness associated with recurrent sub-solar abscessation may have occurred.[2,3,10-12] Neoplastic conditions may be associated with more severe lameness. Distortion of the hoof capsule may have been noted.

CLINICAL SIGNS AND DIAGNOSIS

The hoof capsule, the white line, or both may be distorted but not invariably. Pressure applied with hoof testers may elicit local pain. A variable degree of lameness is present that is alleviated by desensitization of the foot or unilateral palmar block on the side of the lesion. Radiographic examination may reveal a smoothly demarcated radiolucent defect in the margin of the distal phalanx (Fig. 34-9). This characteristic is typical of keratomas or other non-neoplastic space-occupying lesions, whereas neoplastic lesions tend to have more irregular margins and may be associated with new bone formation.[8] Infectious osteitis of the distal phalanx may have a similar appearance, although if the osteitis is chronic, marginal sclerosis with or without new bone formation may be present. The crena at the toe of the distal phalanx should not be confused with a space-occupying lesion. Lesions are best detected in dorsoproximal-palmarodistal oblique, dorsolateral-palmaromedial oblique, or dorsomedial-palmarolateral oblique views. A keratoma also may be present in the absence of radiological change with only distortion of the hoof capsule.[3] If the clinical significance of such a radiographic lesion is in doubt, nuclear scintigraphy may be helpful because such lesions usually are associated with focal increased uptake of radiopharmaceutical. Diagnostic ultrasonography also has been used to identify a keratoma at the coronary band.[13]

TREATMENT

Horses with keratomas and other benign space-occupying lesions respond well to surgical excision and have a good prognosis,[2,3,10-14] but those with neoplastic lesions have a more guarded prognosis. It is best to excise keratomas and other space-occupying lesions through a hoof wall rather than solar approach, if possible.

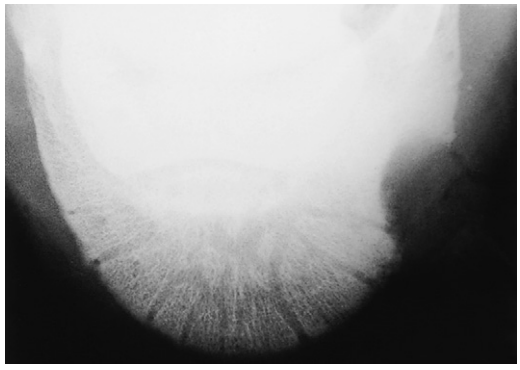

Fig. 34-9 Slightly oblique dorsoproximal-palmarodistal oblique view of a foot. A smoothly outlined semicircular defect is present in the lateral aspect of the distal phalanx, typical of a keratoma. Lateral is to the right.

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FRACTURES OF THE DISTAL PHALANX

• Sue J. Dyson

Fractures of the distal phalanx are a relatively common cause of lameness in horses from all disciplines and often are the result of trauma, through a mis-step or high-speed impact, or kicking a fixed object. Fractures have been classified into six types,[1,2] but other configurations also occur. Fractures of the extensor process of the distal phalanx (type IV) are discussed elsewhere (see page 316). Fractures of the solar margin of the distal phalanx (type VI), which may occur in foals or adults, are considered separately from fractures of the body of the bone. The latter may be articular or non-articular and comprise sagittal (type III), oblique (extending from the midline to the lateral or medial solar margin) (type II), and comminuted fractures (type V), fracture of a palmar process (type I) and, less commonly, fractures of other configurations.

FRACTURE OF THE SOLAR MARGIN OF THE DISTAL PHALANX

Foals

Fractures of the palmar-most aspect of the palmar process of the distal phalanx occur commonly in foals.[3,4] These fractures originate at the incisure (which normally separates the proxi-

mal and distal palmar process angles), continue dorsally toward the toe for 1 to 3 cm and then extend to the solar margin. They are believed to be caused by shear forces generated by tension of the DDFT or by compression of the solar cortical surface and tension on the dorsal cortical surface during weight bearing. One hypothesis is that excessive trimming of the heel, thus increasing tension in the DDFT, or excessive trimming of the sole of the frog, thus increasing concussion on the distal phalanx and forces on the palmar processes, may be predisposing factors to these fractures, but this was not substantiated in a clinical study, perhaps because of the high overall incidence of fractures (34%).[5]

Clinical Signs and Diagnosis

Lameness usually is mild and extremely transitory, lasting only 1 to 2 days, and may precede radiographic identification of a fracture. No consistent response to pressure applied with hoof testers at the heel is present. Diagnosis is based on radiographic identification of a discrete osseous body at the palmar process. Fractures may be identified on lateromedial views, but dorsal 65° proximal-palmarodistal oblique views are more sensitive. Not all fractures are detectable radiographically.

Treatment and Prognosis

Even with normal management, lameness is only transitory and spontaneous healing occurs within 4 to 8 weeks with an excellent long term prognosis. Any type of shoe should be avoided, because shoe application in foals can quickly cause contracted feet.

Adult Horses

Fractures of the solar margin of the distal phalanx in adult horses occur from the quarters toward the more dorsal aspect of the distal phalanx; these fractures also are referred to as *cracking off*.[2,6] These fractures may heal, be resorbed, or persist without signs; therefore radiographic identification of solar margin fragmentation is not necessarily synonymous with identification of the source of pain causing lameness. Solar margin fractures occur almost exclusively in forelimbs. A geographical influence on the incidence of solar margin fractures is apparent, which may reflect the footing on which the horses work or other undefined factors. In the Editors' experience, these fractures are rare, whereas a study in California of distal phalanx fractures in 274 horses identified 132 (48%) horses with solar margin fractures. However, these fractures frequently occurred in association with radiographic evidence of laminitis or another potential cause of lameness, such as navicular disease.[6] Solar margin fractures as the sole potential cause of lameness were identified only in 25 horses. Irregularity and reduced opacity at the solar margin, with widening of the vascular channels, may be a predisposing factor.

Clinical Signs and Diagnosis

Horses with solar margin fractures often have a history of foot soreness. With an acute solar margin fracture, there may be increased sensitivity to hoof testers at the toe, provided that the sole is not excessively hard. Lameness is removed by perineural analgesia of the palmar nerves in the proximal pastern region or at the level of the proximal sesamoid bones. Diagnosis depends on radiographic identification of the fracture or fractures, which may be single or multiple and affect a variable extent of the solar margin. Primary solar margin fractures usually are single, whereas those that occur in association with laminitis or radiographic evidence of demineralization of the solar margin are more likely to be multiple, comminuted, or both. Fractures are identified readily in an appropriately exposed dorsoproximal-palmarodistal oblique radiographic view, but they are overlooked easily if the radiograph is over-exposed. The radiographs should be inspected carefully to detect evidence of any other potential cause of lameness that may influence treatment and prognosis.

Treatment

Horses with lameness associated with a solar margin fracture usually require prolonged rest, especially if radiographic evidence of pre-existing demineralization of the solar margin is present; healing is assessed by periodic radiographic examination. The use of a broad web shoe with a concave solar margin, with or without pads, is recommended. Limited documented long-term follow up information is available for horses with primary solar margin fractures, but the prognosis generally is favorable.

FRACTURES OF THE BODY OF THE DISTAL PHALANX

Fractures of the body of the distal phalanx occur more commonly in forelimbs than hindlimbs, but they are not uncommon in either limb.[1,2,7-9] A fracture of the distal phalanx usually results in acute-onset, severe lameness. If the fracture is articular the distal interphalangeal capsule may be distended. Pain usually occurs when pressure is applied with hoof testers, but this finding may not be a feature of a non-displaced fracture of a palmar process, especially if it has been present for more than several days. Articular fractures invariably result in continuous lameness, but a non-articular fracture of a palmar or plantar process of the distal phalanx may be associated with intermittent lameness, especially in a hindlimb. Clinical signs of an articular fracture usually reflect foot pain and thus regional analgesia often is not required, but with non-articular fractures the clinical signs may be less specific and perineural analgesia may be required to determine the source of pain. Some fractures heal only by fibrous union and not osseous union. The radiographic findings of such fractures, which do not have the appearance of a narrow, clearly defined line, should be interpreted with care because although such fractures may be unstable and therefore are a potential source of pain, the pain does not necessarily originate in the fracture.

Fractures of the distal phalanx are a common racehorse injury in North America. Fractures occur most often in the lateral aspect of the left front foot and the medial aspect of the right front foot in association with the counterclockwise direction of training and racing.

Diagnosis

Diagnosis is based on radiographic identification of a fracture (Figs. 34-10 and 34-11). A very recent non-displaced fracture may be difficult to identify radiographically unless the x-ray beam is completely parallel to the fracture line, and many oblique views may be required. The foot should be cleaned thoroughly before radiographic examination to avoid artifacts. The frog clefts may create confusing lucent lines across the distal phalanx; therefore the foot should be packed with a moldable modeling compound (e.g., PlayDoh; Hasbro, Inc., Pawtucket, RI). Many articular fractures are seen readily on dorsoproximal-palmarodistal oblique views, but fractures of the palmar processes and other less common configurations of fracture may not be apparent. A fracture of a palmar process may be evident on a lateromedial projection, but oblique views of the palmar process are required in many horses. Most of these fractures can be detected in dorsal 30° to 45° lateral (or medial)-palmarodistal oblique views with the foot either bearing weight or in the upright pedal position, but occasionally oblique views are required with the horse standing on the x-ray cassette (e.g., dorsal 45° proximal 45° lateral–palmarodistal medial oblique view). Separate centers of ossification at the palmar aspect of the palmar process should not be confused with a fracture.[10] Rarely a palmar process fracture is seen only in a palmaroproximal-palmarodistal oblique view.

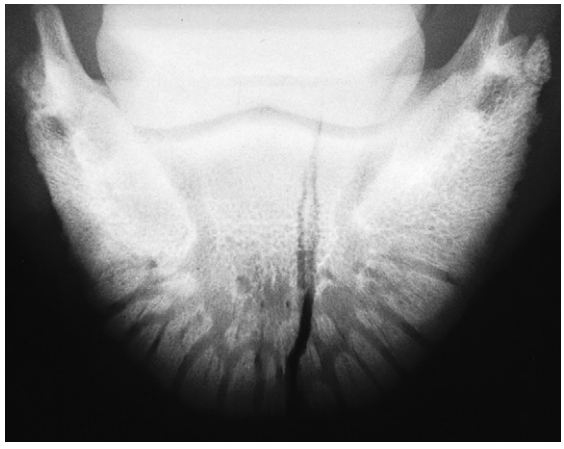

Fig. 34-10 Dorsoproximal-palmarodistal oblique view of a left hind foot. The distal phalanx has a complete, articular, parasagittal fracture.

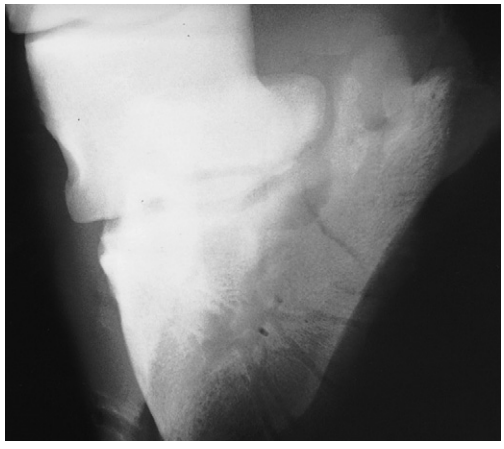

Fig. 34-11 Palmarolateral-dorsomedial oblique view of a left front foot. There is a complete, non-displaced, non-articular fracture of the medial palmar process of the distal phalanx.

Occasionally obscure incomplete, non-articular fractures of the body of the distal phalanx are identified in a palmaroproximal-palmarodistal oblique view.

Nuclear scintigraphic examination usually is unnecessary for the identification of most acute fractures, except those causing episodic lameness, but can be helpful in confirming the likely significance of older fractures.[11] Scintigraphy also is useful for identification of subchondral bone trauma unassociated with detectable radiographic abnormalities (see following text).

Treatment and Prognosis

Conservative treatment of horses with non-articular palmar process fractures, by box rest and use of a bar shoe with five clips or bar rim shoe, has a good prognosis. Complete healing of the fracture may not be evident radiographically for several months but most fractures eventually do heal. Horses with fractures extending into the medial or lateral extremity of the distal interphalangeal joint also have a good prognosis with conservative management. The horse should continue to be shod with a bar shoe when work is resumed to reduce the risks of re-injury. If a bar shoe is replaced by an open shoe, avoidance of maximum work intensity may be preferable until the hoof capsule and distal phalanx have had time to adapt to altered concussive forces. Although sagittal and oblique articular fractures of the distal phalanx often heal well if treated conservatively in horses younger than 3 years of age, the prognosis for adult horses is more guarded and internal fixation using a lag screw technique is recommended.[1,7,9,12] Prognosis depends on whether displacement at the articular margin exists; displacement inevitably results in osteoarthritis and associated lameness. Potential complications include postoperative infection and screw irritation or rejection. The use of a headless titanium Herbert cannulated screw (Zimmer Corp. Warsaw, IN) may result in fewer complications.[13] Complete fracture healing requires 6 to 12 months of convalescence with surgical management.

COMMINUTED FRACTURES

Comminuted fractures of the distal phalanx are not common[1,2,7] but do occur occasionally. Many radiographic views may be required to establish the precise configuration of the fractures and determine possible articular involvement, which results in a more guarded prognosis. The configuration of the fracture determines whether internal fixation is a viable option.[7,12]

FRACTURES OF THE DISTAL PHALANX ASSOCIATED WITH PENETRATING INJURY

A penetrating injury of the foot (see Chapter 28) may result in a fracture of the distal phalanx of any configuration. Treatment is dictated by the fracture configuration. The primary aim is to control infection. Removal of small bone fragments that otherwise may sequestrate may be preferable.

SUBCHONDRAL TRAUMA OF THE DISTAL PHALANX

Some horses have forelimb lameness, usually unilateral, associated with pain localized to the foot but no significant detectable radiological abnormality. Nuclear scintigraphic examination reveals a focal round or semi-circular area of increased uptake of the radiopharmaceutical, seen in a solar view, either medially or laterally, far removed from the distal interphalangeal joint or on a lateral view in the center of the bone in a similar location to fractures.[14] This lameness is believed to reflect subchondral bone trauma and is an injury that occurs particularly in racehorses in North America but also in other types of horses. Lameness usually is unaffected, or less commonly, only partially affected by intra-articular analgesia of the distal interphalangeal joint. In some horses, mediolateral foot balance may be a predisposing factor. Less commonly the onset of lameness is associated with trauma. The failure to identify a fracture radiographically may reflect the inherent limitations of radiography. Alternatively, such reactions may indicate that distal phalangeal fractures, especially in racehorses, may not be single-event episodes but the result of stress remodeling. Treatment comprises rest and correction of any mediolateral foot imbalance. Floating the heel of the affected side may be helpful. The prognosis is favorable given sufficient time, which is proportional to the duration of lameness before diagnosis and treatment. With acute lesions a period of 6 to 8 weeks of box rest and controlled walking exercise usually is sufficient, with a progressive increase in work intensity thereafter, but with more chronic lesions a longer period may be necessary and repeated scintigraphic evaluation may be helpful to determine when the lesion is no longer active.

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PEDAL OSTEITIS: DOES IT EXIST?

• Sue J. Dyson

Pedal osteitis strictly means inflammation of the distal phalanx and has long been suggested as a cause of forelimb lameness. However, it is a poorly defined condition, previously diagnosed radiographically, characterized by focal or general demineralization around the solar margin of the distal phalanx and widening of the vascular channels, with or without abnormal lucent areas in the palmar processes. It is now recognized that considerable variation exists in the radiographic appearance of the distal phalanx in normal horses.[1-3] No good studies correlate foot conformation and the radiographic appearance of the distal phalanx. It is my impression that those horses with a particularly thin sole, especially in association with a horizontal orientation of the solar margin of the distal phalanx, seem prone to foot soreness if worked regularly on hard ground, but the source of pain has been poorly defined. Radiographic changes of the distal phalanx, once established, often persist over the long term and therefore are not synonymous with active inflammation. Radiographs represent a historical record of previous activity or injury. Scintigraphic examination of a large number of horses with foot pain occasionally has revealed evidence of abnormal bone turnover around the solar margins of the distal phalanx or confined to the palmar processes (see page 322).[4] In those horses with increased uptake of radiopharmaceutical around the solar margin of the distal phalanx in solar views, correlation with other clinical signs and radiographic findings frequently is poor unless the condition is localized to the toe, when it often reflects laminitis. The significance of this finding in the absence of localizing clinical signs remains open to question, since it also is seen in clinically normal horses. Pedal osteitis tends to have been used to describe a cause of lameness, when frequently the cause is actually undetermined.[5] The cause of pedal osteitis has not been defined, although abnormal concussion has been suggested.

Therefore I suggest that the term pedal osteitis is inappropriate and should not be used to describe a cause of lameness. Nonetheless, a variety of radiological changes of the margins of the distal phalanx can be identified, the causes of which are poorly defined. With our current state of knowledge, attribution of lameness to these radiological changes seems inappropriate unless localizing clinical signs, or concurrent evidence of ongoing inflammation with or without abnormal bone turnover, are documented scintigraphically.

Chronic laminitis may be associated with modeling of the toe of the distal phalanx and bone resorption with or without new bone on the dorsal aspect at the toe. Mineralization also has been identified on the dorsal aspects of the distal phalanx, midway between the coronary band and the solar margin; mineralization is seen best on lateromedial or slightly oblique views. This may represent mineralization in the dermal laminae or formation of new bone on the dorsal cortex of the distal phalanx. Extensive mineralization has been associated with lameness, but some roughening of the dorsal cortex in the region of the parietal sulci may be an incidental finding. Some horses with a club foot conformation develop focal loss of bone around the solar margin at the toe of the distal phalanx, and new bone on the dorsal aspect of the bone, associated with chronic lameness on hard ground. I suggest that in these conditions the radiological changes should be described and attributed to the primary cause, rather than labeled as pedal osteitis. Many horses with poor foot conformation suffer chronic lameness, probably associated with abnormal concussion to both the soft tissue and bony elements of the hoof. Identification of the primary cause of the problem and admission that the precise source or sources of pain cannot be defined is preferable to use of the term pedal osteitis, which implies a definitive diagnosis.

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OSTEITIS OF THE PALMAR PROCESSES OF THE DISTAL PHALANX

• Sue J. Dyson

ANATOMY

The palmar processes or angles of the distal phalanx are prism-shaped masses that project backward on the medial and lateral aspects of the bone. Each is divided into upper and lower parts by a notch or is perforated by a foramen that leads to the dorsal groove of the distal phalanx. The cartilages of the foot attach to the proximal border of each palmar process. In a normal horse the solar and abaxial surfaces of the palmar processes are relatively smooth.

Osteitis of the palmar processes of the distal phalanx may be part of the pedal osteitis complex, related to the long toe–low heel syndrome. Its origin is poorly understood. It occurs almost exclusively in the forelimbs and may result in irregular roughening of the bone surface (Fig. 34-12, *A*).

HISTORY AND CLINICAL FINDINGS

Lameness often is bilateral, insidious in onset, and tends to be worst on hard ground, similar to navicular disease. Affected horses often have poor conformation of the feet, with low, collapsed heels. The sole may be very flat, thin, and readily compressible. If the sole is readily compressible, pressure applied with hoof testers may be resented, but this usually is not a localized response.

DIAGNOSIS

Diagnosis requires a combination of response to nerve blocks, radiography, and, ideally, nuclear scintigraphy.

Local Analgesia

Lameness is improved by perineural analgesia of the palmar digital nerves but generally is unchanged after intra-articular analgesia of the distal interphalangeal joint or analgesia of the navicular bursa.

Imaging Techniques

Radiography

The orientation of the distal phalanx should be assessed in a lateromedial view. In a normal horse the solar margin is smooth in outline and at a 5° to 10° angle to the sole, sloping proximally toward its palmar aspect.[1] In some affected horses the palmar processes are at the same level, or lower than the toe of the distal phalanx (Fig. 34-12, *B*). The solar aspect of the palmar processes may have a fluffy appearance. The shape may change with elongation of the palmar processes; this change also is seen in dorsal 60° lateral-palmaromedial oblique or dorsal 60° medial-palmarolateral oblique views (Fig. 34-13, *A*). In a dorsoproximal-palmarodistal oblique view, discrete circular lucent areas, 2 to 3 mm in diameter, may be visible in the palmar processes, or the trabecular pattern may be more obvious as a result of generalized demineralization (Fig. 34-12, *C*).

Inversion of the distal phalanx in hindlimbs, with the plantar processes lower than the toe, also has been seen in association with more proximal sites of pain causing hindlimb lameness, such as proximal suspensory desmitis.[2,3] I have not recognized this as a primary cause of hind foot pain.

Nuclear Scintigraphy

If the bony changes are active, nuclear scintigraphic evaluation may reveal increased uptake of the radiopharmaceutical in the affected palmar processes to substantiate the significance of the clinical and radiographic findings (Fig. 34-13, *B*). However, this is sometimes an incidental finding and its significance should be interpreted in light of other clinical observations.

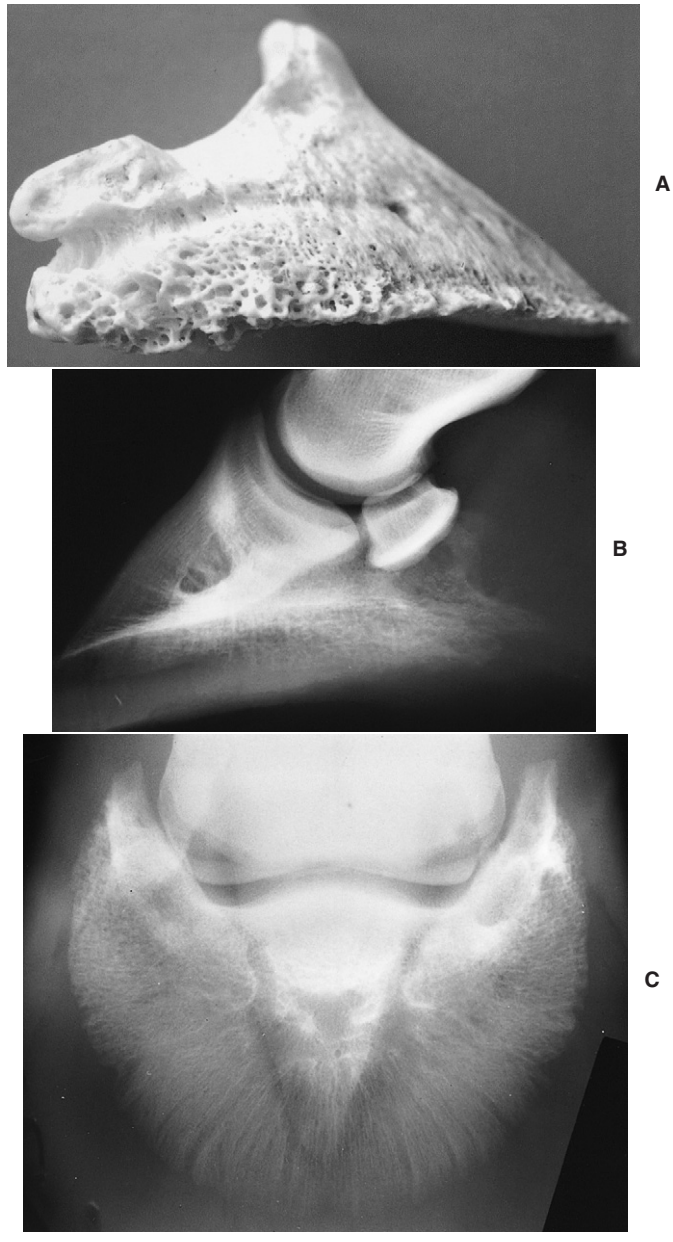

Fig. 34-12 **A,** The distal phalanx of a horse that had chronic heel pain. The palmar process has a roughened surface and new bone is evident on the solar surface. **B,** Lateromedial radiographic view of a foot of an 8-year-old Selle Francais with recurrent heel pain. Note the orientation of the solar surface of the distal phalanx: the palmar process is lower than the toe and the solar aspect of the palmar processes has an irregular contour. **C,** Dorsoproximal-palmarodistal oblique radiographic view of the same foot as shown in **B.** There is a very distinct trabecular pattern in the palmar processes, with many small lucent areas.

TREATMENT AND PROGNOSIS

Effective treatment depends on early recognition and corrective trimming and shoeing to try to restore more normal foot

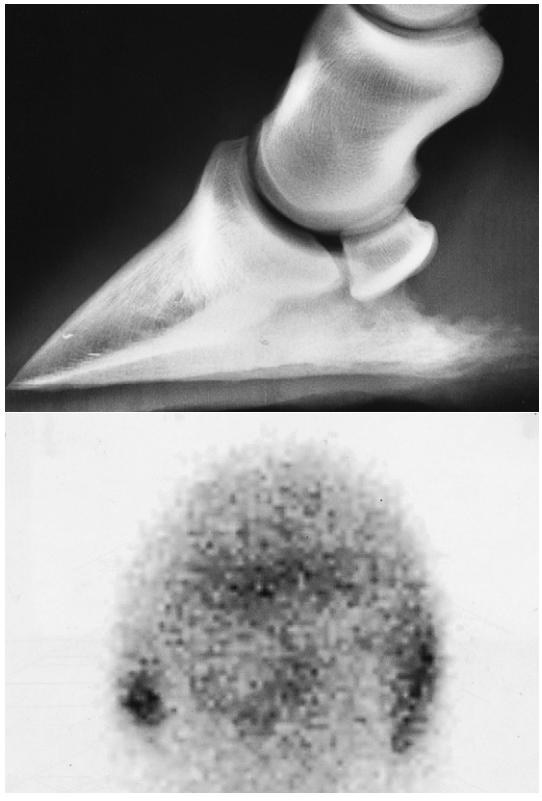

Fig. 34-13 **A,** Lateromedial radiographic view of a front foot of a 9-year-old Warmblood. Features include the abnormally elongated shape of the palmar processes, the horizontal orientation of the distal phalanx, and the thin sole. **B,** Solar scintigraphic image of the same foot in **A.** There is increased uptake of the radiopharmaceutical in the palmar processes of the distal phalanx.

conformation. The response to treatment often is slow, and during the convalescent period work on hard ground should be avoided. Horses with chronic lameness with a markedly distorted hoof capsule have a guarded prognosis. Some horses may benefit from removal of the shoes for 6 months and unrestricted exercise at pasture, provided that the hoof wall quality permits this approach and the ground is not excessively hard.

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DISEASE OF THE CARTILAGES OF THE FOOT

• Sue J. Dyson

ANATOMY

The cartilages of the foot, also referred to as the *collateral cartilages of the distal phalanx*, *ungular (ungual) cartilages,* and *lateral cartilages,* originate as hyaline-type cartilage and become fibrocartilage in adults.[1] They attach to the proximal border of the palmar processes of the distal phalanx. The size and shape vary, as does the degree of ossification. The cartilages have axial extensions and they seem to provide an internal support structure for the back part of the foot.[2] An extensive network of venovenous anastomoses is present within the cartilages. Marked differences in the thickness and tissue composition exist in the front and hind feet. The cartilages of the foot tend to be thicker in forelimbs than in hindlimbs, perhaps reflecting the greater weight-bearing capacity of the forelimbs.[2] The digital cushion has more fibrous or cartilaginous tissue in forelimbs than in matched hind feet, which have more adipose and elastic tissues. There may also be breed differences. An extensive and complex relationship exists between the cartilages of the foot and the digital cushion. The combined role is thought to be energy dissipation, which is dependent on hemodynamic flow.[2]

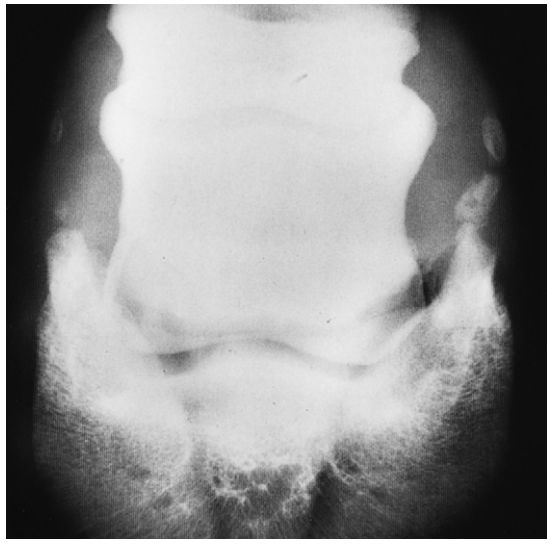

Fig. 34-14 Dorsoproximal-palmaromedial oblique view of a foot of a mature riding horse. Lateral is to the right. Several centers of ossification of both the cartilages of the foot are present, more obvious laterally.

The cartilages of the foot are joined to adjacent structures by a variety of ligaments that vary in size and definition. The chondrocompedal ligament attaches the palmaroproximal aspect of the cartilage with the proximal phalanx, with a medial branch to the distal phalanx.[1] The chondrocoronal ligament connects the dorsal part of the cartilage with the middle phalanx.[1] Ossification of the lateral cartilage of the foot often is more extensive than the medial cartilage.[3] The term *sidebone* has been used to describe extensive ossification of one or both cartilages of the foot. Ossification may occur from more than one center of ossification. Radiolucent lines between separate centers of ossification may persist throughout life (Fig. 34-14).[4] The degree of ossification is greater in mature horses than in horses younger than 2 years of age.[3] Ossification tends to be more extensive in heavier breeds of horse than in lighter-weight horses. Among Finnhorses, ossification was more common and extensive in mares compared with male horses.[3] A correlation also was noted between the extent of ossification of the cartilages of the foot and the height of the heels of the foot.

CLINICAL SIGNS AND DIAGNOSIS

The proximal aspect of the cartilages of the foot can be palpated proximal to the coronary band. However, palpation is an unreliable indicator of both the size of the cartilages and

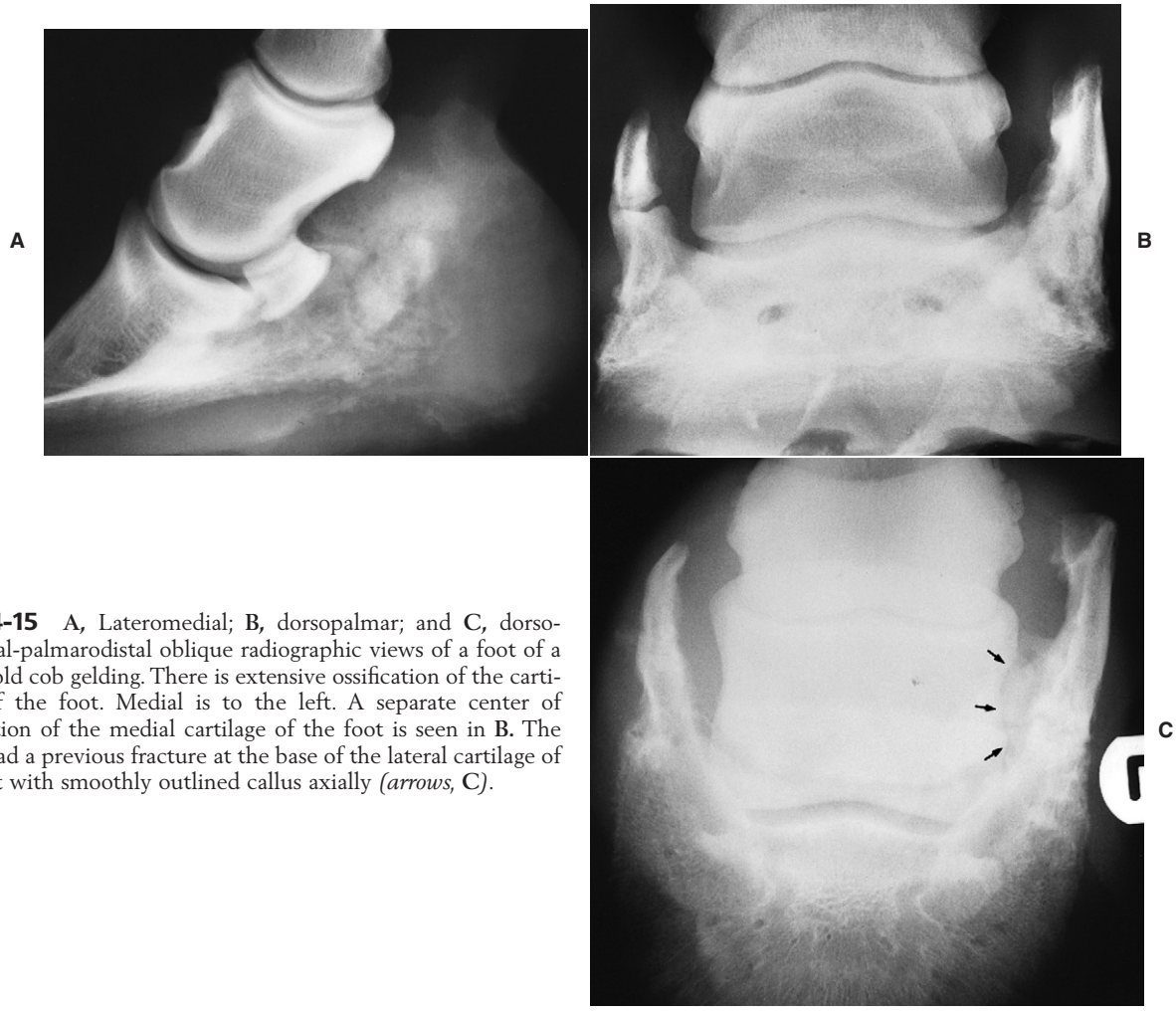

Fig. 34-15 **A,** Lateromedial; **B,** dorsopalmar; and **C,** dorsoproximal-palmarodistal oblique radiographic views of a foot of a 7-year-old cob gelding. There is extensive ossification of the cartilages of the foot. Medial is to the left. A separate center of ossification of the medial cartilage of the foot is seen in **B.** The horse had a previous fracture at the base of the lateral cartilage of the foot with smoothly outlined callus axially *(arrows,* **C***)*.

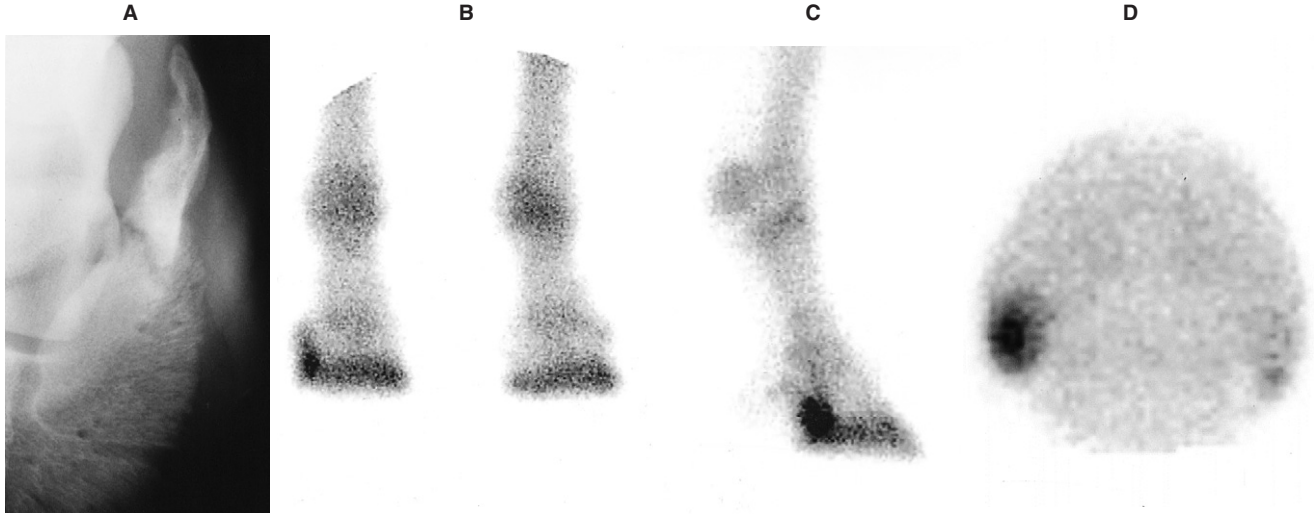

Fig. 34-16 **A,** Dorsoproximal-palmarodistal oblique radiographic view of the right front foot of a Warmblood mare with severe lameness that partially improved with perineural analgesia of the palmar nerves at the base of the proximal sesamoid bones. The lateral cartilage of the foot is fractured. **B,** Dorsal (the right front foot is on the left); **C,** lateral; and **D,** solar scintigraphic images of the same foot in **A.** There is increased uptake of the radiopharmaceutical in the lateral cartilage of the right front foot.

the degree of ossification. The degree of ossification can be established only by radiography. Lateromedial and weight-bearing dorsopalmar views are the most useful (Fig. 34-15).

Ossification of the cartilages of the foot rarely is directly associated with lameness. If ossification extends to the level of the proximal interphalangeal joint, it may be associated with a short-striding gait.

Occasionally a fracture of an ossified cartilage of the foot may be associated with lameness, but care should be taken not to confuse a radiolucent line between separate centers of ossification as a fracture. Nuclear scintigraphy may facilitate a definitive diagnosis (Fig. 34-16).

Desmitis of the chondrocompedal ligament and chondrocoronal ligaments occasionally has been recognized in association with lameness.[5] The clinical features have not been documented.

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CHAPTER • 35

Laminitis

PATHOPHYSIOLOGY OF LAMINITIS

• Christopher C. Pollitt

A tough, flexible, connective tissue suspensory apparatus suspends the distal phalanx to the inside of the inner hoof wall. The surface of the inner hoof wall is folded into leaf-like lamellae (laminae) to increase the surface area of attachment between hoof and bone. A horse has laminitis when this attachment apparatus fails. Without the distal phalanx properly attached to the inside of the hoof, the weight of the horse and the forces of locomotion drive the bone down into the hoof capsule. Important arteries and veins are sheared and crushed, and the corium of the coronet and sole is damaged. Unrelenting pain in the feet and a characteristic lameness occur.

In acute laminitis the tissue suspending the distal phalanx from the inner hoof wall fails at the junction between the connective tissue of the dermis or corium (the bone side) and the basal cell layer of the epidermal lamellae (the hoof side). This junction, the basement membrane zone, appears to be the weak link in an otherwise robust and reliable structure. In acute laminitis, wholesale epidermal cell detachment from and lysis of the lamellar basement membrane occurs,[1,2] leading to failure of the lamellar anatomy and ultimately failure of the suspensory attachment between hoof and distal phalanx. A good correlation exists between the severity, as seen with the microscope (histopathology), and the degree of lameness (using the Obel [1948] grading system[3]) shown by the horse.[1] When a horse first starts to show laminitic pain, the anatomy of the hoof wall lamellae is being destroyed. The higher the lameness grade, the more severe the microscopic damage. Any activity that places stress on an already weakened lamellar attachment apparatus (such as forced exercise) causes further damage and is contraindicated. The use of nerve blocks to eliminate pain encourages locomotion and does more damage.

PATHOPHYSIOLOGY

The disintegration of the lamellar attachment apparatus is initiated during the developmental phase of laminitis. A normally tightly controlled, metabolic process is targeted, causing lamellar-specific pathological conditions during the laminitis developmental period. The continually proliferating hoof wall[4] moves past the stationary distal phalanx by a process of controlled enzymatic remodeling. New evidence suggests that the lamellar attachment apparatus is destroyed when these enzymes are activated out of control. These enzymes, metalloproteinase-2 and metalloproteinase-9 (MMP-2 and MMP-9), destroy key components of the lamellar attachment apparatus.[5]

MMP enzyme is a normal constituent of lamellar cells, and it responds to the stresses and strains of equine life and to constant growth. Sufficient MMP is manufactured locally to release epidermal cell to cell and cell to basement membrane attachment as required, maintaining the correct shape and orientation of the lamellae. From time to time injury to the basement membrane requires its lysis and reconstruction. The controlled release of MMPs and specific MMP inhibitors keeps the remodeling process in equilibrium. In horses without laminitis or those with mild disease the hoof lamellae slowly migrate distally past the stationary basement membrane that is firmly attached to the connective tissue covering the upper surface of the distal phalanx.[6]

HISTOLOGICAL GRADING

The sequences of microscopic events that initiate laminitis follow a consistent pattern, and the stages of histological laminitis can be identified by the degree of severity of these changes. Making the lamellar basement membrane clearly visible is important and requires staining lamellar tissues with periodic acid–Schiff (PAS) and periodic acid–silver methanamine

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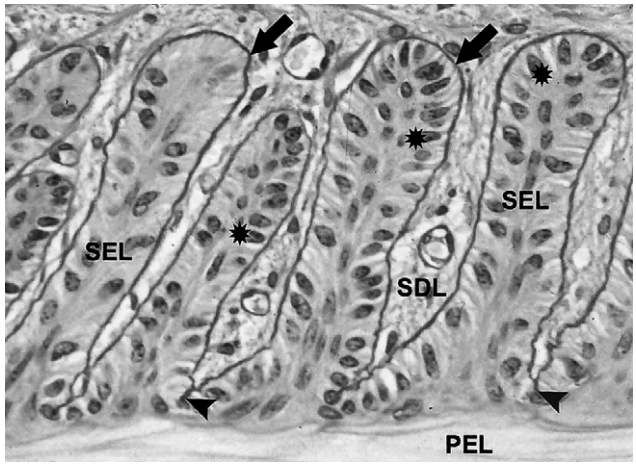

Fig. 35-1 Micrograph of normal hoof lamellae stained to highlight the basement membrane. The basement membrane (indicated by *arrows*) of each secondary epidermal lamella (*SEL*) shows as a dark line closely adhering to the secondary epidermal lamellae basal cells. Between the bases of each secondary epidermal lamellae the basement membrane penetrates deeply (*arrowheads*) and is close to the anuclear, keratinized, primary epidermal lamella (*PEL*). The secondary epidermal lamellae tips are rounded (club-shaped). The basal cell nuclei are oval (*stars*) and positioned away from the basement membrane at the apex of each cell. The long axis of each basal cell nucleus is at right angles to the long axis of the secondary epidermal lamellae. The secondary dermal lamellae (*SDL*) are filled with connective tissue even at their tips, between the secondary epidermal lamellae bases. These parameters of hoof lamellar anatomy form the basis of the histological grading system of laminitis histopathology. (Periodic acid–Schiff stain.)

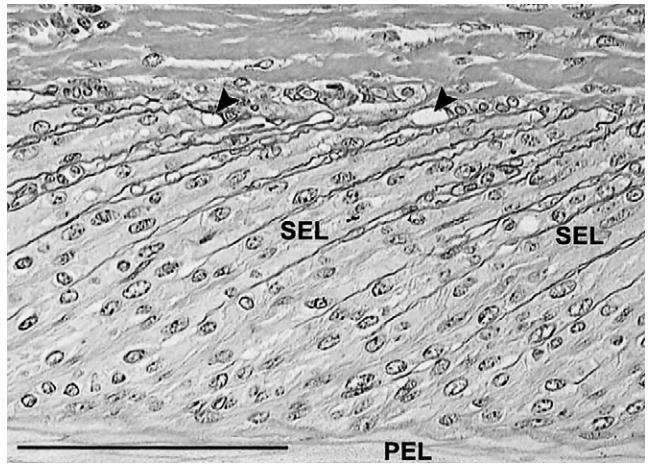

Fig. 35-2 Micrograph showing hoof lamellar tissues (stained to highlight the basement membrane) with histological grade 2 laminitis. The basement membrane is stained dark magenta. At the tips of the now pointed secondary epidermal lamellae (*SEL*s) the basement membrane has continued to lift from the underlying basal cells to form empty, teat-shaped caps (*arrowheads*). The basement membrane has disappeared from the crypts between the secondary epidermal lamellae bases. The lamellar basement membrane is no longer close to the primary epidermal lamella (*PEL*). A reduced amount of connective tissue exists between the secondary epidermal lamellae. Bar is 10 µm. (Periodic acid–Schiff stain.)

(PASM) stains or with immunohistochemical methods using basement membrane–specific antibodies.[1,2]

The normal anatomical characteristics assessed before allocating a laminitis grade to a section of lamellar hoof tissue are as follows:

- The tips of the secondary epidermal lamellae are always rounded (club-shaped) and never tapered or pointed.
- The basal cell nuclei are oval, with the long axis of the oval at a right angle to the long axis of the secondary epidermal lamellae.

These parameters can be satisfactorily assessed using routine hematoxylin and eosin staining of sections. The basement membrane penetrates deeply into the crypts between the secondary epidermal lamellae and outlines the wafer-thin, but connective tissue–filled, secondary dermal lamellae. The basement membrane tightly adheres to the basal cells of each secondary epidermal lamellae. The PAS and PASM stains show this best (Fig. 35-1).

Grade 1

As the developmental phase ends and the acute phase begins, loss of shape and normal arrangement of the lamellar basal and parabasal cells occurs. The basal cell nuclei become rounded instead of oval and take an abnormal position in the cytoplasm of the cell. The secondary epidermal lamellae become stretched, long, and thin, with tapering instead of club-shaped tips.

While the former takes place, the basement membrane of the secondary epidermal lamellae loses its attachment to the basal cells and is first noticeable at the tips of the secondary epidermal lamellae where teat-shaped bubbles of loose basement membrane form. The tissues should be stained with PAS or PASM stains to show this best.

Examination of laminitis tissues with the electron microscope confirms lysis and separation of the lamellar basement membrane. Importantly, the greater magnification shows widespread loss of basal cell hemidesmosomes and contraction of the basal cell cytoskeleton away from the inner cell surface. Electron microscopy shows why the basement membrane separates from the feet of the basal cells. The anchoring filaments that bridge the gap between the hemidesmosome and the lamina densa of the basement membrane are no longer present.[7]

Grade 2

Because the basement membrane is no longer tethered to the basal cells, it slips farther away with each cycle of weight bearing by the horse. The lamellar basement membrane begins to disappear initially at the bases of the secondary epidermal lamellae (Fig. 35-2). The basement membrane retracts from between the secondary epidermal lamellae and takes with it the connective tissue. The basement membrane–free epidermal cells appear not to be undergoing necrosis, at least initially, and clump together to form amorphous, basement membrane–free masses on either side of the lamellar axis.

Grade 3

In laminitis the worst-case scenario is a rapid and total basement membrane separation from all the epidermal lamellae. Sheets of basement membrane peel away to form aggregations of loose, isolated basement membrane in the connective tissue adjoining the lamellae. The epidermal lamellar cells are left as isolated columns with no connection whatsoever with the dermal connective tissue. The lamellar tips slide away from the basement membrane connective tissue attachments, at first microscopically, but as the degree of separation increases, the distance between hoof and distal phalanx becomes measurable in millimeters (Fig. 35-3). This manifests clinically as the sinker. Because the basement membrane is the key struc-

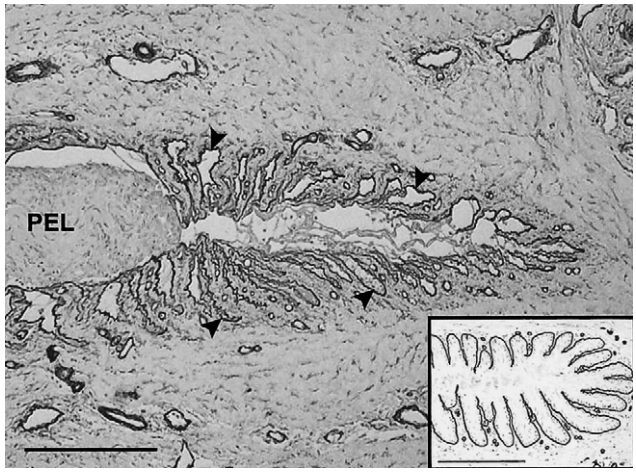

Fig. 35-3 Grade 3 histological laminitis (immunostain). The basement membrane of a lamellar tip is highlighted by type IV collagen immunostaining. The tip of the primary epidermal lamella (*PEL*) has completely detached from its basement membrane. The primary epidermal lamella basal cells are now an unattached, amorphous mass. Collapsed tubes of basement membrane, now empty of epidermal cells, are still attached to connective tissue (*arrowheads*). The primary epidermal lamella has already moved 0.03 mm from its dermal compartment and soon the distance will be measured, using a tape measure, on a radiograph. The inset shows a normal lamellar tip, immunostained the same way. Type IV collagen immunostain was used. Bars are 10 μm.

ture bridging the epidermis of the hoof to the connective tissue of the distal phalanx, wholesale loss and disorganization of the lamellar basement membrane follows and inexorably leads to the failure of hoof anatomy so characteristic of the chronic stage of laminitis.

The laminitis process also affects the lamellar capillaries. As the basement membrane and the connective tissue between the secondary epidermal lamellae disappear, so do the capillaries. They become obliterated, compressed against the edges of the primary dermal lamellae. Without capillaries in the lamellar circulation, blood bypasses the capillary bed through dilated arteriovenous shunts[8] and dramatically changes the nature of the foot circulation. A bounding pulse is detectable by finger palpation of the digital arteries. Furthermore, epidermal cell necrosis, intravascular coagulation, and edema are not present in sections made from tissues in the early stages of laminitis. The vessels in the primary dermal lamella, even the smallest, are predominantly open, without evidence of microvascular thrombi. The gross anatomical appearance of freshly dissected laminitis tissue is dryness. Sometimes the lamellae just peel apart.

ENZYMATIC THEORY OF LAMINITIS

The enzymatic theory of laminitis, based on the triggering of lamellar MMP activity, challenges the alternative view that laminitis develops because the flow of blood is impeded to cause ischemic necrosis of epidermal lamellae.[9]

How do the trigger factors of laminitis reach the lamellae? Strong evidence from three independent international laboratories now exists that the foot circulation during the developmental phase of laminitis is vasodilated.[10-12] Laminitis does not occur if the foot is in a state of vasoconstriction during the developmental phase, suggesting that the trigger factors only cause laminitis if they reach the lamellar tissues at a high enough concentration and over a long enough time.

What are the laminitis trigger factors? Because the carbohydrate overload model of laminitis is characterized by endotoxin production, a reasonable presumption seems to be that endotoxemia plays a key role in initiating laminitis. Tumor necrosis factor, along with other cytokines such as interleukin, is expressed by mononuclear phagocytes within minutes of exposure to endotoxin. The cytokine cascade originating from an inflamed leaky bowel is responsible for most of the pathological effects of endotoxemia. However, laminitis has never been triggered by the experimental administration of endotoxin into the bloodstream or the peritoneal cavity, and the actual trigger factors of laminitis remain unidentified. What appears certain in the light of recent research is that the lamellar disintegration of laminitis is mediated by the inappropriate release of excess activated MMP. But what triggers MMP release and activation?

NATURAL TRIGGER FACTORS

Many of the proposed laminitis trigger factors have been investigated using an in vitro laminitis model. Small explants of tissue were taken from the inner hoof wall of normal, freshly killed abattoir horses. After incubation for 48 hours in tissue culture medium, along with the laminitis trigger factor under investigation, each explant was subjected to tension. The force required to separate epidermal from dermal lamellae was recorded. When dermal-epidermal lamellar separation occurs readily (as occurs in field cases of laminitis), the tissue is deemed to have developed in vitro laminitis. Lamellar explants can be cultured for up to 7 days in normal medium and no lamellar separation occurs. Separating normal lamellar explants is virtually impossible. Normal explants can supports a mass of 900 g. When a non-physiological chemical, known to activate metalloproteinases (the organo-mercurial compound aminophenylmercuric acetate) was added to the explant tissue culture medium, the explants separate when only a small separating mass was applied.

Explant tissues were fixed in formalin and examined histologically for evidence of separation. Histological sections showed a clear zone of complete separation between the basement membrane and the basal cells of the epidermal lamellae. This separation is characteristic of in vitro laminitis and resembles the basement membrane lesion of natural in vivo laminitis. The presence or absence of MMP activation in the explant tissue culture medium is detected zymographically using gelatin polyacrylamide electrophoresis. Analysis of the culture medium from normal hoof explants shows that explants produce two MMPs (gelatinases) of molecular weight 92 and 72 kd. A small amount of the active forms of the MMP-2 is also present in normal horses. Incubation of normal hoof explants with aminophenylmercuric acetate results in the activation of MMP-9 and MMP-2.

Laminitis and Metalloproteinase Activity

Lamellar explants from horses with acute laminitis, cultured in medium under the same conditions, contained not only increased amounts of inactive MMP-2 and MMP-9 but also greatly increased amounts of activated MMPs. Furthermore the genes controlling hoof lamellar MMP-2 and MMP-9 activity are significantly up-regulated in tissues affected by acute laminitis. Together this provides firm circumstantial evidence that MMP activation is a pivotal event in the development of laminitis.

Metalloproteinase Inhibitors

The activity of tissue MMPs recently has been shown to correlate strongly with the degree of malignancy and invasiveness of lethal human tumors, such as malignant melanoma, breast

cancer, and colon cancer. Research in this field has generated a wide range of chemical agents capable of inhibiting MMP activity in vitro and in vivo. BB-94 (Batimastat; British Biotech, Oxford, England) blocks the activity of the laminitis MMPs in vitro and has the potential to be a useful tool in preventing and managing acute laminitis. Whether MMP inhibitors can prevent or ameliorate conditions in horses with naturally occurring laminitis has yet to be established.

Trigger Factors of Bacterial Origin

Equine lamellae, cultured in vitro, have tested resistant to virtually all known cytokines, tissue factors, and prostaglandins. Gram-negative bacterial endotoxin, extract of black walnut (*Juglans nigra*), and even anaerobic culture conditions fail to induce lamellar separation or significant MMP activation. Some notable exceptions occur, however. Factors present in the supernatant of cultures of *Streptococcus bovis* isolated from the equine cecum activate equine hoof MMP-2 and cause lamellar separation.

During grain overload *S. bovis* is the principal microorganism responsible for the rapid fermentation of carbohydrate to lactic acid in the equine hindgut.[13] In the presence of virtually unlimited substrate, the population of *S. bovis* increases exponentially. Possibly *S. bovis* and a number of other bacteria produce laminitis trigger factors[14] that cross the mucosal barrier of the hindgut and enter the circulation. These factors attach to basement membranes throughout the body, but they only cause significant damage to lamellar basement membranes because of the uniquely equine involvement in weight bearing.

Changes in Glucose Metabolism

Hoof lamellar explants kept in tissue culture medium consume glucose and readily separate when glucose is absent from the culture medium.[15] Apparently, after removal from the horse, hoof tissue relies on glucose for maintenance of adhesion between the epidermal lamellae and the basement membrane. The addition of compounds to the culture medium that block lamellar energy production from glucose also cause lamellar separation (in vitro laminitis). Ultrastructural studies of hoof lamellae undergoing glucose starvation in vitro show a specific lesion of the hemidesmosomes that bridge the dermo-epidermal junction. The longer the duration of glucose starvation, the more hemidesmosomes disappear, until lamellar separation occurs.[8] The glucose starvation lesion is quite unlike that induced by MMP activation.

The major feature of the acute metabolic stress that attends acute fulminating diseases such as colitis, metritis, and carbohydrate alimentary overload is the reduction of glucose consumption in many peripheral tissues. The purpose of this change is to maintain glucose, and therefore energy supplies, to the vital organs (heart, lung, and brain) at the expense of other tissues. Metabolic stress is regulated by the hormones insulin, glucagon, cortisol, and adrenaline. Insulin promotes glucose utilization; glucagon promotes glucose production, especially by the liver. Cortisol and adrenaline promote glucose production from other substrates and reduce glucose consumption in peripheral tissues such as skin and hoof. In vitro studies show that hoof tissues rely greatly on glucose, and the rapidly decreasing local concentrations of glucose that accompany severe physiological stress could mimic the in vitro situation and cause lamellar separation. Other epithelia may be similarly weakened, but gross separation would be manifest most readily in the hoof because of the large mechanical forces generated by weight bearing.

Evidence exists that the metabolic changes described previously do occur as a consequence of carbohydrate overload in horses developing laminitis. During the development of laminitis, an increase in blood cortisol, consistent with a metabolic change to conserve glucose, occurs. Failure of the lamellar basement membrane may be occurring via two mechanisms: lamellar MMP activation (because of arrival of bacterial laminitis trigger factors) and hemidesmosome dissolution (because of lamellar basal cell glucose uptake).

Equine Cushing's Disease

Equine Cushing's disease, caused by a pituitary adenoma, results in excess production of adrenocorticotropic hormone, producing a hormone imbalance that creates a tissue resistance to insulin, disturbing glucose uptake in hoof lamellae and causing an insidious, relentlessly developing chronic laminitis. Affected horses and ponies often have higher than normal serum concentrations of glucose, cortisone, and insulin. This laminitis is usually refractory to treatment. However, promising results have been obtained after administration of pergolide mesylate (Permax, Eli Lilly), a drug registered for human use.[16] Doses in the range of 1 to 2 mg/horse/day have been recommended. The drug reduces production in the pituitary gland of the adrenocorticotropic hormone, which controls cortisol production in the adrenal gland. With cortisol under control, insulin responsiveness in hoof lamellae returns and the laminitis stabilizes.

Exogenous Corticosteroids and Laminitis

Clinically an association has been observed between systemic or intra-articular administration of corticosteroids, including triamcinolone and methylprednisolone acetate, and the development of laminitis in otherwise apparently healthy horses.[17] However, a cause and effect has yet to be proved. Intra-muscular administration of 60 mg of triamcinolone daily for 7 days failed to induce laminitis in healthy horses. Nonetheless, clinicians must be aware of the risks associated with exogenously administered corticosteroids.

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DIAGNOSIS OF LAMINITIS

• Sue J. Dyson

Laminitis is characterized by an acute onset lameness of variable severity involving one or more feet. Most often both front feet are affected, with or without the hind feet, but unilateral laminitis does occur, usually caused by excessive load bearing because of severe contralateral limb lameness. Occasionally the hind feet are affected, without involvement of the front feet. The horse may be extremely reluctant to move and, if persuaded to move, tends to land heel first, with a short, pottery gait, with the hindlimbs placed unusually far underneath the body. Lameness may be accentuated as the horse turns. Lameness is worse on hard ground than on soft ground. Although many horses show severe lameness, in those with milder lameness the lameness may be less typical, although suggestive of foot pain.

Usually, but not invariably, a significant increase in digital pulse amplitudes occurs, and in the acute phase the affected feet may be hot. Pressure or percussion applied to the feet, especially in the toe region, usually causes pain, but if the horn is excessively hard, the horse may not react. Careful palpation around the coronary band may reveal an unusual depression associated with sinking of the distal phalanx. An area of unusual softness may herald infection tracking proximally in association with laminitis complicated by sub-mural abscessation.

Clinical signs are usually diagnostic, except in less severely affected horses or those with involvement of only the hind feet, when the characteristics of the lameness may be suggestive of foot pain, but not necessarily pathognomonic for laminitis. The response to perineural analgesia varies and is not associated necessarily with the degree of pain and lameness. Apparent desensitization of the foot with palmar (abaxial sesamoid) nerve blocks may have absolutely no effect on the lameness in some horses, although in others some improvement may occur.

Because laminitis frequently develops secondarily to a primary disease process, it is critical to evaluate the entire horse and to identify any predisposing factors that require treatment, such as endotoxemia, septic metritis, or Cushing's disease.

Radiographic examination is critical for establishing a treatment protocol and prognosis. Although rotation of the distal phalanx often can be managed successfully, sinking warrants an extremely guarded prognosis. Lateromedial views help to determine whether the condition is acute or an exacerbation of a more chronic problem. Abnormal thickness of the dorsal hoof wall, with or without modeling of the toe of the distal phalanx, implies previous disease. Lateromedial views are also important to establish the baseline position of the distal phalanx within the hoof capsule. Dorsopalmar projections may be useful for assessing mediolateral balance in horses with chronic, unstable laminitis. If the foot is grossly misshapen, trimming it first is preferable; otherwise, a false impression of severe rotation of the distal phalanx, which merely reflects the abnormal hoof wall growth, may occur.

Standardizing the procedure (positioning, film-focus distance, and exposure factors) is essential to make meaningful comparisons between examinations. To enhance soft tissue detail, use soft exposures or radiographic film with a large grey scale. A grid is usually unnecessary. A horizontal x-ray beam should be perpendicular to the sagittal plane of the digit, centered between the toe and the heel, about 2 cm distal to the coronary band. Radiodense markers are placed on the dorsal aspect of the hoof wall and on the sole at the apex of the frog. The marker on the dorsal hoof wall should extend from the coronary band distally. This helps to establish the orientation of the distal phalanx and its position relative to the coronary band. Radiographic abnormalities include rotation or sinking of the distal phalanx, increased thickness of the dorsal hoof wall, and radiolucent lines in the dorsal hoof wall, reflecting necrotic tissue or gas caused by infection or hoof wall separation.

MEDICAL THERAPY OF LAMINITIS

• Christoper C. Pollitt

Laminitis often develops because disease is occurring in a body compartment other than the foot. Thus it is of paramount importance that the primary disease is treated urgently and effectively. If the duration and severity of the primary disease can be reduced by intensive therapy, a strong chance exists that the severity of lamellar pathology also may be reduced, thus improving the prognosis for the horse. Severe laminitis is sometimes the outcome despite the best of current therapy, but some horses can exhibit early, mild clinical signs of laminitis yet recover with no long-term ill effects.

Currently no therapeutic regime is able to arrest or block the triggering of laminitis, and damage is what influences the outcome.[1] An effective laminitis preventive may emerge when the mechanism behind the disintegration of the anatomy of the hoof wall lamellae is fully understood. The discovery that a class of enzymes appears to be involved in the lamellar failure of laminitis[2] offers hope that proteinase inhibitor therapy, specifically targeted at hoof wall matrix metalloproteinases, may arrest laminitis development. Horses that were able to keep the feet cool during the laminitis developmental period did not develop laminitis,[3] suggesting that cryotherapy may be a useful preventive, first-aid measure. Bacteria are a source of laminitis trigger factors,[4] so effective anti-microbial therapy is a treatment priority.

Horses diagnosed with toxemia during enteritis, colitis, strangulating colic, pleuropneumonia, retained placenta, infectious metritis, and grain overload are at high risk of developing laminitis, and ideally, medical therapy and mechanical support for the distal phalanx should be initiated before the clinical signs of foot pain appear. Addressing laminitis as soon as it appears in a sick horse should always be regarded as an emergency procedure. Even then treatment may be too late. Anti-endotoxin hyperimmune serum (Polymmune J, Veterinary Dynamics Inc., Templeton, CA) should be included in the intravenous fluid therapy for horses with, or at risk of developing, endotoxemia.

Non-steroidal anti-inflammatory drugs (NSAIDs) are required to reduce inflammation and foot pain. Flunixin

meglumine (0.25 mg/kg tid intravenously [IV] or 1.1 mg/kg bid IV) has a proven anti-endotoxin effect by reducing prostaglandin production via cyclooxygenase inhibition[5] and is valuable. Horses receiving flunixin meglumine that subsequently are given endotoxin had significantly lower blood prostaglandin and lactate concentrations and reduced clinical signs than control horses. However, the effectiveness of flunixin or any NSAID as an anti-laminitis agent has never been tested. A proven cause-and-effect link between endotoxemia and laminitis has never been established.[6]

Phenylbutazone (4.4 mg/kg IV or orally every 12 hours) appears to be a potent NSAID for the control of foot pain and is popular with most clinicians. Phenylbutazone and flunixin meglumine at the lower dose rate can be used concurrently; the former to control severe foot pain and the latter to control the effects of endotoxemia. Intravenously administered ketoprofen (2.2 mg/kg bid) can be used interchangeably with flunixin. Horses with acute laminitis usually require NSAID therapy for at least 2 weeks, and because of its low cost, phenylbutazone (2.2 mg/kg) is the best choice for maintenance therapy. Care must be taken not to over-dose small ponies.

However, NSAIDs have been administered to horses during experimental induction of laminitis without altering the outcome; laminitis still occurred.[7] In vitro studies of laminitis indicate that MMP activation is slightly potentiated when NSAIDs are present in the culture system. When the laminitis process is triggered, virtually nothing by way of drug therapy stops its progress. The administration of phenylbutazone during the developmental and acute stages reduces foot pain and creates a more comfortable-looking horse, but the disease continues unabated. This creates an ethical dilemma: balancing the need to alleviate pain and suffering against the realization that most of what is administered is only palliative. When NSAIDs are in use, the patient should be confined to a stall with deep bedding. Exercise is contraindicated while the horse is under the influence of analgesics.

CRYOTHERAPY

Distal limb cryotherapy to cool the foot and induce digital vasoconstriction may be a useful preventive strategy in the developmental phase of laminitis. Cold-induced, digital vasoconstriction during the laminitis developmental phase may limit exposure to circulating trigger factors and reduce the impact on lamellar anatomy.[3] Cryotherapy may bestow additional protection by slowing the kinetics of lamellar enzyme activity below a threshold that causes damage. Nuclear scintigraphic studies showed cold therapy significantly decreased perfusion of the soft tissues of a horse's foot within 30 minutes of the application of external cold.[8] Limited anecdotal evidence from practicing veterinarians suggests that cryotherapy does halt the development of the disease. An ice water footbath was used successfully to treat a pony in the developmental stage of acute laminitis after experimental cecal catheterization.[9] Cryotherapy for laminitis requires maintaining the limbs from the proximal metacarpal region and distad in a slurry of crushed ice or a circulating cold water (4° C) apparatus continuously for 24 hours or even longer if the period of septic shock, pyrexia, and digital vasodilation persists. Cryotherapy is safe, well tolerated, and economical. Unlike people, horses do not find cold therapy noxious. We have kept the forelimbs of normal horses in a slurry of ice and water continuously for 2 days, with no immediate or long-term ill effect. Initial experiments support the effectiveness of cryotherapy for halting laminitis onset.[7] Keeping a horse with its feet in ice boots requires an extraordinary amount of time and dedication but can be done and, if laminitis is prevented, is worth the effort.

DIGITAL BLOOD FLOW THERAPY

Vasodilatory therapy and hot water footbaths during the developmental phase of laminitis are contraindicated. Drugs with vasodilator action such as isoxsuprine hydrochloride, acepromazine, and glyceryl trinitrate (applied as patches to the pastern) may be beneficial after lamellar damage has occurred, when healing is required, but should be administered with caution during the developmental phase. However, neither orally administered isoxsuprine nor pentoxifylline produced significant improvement in digital or lamellar blood flow when tested under controlled conditions using ultrasound and laser Doppler flowmetry.[6,10,11] Intravenously administered acepromazine maleate, long held to be effective at increasing lamellar microcirculatory blood flow, had no significant effect when tested by laser flowmetry.[12] Glyceryl trinitrate tested in the same way, against a black walnut extract laminitis induction model, had no effect on lamellar blood flow.[13] The continued veterinary use of these drugs in the therapy of laminitis is questionable.

Exercise of an intensity that raises core temperature and local analgesia of the palmar or plantar nerves result in hoof wall heating (and by implication vasodilation) and are contraindicated during the developmental stage of laminitis. Local analgesia of the foot to reduce foot pain and encourage the horse to walk may result in greater lamellar damage than in a rested, confined horse. Forced exercise of any horse with acute laminitis is strongly contraindicated.

FREE RADICAL SCAVENGERS

Dimethylsulfoxide (DMSO) may be given intravenously for its free radical scavenging and anti-inflammatory effects. DMSO (90% solution) mixed with polyionic solutions and 5% dextrose is best administered slowly at about 8 L per hour. The concentration of DMSO must remain below 20% to avoid the risk of intravascular hemolysis. However, despite the potential of DMSO, its promise as an effective laminitis therapy has not been fulfilled. No evidence exists that ischemia, reperfusion injury, and the generation of free radicals are involved in the pathogenesis of most horses with laminitis.

RECOMMENDED TREATMENT STRATEGY

The list of pharmaceuticals that have been administered to horses with laminitis is long, and apart from the NSAIDs none have achieved particular prominence. I recommend aggressive treatment of the primary disease, using fluids and electrolytes, antibiotics and NSAIDs, and uterine lavage for horses with infectious metritis or retained placenta.

The administration of 4 L of mineral oil four times a day may be beneficial in laminitis developing from grain overload. Mineral oil has a laxative effect and is said to block the absorption of toxins in the large intestine.[14] Activated charcoal is an effective adsorbent of a range of toxins and may be useful in cases of grain overload if administered promptly. In Australia, doses of 1 to 5 g/kg/day have been used to treat plant toxicoses in large animals. The higher dose is indicated if a large quantity of grain has been consumed. However, activated charcoal has not been tested against alimentary laminitis, so its true effectiveness is unknown. The application of cold therapy to the front feet, strict confinement to a stall with a deep bedding of sand or shavings and mechanical support for the distal phalanx are also recommended.

Laminitis developing secondary to Cushing's disease is usually refractory to treatment unless treated using pergolide mesylate (1 to 2 mg/horse/day) to reduce adrenocorticotropic

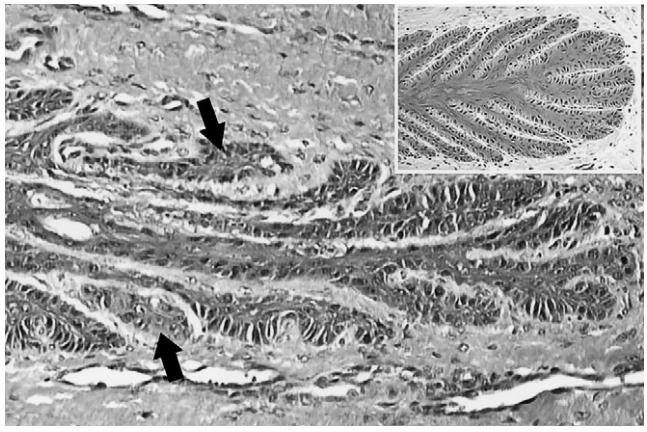

Fig. 35-4 Histopathological section of chronic laminitis in the lamellar tip. Despite an apparent clinical recovery by the horse, biopsies of hoof lamellae show that the normal architecture has not been restored. The lamellar tips are distorted and weak, and some secondary epidermal lamellae (*arrows*) are not connected to the primary epidermal lamella. The *inset* shows the tip of a normal lamella. (Hematoxylin-eosin stain.)

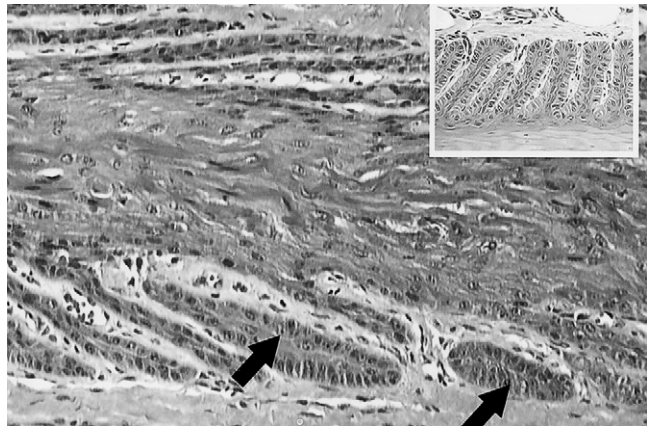

Fig. 35-5 Histopathological section of chronic laminitis in the mid-lamellar region. Some secondary epidermal lamellae (*arrows*) are not connected to the primary epidermal lamella. Laminitis has compromised the surface area of attachment, leaving the horse prone to recurrent bouts of the disease. The *inset* shows normal lamellae. (Hematoxylin-eosin stain.)

hormone production in the pituitary gland and thus reducing adrenal cortisol production.[15] This restores insulin responsiveness, permitting more normal glucose use by the hoof lamellae.

Some horses that show the clinical signs of acute laminitis recover completely if treated promptly using a combination of rational medical therapy and mechanical support. However, horses recovering from even the mildest laminitis should be allowed to rest and observed closely. If no radiographic evidence of palmar displacement of the distal phalanx within the hoof capsule is apparent, and the digital pulse amplitude is not palpably exaggerated 48 hours after treatment has ceased, the horse can be returned to its usual function with caution.

If radiographs do show displacement of the distal phalanx, then the prognosis must be more guarded. Horses with a mild increase in the distance between the distal phalanx and the dorsal hoof wall, with or without rotation of the distal phalanx, often make an apparent recovery and remain sound indefinitely. However, horses with marginally greater displacement and rotation of the distal phalanx make only partial recoveries and often have a history of intermittent lameness, especially after exercise. Histopathological examination of the hoof lamellae of partially recovered horses showed a reduction in the number of secondary epidermal lamellae. Many of the secondary epidermal lamellae had distorted, abnormal shapes even several years after the initial episode of laminitis. Some secondary epidermal lamellae become isolated from the attachment to the primary epidermal lamella and exist as isolated, unattached islands adrift in the lamellar connective tissue (Figs. 35-4 and 35-5). If the surface area of the lamellae of the inner hoof wall is reduced after laminitis, the effectiveness of the lamellar distal phalanx suspensory mechanism must also be reduced. Horses developing laminitis associated with significant initial lamellar destruction, as manifest by radiographic displacement of the distal phalanx, appear never to make a complete anatomical recovery and are prone to recurrent episodes of foot pain.

Ultimately the prognosis is directly proportional to the severity and extent of lamellar pathological condition. Horses with more than 15° of rotation, accompanied by downward displacement of the distal phalanx within the hoof capsule, within 4 to 6 weeks of the initial episode of laminitis have a poor prognosis. Prolapse of the distal phalanx through an already necrotic sole, accompanied by sub-solar and sub-lamellar infection, usually occurs. Pus discharges from the coronet and the heels. Osteitis and lysis of the distal margin of the distal phalanx develop. Such horses require months of expensive supportive care and surgery and although occasionally a horse does make a surprisingly good recovery, most suffer months of crippling foot pain and recumbency and eventually require euthanasia on humane grounds.

The road to recovery after a serious bout of laminitis is a rocky one. The extent of lamellar pathology lies hidden beneath the hoof wall, and we can only guess at what is really going on. Radiographs and the initial degree of pain expressed by the horse, often masked by analgesics such as phenylbutazone, give valuable clues. However, relentless sinking of the distal phalanx in the hoof capsule and involvement of all four feet make recovery unlikely.

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HOOF CARE OF A LAMINITIC HORSE

• Frank A. Nickels

Hoof care in all phases of laminitis is extremely important. The goal is to reduce stress to the damaged lamellae by minimizing the distracting forces affecting the displacement of the distal phalanx (rotation or sinking). These distracting forces are the weight of the horse, the constant pull of the deep digital flexor tendon (DDFT), and the leverage on the toe of the hoof capsule. Nothing can be done about the horse's weight, but absolute stall rest can alter the increased stress associated with movement. Some of the weight of the horse can be partially redirected away from the hoof wall in the resting horse by removing the shoes. Recruiting other parts of the ground surface of the hoof to bear weight can also reduce stress on the hoof wall with the use of particular bedding or sole pads. Elevating the heel reduces the stress on the DDFT. In horses with rapid rotation or penetration through the corium of the sole, a tenotomy of the DDFT is recommended to eliminate any pull on the distal phalanx (see page 333). Finally, beveling (unweighting) the toe with a rasp helps increase ease of breakover, thereby decreasing the stress created by the leverage of the toe. Baseline radiography (see page 329) is important to establish the relationship between the distal phalanx and the hoof capsule. I place both front feet on two 8 × 13 × 18-cm wooden blocks in the surface of which are radiodense markers. I also place small spherical radiodense markers at the dorsal aspect of the coronary band and at the apex of the frog and a linear marker on the dorsal aspect of the hoof capsule (Fig. 35-6).

In the developmental phase the goal is to recognize the possibility of disease and be prepared if the disease occurs. The goal is to prevent and/or minimize distal displacement of the distal phalanx. This can be partially accomplished by transferring weight from the wall to the solar surface of the foot thereby reducing the pull of the DDFT. If the horse is shod, the shoes should be removed carefully by rasping off each nail clinch, then removing each nail separately with a creased nail puller. This helps to reduce mechanical trauma, pain, and discomfort associated with shoe removal. Then the toe of the hoof capsule should be unweighted by beveling it with a rasp back to a point 2.5 to 4 cm in front of the true apex of the frog depending on the size of the hoof. In a normal horse the frog can serve as a reference point for the tip of the distal phalanx, which is located approximately 1.9 to 2.5 cm dorsal to the apex of the frog.

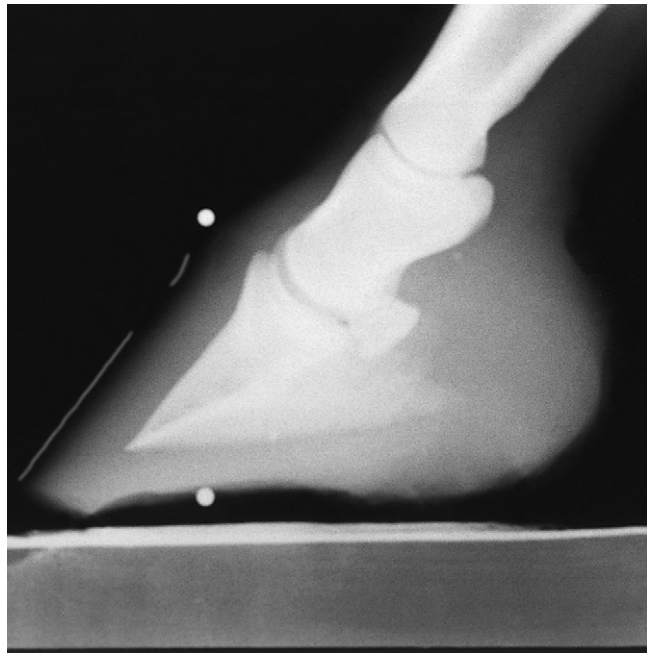

Fig. 35-6 Lateromedial radiographic view of a normal foot. There are circular radiodense markers at the coronary band and at the apex of the frog and a radiodense tape on the dorsal aspect of the hoof capsule. Radiodense wire is also embedded in the surface of the wooden block on which the foot is resting.

Soft bedding materials, such as sand or damp peat, provide good support and relief, but they may be difficult and expensive to maintain. Alternatively, frog or sole supports can be used. Frog supports using Lily pads (Kentucky Blacksmith, Shelbyville, KY) or roll gauze taped to the frog were commonly used in the past, but they are being replaced by pads that adapt to the entire solar surface, providing more support and comfort to the entire foot.

High-density foam is now one of the most practical and economical methods for providing solar support. Sheets of 5-cm extruded foam house insulation (Styrofoam scoreboard, Dow Chemical, Midland, MI) can be purchased at home centers or building supply companies. For foam application the clinician stands the horse on small precut rectangular blocks of the foam insulation, then cuts and shapes it to the foot using a serrated bread knife. The clinician then attaches the foot-formed pad with duct tape (Fig. 35-7). In the acute phase, two layers of foam are used to provide more comfort and to elevate the heel. One layer is applied and then removed within 24 hours when it becomes compressed. Ideally, this layer will compress evenly to approximately 2 cm. If the foam compresses more unevenly at the toe than the heel, the clinician should remove the excess from the ventral surface with a rasp to make it uniform. Removing the dorsal portion of this pad reduces the pressure over the most painful area of the sole. This trimmed pad and another foam layer are then attached to the foot with duct tape to raise the heel and provide more cushion to an area of the foot that can tolerate weight bearing (Fig. 35-8). The foam pads should be replaced every 4 to 5 days or as needed. A commercial wedge pad system (Redden Ultimate, Kentucky Blacksmith) using rubber impression material (Advanced Cushion Support, Kentucky Blacksmith) is available and works well in horses that are not getting adequate support from the foam insulation. These

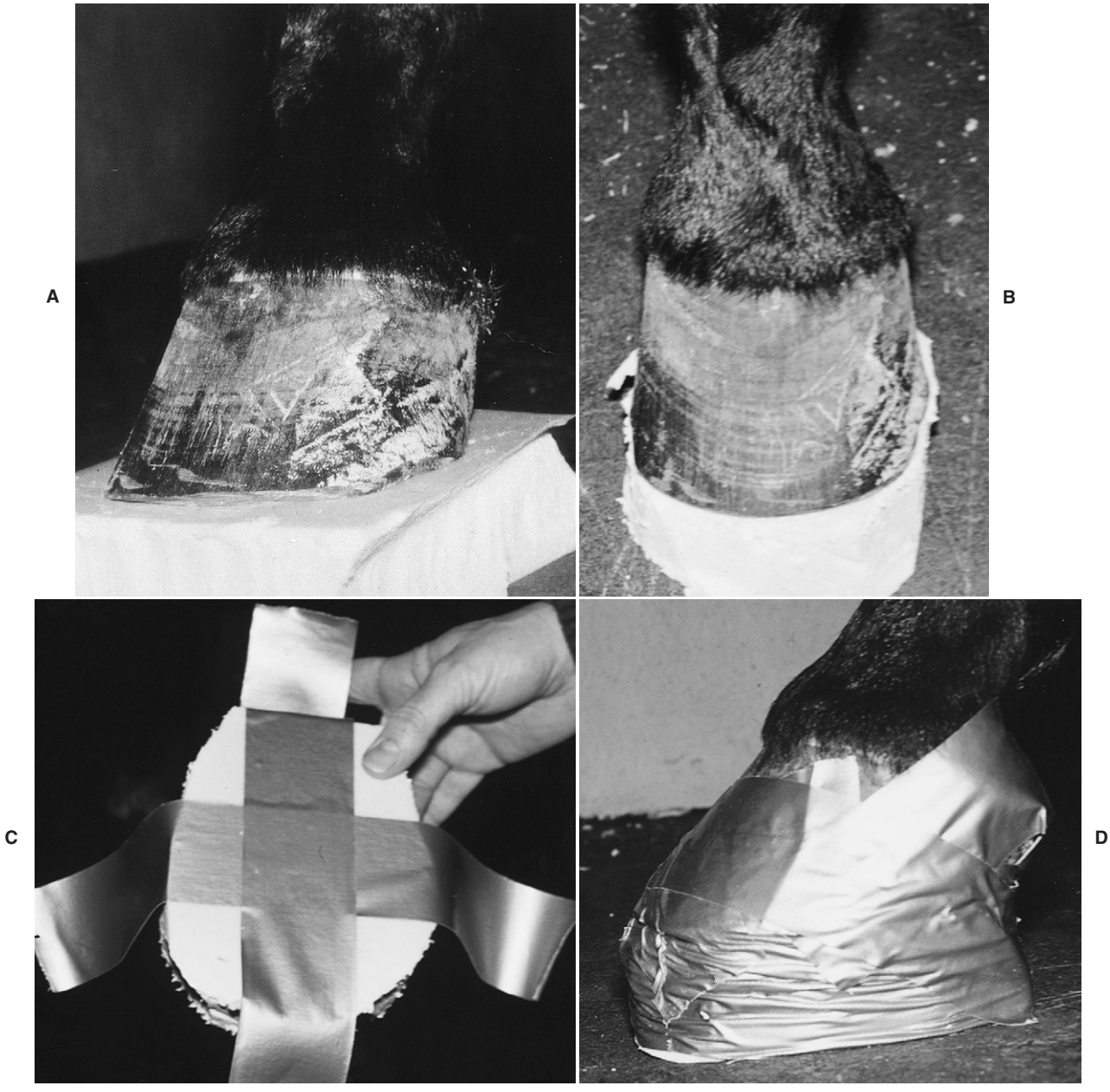

Fig. 35-7 The use of styrofoam insulation for making sole pads. With the horse standing on a precut block of foam **(A),** the foam pad is trimmed to the shape of the foot **(B),** duct tape applied to the bottom of the pad to help adhere it to the hoof **(C),** and the pad is completely secured to the hoof with duct tape **(D).**

commercial pads are taped or glued to the foot (Fig. 35-9). The system combines a cuff with two attached 5° wedge pads with a built-in dorsal to palmar breakover. The bottom wedge also has beveled edges for ease of medial to lateral breakover; this bottom wedge can be removed separately. The guidelines for the removal of the wedge pads are the absence of medication and pain for at least 10 days, indicating the treatment is progressing successfully. If these parameters remain unchanged, the first wedge should be removed. If no change occurs during the next 10 days, the last wedge is removed. It is important to remember that clinical improvement occurs before lamellar healing. Therefore if the lamellae become unduly stressed, re-injury to the lamellae will occur.

The goal of hoof care in the chronic phase is to minimize further rotation of the distal phalanx. The following principles should be followed when preparing the hoof and selecting a therapeutic horseshoe:

1. The palmar aspect of the hoof should be supported using all structures (sole, bars, and frog, including the central and lateral sulci).
2. The sole should be protected by not removing any portion of the sole.
3. The hoof wall should be trimmed from the quarters to the heel only, lowering the heel to reestablish the normal relationship between the solar surface of the distal phalanx and the sole.

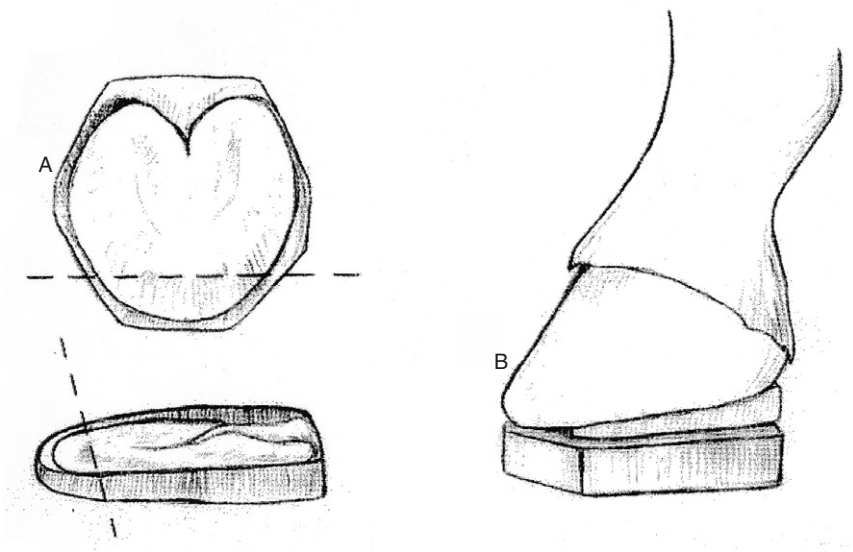

Fig. 35-8 Trimming *(dashed line)* of the previously used compressed foam pad **(A)** to be added to a new layer of foam **(B)** before attaching the two to the hoof with duct tape.

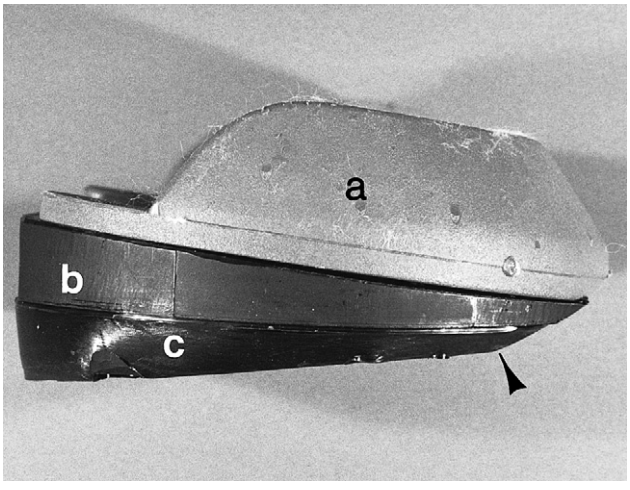

Fig. 35-9 The Redden Ultimate wedge system can be attached to the hoof by a cuff *(a)* with special glue or adhesive tape. There are two 5° wedge pads *(b* and *c)* attached to each other with screws. The bottom pad *(c)* has round edges for ease of lateral to medial breakover. The *arrow* depicts how the system eases the dorsal to palmar breakover.

4. The dorsal aspect of the hoof capsule should be protected by bringing the point of breakover back to between 2.5 and 4 cm (depending on hoof size) from the apex of the frog.
5. In some horses the heel should be raised to reduce tension of the DDFT on the distal phalanx.

Specific hoof care management in this phase depends on whether this chronic laminitic condition is stable or unstable. In horses maintaining a stable condition, trimming alone may be all that is necessary, carefully following the stipulated principles. Generally, the traditional therapeutic horseshoes (heart bar shoe, reverse shoe with a wedge, reverse shoe with frog support, and the egg bar shoe with a treatment plate) advocated for chronic laminitis are beneficial if the condition is reasonably stable (see page 338).

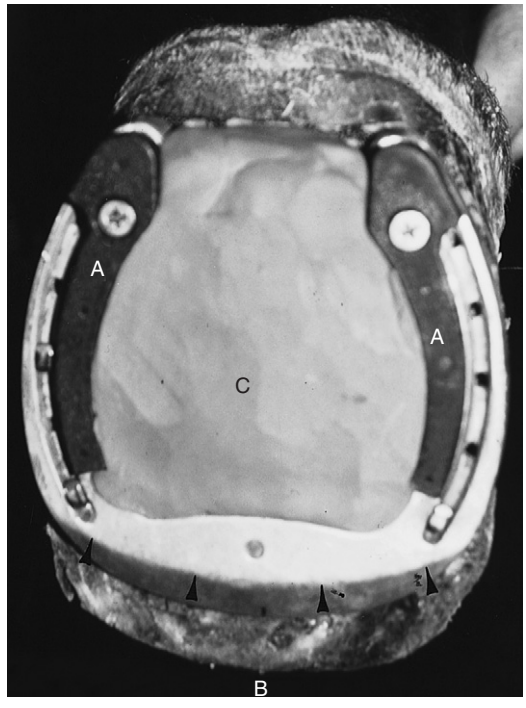

Fig. 35-10 The Ovnicek aluminum shoe (EDDS, Penrose, CO). has interchangeable plastic rails *(A)*. It is specially machined to pull the point of breakover *(small arrows)* back. The cushion elastomer is set back from the toe *(B)*. Sole support is provided with cushion elastomer *(C)*.

Horses with instability of the distal phalanx require a custom-made (International Podiatry Center, Versailles, KY) or commercially available (Equine Digit Support System, Penrose, CO) aluminum rail shoe (Fig. 35-10) to provide the additional required foot support. The new design of these shoes automatically pulls the breakover back, raises the heel, and provides palmar support. Ideally, the breakover should be close to the tip of the distal phalanx. A cushion elastomer (Advanced Cushion Support) is used between the palmar

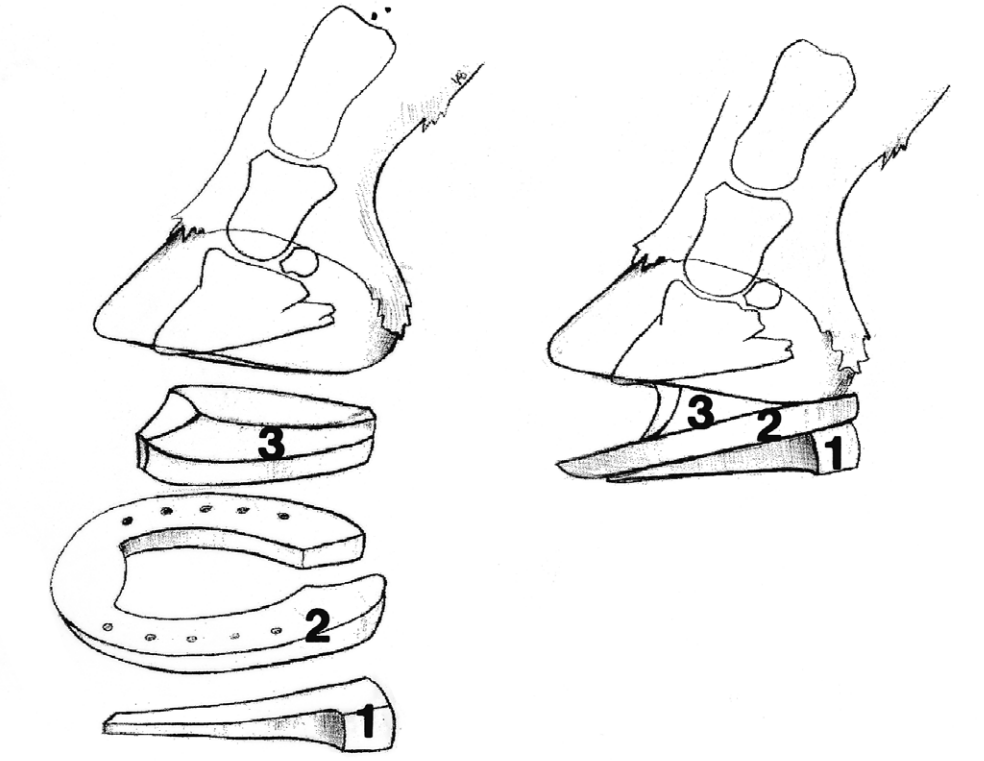

Fig. 35-11 This drawing illustrates the special shoeing technique for a horse with insufficient heel to remove for reorientating the foot and when there is penetration of the sole by the corium. The components of this technique are *(1)* plastic rails, *(2)* the EDSS shoe, and *(3)* cushion elastomer. The cushion elastomer is used as a spacer to keep the contact surface of the shoe parallel to the solar surface of the distal phalanx. The shoe is attached with a hoof acrylic and fiberglass cloth because it cannot be attached to the hoof conventionally. Tenotomy of the deep digital flexor tendon is mandatory with this technique.

structures of the sole and the shoe to provide support of the entire foot.

Horses with severe rotation, penetration of the sole, or both conditions usually have insufficient heel to re-establish the normal relationship between the solar surface of the distal phalanx and sole by removing the heel alone. A special technique is required. The cushion elastomer is used to provide sole support and act as a spacer between the weight-bearing surface of the hoof wall and the therapeutic shoe (Fig. 35-11). These shoes cannot be attached to the hoof conventionally; they are fastened to the hoof using an acrylic adhesive and fiberglass cloth (Equilox and Equilox Composite Cloth, Equilox International, Pine Island, MN). Routine clinical and radiographic evaluations provide useful information to determine the patient's progress every 5 to 6 weeks when the shoes are reset.

In horses with unstable feet exhibiting progressive rotation or penetration of the corium through the sole, a deep digital flexor tenotomy is also recommended to reduce the active forces influencing separation of the lamellae and of the distal phalanx on the sole. This procedure relieves pain, prevents or minimizes rotation, and allows reestablishment of the normal relationship between the solar surface of the distal phalanx and the sole. Severing the DDFT causes partial luxation and hyperextension of the distal interphalangeal joint, as well as overloading of the superficial digital flexor tendon. This abnormal joint position may cause palmar heel pain and usually results in osteoarthritis of the distal interphalangeal joint. Post-operative management of these horses is very important to prevent or minimize these complications. Shoeing is crucial to protect the distal interphalangeal joint and reduce the load on the tendons. The horse is shod with a slight flexion of the distal interphalangeal joint with a shoe in which the heels are raised and extend caudally approximately 1 to 1.5 cm (Fig. 35-12).

Treatment of horses with complicated laminitis may take months or years and requires perseverance and team commitment. It can be an emotional roller coaster for the owner who must be made aware of these realities.

DEEP DIGITAL FLEXOR TENOTOMY FOR MANAGING LAMINITIS

• Robert J. Hunt

Transection of the DDFT has been advocated in treatment of horses with chronic refractory laminitis.[1-4] The procedure initially appeared to have merit when used in horses with acute laminitis, but the long-term results were poor because of the severe and unstable nature of the laminitis.[2] The procedure attenuated pain, but most horses had ongoing displacement of the distal phalanx and suffered recurrent abscessation, and ultimately humane destruction was necessary.

The rationale for surgery is based on the biomechanical forces in the foot, which include the attachments of the dermal lamellae between the distal phalanx and the hoof wall, the downward vertical load through the bony column of the limb, the proximal palmar traction of the DDFT on the distal phalanx, the proximal pull of the common digital extensor

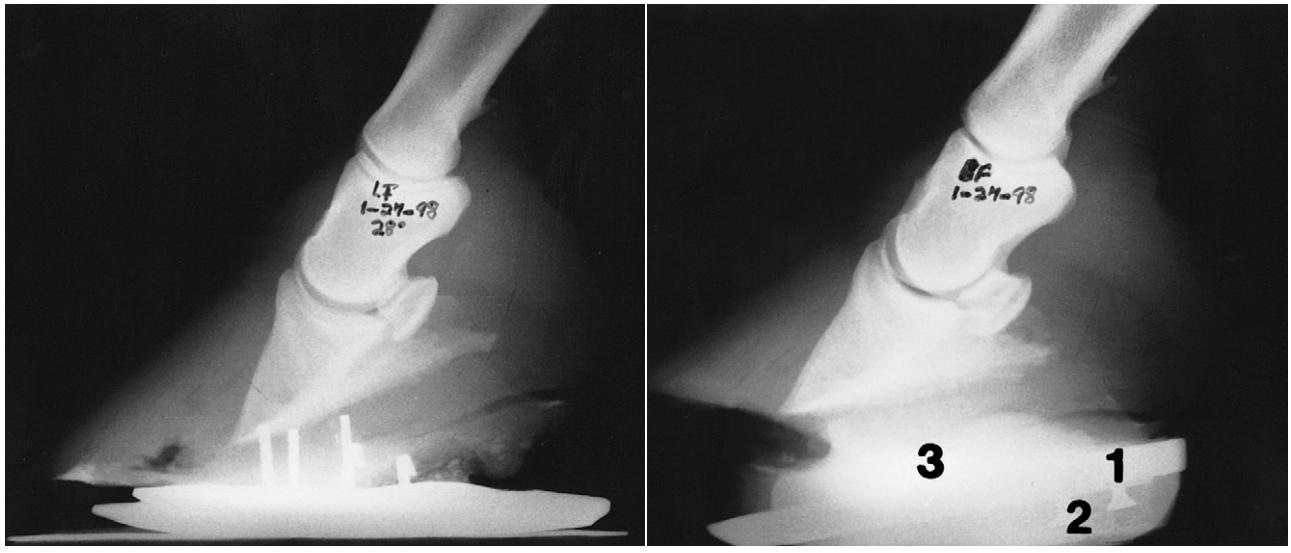

Fig. 35-12 Lateromedial radiographic views of a foot of a horse with severe rotation of the distal phalanx, which clinically had penetrated the corium of the sole. **A,** The horse was shod unsuccessfully with a reverse shoe with frog support. **B,** Note the phalangeal axis and the relationship between the solar surface of the distal phalanx and the contact surface of the shoe immediately after tenotomy of the deep digital flexor tendon had been performed and the special shoeing technique. The foot is shod with the distal interphalangeal joint in slight flexion to prevent partial luxation of the joint. *1*, The EDSS shoe; *2*, plastic rails; *3*, cushion elastomer.

tendon, and the digital cushion. The balance of these forces maintains a functional unit.

During a non–weight-bearing state, essentially no force is imposed on the digit by the DDFT. When the limb is placed under load, the DDFT serves as a tension band and places shearing forces on the dorsal lamellae of the digit from its proximal palmar pull. The predominant force on the digit is the vertical load produced by the weight of the horse. Neither of these forces is necessarily detrimental under normal conditions; however, with compromise of the dorsal lamellae during laminitis, these forces result in distraction of the distal phalanx from the hoof capsule. The type and extent of displacement of the distal phalanx is determined by the degree of laminar damage and the load placed on the foot.

The rationale for tenotomy is to reduce the proximal palmar pull of the DDFT on the distal phalanx and therefore decrease the shearing forces on the lamellae of the dorsal aspect of the foot. Transecting the tendon also permits lowering the heels if indicated to allow more normal alignment of the distal phalanx. The procedure generally is considered a salvage procedure for breeding animals or horses not intended for athletic endeavors, although some horses have returned to successful athletic careers. Generally the degree of damage associated with the laminitis, rather than the surgery, limits future soundness.

No clear-cut guidelines dictate when and if surgery should be performed. In general, surgery is reserved for horses with chronic recurring laminitis that suffer from periodic hoof abscesses and show varying degrees of distal phalangeal rotation, with minimal to no sinking, and that produce a reasonable dorsal hoof wall.

Tenotomy may be repeated with recurrence of clinical signs or with the development of a metacarpophalangeal joint flexural deformity subsequent to a mid-pastern tenotomy. Performing repeat tenotomy is easier after a mid-metacarpal tenotomy, although repeat tenotomy is also possible to perform following a mid-pastern tenotomy. I have performed tenotomy of the DDFT in the same horse five times in a 4-year period. Clinical improvement was seen after each procedure, and subjectively the effects were not as beneficial or prolonged as in the previous tenotomy. This may have been associated with progressive deterioration of the digit associated with laminitis or from adhesions of the DDFT preventing tension relief after transection. This experience is similar to that in three horses with repeat tenotomies (one to three times).

Deep digital flexor tenotomy is performed in the mid-metacarpal or mid-pastern region (Figs. 35-13 and 35-14). When performed in the mid-metacarpal region, the tenotomy may be done with the horse standing, under local analgesia, or under general anesthesia. An audible pop often occurs once transection is complete. A 1- to 2-cm gap in the transected tendon ends is immediately palpable. The stab incisions do not require closure, but a firm pressure bandage is applied.

Mid-pastern tenotomy is almost always performed with the horse recumbent, with the limb flexed to relax the DDFT (see Fig. 35-14). Performing the procedure in a standing horse is possible if general anesthesia is prohibited, such as in a late-term pregnant mare. A higher likelihood of contamination of the surgery site that involves the digital flexor tendon sheath (DFTS) is possible with the horse standing; therefore extreme caution should be taken.

It is mandatory to apply an extended and optimally elevated heel shoe in horses with mid-pastern tenotomy before or during surgery. The shoe should be worn for a minimum of 8 to 10 weeks to prevent subluxation of the distal interphalangeal joint. Although this shoe is not mandatory for all horses with mid-metacarpal tenotomy, if clinical evidence exists of distal interphalangeal joint subluxation or no improvement clinically after surgery, an extended and elevated heel shoe should be applied. After tenotomy a horse should be confined to a restricted stall-size area. Bandages should be changed every 2 to 4 days for 8 to 10 weeks to minimize swelling.

The amount of tension release is greater after mid-pastern tenotomy because of the anatomical approximation to the

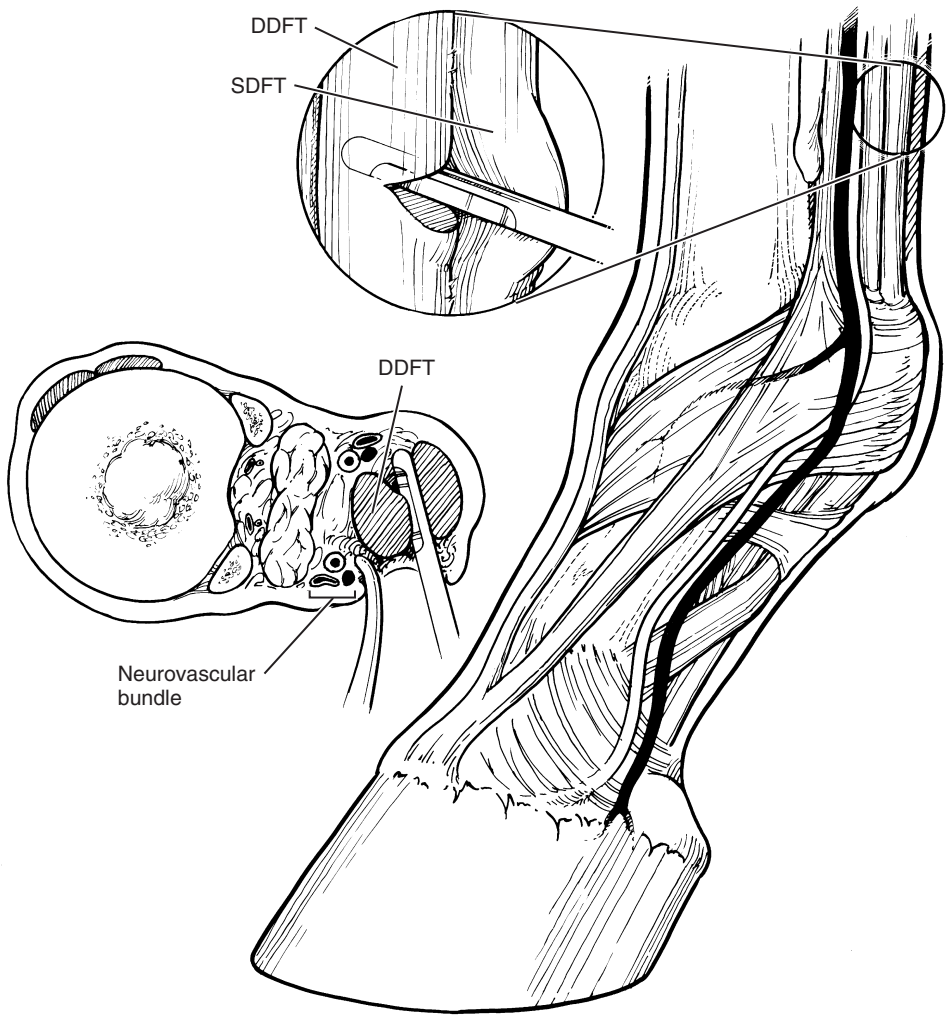

Fig. 35-13 Deep digital flexor tenotomy can be performed in the metacarpal region in a standing horse or with the horse under general anesthesia. *DDFT,* Deep digital flexor tendon; *SDFT,* superficial digital flexor tendon.

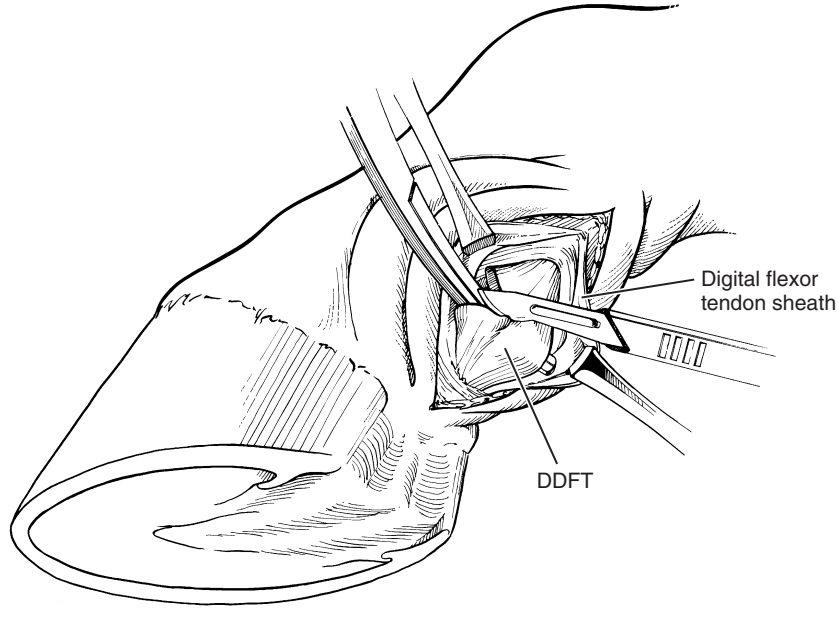

Fig. 35-14 Deep digital flexor tenotomy can be performed in the mid-pastern region with the horse under general anesthesia. *DDFT,* Deep digital flexor tendon.

insertion of the DDFT on the distal phalanx and unrestricted separation within the DFTS. The tendon may separate 6 to 10 cm after transection in this region. In contrast, separation after mid-metacarpal tenotomy is limited by peritenon attachment to the subcutaneous tissue.

Clinical improvement is generally seen within 2 to 3 days of surgery. In general, clinical effects after tenotomy appear to be beneficial for several months. A flexural deformity of the metacarpophalangeal joint may develop from chronic pain, resulting in unweighting of the limb and contracture of scar tissue at the tenotomy site. Chronic pain may result from overloading the superficial digital flexor tendon before healing, osteoarthritis of the distal interphalangeal joint, or chronic infections of the digit. Chronic infection may be a major complication, especially if infectious osteitis of the distal phalanx develops. Systemic antibiotics, local debridement, topical anti-microbials, and bandaging are important in resolving infection. Treatment of such complicated conditions may take many months to resolve at great emotional and financial cost, and everybody involved must be aware of this.

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OTHER MANAGEMENT ASPECTS OF LAMINITIS

• **Sue J. Dyson and Mike W. Ross**

Medical management of laminitis (see page 329) and some aspects of hoof care focused on a horse with progressive rotation of the distal phalanx (see page 332) are discussed elsewhere. This section discusses some additional aspects of management, routine care, monitoring of the patient, and prevention of recurrence. It must be acknowledged that in equine practice, ideal management is not always possible and compromises must sometimes be made to adapt to the prevailing circumstances.

In a horse with acute laminitis, some clinicians favor removal of the shoes, assuming that the horse can stand on soft bedding. However, in our experience removal of the shoes often results in increased discomfort, and in the acute stages of laminitis, we prefer to leave the shoes in place. Alternatively, the normal shoes may be replaced with glue-on heart bar shoes, foam pads, frog pads, or custom-made boots,[1] which seem to enhance comfort. Not all affected horses develop rotation or sinking of the distal phalanx; however, even if baseline radiographs reveal no evidence of rotation, each horse should be monitored carefully. If there is any evidence of increased discomfort, the feet should be re-examined radiographically. The client must be instructed carefully about observing the horse's stance and mobility and the amount of time spent lying down and advised that veterinary advice should be sought if there is any evidence of deterioration.

Ideally a horse with acute laminitis should be restricted to stable rest, but this is not always possible, especially for a child's pony that lives outdoors throughout the year. In these circumstances a small area of the field should be fenced off using electric fencing. This area should be tightly mown and the grass clippings should be removed. Spreading a thick layer of wood shavings over the entire surface may be beneficial.

Many horses and ponies that develop laminitis are grossly overweight, and reduction of body weight is crucial for successful management. Owners are often reluctant to admit that the horse is overweight and find it difficult to restrict the diet adequately. It is helpful to weigh the horse or make an estimate of body weight using a height-specific weigh tape[2] and then set a target for the expected weight loss with monitoring at intervals. Strict instructions concerning dietary management should be made, stipulating the amounts of food, preferably by measured weights, that should be fed. If a pony is living outdoors and cannot be stabled, then a strip grazing system should be established; each strip should be finely mown before the animal has access to it.

In horses with chronic laminitis, even if rotation of the distal phalanx occurred previously, the position of the distal phalanx may actually be stable. The heel tends to grow more quickly than the toe, and regular trimming of the heel and shortening the toe are imperative to try to re-establish correct alignment of the distal phalanx within the hoof capsule. The heart bar shoe recruits part of the frog to share load bearing and if correctly fitted, shifts weight-bearing load away from the toe, thus sparing the dorsal structures of the foot. This approach, combined with removal of the lamellar wedge, often results in improved alignment of the hoof wall and distal phalanx and improved comfort. Correct positioning of the frog support plate is critical so that even, mild pressure is applied over the length of the frog. Reverse shoes or W-shoes (open toe shoe with a heart bar) nailed on with one or two nails both medially and laterally, or glued or taped in place, may provide comfort in some horses. Often what works in one horse may not work in another and a different strategy must be used.

The term *derotation* of the distal phalanx has become popular; however, we believe that this is an inappropriate term. Although lowering the heel changes the angle of the distal phalanx with respect to the middle phalanx, it cannot acutely change the position of the distal phalanx relative to the hoof wall. The position of the distal phalanx within the hoof capsule often appears improved after removal of excessive toe.

It is critical for the farrier and veterinarian to work together closely. If the veterinarian believes that the farrier is not trimming the foot sufficiently aggressively, obtaining lateromedial radiographs may be helpful to demonstrate excess toe, heel, or both. Lateromedial radiographs are also useful for demonstrating excessive separation of the dorsal hoof wall or accumulation of fluid and when limited dorsal hoof wall resection may be indicated. If sinking of one or more of the distal phalanges is evident on radiographs, the owner must be advised that the prognosis for recovery is extremely guarded.

If pain is not adequately controlled by systemic analgesics, daily patient monitoring, preferably by a veterinarian, is crucial to identify signs of impending penetration of the sole, such as increased softening of the sole dorsal to the apex of the frog and red discoloration. The coronary bands of all feet should be assessed carefully; depression at the coronary band may herald sinking of the distal phalanx. Increased patient discomfort may be associated with a sub-solar abscess that requires establishment of drainage. A horse that has developed or is at risk of development of mechanically induced laminitis because of a painful condition in the contralateral limb must be monitored particularly vigilantly. In these horses, subtle evidence of mechanical laminitis is easily missed and usually occurs between 4 and 6 weeks after injury to the contralateral limb. However, in some horses, usually racehorses, mechanical

laminitis occurs early, even within the first few days, and is inexplicable. Often the first sign of contralateral mechanical laminitis is increased weight bearing on the injured limb, or treading. Increased weight bearing on the injured limb may be a sign of improved comfort, but the clinician should investigate the contralateral limb carefully for the development of mechanical laminitis.

Careful assessment of digital pulse amplitudes and foot temperature may be useful for detecting the development of laminitis or deterioration of pre-existing laminitis. In older horses in which laminitis cannot be stabilized, consideration should always be given to the existence of primary Cushing's disease.

It is critical that clients be kept well informed of the likely outcome and are well appraised of the long convalescent periods that often are required after substantial rotation of the distal phalanx. They must be warned that unexpected relapses may occur despite steady progress. Nonetheless, it is important to recognize that with early aggressive treatment some horses can make a complete recovery to athletic function despite marked rotation of one or more distal phalanges. The prognosis is generally more favorable in ponies than in horses. Clients must be advised that continued long-term dietary management may be crucial to prevention of recurrent laminitis.

The prognosis in heavy breeds of horse with sinking of the distal phalanges is generally hopeless. These horses seem particularly prone to the development of laminitis secondary to retention of the placenta, and although with aggressive management they can be salvaged until the foal is weaned, longer-term treatment is invariably unsuccessful.

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CHAPTER • 36

The Proximal and Middle Phalanges and Proximal Interphalangeal Joint

Alan J. Ruggles

ANATOMICAL CONSIDERATIONS

The term *pastern* originated from the shackle that was secured below the metacarpophalangeal or metatarsophalangeal joints to tether a horse to the pasture.[1] The proximal interphalangeal joint or pastern joint is a diarthrodial joint, which is formed from the distal aspect of the proximal phalanx and the proximal aspect of the middle phalanx. The pastern region is bounded dorsally by the common digital extensor tendon and on the palmar/plantar border by the distal sesamoidean ligaments, digital flexor tendons, digital flexor tendon sheath (DFTS), and the proximal and distal digital annular ligaments. The superficial digital flexor tendon (SDFT) inserts on the distal palmar/plantar aspect of the proximal phalanx and the proximal palmar/plantar aspect of the middle phalanx. The straight distal sesamoidean ligament arises from the base of the proximal sesamoid bones (PSBs) and extends distally to insert on the proximal palmar/plantar aspect of the middle phalanx. The oblique (middle) distal sesamoidean ligaments arise from the base of the PSBs and attach to the triangular region on the palmar/plantar region of the middle portion of the proximal phalanx. The medial and lateral collateral ligaments of the proximal interphalangeal joint provide support in the sagittal plane and attach to the collateral tubercles of the distal proximal phalanx and proximal palmar/plantar aspect of the middle phalanx. The paired smaller abaxial and axial ligaments are located just palmar/plantar to the collateral ligaments. The neurovascular bundle of the digit runs just abaxial to the deep digital flexor tendon on the medial and lateral sides.

The palmar/plantar eminence of the middle phalanx extends proximal to the horizontal axis of the joint surface and blends proximally with a fibrocartilaginous cap at the most proximal extent of the middle phalanx. In the adult horse this is dense, and it is important to engage this portion of the bone during internal fixation of fractures or arthrodesis procedures.

The proximity of the pastern region to the ground, the paucity of soft tissue coverage over the dorsum and sides, and the important soft tissue structures on the palmar/plantar surfaces make it especially vulnerable to external trauma. Wounds in this region may involve the DFTS and warrant careful investigation. Angular deformities from growth disturbances at the pastern are uncommon. Radiographic closure of the proximal physis of the proximal and middle phalanges occurs by 6 to 9 months of age, but functional closure is earlier, usually by 8 weeks of age.[2] Radiographic closure of the distal physis of the proximal phalanx occurs by 1 month of age. Radiographic closure of the distal physis of the middle phalanx occurs by the time of birth.

LAMENESS EXAMINATION

Physical derangements usually are obvious because of minimal soft tissue coverage of the pastern. Phalangeal fractures usually are associated with soft tissue swelling and focal pain. Effusion of the DFTS is typical of penetrating wounds of the sheath, damage to the enclosed flexor tendons, or injury of the sesamoidean ligaments located dorsad. In horses with osteoarthritis, new bone formation on the dorsomedial and dorsolateral aspects of the proximal interphalangeal joint often occurs. Angular deformity of the digit often is present in horses with complete fractures of the proximal or middle phalanx. Effusion in the metacarpophalangeal or metatarsophalangeal joint is common in horses with fractures of the proximal articular surface of the proximal phalanx.

Lameness from the pastern region ranges from severe to subtle depending on the injury. Generally, disorders involving the proximal interphalangeal joint or DFTS cause obvious lameness, but lameness from early osteoarthritis of the proximal interphalangeal joint may be mild and the clinical signs and blocking pattern may be confused with foot or occasionally fetlock lameness. Pain in the pastern region is exacerbated by distal limb flexion. There is often a weight-bearing lameness that is worse if a horse is lunged or trotted with the affected limb on the inside of a circle.

Response to intra-articular analgesia varies depending on the injury, but improvement of lameness by 50% or more implicates the proximal interphalangeal joint as an important source of pain. Entry into the proximal interphalangeal joint can be difficult, especially if new bone is present as a result of osteoarthritis. Techniques for intra-articular and perineural analgesia are described in Chapter 10. Complete analgesia of the proximal interphalangeal joint is not always accomplished by perineural analgesia of the palmar nerves at the level of the PSBs, and a low four-point (palmar and palmar metacarpal nerves) block may be necessary. However, in some horses lameness improves with palmar/plantar digital analgesia. Intra-articular analgesia of the proximal interphalangeal joint should be considered in a horse with suspected metacarpophalangeal or metatarsophalangeal joint pain that has not responded to treatment. Complete analgesia of the DFTS requires intrasynovial or a low four-point block.

IMAGING CONSIDERATIONS

Standard radiographic examination for evaluation of the proximal phalanx includes lateromedial, dorsopalmar (plantar), dorsal 45° lateral-palmaromedial oblique, and dorsal 45° medial-palmarolateral oblique views. Oblique radiographs that are angled distally are helpful to identify osteochondral fragments on the proximal palmar/plantar aspect of the proximal phalanx.[3] If a sagittal plane fracture of the proximal phalanx is suspected, dorsopalmar/plantar radiographs that are 5° off the mid-sagittal plane are useful. Slight underexposure of the lateromedial projection helps to identify callus on the proximodorsal aspect of the proximal phalanx associated with chronic, short, incomplete fractures. Radiographic examination of the proximal interphalangeal joint includes lateromedial,

dorsopalmar/plantar, and oblique views. Flexed oblique views are particularly helpful for evaluation of the articular margins.[3] A well-positioned and exposed dorsopalmar/plantar projection may reveal subtle joint space narrowing and sclerosis of the subchondral bone consistent with early osteoarthritis. Comparative views of the contralateral limb may be helpful.

Contrast radiography may be used to determine the course of fistulous tracts or to identify communication between wounds and the DFTS or areas of proliferative synovium within the DFTS. Contrast radiography is performed by placement of a 50% solution of diatrizoate preglumine and diatrizoate sodium (Hypaque-76; Nyromed, Princeton, NJ) within a draining tract or directly into the synovial structure, after aseptic preparation. Standard radiographs are obtained to identify communication with draining tracts or filling defects.

Computed radiography is helpful in detecting subtle changes in trabecular bone and bone density. As computed tomography of the distal limb becomes more common, new information on development of pastern injury will become available.

Ultrasonographic evaluation of the pastern region is helpful in identifying abnormalities associated with the soft tissue structures[4] and is discussed in detail in Chapters 16 and 83.

Nuclear scintigraphy is useful in identifying incomplete fractures of the proximal phalanx. Incomplete fractures of the proximal articular margin of the proximal phalanx can occur in all breeds, but they are most commonly seen in Standardbred (STB) and Thoroughbred (TB) racehorses. Clinical signs and response to perineural analgesia varies. Nuclear scintigraphy should be considered to eliminate fracture in horses with lameness referable to the fetlock and clinical signs consistent with an incomplete fracture of the proximal phalanx. Nuclear scintigraphy can be helpful to identify bone remodeling in the early phases of osteoarthritis of the proximal interphalangeal joint. Other bone-related abnormalities in the pastern region often are evident radiographically and scintigraphy is unnecessary.

BREED PREDILECTION

Differential diagnoses of disorders of the pastern region of all breeds include osteoarthritis, osteochondrosis, fractures, and infection. The types of fractures tend to be breed or use specific. Fractures of the proximal phalanx occur most often in racing breeds, with the STB most often affected. Fractures of the middle phalanx occur most commonly in horses used for Western-type activities such as reining, but are seen in all breeds during lunging or after kicks. Osteochondrosis of the proximal aspect of the proximal phalanx is common in yearling and 2-year-old TBs and STBs. Osteochondrosis of the distal aspect of the proximal phalanx and proximal aspect of the middle phalanx is less common with no breed predilection. Osteoarthritis of the proximal interphalangeal joint occurs most often in older horses used for riding or Western-type activities. It may be seen in young horses secondary to osteochondrosis.

FRACTURES OF THE PROXIMAL PHALANX

Fractures of the proximal phalanx are important causes of lameness in all breeds. Racing breeds are particularly prone. Clinical signs vary from subtle to obvious. In horses with complete fractures, lameness is severe and limb swelling and deformity may be present. In horses with incomplete fractures, lameness may be subtle, and careful clinical and radiographic examination may be necessary to define the fracture. Serial radiographs may be needed to detect radiolucency and callus formation associated with short incomplete fractures of the proximal phalanx. Nuclear scintigraphy may be helpful.

Fractures of the proximal phalanx include the following types:

- Short and long incomplete sagittal
- Complete fractures that exit the lateral cortex
- Simple complete
- Comminuted
- Dorsal frontal
- Proximal palmar/plantar avulsion fractures associated with the collateral ligament

Incomplete fractures of the proximal phalanx occur most commonly in racing breeds. Clinical signs include mild to severe lameness. Fetlock joint effusion and mild swelling and pain over the dorsal aspect of the proximal phalanx are common. Most fractures are readily identified radiographically, but in some horses dorsopalmar/plantar projections that are slightly off the sagittal plane are useful. In addition, obtaining follow-up radiographs 7 to 10 days after the fracture allows time for osteoclastic resorption along the fracture line to occur so that the fracture line becomes more obvious. Treatment for incomplete sagittal fractures includes stall rest or internal fixation, depending on the degree of lameness, concern about catastrophic fracture of the bone, and economic factors. The advantages of surgical repair are well documented, but incomplete short fractures of the proximal aspect of the proximal phalanx can often be managed with rest alone. In some circumstances a bone screw is placed to provide compression for short fractures to reduce the risk of fracture recurrence.

Indications for internal fixation of incomplete fractures include fractures that extend more than 15 mm from the proximal articular surface, those with potential for catastrophic failure, and those that occur in horses with severe lameness. Internal fixation is accomplished by placement of 4.5- or 5.5-mm cortical bone screws in lag fashion. Although 4.5-mm screws generally are used and provide ample compression, 5.5-mm screws provide maximal fracture compression and can be used to replace a stripped 4.5-mm thread. Because these fractures are non-displaced, open reduction is not required and the screws can be placed through minimal incisions. Horses with incomplete fractures are reported to have a 67% to 89% chance to return to racing soundness after treatment. However, STBs returned to racing with slower racing times and reduced performance indices.[5-7]

Horses with complete fractures of the proximal phalanx are candidates for internal fixation to provide anatomical reduction and compression of the articular surfaces, improved comfort, reduced risk of osteoarthritis, and early return to use. In horses with acute, minimally displaced fractures, reduction may be achieved by percutaneus placement of bone reduction forceps under radiographic control. Open reduction, arthrotomy, or both are required in horses with moderately to severely displaced fractures, chronic fractures, or fractures in which fragments of bone prevent reduction of the fracture fragments. After debridement of the fracture bed and reduction of the fracture, compression of the fracture fragments is accomplished using 4.5- or 5.5-mm cortical bone screws. External coaptation with a half-limb cast may be required for recovery or the immediate post-operative period. Prognosis for horses with non-comminuted complete fractures depends on whether the proximal interphalangeal joint is involved. Horses with fractures that enter the proximal interphalangeal joint had an approximate 50% chance to return to racing. Of those with complete fractures that exited the lateral cortex, 71% returned to racing.[5,7]

Salter-Harris type II fractures of the proximal aspect of the proximal phalanx occur in foals and weanlings. Reduction of the fracture is accomplished with the horse under anesthesia. Methods of coaptation include bandaging alone, bandaging with splinting, or half-limb cast application. I prefer to use a cast for 4 weeks, but it is important that the cast be changed

at 2 weeks. Other clinicians prefer less aggressive coaptation to avoid complications of flexor laxity and osteoporosis of the PSBs.

Dorsal frontal fractures of the proximal aspect of the proximal phalanx occur in three general types: incomplete proximal dorsal articular fractures, complete proximal dorsal articular fractures, and fractures that originate in the midportion of the proximal articulation and extend distally into the proximal phalanx. These fractures usually are incomplete. Incomplete proximal dorsal frontal fractures occur most commonly in STB racehorses. The right hindlimb is most commonly affected. Proximal dorsal articular fractures can be treated with rest alone or internal fixation with a 3.5-mm cortical bone screw. The prognosis for horses with proximal fractures is good.[8] Horses with large fractures that originate in the central portion of the metacarpophalangeal or metatarsophalangeal joint need internal fixation. In my experience, osteoarthritis tends to develop despite anatomical reduction and internal fixation of the fracture, and the prognosis for athletic soundness is poorer.[9]

Horses with comminuted fractures of the proximal phalanx have acute, non–weight-bearing lameness and often limb deformity. Appropriate first aid is required to prevent further injury to the soft tissues, with or without disruption of the digital artery blood supply. A half-limb cast with a dorsally incorporated splint is appropriate to realign the bony column for transport to a surgical facility. Comminuted fractures can be divided into two categories for treatment: those with and those without an intact column of bone extending from the proximal to distal articular surfaces. This intact strut of bone allows reconstruction of many comminuted fractures of the proximal phalanx by lag screw fixation of the fracture fragments to the strut. Treatment strategies include the following:

- Complete reconstruction via bone screws, plates, or both by open reduction, partial reconstruction, and transfixation cast or external fixator
- Transfixation cast alone
- Use of external skeletal fixation (see Chapter 88)

In my opinion, the use of half-limb casts alone is not the best method of sole treatment for most horses with comminuted fractures of the proximal phalanx, although successful outcomes have been reported. Half-limb casts significantly reduce axial loading in the intact skeleton,[10,11] but the risks of fracture compression, skin injuries leading to open fractures, and contralateral limb laminitis are substantial.

Horses with comminuted fractures with an existing strut of bone are candidates for open reduction and reconstruction with bone screws. An initial study reported an unacceptably high risk of infection after open reduction and internal fixation,[12] but a more recent report described a good prognosis for pasture soundness.[13] If the strut has a transverse fracture but enough bone stock proximal and distal to the transverse fracture, I have reconstructed the bone with two 4.5-mm narrow dynamic compression plates and bone screws. With both screw fixation alone and plate and screw fixation, external coaptation with a half-limb cast is required. Partial reconstruction of the articular surfaces and placement of a transfixation cast or external fixator is elected when possible in horses with severely comminuted fractures to reduce degenerative changes, improve long-term comfort, and reduce the requirement for subsequent arthrodesis. A transfixation cast is used when comminution is severe enough to prevent anatomical reconstruction and protection of the fracture from collapse is required. Transfixation casts have been shown to significantly improve axial stability compared with standard casts in an osteotomy model.[14] External fixators are used in place of transfixation casts if the fracture is open to allow direct access to the injury site (see Chapter 88).[15] The use of transfixation casts and external fixators carries the risk of catastrophic failure of the third metacarpal (metatarsal) bone, and the overall prognosis for salvage is only fair (Fig. 36-1).

Palmar/plantar avulsion fractures of the proximal phalanx usually occur after kicks, falls, or stall injury. Lameness is variable and fetlock effusion is usually present. Differential diagnosis includes fracture and osteochondritis dissecans fragmentation. Because this region is the distal attachment for the collateral ligaments, surgical treatment is recommended to improve bony union and prevent osteoarthritis. Treatment options include lag screw fixation, with one or preferably two 3.5-mm cortical bone screws. Removal is also possible for chronic or small fractures or those that cannot be properly reduced. After removal, external coaptation for recovery from general anesthesia and then for an additional 4 weeks is recommended. The prognosis is considered good with either treatment in the absence of osteoarthritis.

DORSAL OSTEOCHONDRAL FRAGMENTS IN THE METACARPOPHALANGEAL/ METATARSOPHALANGEAL JOINT

See Chapters 37 and 38 for a discussion of dorsal osteochondral fragments in the metacarpophalangeal/metatarsophalangeal joint.

OSTEOCHONDROSIS OF THE PROXIMAL INTERPHALANGEAL JOINT

Osteochondrosis of the proximal interphalangeal joint is less common than that of the metacarpophalangeal or metatarsophalangeal joints. As with other sites, osteochondrosis involves fragmentation (osteochondritis dissecans), or osseous cyst-like lesions. Sites include the distal aspect of the proximal phalanx and proximal aspect of the middle phalanx. A radiolucent area at the distal central aspect of the proximal phalanx usually is an incidental radiographic finding. Radiolucent areas or fragmentation in the condylar regions of the distal aspect of the proximal phalanx tend to cause clinical signs. In the most severe circumstances, these subchondral lucencies in weight-bearing portions of the joint may cause significant osteoarthritis and lameness requiring arthrodesis of the proximal interphalangeal joint. Osseous cyst-like lesions are more common in the hindlimb than in the forelimb and can occur bilaterally. Osseous cyst-like lesions can communicate with the proximal interphalangeal joint, and if lameness is present, radiographic evidence of osteoarthritis can be substantial. Subtle clinical signs often are seen in horses with osseous cyst-like lesions that do not communicate with the proximal interphalangeal joint. Although unusual, sudden-onset severe lameness can occur as a result of osseous cyst-like lesions even though the radiographic abnormality was present for several months or years. Clinical relevance of osseous cyst-like lesions must be established using diagnostic analgesia, and in some horses scintigraphy is required. Management of horses with osseous cyst-like lesions may include intra-articular injections, or in some horses arthrodesis of the proximal interphalangeal joint. Arthrodesis of the proximal interphalangeal joint can yield a good prognosis for soundness in Western performance horses or other types of sport horses that are not expected to perform at advanced levels. A STB pacer that underwent arthrodesis of the left hindlimb proximal interphalangeal joint raced successfully.[16] A peri-articular drilling procedure can be used in horses with osseous cyst-like lesions that do not communicate with the proximal interphalangeal joint. A small drill bit is used under radiographic guidance to approach the cystic cavity. Either methylprednisolone acetate or liquid bone marrow can be injected. Alternatively,

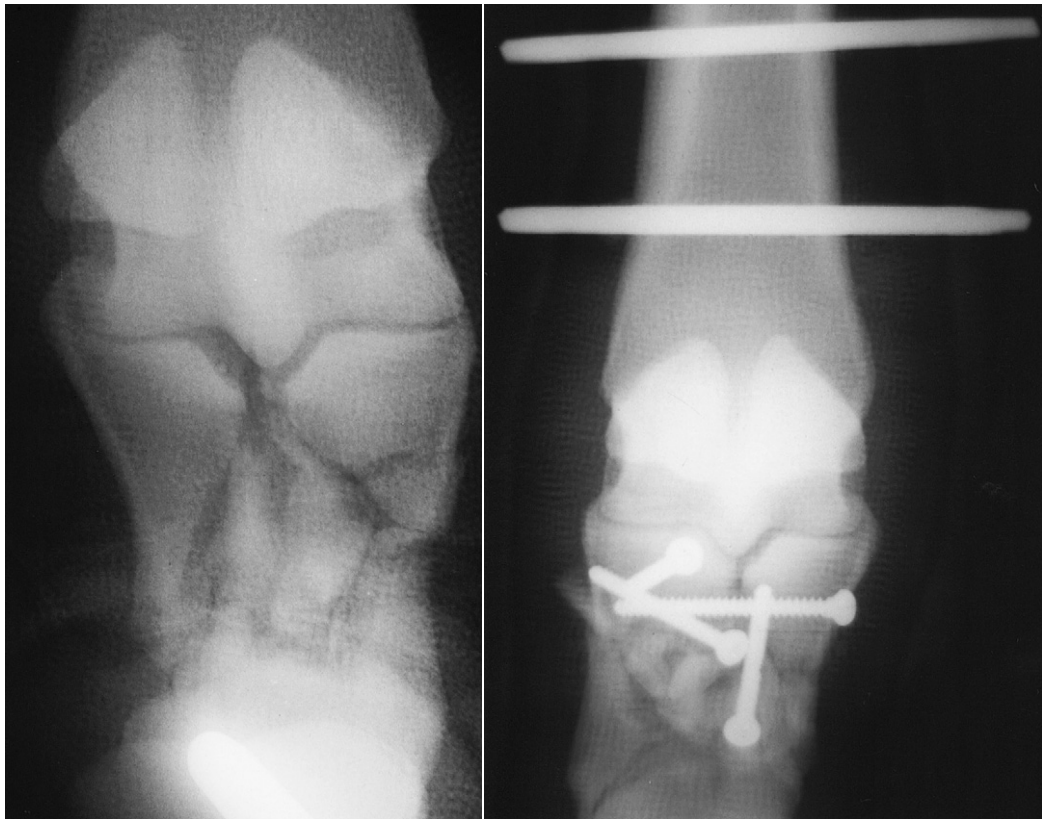

Fig. 36-1 A and B, Dorsoplantar radiographic projections of a 3-year-old Standardbred filly with a severely comminuted fracture of the proximal phalanx before **(A)** and after **(B)** treatment by partial reconstruction of the proximal articular portion of the fracture and a transfixation pin cast.

cancellous bone can be packed into the cyst cavity. Radiographic evidence of healing of the cyst cavity is variable, and in some horses residual lameness remains. Osteochondral fragments associated with osteochondrosis tend to occur at the palmar/plantar eminence of the proximal aspect of the middle phalanx and cause variable lameness. They often are incidental findings found on radiographs taken before sale, but they certainly can complicate the sales process. Prognosis is difficult to assess in yearlings that have not been trained. Osteochondrosis fragments are more common in the hindlimbs than in the forelimbs. There is often radiographic evidence of advanced osteoarthritis, even if lameness is mild. Because these fragments are difficult to remove, it is important to document the true source of lameness before surgery is considered. Arthroscopic techniques for the pastern have been described, but because of limited surgical access, I generally perform an arthrotomy to remove the fragments, which often are large.[17,18]

OSTEOARTHRITIS OF THE PROXIMAL INTERPHALANGEAL JOINT

Osteoarthritis of the proximal interphalangeal joint is also known as *high ringbone*. Horses used for jumping, dressage, and Western-type activities seem to be prone to high ringbone. It can also be a consequence of articular fracture, infection, or osteochondrosis. The clinical signs of osteoarthritis of the proximal interphalangeal joint include mild to severe lameness. Lameness is exacerbated by distal limb flexion. Obvious bony formation or angular deformity may be present in horses with advanced lameness. Diagnosis is based on clinical signs, response to perineural or intra-articular analgesia, and radiographic findings. Radiographic abnormalities commonly include new bone formation, periarticular sclerosis, and loss of joint space typically on the medial aspect of the proximal interphalangeal joint. Angular deformity is typically only present in horses with advanced osteoarthritis (Fig. 36-2). Comparative radiographs of the contralateral limb should also be obtained because the condition may be bilateral. Pain associated with the proximal interphalangeal joint may be present without any radiographic signs of osteoarthritis, and scintigraphic examination is useful in documenting active bone modeling. On the other hand, horses may develop acute severe lameness with radiographic evidence of existing osteoarthritis that predated any clinical evidence of pain. Lameness improves after perineural or intra-articular analgesia.

In horses with mild osteoarthritis of the proximal interphalangeal joint that is unrelated to fracture, conservative treatment includes therapeutic shoeing aimed at reducing the toe length, and in some horses, elevating the heel and easing breakover. Non-steroidal anti-inflammatory drugs are quite useful. The therapeutic value of administering intramuscular glycosaminoglycans and intravenous hyaluronan is questionable. Intra-articular injections are of considerable value. A combination of hyaluronan and corticosteroids or corticosteroids alone is most effective in horses with mild or moderate osteoarthritis. However, in horses with advanced osteoarthritis, particularly those with extensive periarticular proliferation and extensive loss of joint space, arthrodesis should be considered. The surgical procedure is invasive and expensive but is generally considered a better solution over the long term. Natural ankylosis of the proximal interphalangeal joint can occur, but it is a long, painful process and one

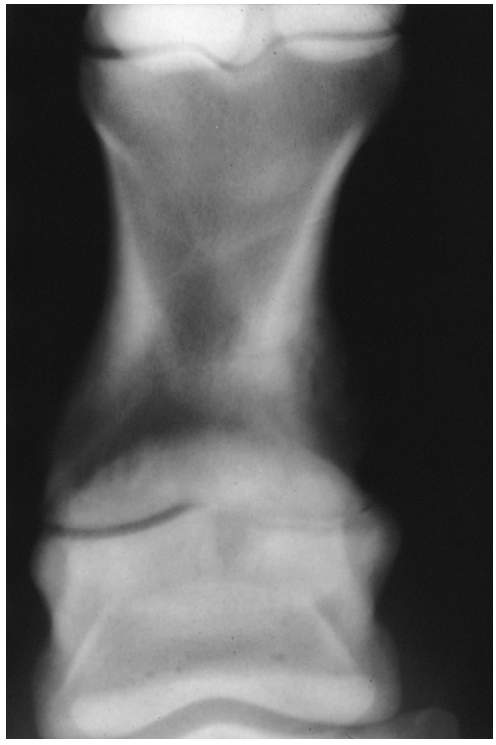

Fig. 36-2 This dorsoplantar radiographic projection demonstrates proliferative and radiolucent changes associated with advanced osteoarthritis of the proximal interphalangeal joint. There is complete loss of joint space on one side and angular deformity of the proximal interphalangeal joint.

that is not necessarily complete. Surgical arthrodesis is best. Tibial neurectomy can be helpful in horses with advanced hindlimb proximal interphalangeal osteoarthritis.[19]

ARTHRODESIS OF THE PROXIMAL INTERPHALANGEAL JOINT

Arthrodesis of the proximal interphalangeal joint is indicated in horses with advanced osteoarthritis or an articular fracture. Lag screw fixation of simple fractures can be performed, but because of difficulty in obtaining anatomical reduction of the proximal interphalangeal joint surface and resisting tension forces at the palmar/plantar eminences, lag screw fixation alone is usually unsuccessful in preventing osteoarthritis and returning horses to athletic soundness. Therefore I recommend proximal interphalangeal joint arthrodesis in horses with articular fractures.

Many methods of arthrodesis of the proximal interphalangeal joint are described, and opinions vary as to which is preferred. Current preferred methods involve the insertion of three parallel 4.5- or 5.5-mm screws or dorsally applied plate or plates with additional transarticular screws. I have used both methods and prefer the plating technique because it improves comfort in the immediate post-operative period and reduces the necessity for prolonged cast application. This method requires cast coaptation for 2 to 3 weeks to protect the incision site. The plating technique provides improved stability compared with the three 5.5-mm parallel screw technique in fatigue testing.[20] After arthrodesis of the proximal interphalangeal joint with screws alone, approximately 80% of horses with hindlimb and 46% to 67% of horses with forelimb lameness returned to athletic soundness.[21,22] Long-term follow-up data for the plating technique are currently unavailable, but in my opinion, the results are at least similar, if not superior.

Horses with comminuted middle phalanx fractures or those that involve the distal interphalangeal joint should undergo arthrodesis of the proximal interphalangeal joint with single or double plating and potentially transfixation casts or external skeletal fixation (see Chapter 88). Comminuted fractures of the middle phalanx should be repaired if possible to preserve the distal interphalangeal joint surface, and if possible, the proximal interphalangeal joint surface. Displacement of the distal articular surface considerably worsens prognosis. Single or double plating can be used depending on the configuration of the fracture. The use of additional screws placed in lag fashion is usually required to reduce large fragments and reconstruct the articular surface. Another plate technique using a Y-plate has been tested experimentally and shows similar biomechanical properties to the double-plate technique.[23] Cast coaptation is required for 6 to 8 weeks after surgery. Transfixation pins are used to prevent collapse of the fracture in horses with severely comminuted fractures. This technique may be combined with plate and screw fixation. Prognosis after double-plate fixation of 10 horses with comminuted fracture of the middle phalanx was good for pasture soundness, and 5 horses were able to be ridden or shown, but mild lameness persisted.[24]

SOFT TISSUE INJURIES

Soft tissue injuries in the pastern are discussed in Chapter 83.

SUBLUXATION OF THE PROXIMAL INTERPHALANGEAL JOINT

Subluxation of the proximal interphalangeal joint is uncommon and can occur in the palmar/plantar direction or dorsally. Palmar/plantar subluxation usually is seen after severe, traumatic soft tissue injury, such as complete tearing of the distal sesamoidean ligaments, SDFT branch injury, or a combination of soft tissue injuries (Fig. 36-3). Treatment options for palmar/plantar subluxation include both conservative and surgical management. External coaptation often is successful in adult horses managed acutely. The application of a Kimzey splint (Kimzey Leg Saver Splint; Kimzey, Inc., Woodland, CA) is an excellent method of external coaptation with this injury. Cast immobilization can be successful, but instability of the proximal interphalangeal joint may preclude successful realignment with this method. Arthrodesis of the proximal interphalangeal joint is always an option. Dorsal subluxation can occur after traumatic disruption of the suspensory apparatus and arthrodesis of the fetlock joint to manage this problem, but it most commonly occurs in horses with progressive, severe suspensory desmitis. This is particularly true in the STB or TB racehorse in which suspensory desmitis is common. Swelling along the dorsal aspect of the pastern region is the first clinical sign recognized, but progressive hyperextension (dropping) of the fetlock joint is usually present. The appearance of dorsal subluxation in any horse with chronic suspensory desmitis is a negative prognostic sign.

Dorsal subluxation of the proximal interphalangeal joint can occur without any identifiable structural abnormality of the pastern or metatarsal soft tissue structures. This occurs primarily in the hindlimbs in young horses, and lameness is usually absent or mild. Dorsal subluxation is most often dynamic in nature and resolves during full weight bearing. The presence of changes consistent with osteoarthritis worsens prognosis. Dorsal subluxation of the proximal inter-

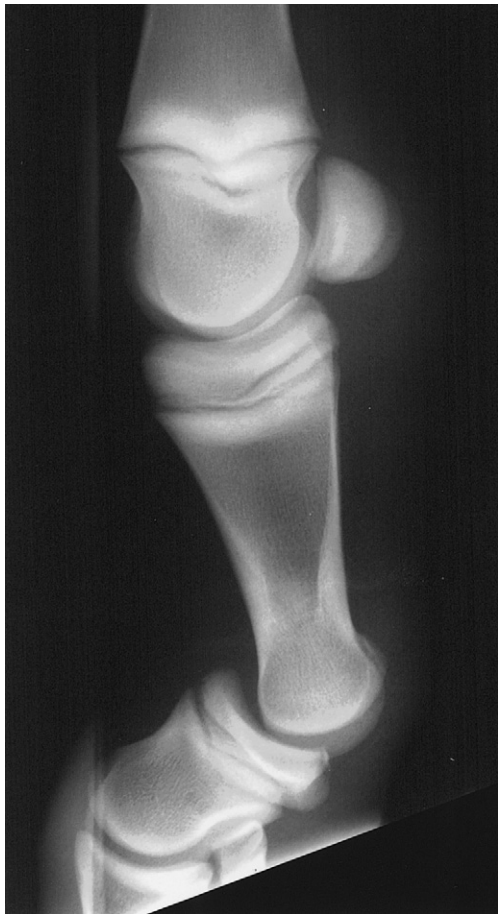

Fig. 36-3 Lateromedial radiographic projection of the pastern in a foal with plantar subluxation from avulsion of the attachments of the superficial digital flexor tendon and subsequent loss of plantar support. This foal underwent arthrodesis of the proximal interphalangeal joint.

phalangeal joint as a result of mild flexor deformity was reported in three horses.[25] In these horses, ranging in age from 5 months to 4 years, subluxation was believed to be caused by mild contraction of the DDFT without compensatory contraction of the SDFT.[2–5] Subluxation was seen primarily during early weight bearing, but the condition resolved during full weight bearing. Tenotomy of the medial head of the DDFT in the proximal metatarsal region resolved dynamic dorsal subluxation.[25]

WOUNDS IN THE PASTERN REGION

Lacerations and puncture wounds in the pastern region have a high propensity to involve the proximal interphalangeal joint or DFTS. Careful inspection of the wound, radiography, ultrasonography, and analysis of synovial aspirates are often required to determine the extent of the wounds. Wounds that involve the proximal interphalangeal joint or DFTS require aggressive management to prevent synovial infection. Treatments include synovial lavage, wound debridement, drain placement, and antimicrobial therapy with or without tenoscopic examination. Intrasynovial, systemic, and regional perfusion of antimicrobial agents can be used. Sequelae of proximal interphalangeal joint infection include osteoarthritis. Sequelae of infection of the DFTS include intrasynovial adhesion formation and infectious osteitis of the PSBs (see Chapter 75).

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CHAPTER • 37

The Metacarpophalangeal Joint

Dean W. Richardson

ANATOMICAL CONSIDERATIONS

The metacarpophalangeal (fetlock) joint is an intensely loaded, high-motion joint that is frequently injured in athletic horses. Fetlock region lameness can occur in horses of any occupation, but the joint is at particularly high risk in horses performing at maximal speed. Countering the high load experienced by the joint is the elastic suspensory apparatus that constrains the range of extension. Both the superficial and deep digital flexor muscle tendon units additionally serve to actively support the fetlock joint because of the position on the palmar aspect of the joint. Loss of this active support by the flexors may lead to overload of the suspensory apparatus support and some degree of hyperextension. Extreme extension of the metacarpophalangeal joint results in impingement of the proximal rim of the proximal phalanx against the dorsal aspect of the third metacarpal bone (McIII), high compressive forces on the distal, palmar aspect of McIII opposing the proximal sesamoid bones (PSBs), and both tensile and bending forces on the PSBs intercalated within the suspensory apparatus. Complex forces of torsion on the loaded fetlock joint result in a myriad of injuries.

The metacarpophalangeal joint is anatomically composed of the distal McIII, the proximal phalanx, and both PSBs. A single synovial space extends on its palmar aspect at least 3 cm proximal to the apex of the PSBs. Dorsally, a bilobed synovial "pad" is located on the proximal dorsal articular rim of the distal McIII; this pad presumably functions to help cushion the impingement of the McIII and proximal phalanx. The ligamentous elements of the fetlock joint are complex and important. Well-developed medial and lateral collateral metacarpophalangeal and metacarposesamoidean ligaments constrain the almost purely sagittal motion of this joint. The suspensory branches insert on the proximal, palmar abaxial margins of the PSBs and functionally continue through the distal sesamoidean ligaments to attach the distal portion of the sesamoids to the proximal phalanx (cruciate or deep and oblique or middle) and the middle phalanx (straight or superficial). The intersesamoidean ligament attaches the axial aspects of the PSBs.

The digital flexor tendon sheath and its contents are described elsewhere (see Chapter 75). The common digital extensor tendon is unsheathed as it passes over the dorsal aspect of the joint. Branches of the medial and lateral palmar and metacarpal nerves primarily supply innervation to the fetlock joint,[1] but small subcutaneous branches of the ulnar nerve supply a minor amount of dorsal sensory innervation.

DIAGNOSIS

Careful physical examination to detect heat, swelling (effusion with or without periarticular fibrosis), and pain with palpation or manipulation is critical for diagnosis of lameness of the metacarpophalangeal joint. However, physical findings may be subtle or seemingly non-existent, whereas diagnostic analgesia often pinpoints the joint or region as the source of pain. Interpretation of fetlock flexion test results should be made with caution, because many horses have false-positive responses to forced flexion of this joint, especially if the toe is used to increase leverage. Pain originating in the limb distal to the fetlock joint often is exacerbated by fetlock or lower limb flexion tests. False-negative responses to fetlock joint flexion also are quite common, especially in horses with subchondral bone injury and chip fractures. Increased fluid in the fetlock joint can also be a false localizing sign. The presence of an effusion in the absence of heat and pain may indicate some derangement in synovial function, but not necessarily point to the source of a clinically relevant lameness. Lameness characteristically occurs with weight bearing and usually, but not always, is worse with the limb on the inside of a circle. If clinical signs do not adequately localize lameness, perineural or intrasynovial analgesia can specify the region relatively easily. In the absence of localizing clinical signs the foot and pastern regions should first be eliminated as sources of pain by a mid-pastern digital nerve block with a dorsal subcutaneous ring. Ideally this is followed by intra-articular analgesia. If the lameness does not improve within 15 to 20 minutes of intra-articular analgesia, the clinician should perform a low palmar block or palmar and palmar metacarpal nerve blocks. In most horses with intra-articular chip fractures, synovitis, capsulitis, and osteoarthritis, lameness improves after intra-articular analgesia. Horses with major fractures, non-articular fractures, subchondral bone injuries, and tendon and tendon sheath lesions usually are only markedly improved with perineural analgesia. Perineural techniques are superior in abolishing the pain associated with the fetlock joint. Pain in horses that do not respond to intra-articular analgesia should not necessarily be presumed to originate elsewhere.

Several techniques are used for intrasynovial analgesia of the fetlock joint. I prefer to inject the local anesthetic solution with the horse's limb on the ground. The dorsal aspect of the fetlock is readily accessible when the horse is bearing weight. As the clinician faces forward and uses the back of his or her arm to hold the horse's carpus, a 22-gauge 2.5-cm needle

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CHAPTER • 37

The Metacarpophalangeal Joint

Dean W. Richardson

ANATOMICAL CONSIDERATIONS

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attached to a 75-cm extension set is inserted horizontally into the dorsal joint just proximal to the margin of the proximal phalanx and deep to the common digital extensor tendon. I use 10 to 15 ml of 2% mepivacaine, but other practitioners generally use no more than 6 to 10 ml.

IMAGING CONSIDERATIONS

The fetlock joint is easy to image accurately because of the size and accessibility. Numerous flexed and oblique radiographic views to specifically silhouette or separate structures help to identify subtle lesions. The same advantages apply to scintigraphic and ultrasonographic diagnostic imaging. For example, flexed scintigraphic images can help determine whether a lesion in the palmar fetlock involves the base of a PSB or the palmar surface of McIII. A minimum set of radiographs should include dorsopalmar, lateromedial, flexed lateromedial, and both oblique views. The size of the region and absence of overlying soft tissues results in excellently detailed radiographs, even with portable x-ray machines, if suitable screen-film combinations are used. A number of specific radiographic views should be used if certain lesions are suspected. For example, if a subchondral injury involving the distal palmar McIII is suspected, the oblique views should be taken in a more proximal to distal direction than usual. If a lateral condylar fracture is identified, a partially flexed dorsopalmar view should be used to help identify comminution along the distal, palmar aspect of the fracture line. Osteochondrosis of the sagittal ridge of the McIII and incomplete sagittal fractures of the proximal phalanx are lesions that can be difficult to identify in over-exposed lateromedial views or under-exposed dorsopalmar projections.

TYPES OF FETLOCK JOINT LAMENESS

Most fetlock joint lamenesses can be categorized into one of three types:

1. Acute or repetitive overload injuries without specific fracture or fractures: capsulitis/synovitis, chronic proliferative (villonodular) synovitis, osteoarthritis, subchondral bone injury without obvious fracture, and sesamoiditis.
2. Articular fragments that can be removed (traumatic and developmental lesions) from the dorsal or palmar aspects of the proximal phalanx; apical, abaxial, and basilar sesamoid fractures; and fragments from the sagittal ridge of the McIII.
3. Major articular fractures (i.e., those that should be repaired), including sagittal and dorsal frontal fractures (mostly seen in the hindlimbs); collateral avulsion injuries (mostly foals and yearlings) of the proximal phalanx, mid-body; large abaxial or basilar fragments of the PSBs; and condylar fractures of McIII.

Conditions specific to the metatarsophalangeal joint and a detailed description of sagittal fractures of the proximal phalanx are included elsewhere (see Chapters 36 and 43).

ACUTE OR REPETITIVE OVERLOAD INJURIES

Capsulitis/Synovitis

Clinical Signs

Virtually every young racehorse has one or more episodes of metacarpophalangeal capsulitis or synovitis that is characterized by heat, effusion, and pain with flexion. Overt lameness usually is mild and often not evident after the horse warms up. Signs typically manifest as the horse increases the speed and distance of its exercise regimen. If the typical localizing signs are missed, the major indication may be a decrease in performance. In horses with chronic disease, visible thickening of the periarticular tissues usually is noticeable, with decreased range of motion of the joint.

Diagnosis

A diagnosis of primary capsulitis or synovitis usually is made by clinical observations and the absence of radiographic scintigraphic bony abnormalities. In most horses, synovitis and capsulitis accompany other causes of fetlock joint lameness, such as chip fractures. Synovial fluid analysis has not been particularly useful in diagnosis or prognosis for most fetlock joint injuries except to help identify sepsis.

Treatment

In most young horses the condition resolves with nonsteroidal anti-inflammatory drugs (NSAIDs), adjustments in training, and increased fitness. Intra-articular hyaluronan (20 mg) is helpful in horses with acute, mild to moderate synovitis, but a combination of hyaluronan with a low dose of corticosteroid (e.g., 3 to 5 mg of triamcinolone or 20 to 30 mg of methylprednisolone acetate) is more consistently effective in resolving the clinical signs of inflammation. Injection should be followed by a decrease in exercise intensity for 1 to 2 weeks if possible. Post-exercise icing of the involved fetlock joints can be helpful. Generic osteoarthritis treatments, including oral glucosamine with or without chondroitin sulfate, intramuscular polysulfated glycosaminoglycan, and intravenous hyaluronan merit consideration, but they do not usually yield the consistent response seen after intra-articular therapy.

Chronic Proliferative (Villonodular) Synovitis

Clinical Signs

Chronic capsulitis/synovitis is caused by repetitive injury of the dorsal aspect of the fetlock joint. The lesion is defined by a thickening of the normally dorsally located bilobed synovial pad that hangs down on either side of the sagittal ridge of the McIII. With extreme extension of the fetlock joint the dorsal rim of the proximal phalanx impinges on the synovial pad, and repetitive trauma results in its inflammation and subsequent fibrosis. The tissue can become so thick that the dorsal profile of the joint is visibly disfigured. The characteristic swelling is asymmetrical on the mid-proximodorsal aspect of the fetlock (Fig. 37-1) rather than simply spherical in outline as in a typical osteoarthritic fetlock joint (this is called an *osselet*). Exercise inflames the tissue further, and clinical signs of lameness or diminished performance can result.

Diagnosis

The diagnosis of proliferative synovitis is based on physical examination, radiography, ultrasonography, or a combination of these modalities. The most common radiographic sign of the lesion is a crescent-shaped, radiolucent "cut-out" on the dorsal aspect of the McIII at the level of the joint capsule attachment (Fig. 37-2). The proliferative lesion may undergo dystrophic mineralization and be radiographically visible. Radiographic contrast studies can be used, but ultrasonography is simpler and more reliable (Fig. 37-3). However, many horses have a substantial synovial pad without any concomitant discomfort. Mere identification of a slightly thicker than average structure is certainly not an indication for surgical excision. Horses with extremely severe osteoarthritis have proliferative synovitis in the palmar pouch, and a large concave outline of the distal palmar aspect of the McIII is seen proximal to the PSBs. A crescentic concavity in this region is a strong indication of severe osteoarthritis and is associated with a poor prognosis.

Treatment

Treatment for proliferative synovitis usually consists of aggressive intra-articular therapy (e.g., hyaluronan and corticosteroids), rest, and alterations in training. Many horses require frequent medication until the joint becomes stiff enough to

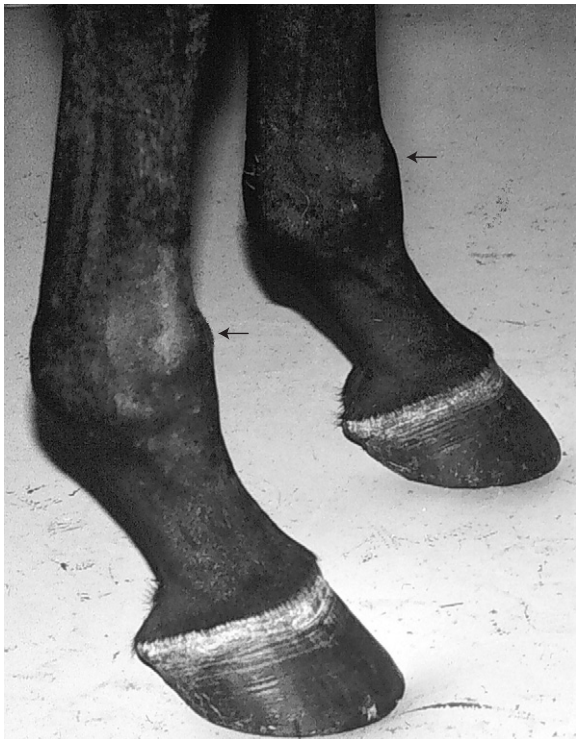

Fig. 37-1 An older Thoroughbred racehorse with chronic proliferative synovitis. The dorsal swelling is firm and localized in the proximodorsal aspect of the joint *(arrows)*.

prevent dorsal impingement. Horses appear to be able to work through this problem as they develop increasing strength and fitness. If the horse does not respond to medical treatment, surgical excision using an arthroscopic technique is recommended.[2,3] Surgical debridement must be followed with continued medical treatment and careful attention to the training regimen. It is important to recognize that the thickening of the proximal dorsal synovial pad may be just one part of a chronic, osteoarthritic joint. In such horses, any therapy such as surgery directed solely at this lesion probably will fail.

Subchondral Bone Injury

Clinical Signs

Subchondral bone injury is an extremely important cause of lameness involving the metacarpophalangeal joint, especially in racehorses. There are no specific localizing clinical signs in many horses other than variable lameness and an observable diminution in performance. Signs such as heat, swelling, or response to flexion can be absent or extremely subtle, even in horses with overt lameness. Although subchondral bone injury is far more common in racehorses, it appears that single-step overload injuries that focally damage a portion of the proximal phalanx can occur in non-racehorses. Such injuries may result in radiographically visible sclerosis or lysis. The severity of the lameness in such horses can be surprising and infection is often a differential diagnosis.

Diagnosis

The inconsistent response to local analgesia can make diagnosis difficult unless a reasonably high index of suspicion for these lesions is maintained. Many horses do not achieve soundness with intra-articular analgesia. Horses with full-thickness cartilage injury overlying the subchondral injury may be most likely to experience improved outcomes because of the access of the anesthetic agent to the site. Lameness in most improves dramatically with low palmar analgesia.

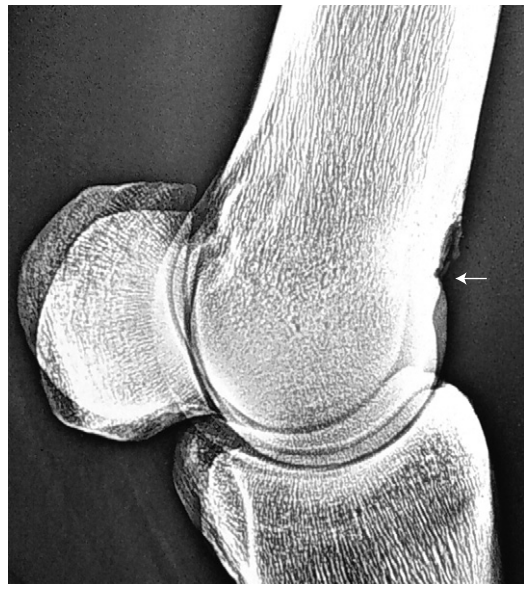

Fig. 37-2 Chronic proliferative (villonodular) synovitis of the metacarpophalangeal joint with a typical erosive lesion *(arrow)* along the proximal dorsal margin of the third metacarpal bone seen in a lateromedial radiographic view. The fibrotic tissue may have small fragments of bone and cartilage or dystrophic mineralization.

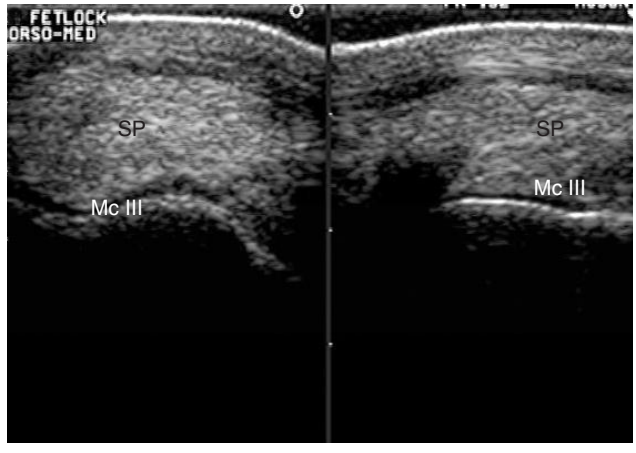

Fig. 37-3 Transverse *(left)* and longitudinal *(right)* ultrasonographic images of the distal dorsal metacarpal region. Ultrasonographic examination can confirm a thickened synovial pad, but the diagnosis usually is made on the basis of physical examination and radiographic findings. *McIII*, Third metacarpal bone; *SP*, synovial pad.

The two most common locations for subchondral injury of the fetlock are the distal palmar aspect of the McIII and proximal phalanx under the center of the weight-bearing portion of the medial or lateral articular surfaces. The palmar metacarpal lesion appears to develop as a focal overload injury where the base of the PSB impacts during maximal weight bearing.[4,5] In this location, linear or crescent-shaped lucencies are the most common indications of a major subchondral bone injury (Fig. 37-4). These often can be seen on a lateromedial projection, but special views may improve the likelihood of seeing a lesion. A slightly flexed, horizontal beam dorsopalmar projection silhouetting the distal palmar aspect of the McIII can help define an irregular subchondral outline

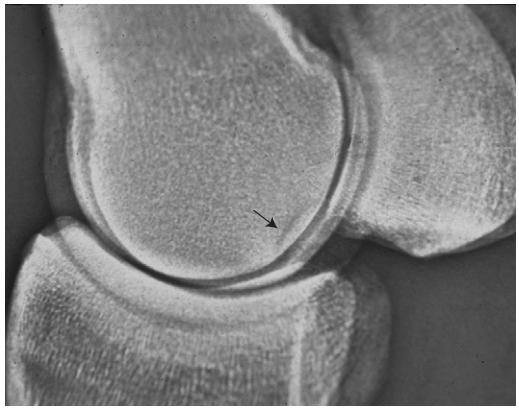

Fig. 37-4 Lateral radiographic view of a metacarpophalangeal joint. Subchondral crescent-shaped *(arrow)* or linear lucencies in the distal palmar third metacarpal bone are important radiographic findings. Most horses with such lesions have clinical signs of fetlock pain, especially with hard training. The cartilage overlying such lesions is damaged and often unstable.

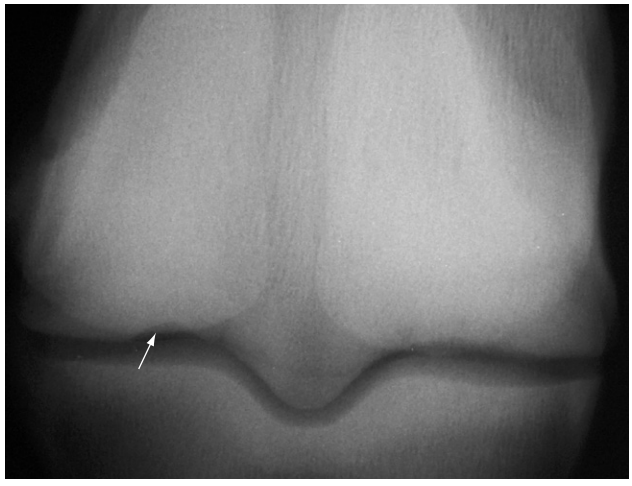

Fig. 37-5 Slightly flexed dorsopalmar radiographic view of the metacarpophalangeal joint is helpful to define *lesions (arrow)* of the distal palmar third metacarpal bone.

(Fig. 37-5).[6,7] Oblique projections obtained in a slightly (25° to 30°) proximal-distal direction also allow a better evaluation of the palmar condyle and the presence of radiolucent defects (Fig. 37-6).[8] With lesions of the proximal phalanx an increase in subchondral density may be evident before lytic lesions develop, but sclerosis in the McIII is much more difficult to recognize.

Scintigraphy is the most sensitive diagnostic tool and can identify lesions well before radiographic lesions are evident. Even arthroscopic diagnosis can be difficult in the fetlock joint, because the anatomical location of these lesions makes evaluation quite difficult. If the lesions are accessible, the overlying cartilage may be thin or discolored. The amount of cartilage fibrillation or crazing of the joint surface is variable.

Treatment

Rest is the only currently accepted treatment for subchondral bone injury, although medications such as isoxsuprine and aspirin are frequently recommended. Damaged bone can heal if the horse is rested, but prognosis depends on the amount of structural damage to the overlying cartilage and any loss in the normal architecture of this critical area.

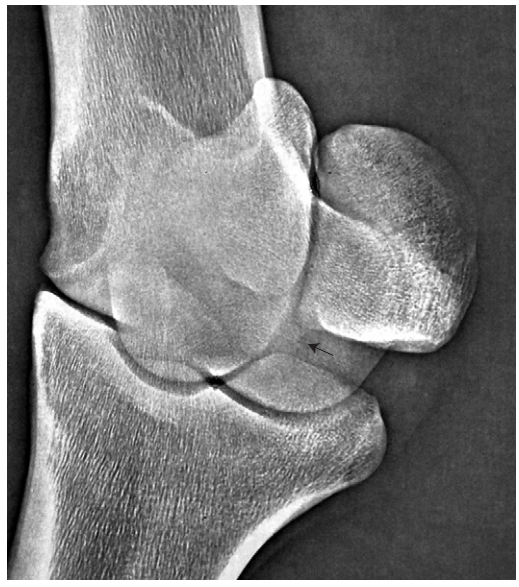

Fig. 37-6 Oblique xeroradiographic view taken at a proximal to distal 30° angle will allow identification of the radiolucent changes of the third metacarpal bone *(arrow)* otherwise obscured by the proximal sesamoid bone.

INJURIES TO THE PROXIMAL PHALANX

Osteochondral Fragments: Dorsoproximal Aspect of the Proximal Phalanx

Clinical Signs

The most common chip fracture in Thoroughbred (TB) racehorses involves the proximal dorsal aspect of the proximal phalanx and is an important differential diagnosis in any young TB with acute swelling or heat in a metacarpophalangeal joint. Affected horses rarely are lame for more than 2 days after a fracture and may never show overt lameness. An observant groom or trainer may notice an effusion with some heat, and forced flexion usually causes a painful response. Common historical findings include diminished performance, refusal to change leads, and bearing in or out, especially in the stretch.

Dorsal chip fractures can also be seen in yearlings and nonracehorses as an incidental finding. Most presumably occur as a traumatic event during early development and simply go unrecognized until presale radiographs are obtained. Such fragments occur equally in the forelimbs and hindlimbs, whereas traumatic injuries in active racehorses are most commonly seen in the forelimbs.

Diagnosis

Chip fractures of the proximodorsal aspect of the proximal phalanx of any size are not difficult to diagnose if they are displaced, but non-displaced fragments may require nearly perfect radiographs. Most chips occur on the prominence just medial to the median sagittal groove of the proximal phalanx. The left forelimb is slightly more commonly affected than the right forelimb in North American TBs. Bilateral chips are fairly common, and thus both fetlocks should be radiographed before surgery is considered. The fragments generally are seen best on a dorsolateral-palmaromedial oblique (Fig. 37-7) or lateromedial projection, depending on the obliquity of the fracture line and projection. Correct radiographic technique is especially important for small chips, because over-exposure burns out the lesion on a lateromedial view and under-exposed oblique views do not have enough penetration. Although most radiographically diagnosed fractures are dorsomedial, it is

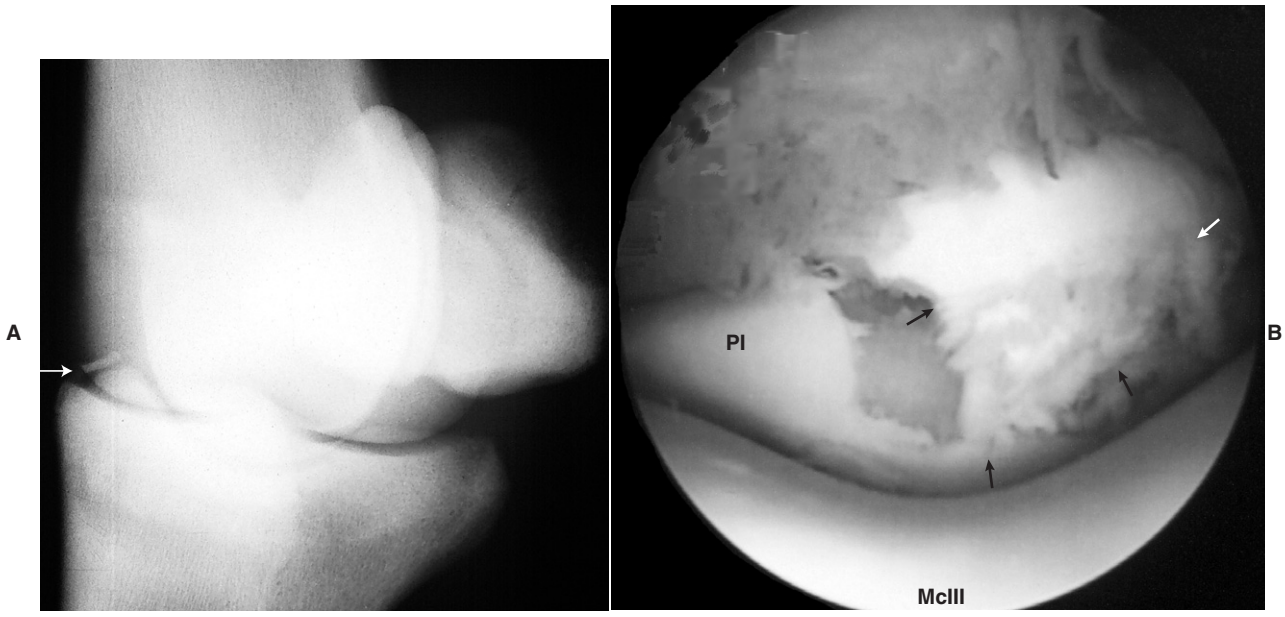

Fig. 37-7 **A,** Proximal dorsomedial osteochondral fragments (chip fractures; *arrow)* of the proximal phalanx are the most common chip fracture in Thoroughbred racehorses and can be seen on this dorsolateral-palmaromedial oblique view. **B,** Ideal treatment is early arthroscopic removal that allows a rapid return to training. The fragment *(arrows)* can be seen in this intraoperative view. *McIII*, Third metacarpal bone condyle; *PI*, proximal phalanx

important to evaluate arthroscopically the entire dorsal rim of the proximal phalanx, because small chondral fractures are common. Horses in which a dorsolateral fragment is seen radiographically usually also have a lesion on the dorsomedial side.

Treatment

Treatment for these chip fractures is less controversial since the advent of arthroscopy. The previous morbidity associated with large incisions on the dorsal aspect of the fetlock to remove rather small fragments made surgical treatment much less attractive. With arthroscopic technique, however, the fragments can be easily and atraumatically removed. An excellent prognosis after a short (approximately 4 to 6 weeks) postoperative convalescence can be given if there are no other degenerative changes in the joint.[9-11] In economically unworthy animals a period of 3 to 4 months' rest, followed by intra-articular medications as needed, also affords a favorable prognosis, especially if the fragment is not markedly displaced. In my opinion, superior conditioning when these horses return to work helps prolong careers. Injecting a horse with a corticosteroid after an acute chip fracture also can achieve a favorable short-term result and is always a consideration if the horse's immediate racing demands will not allow a significant period of rest. However, training and racing a horse with an unstable chip fracture may lead to more rapid degenerative changes in the fetlock. Such horses often have extensive score lines, thin cartilage, and more advanced arthritic changes if the fragment is removed arthroscopically at a later date.

Short Sesamoidean Avulsions (Osteochondral Fragments) from the Palmar Aspect of the Proximal Phalanx

Clinical Signs

Characterization of osteochondral fragments arising from the proximal palmar aspect of the proximal phalanx is difficult. Many consider these fragments to be manifestations of osteochondrosis because they are recognized in very young, untrained animals. It seems most probable that the fragments result from avulsion of a portion of the incompletely ossified proximal phalanx followed by development of a traumatic secondary ossification center[12,13] (Fig. 37-8). These fragments are unusual in the forelimbs, much more common in the hindlimbs (see Chapter 43), and most common in the Standardbred (STB) racehorse. They are also commonly recognized in European Warmbloods. Medial fragments are more common than lateral, but fragments can be biaxial and occur in several limbs. These fragments often are found on presale radiographs, and the relevance to lameness should always be questioned. Many successful athletic horses perform with these lesions. Racehorses in which the fragments affect performance rarely show overt lameness. TBs may be unable to maintain a straight path or fail to change leads properly, and STBs tend to ride a shaft or take a line. Horses in which the lesions cause significant lameness usually have an effusion. Most are not painful to fetlock manipulation.

Diagnosis

Oblique radiographic views are best for delineating fragments arising from the palmar, proximal aspect of the proximal phalanx. A flexed view helps to further separate the fragment from the base of the PSBs. This separation is useful with proliferative change at the distal dorsal margin of the PSBs or small, basilar sesamoid fractures. Proximal phalangeal fragments usually are well rounded and often are asymptomatic. It appears that many of these fragments develop in foals without manifestation of clinical signs until training or racing begins.

Training a horse after analgesia of the joint and recognition of improvement in gait should be diagnostic, but the subtle nature of the lameness can make this difficult. Clinical improvement after empirical intra-articular medication also supports the relevance of a radiographic lesion. Scintigraphic localization of a lesion may also help confirm the diagnosis, but in many racehorses, the radiopharmaceutical uptake may be increased in other parts of the joint.

Treatment

Treatment consists of arthroscopic removal of the lesion if the clinician is confident that the fragment or fragments are

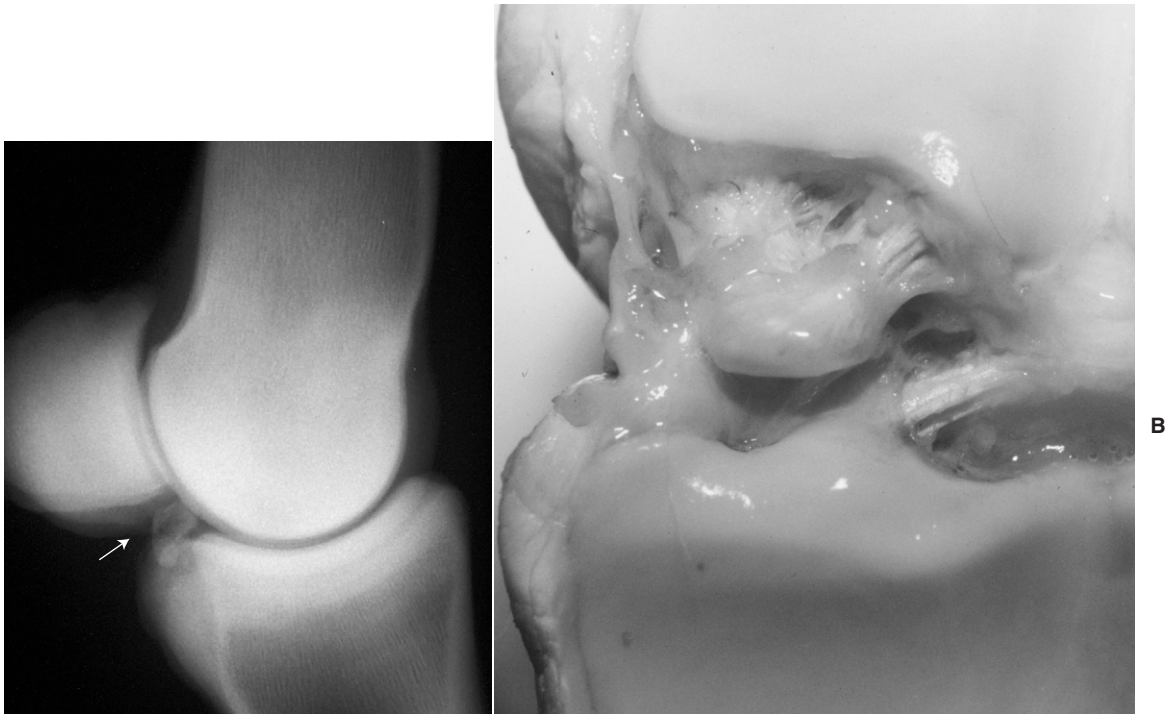

Fig. 37-8 **A,** Lateromedial radiographic view of a typical osteochondral fragment *(arrow)* avulsed from the proximal palmar margin of the proximal phalanx. Some consider this a form of osteochondritis dissecans. **B,** In the dissection the attachment of the fragment to the base of the proximal sesamoid bone by the short sesamoidean ligament is evident, as is the smoothly remodeled defect in the rim of the proximal phalanx.

the source of lameness or if the horse is going to public sale. The most common situation is that the fragments are identified before a horse enters training and the fragments are removed prophylactically. Arthroscopic removal is much less traumatic than arthrotomy for this type of lesion. The prognosis is excellent if the lameness diagnosis is correct.[14] If good arthroscopic technique with minimal soft tissue trauma is used, horses require a short convalescence (4 to 6 weeks). Intra-articular injection of corticosteroids with or without hyaluronan can also improve the clinical signs, but surgery is preferable.

Larger Palmar Fragments of the Proximal Phalanx

Large fracture fragments often cause a more obvious lameness than the more common rounded fragments from the proximal palmar rim of the proximal phalanx. They must be distinguished from more palmar fragments that involve the insertions of the true distal sesamoidean ligaments, because the latter are not equally good candidates for surgical removal. Very large, acute fractures of the palmar process of the proximal phalanx can be repaired with screws if recognized early (see Chapter 36). Large fragments occur in foals and weanlings but clinical signs may abate quickly or be non-existent. Lameness may be evident only when training or racing ensues. In foals, these fragments may be recognized radiographically if there is effusion, mild to moderate lameness, and obvious peri-articular swelling. Lameness is seldom commensurate with fragment size. The lesions in foals are largely cartilaginous and can be followed over time as they develop as a separate center of ossification. These fractures should be differentiated from an avulsion fracture at the site of the medial (or, less commonly, the lateral) collateral ligament (see page 359).

Major Fractures of the Proximal Phalanx: Sagittal Fractures

The most common major fracture of the proximal phalanx is a sagittal crack propagating from the proximal sagittal groove. Horses with long sagittal, displaced sagittal, and comminuted fractures developing from an original fracture in the sagittal or frontal plane have obvious localizing signs of swelling and pain. Horses with shorter sagittal fractures may show more subtle signs. Degree of lameness with short, incomplete sagittal fractures is remarkably variable; some horses are almost unable to bear weight, whereas in others the lameness is barely perceptible (see Chapter 36). There is usually effusion of the fetlock joint, pain on manipulation (flexion and twisting), and pain with firm pressure over the mid-dorsal aspect of the proximal phalanx. Sagittal fractures of the proximal aspect of the proximal phalanx are obvious radiographically if they extend more than a few centimeters from the joint surface, but a short, incomplete fracture is very frequently missed without good-quality radiographs. If a fracture is more than 10 days old, there is usually a definite periosteal change on the dorsal cortex seen on a lateromedial view just distal to the capsular attachments. Initially the change is very indistinct, but within a few more weeks, distinct periosteal new bone is obvious. Whenever proliferative change is seen in this area, the dorsopalmar views should be carefully evaluated for a sagittal fracture. Underexposure results in lesions being missed. These fractures are more common in STBs than TBs and frequently involve more than one limb. Even though these fractures are short and indistinct, they may cause surprisingly severe lameness. The clinician should always suspect such fractures, because blocking followed by high-speed training or pasture exercise may catastrophically propagate the fracture. For management and prognosis, see Chapter 36.

Dorsal Frontal Fractures of the Proximal Phalanx

See Chapters 36 and 43 for a discussion of dorsal frontal fractures of the proximal phalanx.

CONDITIONS OF THE PROXIMAL SESAMOID BONE

Sesamoiditis

Clinical Signs

The PSBs are an integral part of the suspensory apparatus and metacarpophalangeal articulation. They are susceptible to injury in all athletic horses, but particularly in those that perform at speed. Sesamoiditis is a clinically distinctive condition, although it is poorly characterized pathologically. There is usually pain with direct, firm palpation over the abaxial aspect of the affected PSB(s) and sometimes pain with fetlock flexion. Often, however, the lameness is manifest only after hard exercise.

Diagnosis

The lameness is eliminated with perineural, but not intra-articular, analgesia. Radiographic evidence of sesamoiditis involves four basic changes: marginal osteophytes, entheseophytes, enlarged vascular channels, and focal osteolysis. Marginal osteophytes occur at the proximal dorsal and distal dorsal extremities of the PSBs. Best seen on lateromedial views, these lesions represent later changes of generalized osteoarthritis of the fetlock joint and usually can be considered a poor prognostic sign. Entheseophytes occur along the palmar aspect of the proximal half of the abaxial ridge of the PSB or the distal third of the bone at the origin of the sites of the distal sesamoidean ligaments. These changes are frequently abaxial in location and therefore seen best on oblique projections. Interpretation of vascular channels in the PSBs is always subjective. The direction of any linear radiolucency is important to note; vascular channels have a radial orientation, whereas hairline fractures usually are closer to transverse (parallel to the ground).

True lytic change that is not associated with the normal trabecular pattern or vascular channels frequently indicates infection (see Chapters 73 and 75). Nuclear scintigraphy is exceptionally useful for diagnosis of sesamoiditis, can identify early changes, and can help confirm whether a chronic radiographic change is currently active. A complete series of scintigraphic views, including standing lateral, flexed lateral, and dorsal views can aid in exact localization of a lesion within a particular PSB.

Treatment

Treatment of horses with sesamoiditis is difficult and usually involves enforced rest and symptomatic treatment to counter inflammation and pain. Many racehorses with sesamoiditis are clinically helped by post-exercise icing and altered training regimens, such as swimming. It seems to be easier to manage older, proven racehorses with sesamoiditis by such methods than younger, unraced horses. Aspirin and isoxsuprine have been used, but there is no reliable evidence for efficacy. Shock wave therapy is probably the best current therapeutic option for active sesamoiditis, although its efficacy and mechanisms of action are still debatable.

Proximal Sesamoid Bone Fractures

Clinical Signs

Because the PSBs are such important elements in the suspensory apparatus and support of the fetlock joint, fractures are associated with obvious clinical signs of heat, swelling, and pain with flexion or direct pressure, as well as an obvious weight-bearing lameness.

Diagnosis

Lameness caused by most PSB fractures is eliminated or greatly reduced by intra-articular or perineural analgesia. Apical, mid-body, and basilar fractures of the PSBs usually are easy to diagnose radiographically with routine views. Abaxial fractures more frequently are missed and are best demonstrated by a lateral proximal-medial distal or medial proximal-lateral distal oblique projection that is tangential to the abaxial surface of the suspect PSB. This projection also is useful in determining whether the fracture is articular. Axial fractures usually are associated with displaced condylar fractures of the McIII or third metatarsal bone (MtIII). Diagnosis requires a well-penetrated dorsopalmar view that is well positioned. Even slight obliquity obscures the fracture line. Mid-body fractures frequently have wedge-shaped, comminuted fragments present at the abaxial margins. They should not be confused with the overlapping lines of the palmar and dorsal cortices that occur because of fracture displacement and failure to obtain the radiograph directly through the fracture line.

Ultrasonography can be a valuable adjunct in evaluating PSB fractures, because soft tissue insertions of the suspensory ligament (SL), intersesamoidean ligament, and distal sesamoidean ligaments can be simultaneously injured.

Apical fractures. Apical fractures comprising less than 30% of the bone occur primarily in racehorses, but they can occur in all types of horses. These injuries are caused by a combination of bending and suspensory tensile forces. In young STBs the fracture is particularly common in the right hind lateral PSB. Horses with apical PSB fractures are usually mildly to moderately lame unless a concomitant SL injury is present. Although lameness in some horses has been managed successfully with rest alone, arthroscopic removal of the fractured fragment generally is recommended. Both prognosis and convalescent time are dependent on fragment size and presence of suspensory desmitis. Horses with small, apical fragments without major SL ligament injury can resume training within 4 to 6 weeks after surgery, especially if they were fit at the time of injury. Horses with large fragments usually associated with suspensory branch injury may need 6 to 12 months of convalescence and have a poor prognosis.

Abaxial fractures. Abaxial fractures are true avulsion injuries involving the insertion of the SL on the abaxial surface. Most involve a narrow rim of the articular surface and can be removed by an arthroscopic approach. Displaced fragments should be removed, whereas extremely large fragments can be repaired with 3.5-mm screws. Horses with non-displaced fragments sometimes can be treated with rest. The prognosis depends on the size of the fragment and the concomitant suspensory compromise.

Basilar fractures. Basilar fractures of the PSBs are seen in all athletic horses and are treated according to configuration.[15,16] Smaller, wedge-shaped fragments that do not extend more than 50% to 75% of the dorsopalmar width of the PSB are clear candidates for surgical excision of the fragment (Fig. 37-9). Such fragments do not heal with rest and cause persistent lameness, presumably because of instability and incongruity at the articular surface. The prognosis with arthroscopic technique is at least 50% for return to athletic function because the distal sesamoidean ligaments are not disrupted. Horses are usually given 4 to 6 months of rest after removal of such fragments (3 months with very small pieces).

Horses with larger, basilar PSB fractures that extend to the palmar aspect of the bone have the poorest prognosis, because fractures often are significantly displaced, involve a greater proportion of distal sesamoidean ligament origin, and have more metacarpal or metatarsal injury (Fig. 37-10). They also are more difficult to repair than mid-body fractures because of the size and shape of the fracture. The prognosis for horses with large, displaced, or comminuted basilar PSB fractures is very poor for return to any significant level of athletic activity.

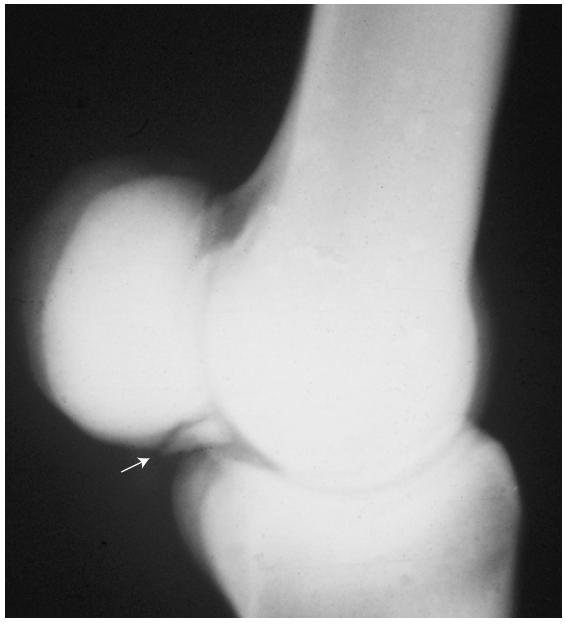

Fig. 37-9 Lateromedial view of a metacarpophalangeal joint. There is a basilar sesamoid fracture *(arrow)*. Small, triangular, basilar sesamoid fragments that do not involve the major distal sesamoidean ligaments are appropriate candidates for arthroscopic removal.

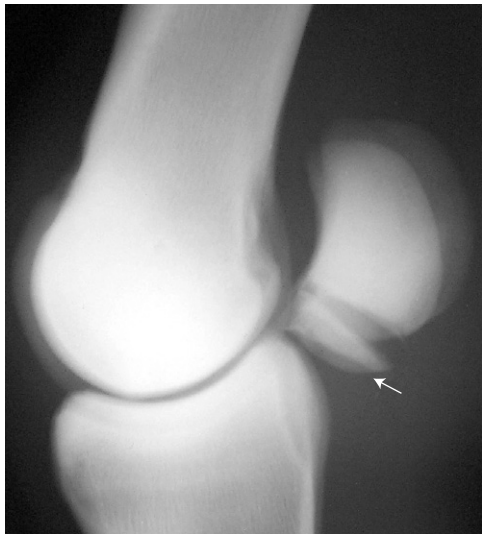

Fig. 37-10 Flexed lateromedial radiographic view of a metacarpophalangeal joint. There is a large displaced basilar sesamoid fracture *(arrow)*. Large basilar fractures are very difficult to successfully repair. Because there are extensive attachments of the distal sesamoidean ligaments, the prognosis associated with surgical removal is poor.

Some horses with severe osteoarthritis develop small basilar fractures. These fractures typically are more rounded or multiply fragmented than acute chip fractures (Fig. 37-11). Acute chip fractures have a sharply demarcated triangular shape. Horses with these degenerate basilar fractures are very poor candidates for any surgical treatment, and horses have a grave prognosis for return to athletic activity. Radiographic narrowing of the joint space between the PSB and McIII (Fig. 37-12), as well as the development of a radiographic "waist" in the distal aspect of McIII, are associated signs of severe osteoarthritis (Fig. 37-13).

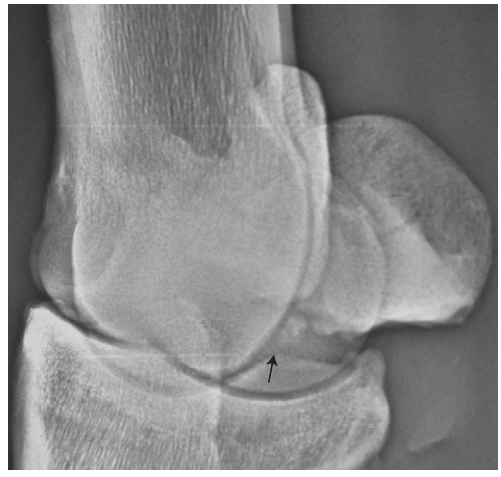

Fig. 37-11 Dorsomedial-palmorolateral radiographic view of a metacarpophalangeal joint. There are fragments distal to the proximal sesamoid bone *(arrow)*. Irregular, "crumbled" fragments from the base of the sesamoid usually are seen in joints with moderate to severe osteoarthritis. Note also the modeling of the articular margins of the proximal phalanx.

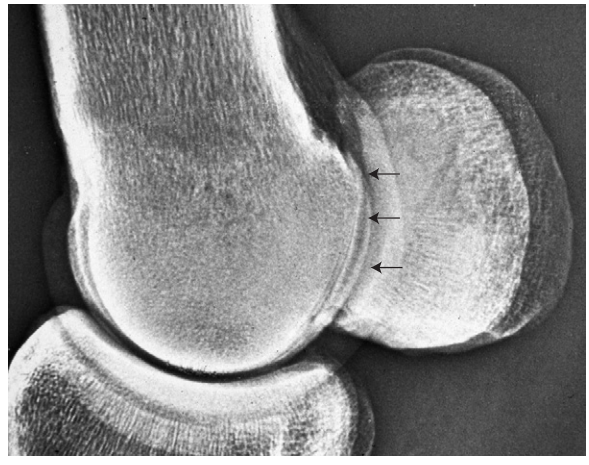

Fig. 37-12 Lateromedial radiographic view of a metacarpophalangeal joint. Note the loss of space between the proximal sesamoid bones and the third metacarpal bone *(arrows)*. Near complete loss of joint space between the third metacarpal bone and the proximal sesamoid bones can precede that between the distal third metacarpal bone and the proximal phalanx. Horses with this degree of cartilage damage are often candidates for euthanasia or arthrodesis.

Mid-body fractures. Mid-body fractures of the PSB can occur in any horse, but they usually are seen in racehorses. In North American TBs the medial bones of the forelimb are most commonly affected. In the STB, hind lateral PSB fractures are most common. Numerous surgical techniques have been used to repair such fractures, including cast coaptation alone, bone grafting and casting, lag screws (distal to proximal or proximal to distal) with or without cancellous grafting, and hemicerclage wiring and graft.[17,18] Regardless of the technique used, mid-body PSB fractures are serious injuries and require an extended period of rest after surgery. With surgery, 60% to

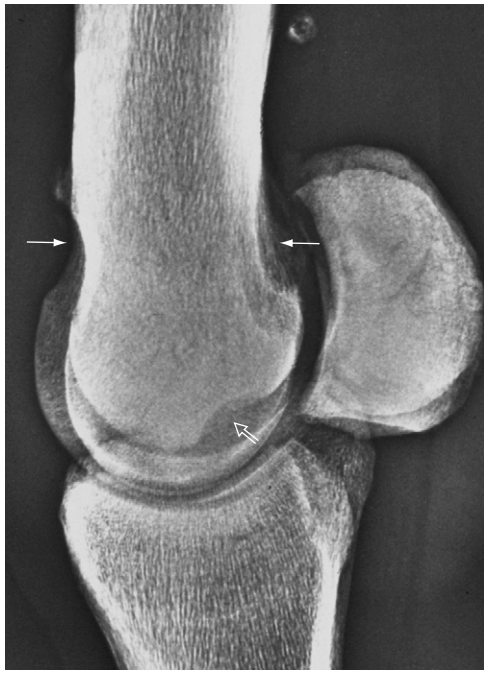

Fig. 37-13 Slightly oblique flexed lateromedial radiographic view of a metacarpophalangeal joint. Development of a "waist" around the proximal joint margin on the third metacarpal bone, both dorsally and palmarly *(closed arrows)*, is indicative of severe, chronic osteoarthritis. There is a pronounced defect of the distal palmar aspect of the third metacarpal bone *(open arrow)*.

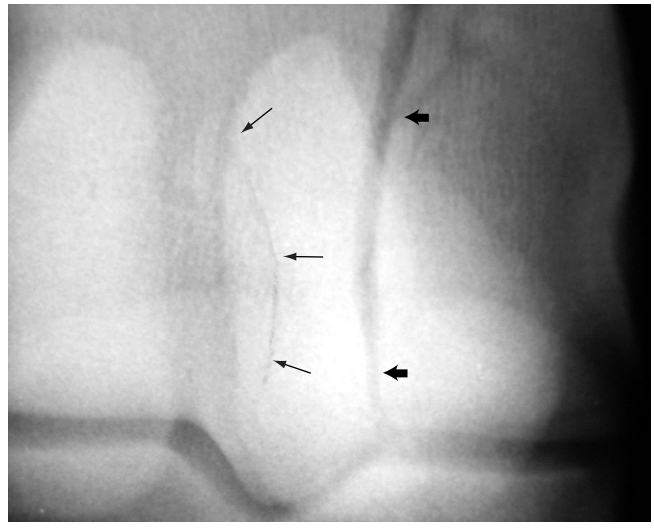

Fig. 37-14 Dorsopalmar radiographic view of a metacarpophalngeal joint. There is a displaced lateral condylar fracture of the third metacarpal bone *(short bold arrows)* and an axial fracture of the lateral proximal sesamoid bone *(arrows)*. Clinicians should look for axial proximal sesamoid bone fractures in every horse with a displaced lateral condylar fracture. This unusual type of fracture is associated with a poor prognosis for return to racing.

70% of horses with mid-body fractures can return to athletic function, but most horses drop substantially in class. The owner or trainer should be advised that almost a year of convalescence is required for horses with such injuries.

Axial PSB fractures. Axial PSB fractures are nearly always seen in combination with displaced lateral condylar fractures of the McIII or MtIII. They are recognized radiographically only on a dorsopalmar view and it is difficult to determine fracture depth (Fig. 37-14). Some fractures involve deep gouges in the articular surface, and most are avulsions by the intersesamoidean ligament. They usually are not treated specifically because of inaccessibility and the likelihood that the condylar fracture and overall joint damage will limit return to function. Recognition of an axial PSB fracture is important primarily as an indicator of more severe trauma to the joint and a poorer prognosis. The prognosis for a horse returning to race after this injury is extremely poor.[19] Early metacarpophalangeal arthrodesis should be seriously considered in horses with a displaced axial sesamoid fracture.

Intersesamoidean ligamentous injury without fracture is difficult to diagnose unless it is accompanied by radiographic lysis along the axial aspect of one or both PSBs (see Fig. 73-18, *A*). Affected horses often are extremely lame even at a walk. Scintigraphy frequently identifies an area of intense, focal increased radioisotope uptake. The intersesamoidean ligament also can be evaluated ultrasonographically to a limited extent. The ligamentous lesion may be visible and debrided arthroscopically, but it is not known if this alters the outcome. Horses generally are treated with support bandages and stall rest for several months. The prognosis for return to high-level athletic activity appears to be guarded, but a large series of horses has not been reviewed. A differential diagnosis for lytic lesions in the intersesamoidean region also includes osteomyelitis.[20] If an infection is the cause of the bone lysis, horses typically have more dramatic localizing signs, and the prognosis is extremely poor.

PSB fractures in young foals. PSB fractures in young foals (apical, abaxial, mid-body, and basilar) can occur at pasture and appear to be underdiagnosed, because clinical signs are surprisingly subtle and foals often are not thoroughly examined for mild lameness. Because most fractures involve only one PSB and the suspensory apparatus is still functional, severe lameness does not develop. Clinicians should look for subtle clinical signs of fetlock region swelling, pain with flexion, and pain with pressure over the PSBs in any foal with mild to moderate lameness. Several bones can be affected. Signs often abate quickly, especially in young foals (<2 months), in which these fractures are most common. Good-quality radiographs are essential, because the fractured apical, basilar, or abaxial fragments are often poorly mineralized. Unlike older horses, PSB fractures in foals frequently progress to form a bony union (Fig. 37-15). If the fragments have any displacement, however, an enlarged PSB can result. These oversized PSBs often are diagnosed in yearlings or 2-year-olds on prepurchase or presale radiographs. Surprisingly, enlargement of the fetlock joint is usually difficult to detect. Although some horses have raced successfully with enlarged PSBs, this abnormality can be a source of lameness as the horse proceeds in training.

PSB fractures in foals are essentially a management problem. Avoidance of situations in which a young, weak foal has to chase its dam around a large pasture to the point of exhaustion can prevent many fractures. It is essential to slowly return foals back to pasture if they have been stall confined for any reason. Early recognition of lameness in foals can allow diagnosis before the fragments become markedly displaced. Early diagnosis improves prognosis considerably, because minimally displaced fractures nearly always heal completely with rest alone. Uncontrolled exercise by the foal must be avoided, because the degree of displacement dictates the eventual PSB deformity.

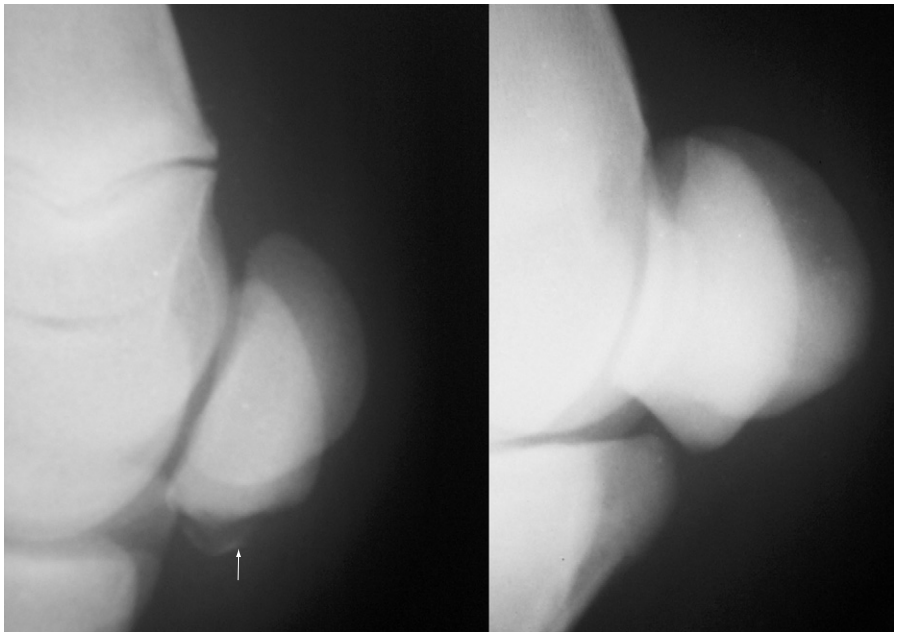

Fig. 37-15 Lateromedial radiographic views of a metacarpophalangeal joint of a foal. There is a displaced basilar fracture *(arrow)* from one of the proximal seasamoid bones on the first radiograph *(left)*. Radiographs obtained 4 months later *(right)* reveal healing of the fracture. Proximal sesamoid bone fractures in foals can heal, although displaced fractures result in an enlarged bone.

FRACTURES OF THE THIRD METACARPAL BONE

Condylar Fractures

Clinical Signs

The most important fracture of the fetlock joint is that involving the condyles of the McIII. They occur almost exclusively in racehorses and usually only when horses are running at racing speeds. Because the fractures involve a major weight-bearing surface, most horses are overtly lame, with clear localizing signs of pain and swelling. Most are very positive to any manipulation of the fetlock. Degree of lameness often does not directly correlate with the degree of displacement. Many horses with incomplete, acute fractures are more lame than one with a clearly displaced, complete fracture. This is particularly true with incomplete medial fractures. Although it appears that many horses that develop condylar fractures have preexisting subchondral damage of the distal, palmar condyle,[5] the majority do not have a strong history of previous fetlock lameness. (See Chapter 43 for fractures of the MtIII.)

Diagnosis

Radiographic diagnosis is not difficult in most horses. A dorsopalmar view should be centered over the condyle and the beam directed 15° to 20° downward. If the beam is too horizontal, the PSBs may obscure the distal articular surface of the McIII. Lesions may be missed if films are under exposed. The most common fractures involve the lateral condyle and clearly propagate toward the lateral cortex. Some extend only 1 to 2 cm proximally from the joint surface, and these can be particularly difficult to see without good-quality radiographs. Lateral condylar fractures rarely spiral or extend into the mid-diaphysis. Medial condylar fractures, however, usually extend up the diaphysis in a spiraling fashion or have an occult Y-shaped configuration at the mid-diaphysis. *It is essential to remember the difference between lateral and medial fractures*. Horses with medial condylar fractures *must* have a complete, high-quality set of radiographs that encompass the entire length of the McIII in an attempt to identify fracture configuration. Most importantly, the owner or trainer must be informed immediately of the significant risk associated with medial fractures.

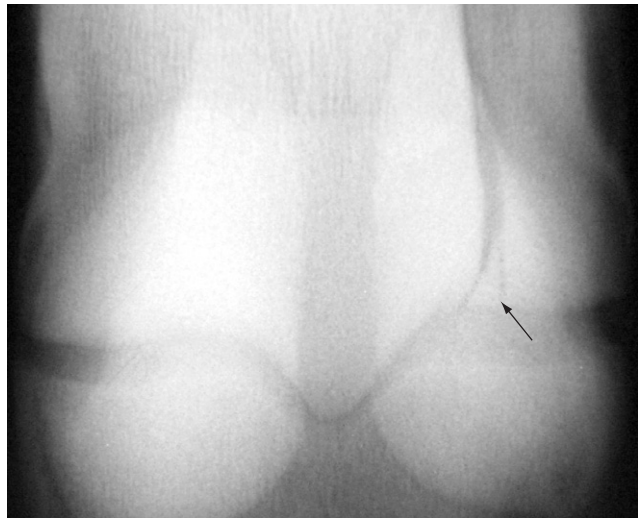

Fig. 37-16 Flexed dorsopalmar radiographic view of the metacarpophalangeal joint that shows a triangular-shaped defect *(arrow)* at the distal aspect of a lateral condylar fracture of the third metacarpal bone. Comminution can be seen only in this radiographic view and worsens the prognosis.

The prognosis for horses with displaced, lateral condylar fractures, regardless of treatment, is much poorer for return to function compared with those with non-displaced fractures. This is almost certainly the result of a combination of preexisting disease of the fetlock joint and the magnitude of damage done to the joint when the fracture occurs. Accurate prognosis requires radiographic examination of the palmar distal aspect of the McIII. The radiographer should slightly flex the fetlock and use a horizontal beam to obtain a dorsopalmar view (Fig. 37-16). This helps to define comminution of the condyle along the fracture margin, as well as subchondral lysis that may have preceded the fracture.

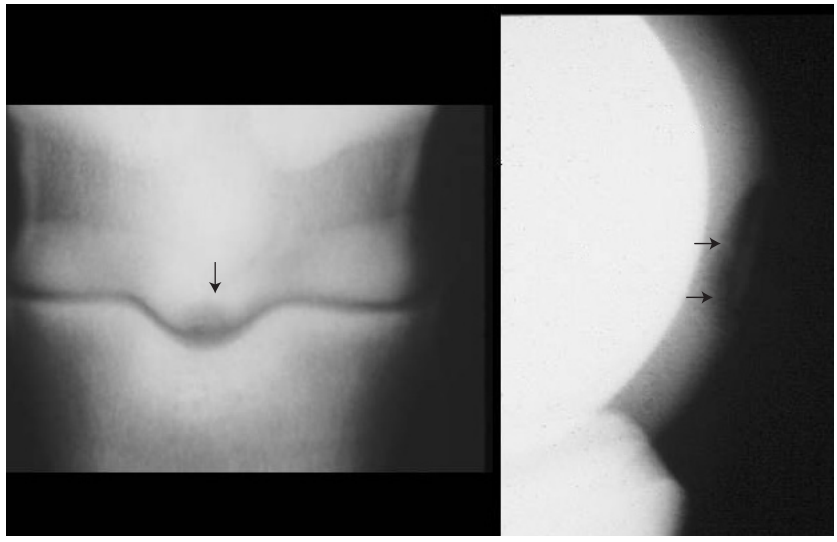

Fig. 37-17 Dorsopalmar *(left)* and flexed lateromedial *(right)* views of metacarpophalangeal joints. Note the radiolucent defect *(arrow)* in the sagittal image in the dorsopalmar view and the smoothly outlined defect in the dorsal aspect of the sagittal ridge *(arrows)* of the third metacarpal bone in the lateromedial view. Osteochondritis dissecans lesions of the sagittal ridge usually can be identified on dorsopalmar and flexed lateromedial views.

Most horses with condylar fractures are best treated with lag screws. Non-displaced or minimally displaced fractures can be repaired through stab incisions alone, but arthroscopic evaluation of the joint should allow a more accurate assessment of the prognosis. Displaced fractures require accurate reduction of the distal articular surface, so they must be reduced under arthroscopic guidance or through an open incision. Horses with medial condylar fractures should undergo surgery with extreme caution, because catastrophic failure in the mid-diaphysis can occur during anesthetic recovery or even several days to weeks after repair (see Chapter 43). Spiral fractures extending the length of the diaphysis may be successfully repaired with carefully positioned lag screws alone, but medial fractures that disappear in the mid-diaphysis should be repaired with a bone plate combined with lag screws across the condyle. Most complications of condylar fractures occur because of a failure to recognize potential problems before surgery. Even if certain complications cannot be entirely avoided, forewarning the owner of the risks is advisable.

Osteochondrosis

Sagittal Ridge of the Third Metacarpal Bone

The most common site of osteochondrosis in the metacarpophalangeal joint (excluding proximal palmar lesions) is the dorsal sagittal ridge of the McIII/MtIII (Fig. 37-17).[21] These lesions often are clinically silent, but mild to moderate effusion and lameness may be noted. They are often diagnosed in weanlings or on yearling presale radiographs. They are best seen on a dorsopalmar view or a slightly under-exposed flexed lateromedial projection. If no fragment is obvious and clinical signs are minimal, it is advisable to rest the horse for a couple of months, because some healing may occur, especially in younger animals. Arthroscopic debridement is an option, but the resulting bony defect will remain evident and the likelihood of a rewarding sale price is thus lessened. If the lesion does not fill in radiographically by the spring of its yearling year, arthroscopic evaluation may be suggested. A completely conservative (i.e., non-surgical) approach is justified if the clinical signs are minimal. The prognosis likely depends on the size of the lesion. Lesions more proximal on the sagittal ridge may have a better outcome than those closer to its distal aspect.

In older horses, traumatic chip fractures may occur on the distal sagittal ridge in a similar location to that of typical osteochondrosis (Fig. 37-18). This injury may occur in jumpers striking an obstacle with the fetlock joint in a palmarly flexed position. These fragments usually are unstable and should be arthroscopically removed. The prognosis is favorable if the fragment is fairly small.

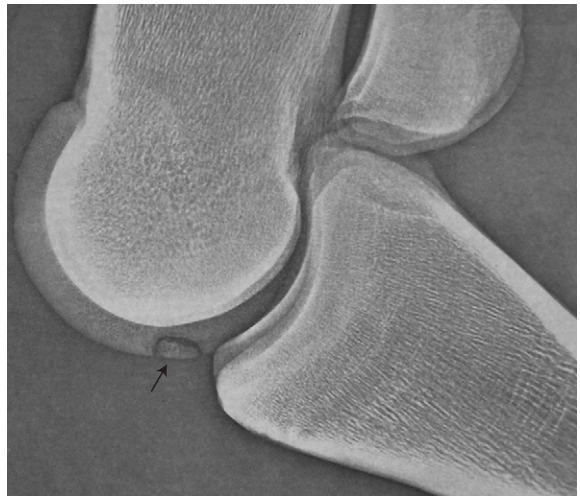

Fig. 37-18 Flexed lateromedial radiographic view of a metacarpophalangeal joint. There is a chip fracture on the distal dorsal aspect of the sagittal ridge *(arrow)* of the third metacarpal bone. Chip fractures of the sagittal ridge in older horses occur occasionally from direct trauma. In this radiograph the osteochondral fragment is located somewhat more distal than most, and they may be missed in a weight-bearing projection.

Osseous Cyst-Like Lesions of the Third Metacarpal Bone

Osseous cyst-like lesions of the distal Mc III/Mt III occur on the weight-bearing surface of the condyle and may be a manifestation of osteochondrosis. Most occur in the medial condyle and are diagnosed in yearlings or early in training as a 2-year-old. The lesions are easy to recognize radiographically, and horses have obvious lameness exacerbated by lower limb flexion. Perineural analgesia and scintigraphy also localize the problem. Surgical debridement has been the typical treatment, and the reported results are unexpectedly favorable.[22]

Collateral Ligament Injury

The fetlock joint has strong anatomical constraints, and the equine limb and gait have a strongly sagittal orientation; therefore mediolateral instability is relatively uncommon except after severe trauma (e.g., foot in a hole or lower limb in a gate or fence). Mild forms of collateral desmitis are suspected in horses with pain on fetlock manipulation, focal pain, and swelling over the affected ligament or lameness exacerbated with valgus or varus stress. The diagnosis is confirmed by ultrasonographic examination. Radiographs usually are not helpful unless there is an avulsion fracture or obvious asymmetry of the joint surface. Asymmetry is more likely to be demonstrated on non–weight-bearing, valgus or varus stressed dorsopalmar (plantar) radiographs.

Treatment of collateral ligament injury usually consists of rest and adequate external coaptation. The fetlock is an inherently stable joint in the sagittal plane; thus minimal support is necessary unless the injury is severe. If the joint is very unstable, a cast extending to the proximal metacarpal region is maintained for 4 to 6 weeks. If the injury is not overtly unstable, a single polyvinyl chloride pipe splint applied on the side ipsilateral to the collateral injury should be adequate. The duration of rest should be based on clinical soundness, palpable stability, and the ultrasonographic appearance of the ligamentous tissues (see Chapter 17). Shock wave therapy for insertional desmopathies may be beneficial, but firm data are still unavailable.

In young horses (usually yearlings or 2-year-olds), fragments can be avulsed from the collateral ligament's insertion on the proximal phalanx. These sometimes are mistaken for dorsal chip fractures on oblique views, but careful scrutiny reveals that the fragment is too far abaxial for a typical chip fracture (Fig. 37-19). The fracture can also usually be seen on a dorsopalmar (dorsoplantar) view. These fragments often are along the articular margin and do cause lameness. Arthroscopic removal should be performed, but it can be difficult because of the limited space on the medial or lateral aspects. The arthroscope portal should be dorsoproximal on the same side as the lesion. The instrument portal should be just dorsal to the edge of the collateral ligament. The fragments may be tightly attached along the abaxial margin, and an arthroscopic scalpel or other sharp dissection tool may be needed to separate them.

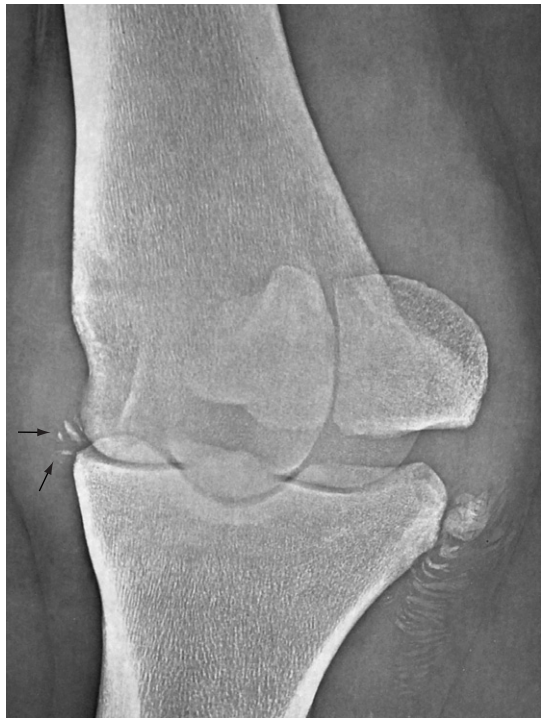

Fig. 37-19 Dorsomedial-palmarolateral oblique radiographic view of a metacarpophalangeal joint. Note the extensive periarticular soft tissue fragments *(arrows)* on the dorsal aspect of the joint. Collateral ligament avulsion results in dramatic swelling of the fetlock region. Removal of loose articular fragments and external coaptation can result in athletic soundness. This horse became a highly successful steeplechaser.

Traumatic Disruption of the Suspensory Apparatus

Traumatic disruption of the suspensory apparatus is almost exclusively an injury that occurs in the forelimbs of TB racehorses. It occurs more often in North America than Europe, although any horse running at high speed may suffer it, including young foals that are chasing the dams in the pasture. Speed alone does not account for the injury, and it is likely that fatigue of the flexor muscles supporting the fetlock and digit leads to higher stresses in each component of the suspensory apparatus. There also may be a history of the horse being bumped or making a misstep.

The history and clinical presentation of traumatic disruption of the suspensory apparatus in adult horses are straightforward, since the horses are either breezing or racing and come up acutely and severely lame. The fetlock joint drops as the horse attempts to bear weight. Many horses become anxious or even frantic as they attempt to control the injured limb. There is obvious swelling and pain over the site of the injury. In foals the diagnosis is often not made as quickly.[23] The typical history of a foal with traumatic disruption of the suspensory apparatus (or lesser injuries of the suspensory apparatus) is of being turned out in a large field with its dam and other mares and foals shortly after birth, or after confinement to a box stall for an extended period. As the mare runs with the other horses, the foal attempts to keep up, running at speed and to the point of exhaustion. This results in the same combination of speed and fatigue that leads to this injury in the racehorse. The clinical signs of complete disruption of the suspensory apparatus are similar, but less dramatic than in adult horses.

Fetlock drop and severe lameness are seen with complete disruption of any portion of the suspensory apparatus. The most common injury in both adults and foals is fracture of both PSBs. The fractures are often comminuted, especially in the basilar portions. The second most common type of traumatic disruption of the suspensory apparatus is complete avulsion of the distal sesamoidean ligaments. This is easily recognized radiographically by the proximal displacement of the intact PSBs (Fig. 37-20). The least frequently recognized type of traumatic disruption of the suspensory apparatus is a complete tear of the SL body or both branches. The Paso Fino, Peruvian Paso, and some other older horses have a degenerative condition of the SL that leads to suspensory apparatus failure that is particularly problematic in the hindlimbs. These horses do not have acute severe lameness (see Chapter 73). In horses with any traumatic disruption of the suspensory apparatus, the excessive extension of the fetlock may result in stretching and damage to the digital vessels, which may cause avascularity or hypovascularity of the digit. Simultaneous damage to the superficial and deep digital flexor tendons is also common and should be assessed ultrasonographically. Although PSB fractures and suspensory injuries are both common hindlimb injuries, the severe, breakdown injuries usually involve forelimbs.

Traumatic disruption of the suspensory apparatus is virtually always a career-ending injury. The only exceptions are

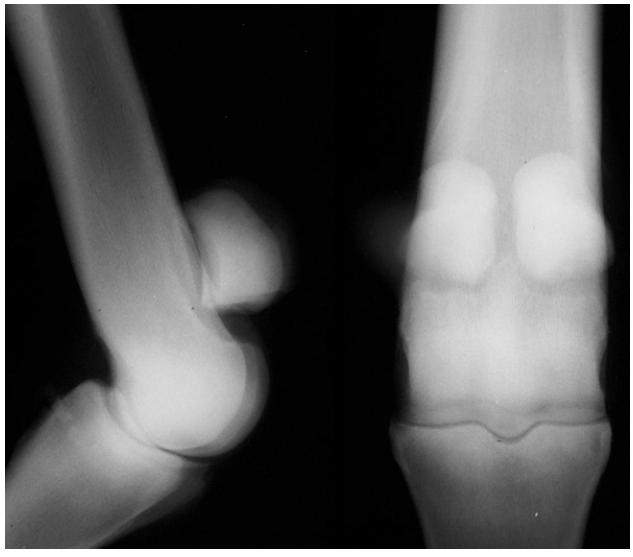

Fig. 37-20 Lateromedial *(left)* and dorsopalmar *(right)* radiographic views of a metacarpophalangeal joint. Note the overextension of the joint in the lateromedial view and the extreme proximal displacement of the proximal sesamoid bones, which are intact. This is the result of disruption of the distal sesamoidean ligaments, which is a clear-cut indication for metacarpophalangeal arthrodesis.

simple, displaced mid-body fractures of both PSBs that can be individually repaired and suspensory body or branch injuries in some STBs. It is important to recognize the severity of the injury so that an informed decision can be made concerning treatment. Most horses can be saved as pasture sound or breeding animals, but treatment often is prolonged and expensive regardless of the therapeutic approach selected.

First Aid

The most important first aid treatment is application of an appropriate splint that aligns the McIII and phalanges in a column to prevent bending of the fetlock and loading of the suspensory apparatus. Although homemade splints can be fashioned to accomplish the same objective, commercially available splints are quick, easy to apply, and very effective for temporary immobilization (Kimzey LegSaver splint; Kimzey, Inc., Woodland, CA). Most horses are less anxious and able to move comfortably immediately after the splint is applied. If a prefabricated splint is not available, a lightly padded bandage with a splint applied to the dorsal aspect of the metacarpal region, phalanges, and hoof serves the same purpose if the heel is kept elevated. This can be done by taping the heel and a heel wedge with non-elastic tape (e.g., duct tape) to the dorsal splint. Excessive padding should be avoided because it allows dorsal flexion within the bandage and shifting of the splint. Other first aid measures include the administration of NSAIDs, intravenous fluids to replace water and electrolytes lost through sweating, and broad-spectrum antibiotics. Antibiotics should be given even if the fracture is ostensibly closed. High concentrations of antibiotics in the fracture hematoma are desirable, because skin abrasions and lacerations over hypovascular tissue are common. The vascularity of the foot should be assessed by palpation of the digital vessels and assessment of hoof temperature. Horses that are extremely anxious and difficult to calm should be given xylazine, with or without butorphanol, as needed for restraint and to prevent further self-injury by the horse.

Non-Surgical Management

Non-surgical management involves long-term splinting of the injury in an attempt to achieve sufficient fibrosis of the injured tissues so that satisfactory support of the fetlock returns. The primary advantage of this approach is the avoidance of the obvious risks associated with operating in a hypovascular and possibly contaminated site. The surgical and technical expertise and equipment required are modest, and the expense of *initial* treatment is relatively small. There are some disadvantages of non-surgical management. The fibrosis that develops may not be adequate to support the fetlock in a horse exercising at pasture; this is more problematic with distal sesamoidean ligament avulsions than fractured PSBs. Many horses are uncomfortable on the splinted limb and unable to use it normally because of the abnormal posture, and they are at risk of supporting limb breakdown and laminitis. Long-term splinting also demands meticulous daily bandage changes to prevent rub sores at the proximal dorsal metacarpal region and suppurative dermatitis of the palmar pastern and heel bulbs.

The technique of successful long-term splinting of horses with traumatic disruption of the suspensory apparatus primarily involves meticulous nursing care. Most clinicians use a prefabricated splint because of the ease of its removal and application. The Kimzey splint can be improved for long-term use by welding a heel extension on it to increase its bearing surface area. This helps minimize the tendency to develop rub sores at the proximal, dorsal edge of the splint. The prefabricated splints are not perfectly fitted for every horse. Therefore some modifications in padding may be required to avoid excessive pressure on the proximal, dorsal metacarpal region. Regardless of which type of splint is used, the leg should be checked daily for developing sores or dermatitis, particularly in the palmar pastern. If the leg is washed, it should be dried before applying the bandage. If skin infection develops, it can be very difficult to manage. Both systemic and local antibiotics are usually necessary.

The most critical decisions in management of a traumatic disruption of the suspensory apparatus with splints concern removal of the splints and a gradual return to more normal fetlock and digit joint angles. Upright splinting is used for most horses for approximately 6 weeks but many, particularly those with distal sesamoidean ligament avulsions, may require much longer. The process of dropping the fetlock should be gradual. This can be done by splinting at decreasing angles (which is difficult with prefabricated splints), or using a fetlock support shoe incorporating an adjustable sling. Horses should be confined to a stall for a prolonged period (4 to 6 months), because a single misstep can lead to tearing of the still maturing scar tissue. Although external coaptation has worked well in individual horses and in some clinicians' hands, the long-term results do not appear to be as good as surgical arthrodesis. Although surgical fusion has considerable potential complications, the solid fusion of the joint is not likely to slowly fail and cause progressive discomfort when a mare or stallion enters the breeding shed. Horses with conservatively managed injuries appear to have more problems with long-term comfort.

Surgical Management

Surgical management of traumatic disruption of the suspensory apparatus generally involves fetlock arthrodesis, although repair of simple, displaced mid-body proximal sesamoid bone fractures can be performed to preserve some joint function. The major advantage of arthrodesis is that the procedure usually affords the horse immediate, comfortable use of the limb and avoids the complications of prolonged overuse of the contralateral limb. Because the fixation is usually very stable, post-operative immobilization is minimal and the various complications of casts and splints can be avoided. The primary disadvantage of surgical arthrodesis is the increased risk of infection. An additional consideration is that successful

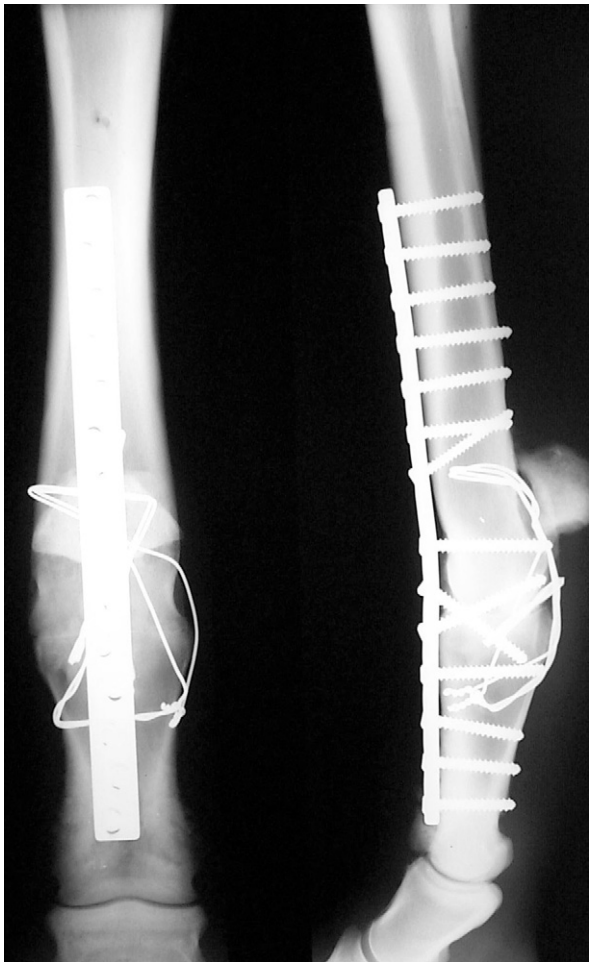

Fig. 37-21 Dorsal plating with a palmar tension band is the standard technique for metacarpophalangeal arthrodesis after traumatic disruption of the suspensory apparatus.

arthrodesis requires both surgical expertise and investment in equipment and facilities.

The most widely used technique for fetlock arthrodesis is that of a dorsal plate with creation of a tension band on the palmar aspect (Fig. 37-21).[24] Dorsal plating without a tension band on the palmar aspect of the fetlock is ill advised because the plate is cycled in bending on the joint's dorsal surface and fatigues and eventually breaks. The specific technique used to create the tension band depends on the type of traumatic disruption of the suspensory apparatus, but both methods involve the same surgical approach. After luxation of the metacarpophalangeal joint, the articular cartilage is removed and multiple holes are drilled into the subchondral bone. Holes are drilled in the McIII and proximal phalanx, through which wires are passed to form a figure-eight tension band. A 14-hole dynamic compression plate is pre-bent to a fetlock angle of 150° to 160° and positioned dorsally using 5.5-mm screws.

If the distal sesamoidean ligaments are intact and the attached PSB fragments are large enough, the tension band wiring can be avoided. Instead, the fetlock is positioned in approximately 5° of palmar flexion and a 5.5-mm lag screw is placed through the McIII into each PSB. When the pre-bent plate is placed, the distal sesamoidean ligaments then serve as the tension band that prevents the plate from bending and eventual fatigue failure. This is the preferred surgical technique in horses with the less common injury involving disruption of the suspensory ligament body or both branches.

A fiberglass cast is placed for anesthetic recovery and is left in place for 10 to 14 days if the horse wears it comfortably. If the cast creates obvious discomfort, it should be removed immediately because cast sores can be a serious problem and the fixation is secure enough that the cast is unnecessary. If the horse wears the cast well, it should be changed with the horse standing at 10 to 14 days. The second cast is left in place 4 to 5 weeks if the horse wears it comfortably. If there is any evidence of a cast complication, it should be replaced with a bandage. Radiographs are obtained at regular (approximately 6-week) intervals. If healing is routine, most horses have a solid fusion by 3 to 4 months after surgery and can begin some limited turnout in a small paddock.

The major complication of bone plating for fetlock arthrodesis is infection. However, because this repair is very strong and stable, healing can occur even in the face of infection. The combination of instability and infection, however, rarely succeeds and reoperation of unstable, infected sites is usually necessary to achieve fusion. Implant failure also is a concern, but proper technique and the use of 5.5-mm screws prevent this complication. Additional strength and fatigue life can be obtained by luting the plate, although this is not usually necessary.

A common long-term complication associated with traumatic disruption of the suspensory apparatus is osteoarthritis of the proximal or distal interphalangeal joints. The joints are stressed more by the fusion of the fetlock joint and often by the presence of serious injury to the flexor tendons and distal sesamoidean ligaments that insert on the phalanges.

Many horses with traumatic disruption of the suspensory apparatus sustain open, contaminated fractures or such extensive soft tissue damage that internal fixation may carry an unacceptable risk of infection. External skeletal fixation is an alternative means of managing such injuries (see Chapter 88).[25]

The prognosis for any horse with traumatic disruption of the suspensory apparatus depends on the specific nature and severity of the injury. Horses with distal sesamoidean ligament avulsions have a poorer prognosis than those with displaced PSB fractures, since the latter tend to form fibrous scar tissue more quickly. Horses with the relatively uncommon suspensory body or bilateral branch tears have the best prognosis, because they seem to heal more quickly and have less fetlock joint instability. At least 60% to 75% of horses with closed disruptions of the suspensory apparatus and an intact blood supply should be saved with proper treatment. Open injuries obviously have a much poorer prognosis regardless of the therapy chosen, and open injuries with vascular compromise have a very poor prognosis.

Complications of traumatic disruption of the suspensory apparatus, regardless of treatment, include laminitis or breakdown of the contralateral limb. The primary complications of long-term splinting are rub sores and inadequate fibrosis, which lead to instability and chronic pain. Surgical arthrodesis offers the advantage of comfort more quickly, but the risks of infection are much greater. Incorrect surgical technique also can lead to mechanical failure of the internal fixation.

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CHAPTER • 38

The Metacarpal Region

Sue J. Dyson

ANATOMY

This chapter discusses the examination and diagnosis of injuries to the metacarpal region of the horse. The detailed anatomy of the deep digital flexor tendon (DDFT) (see Chapter 71), the accessory ligament of the deep digital flexor tendon (ALDDFT) (see Chapter 72), the superficial digital flexor tendon (SDFT) (see Chapter 70), and the third interosseous muscle or suspensory ligament (SL) (see Chapter 73) is discussed elsewhere.

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The proximal aspect of the third interosseous muscle or SL attaches to the proximal palmar aspect of McIII and the palmar carpal ligament and lies between McII and McIV. Large exostoses on the axial aspect of McII or McIV have the potential to impinge on the abaxial border of the SL. The carpal sheath (see Chapter 76) extends through the proximal one third of the metacarpal region. The amount of fluid in the carpal sheath varies between individuals but is usually bilaterally symmetrical.

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metacarpophalangeal joint the sheath passes through the inelastic canal created by the palmar annular ligament (PAL), the palmar fibrocartilaginous surfaces of the PSBs, and the intersesamoidean ligament. Within the DFTS, proximal to the sesamoidean canal, the SDFT forms a ring (the manica flexoria) around the DDFT. In the palmar mid-sagittal plane a synovial reflection, the vincula, attaches the SDFT to the DFTS wall. Proximal to the manica flexoria the DDFT is attached medially and laterally by mesotendon to the DFTS wall. The lateral mesotendon is more substantial and extends farther distad.

The palmar and palmar metacarpal nerves innervate the palmar metacarpal region. The SL is innervated by the medial and lateral palmar metacarpal nerves, branches of the deep branch of the lateral palmar nerve. This nerve receives contributions from the ulnar and median nerves. The medial palmar metacarpal nerve has fibers only from the median nerve, whereas the lateral palmar metacarpal nerve has fibers from the ulnar and median nerves.[1] The SDFT and DDFT are innervated by the palmar nerves.[2]

In the proximal metacarpal region the palmar nerves lie beneath relatively thick fascia, whereas farther distally they are more superficial. In the proximal 5 cm of the metacarpal region the palmar metacarpal nerves and the distal palmar outpouching of the carpometacarpal joint capsule are in close proximity.

DIAGNOSIS

Clinical Examination

The metacarpal region should be examined visually from all angles to identify any changes in contour caused by swelling. If the limb is hairy, subtle swellings may easily be missed, especially in heavier breeds of horses, and if injury to the metacarpal region is suspected, then clipping the hair to facilitate examination can be useful. Palpation of the metacarpal region should be performed systematically with the limb bearing weight and with the limb semi-flexed. With the limb bearing weight the contour of the dorsal and palmar aspects of the limb should be straight. Distention of the medial palmar vein may reflect local inflammation. The size of the SL and its branches is assessed by running both thumbs down the dorsal and palmar borders from proximally to distally, medially and laterally. With the limb lifted the margins of each of the SDFT, DDFT, ALDDFT, and SL should be carefully palpated to detect rounding of the margins or enlargement. Each structure should be squeezed, starting proximally and working distally, gently at first and then with increasing pressure to determine whether pain can be elicited. Careful comparison should be made with the contralateral limb, bearing in mind that lesions may be present bilaterally. Assess the response in light of the horse's temperament and the recent work history. Abnormal stiffness of a structure may reflect previous injury. It is necessary to roll away the flexor tendons and compress the SL against the palmar aspect of the third metacarpal bone to assess the most proximal part of the SL.

The axial and palmar margins of McII and McIV should be palpated to identify any new bone formation (a splint) and to determine whether applied pressure causes pain. It is also important to assess whether the axial margin of the splint is clearly demarcated from the SL. Firm pressure should be applied to the dorsal aspects of McIII to identify pain.

Local Analgesic Techniques

When local analgesic techniques are performed in the metacarpal region, it is important to recognize the potential for local anesthetic solution to diffuse proximally from the site of injection and thereby desensitize structures farther proximally, partially or completely. Minimum volumes of local anesthetic solution should be used (maximum 2 ml per site and less in ponies) to minimize the risks of misinterpretation. The horse should stand still after injection before reassessment of the gait and should be re-evaluated no more than 10 minutes after injection. Even then false-positive results may be seen.

The metacarpal region seems particularly prone to development of swelling at the site of injection, which potentially may be permanent. The limb should be thoroughly scrubbed with chlorhexidine before injection to minimize the risk of adverse reaction and because of the potential for inadvertent injection into a synovial cavity (the DFTS, carpal sheath, or carpometacarpal joint capsule). The use of non-irritating local anesthetic solution (mepivacaine) is strongly recommended. It is also suggested that a stable bandage be applied to the limb for about 18 hours after injection.

Perineural analgesia of the palmar nerves at the level of the base of the PSBs desensitizes the foot and pastern regions, but in some horses it may also alleviate pain from the metacarpophalangeal joint, PAL, and DFTS. In horses with pain arising from the metacarpal region, lameness often appears paradoxically worse after palmar (abaxial sesamoid) nerve blocks. Complete desensitization of the distal third of the metacarpal region requires perineural analgesia of the palmar nerves in the mid-metacarpal area and the palmar metacarpal nerves distal to the distal aspect of McII and McIV, the so-called low palmar or four-point block. Care should be taken to avoid inadvertent injection into the DFTS. Proximal diffusion may result in partial alleviation of pain associated with a proximal metacarpal lesion, especially proximal suspensory desmitis. If a unilateral lesion is suspected, the block may be performed medially or laterally alone.

Desensitization of one specific PSB can be achieved by blocking the sesamoidean nerve by introducing a needle between the insertion of the SL and the dorsal aspect of the abaxial surface of the sesamoid bone. The needle is directed toward the apex of the bone, and 0.5 ml of local anesthetic solution is injected.[3]

Elimination of pain from the entire metacarpal region requires blocking the palmar nerves immediately distal to the carpus and the deep branch of the ulnar nerve (and thus the palmar metacarpal nerves). However, often it is useful to be more specific and desensitize the deep structures (the palmar aspect of McIII and the SL) or the more superficial structures (SDFT, DDFT, and ALDDFT). Perineural analgesia of the deep branch of the lateral palmar nerve or of the palmar metacarpal nerves by subcarpal injection should not remove pain from the fetlock or distal limb. Blocking the palmar metacarpal nerves immediately distal to the carpus runs the risk of inadvertent injection into the distopalmar outpouchings of the carpometacarpal joint or spread of the local anesthetic solution by diffusion. Theoretically the risk of failure to desensitize the most proximal aspect of the SL exists, but diffusion of local anesthetic solution usually removes pain. The response should be compared with that following intra-articular analgesia of the middle carpal joint. Occasionally subcarpal analgesia of the palmar metacarpal nerves relieves pain associated with a primary middle carpal joint lesion better than intra-articular analgesia. Intra-articular analgesia of the middle carpal joint may relieve pain associated with proximal suspensory desmitis or a palmar cortical fatigue fracture of McIII.

Perineural analgesia of the lateral palmar nerve[4] entails less risk of affecting the middle carpal joint, but it does not eliminate it totally. The potential to remove pain from the lateral aspect of the more distal part of the limb also exists.

Theoretically, blocking the ulnar nerve should not completely remove pain associated with the proximal SL or the

proximal palmar aspect of McIII because of the contribution of fibers from the median nerve to the medial and lateral palmar metacarpal nerves. However, in practice it generally does.

Perineural analgesia of the palmar nerves (2 ml per site) should not desensitize the deeper structures (McIII, SL) but should alleviate pain from the more superficial structures (SDFT, DDFT, and ALDDFT). Local infiltration around a painful exostosis of McII or McIV seems to be the most effective way of determining whether pain from the exostosis is contributing to the lameness observed.

Intra-thecal analgesia of the DFTS usually results in improvement in lameness associated with pain from within the sheath, but a better response is frequently seen after perineural analgesia of the palmar and palmar metacarpal nerves. Injection into the DFTS is most easily performed on the palmar midline of the pastern region, distal to the proximal digital annular ligament. The likelihood of inducing iatrogenic hemorrhage at this site is small, and retrieval of synovial fluid usually is easier. If distention of the DFTS is only mild, compression of the proximal part of the sheath by an assistant to increase distention in the palmar pouch in the pastern can be helpful. Injection into the carpal sheath is usually only indicated if the sheath is distended (see Chapter 76).

IMAGING

Radiography

Radiographic examination of the metacarpal region is usually tailored to each particular horse concerning the views required and the exposure factors used. With localized periosteal new bone, often finely coned-down views, use of soft exposures (low kilovolts [peak]) is required to demonstrate the lesion best. Several similar views varying slightly in obliquity may be required, rather than a full series of dorsopalmar, lateromedial, dorsolateral-palmaromedial oblique, and palmarolateral-dorsomedial oblique projections. Therefore a flexible approach must be used, depending on the initial clinical signs. Little soft tissue covers the metacarpal region; therefore a grid is not required. A high-definition screen with slow-speed film provides the best detail. Diffuse swelling in the metacarpal region makes images appear flatter, lacking contrast. Exposure factors may need to be increased slightly.

Superimposition of McII and McIV over McIII can create confusing radiolucent Mach lines (see page 158). The nutrient canal in McII and McIV varies in prominence[5] and should not be confused as a fracture. Ossification varies between McII and McIV and McIII in normal horses.

Ultrasonography

Ultrasonographic examination of the metacarpal region is discussed in detail elsewhere (see Chapters 16 and 70 to 73). In large, cob-type animals the skin and underlying subcutaneous tissues in the metacarpal region may be thick, making it difficult to obtain high-resolution images. Dense stubble may persist after fine clipping of the hair coat, making it difficult to maintain good contact. Deep skin folds may further complicate the issue. For these horses it may be necessary to amplify the power and gain controls of the ultrasound machine and increase the focal depth of the transducer. Image quality may be enhanced by application of copious amounts of the ultrasound coupling gel to the skin for at least 15 minutes before imaging.

Nuclear Scintigraphy

Blood pool (soft tissue phase) and bone-phase (delayed) images of the metacarpal region are particularly useful in horses in which pain has been localized to the metacarpal region but in which no significant radiological or ultrasonographic abnormalities have been identified. Many exostoses involving McII and McIV, although clinically inactive, are associated with moderately increased uptake of the radiopharmaceutical. Clinically silent enostosis-like lesions also may have increased radiopharmaceutical uptake.

DIFFERENTIAL DIAGNOSIS

Bucked or Sore Shins and Saucer (Dorsal Cortical) Fractures of the Third Metacarpal Bone

See Chapter 109 for a discussion of bucked shins and saucer fractures of the third metacarpal bone.

Medial and Lateral Condylar Fractures of the Third Metacarpal Bone

See Chapter 37 for a discussion of medial and lateral condylar fractures of the third metacarpal bone (page 357).

Incomplete, Longitudinal Palmar Cortical Fatigue Fractures of the Third Metacarpal Bone and Stress Reactions

Incomplete palmar cortical fatigue fractures of McIII are relatively common and invariably involve the medial aspect of the bone,[6-9] may involve the metaphyseal and proximal diaphyseal region, and sometimes extend proximally to involve the carpometacarpal joint. They are believed to be fatigue or stress fractures, because sclerosis may be present radiographically when lameness is first recognized, indicating previous bony reaction.

These fractures occur most commonly in young horses, but they also occur in skeletally mature horses. In some horses a recent increase in work intensity can be identified, which may be a predisposing factor. These fractures have been identified in a variety of sport horses, including horses used for flat and harness racing, National Hunt racing and point-to-point racing, dressage, horse trials, and endurance riding.

Clinical Signs

Lameness usually is sudden in onset and may be unilateral or bilateral. In some horses with bilateral injury, lameness is insidious in onset and the horse has loss of forelimb action. Lameness varies from moderate to severe and tends to be worst on hard ground, and often the horse appears to become lamer the farther it trots. After the horse turns at the walk, the lameness then improves and again deteriorates as the horse trots. If the lameness is bilateral and similar in degree in each limb, the horse moves with a short striding, stilted gait.

Usually no localizing clinical signs suggest the source of pain. Often the horse does not react to palpation of the proximopalmar aspect of McIII, unless the injury is acute.

Diagnosis

Lameness is substantially improved or alleviated by palmar metacarpal (sub-carpal) nerve blocks or perineural analgesia of the deep branch of the lateral palmar nerve. Lameness may be improved in some horses by intra-articular analgesia of the middle carpal joint.

Dorsopalmar radiographic views of the proximal metacarpal region may reveal sclerosis of the proximal medial aspect of McIII, with or without a longitudinal lucent line extending a variable distance proximodistally (Fig. 38-1). If present, the radiolucent line is invariably located medial to the axis of McIII. Generally no radiological abnormality is detectable in other views. In some horses, no detectable radiological abnormality exists.

Diagnostic ultrasonography usually reveals no detectable abnormality. Nuclear scintigraphic images usually demon-

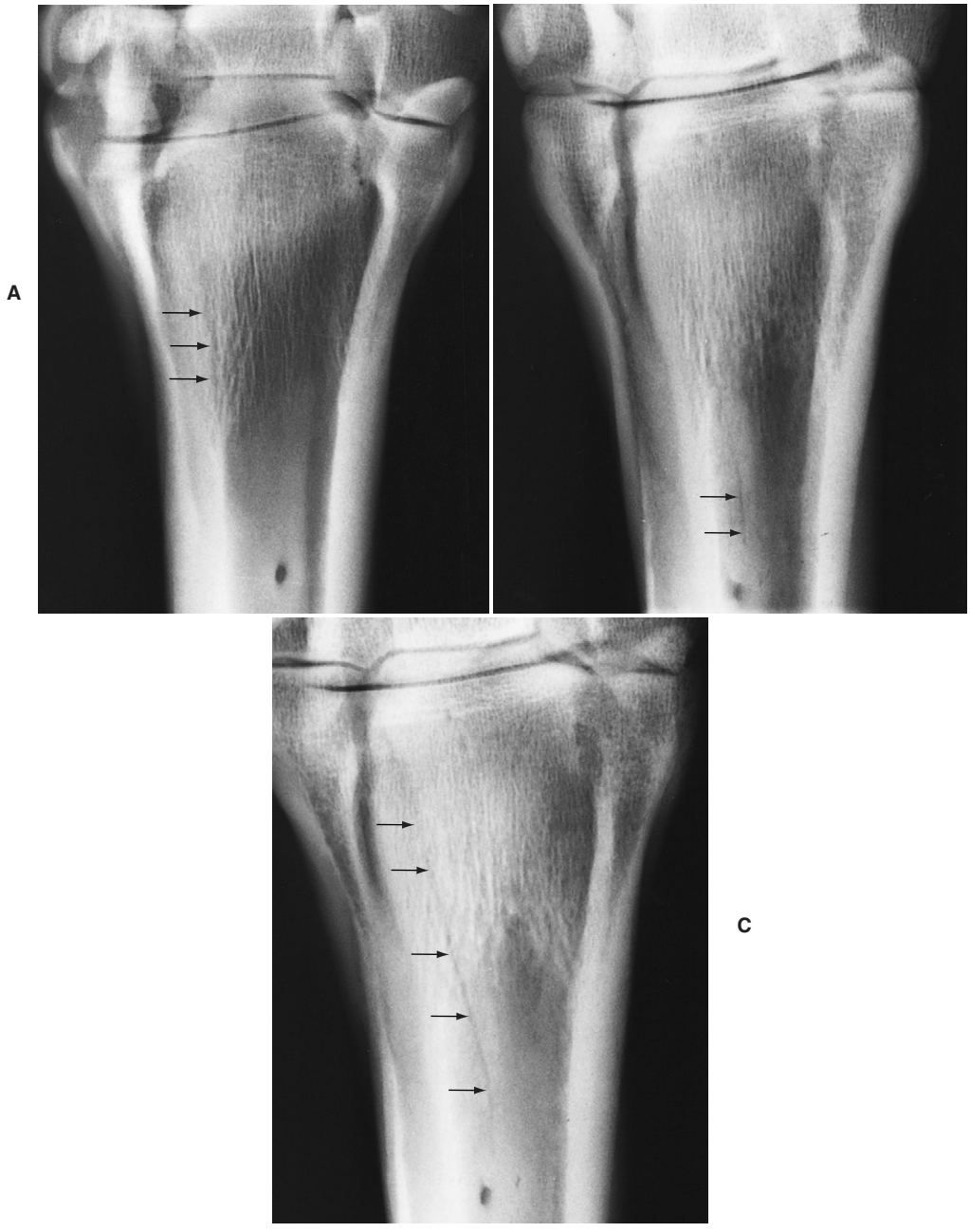

Fig. 38-1 **A,** Dorsopalmar radiographic view of the right third metacarpal bone of a 6-year-old Thoroughbred hurdler with moderate right forelimb lameness recognized 7 days previously and alleviated by subcarpal analgesia of the palmar metacarpal nerves. Medial is to the left. The medial aspect of the metaphyseal and proximal diaphyseal regions of the third metacarpal bone is sclerotic. A poorly defined longitudinal radiolucent line *(arrows)* represents an incomplete longitudinal palmar cortical fatigue fracture. **B,** Dorsopalmar radiographic view of the right proximal metacarpal region of a 6-year-old Arab endurance horse with a moderate right forelimb lameness of 3 weeks' duration. Lameness was alleviated by perineural analgesia of the deep branch of the lateral palmar nerve. Well-defined sclerosis immediately proximal to the nutrient foramen surrounds a longitudinal lucent line in the third metacarpal bone *(arrows)*. **C,** The same horse as in **B** after 4 weeks of box rest. The radiolucent line extends further proximally *(arrows)*.

strate abnormal patterns of uptake of ^{99m}Tc–methylene diphosphonate (^{99m}Tc-MDP) in dorsal and lateral projections. Even in the pool phase, uptake in the proximal palmar aspect of McIII may be abnormal. Bone-phase images show moderate to intense increased uptake of the radiopharmaceutical in the proximal aspect of McIII (Fig. 38-2). This pattern of uptake is indistinguishable from that associated with an avulsion fracture of McIII at the attachment of the SL. Currently unknown is whether horses without radiological change, but with scintigraphic evidence of a stress reaction (i.e., abnormally accelerated bone modeling) in the proximal aspect of McIII, would develop a radiographically evident incomplete

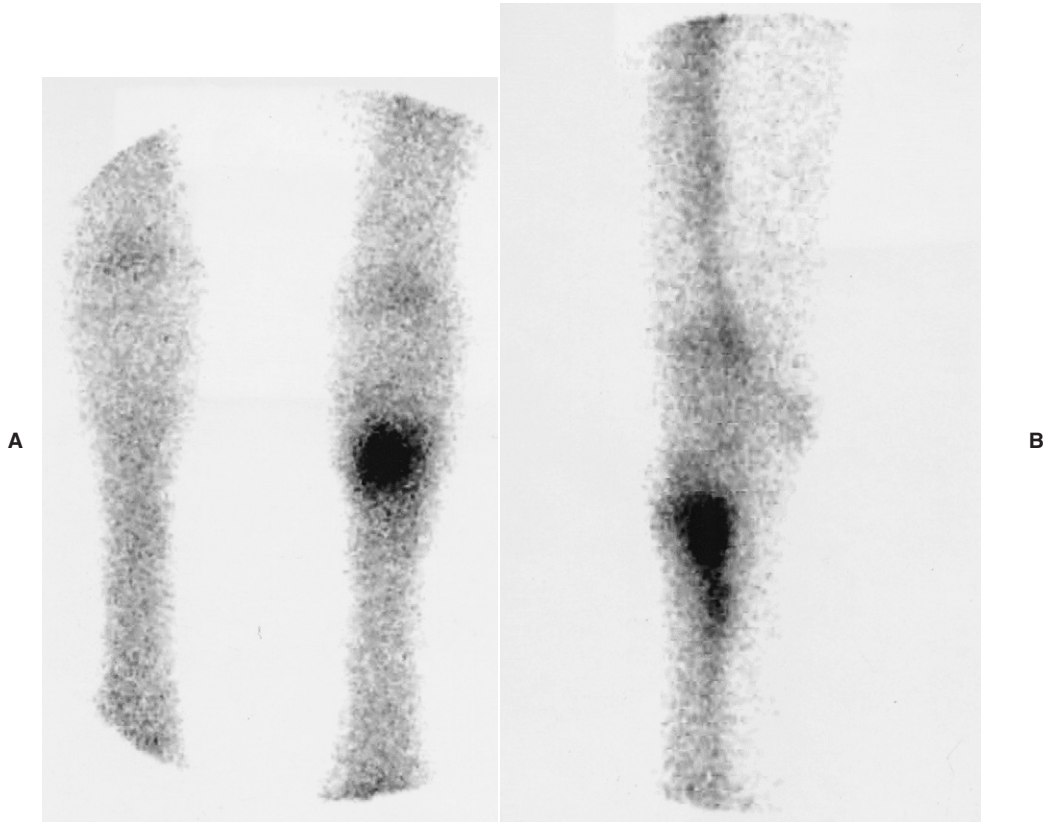

Fig. 38-2 Dorsal (**A**) and lateral (**B**) scintigraphic images of the carpal and proximal metacarpal regions of the forelimbs of an 8-year-old advanced event horse with acute-onset moderate left forelimb lameness of 3 weeks' duration. The left forelimb is on the right in the dorsal view (**A**). Lameness was alleviated by subcarpal analgesia of the palmar metacarpal nerves. The character of the lameness was typical of a palmar cortical fatigue fracture, but no radiographic or ultrasonographic abnormality was detected. Intense focal increased uptake of radiopharmaceutical in the proximal, medial, palmar aspect of the third metacarpal bone is compatible with a stress or fatigue fracture.

palmar cortical fracture of McIII if the horses were kept in work, or whether this is a different manifestation of the response of bone to exercise.

Treatment

Most horses respond well to rest, with box rest for 1 month and then box rest and controlled walking exercise for another 2 months. Horses are then able to slowly and progressively resume normal work. The incidence of recurrent injury is small. Every attempt should be made to identify any problem with the previous training program that may have predisposed the horse to injury.

Transverse Stress Fractures of the Distal Metaphyseal Region of the Third Metacarpal Region

Transverse stress fractures of the distal metaphyseal region of McIII are relatively uncommon. I have seen this fracture in horses of 4 to 7 years of age in the first season of racing over fences and in pleasure horses that have galloped on the beach. These fractures are believed to be stress fractures because endosteal and periosteal callus has been identified radiographically at the first recognition of the lameness.

Clinical Signs

Lameness is usually acute in onset after fast work, is moderate to severe, and may be unilateral or bilateral. Lameness may improve rapidly with the horse appearing sound within a few days. In horses with long-standing lameness a change in contour of the distal dorsal aspect of McIII is visible and is associated with periosteal callus formation. In these horses, pain may be elicited by firm pressure applied to the distal dorsal or palmar aspects of McIII. Twisting McIII may also induce pain. However, horses with acute lameness often have no localizing signs.

Diagnosis

If lameness persists, local analgesic techniques can be used to isolate the pain, but in some horses this is not possible because of the rapid resolution of lameness, unless the horse is maintained in full work. Lameness is eliminated by palmar (midcannon) and palmar metacarpal (distal to the button of McII and McIV) nerve blocks, but it is not influenced by intra-articular analgesia of the metacarpophalangeal joint.

Dorsopalmar, dorsolateral-palmaromedial oblique, dorsomedial-palmarolateral oblique and lateromedial radiographic views of the fetlock region should be obtained. Flexed lateromedial views are preferable, because this technique lifts the PSBs away from the palmar cortex of McIII, allowing better evaluation. Radiographic abnormalities may include a horizontal fracture line and endosteal and periosteal callus (Fig. 38-3). Some horses, in particular those with acute lameness, may have no detectable abnormality.

Nuclear scintigraphy is invaluable for determining the likely presence of a stress fracture in those horses that lack radiographic abnormalities or in which lameness rapidly resolves. Increased uptake of ^{99m}Tc-MDP appears in the distal metaphyseal region of McIII.

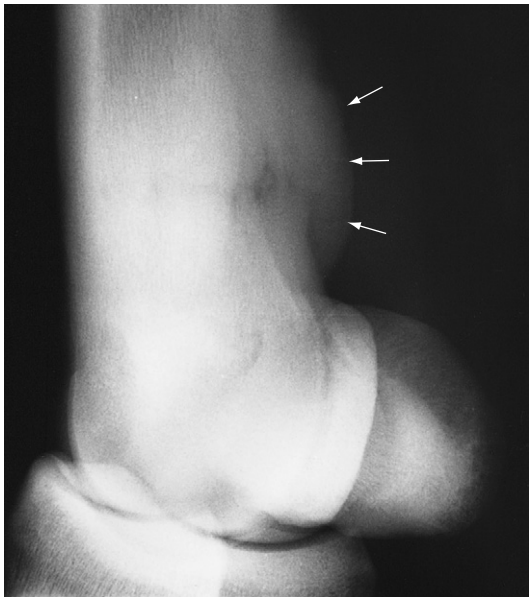

Fig. 38-3 Dorsolateral-palmaromedial oblique radiographic view of fetlock region of 6-year-old Thoroughbred used for point-to-point racing. There is a transverse stress fracture of the distal metaphyseal region of the third metacarpal bone with periosteal callus *(arrows)*.

Treatment

Most horses respond well to 3 months rest, with 1 month of box rest and then box rest combined with walking exercise. Work intensity can then be progressively increased. The previous training program should be reviewed to try to identify any features that may have predisposed the horse to fracture.

Dorsomedial Articular Fractures of the Third Metacarpal Bone

Dorsomedial articular fractures of McIII have only been recorded in the Standardbred racehorse,[10] although similar fractures have been identified in McIII of Thoroughbreds. The condition usually affects horses 2 to 4 years of age and occurs most commonly in pacers.

Clinical Signs

Lameness is acute in onset, after racing or training, and severe. Lameness persists despite rest. A bony swelling may be palpable on the proximal, dorsomedial aspect of McIII. Direct pressure may elicit pain.

Diagnosis

Lameness is generally improved by intra-articular analgesia of the middle carpal joint. Diagnosis is based on radiographic identification of the fracture, best viewed in a dorsolateral-palmaromedial oblique view. The fracture is articular, nondisplaced, and usually incomplete (Fig. 38-4). Active periosteal new bone invariably exists at the distal aspect of the fracture, close to the insertion of the extensor carpi radialis tendon, even in an acute injury.

Treatment

Treatment consists of rest for a minimum of 3 months. The fractures usually heal with modeling of the periosteal reaction and progressive loss of distinction of the fracture line. Lameness resolves, and horses are able to withstand training before the fracture line completely disappears radiographically.

Stress Reactions in the Condyles of the Third Metacarpal Bone

See Chapter 37 for a discussion of stress reactions (see page 350).

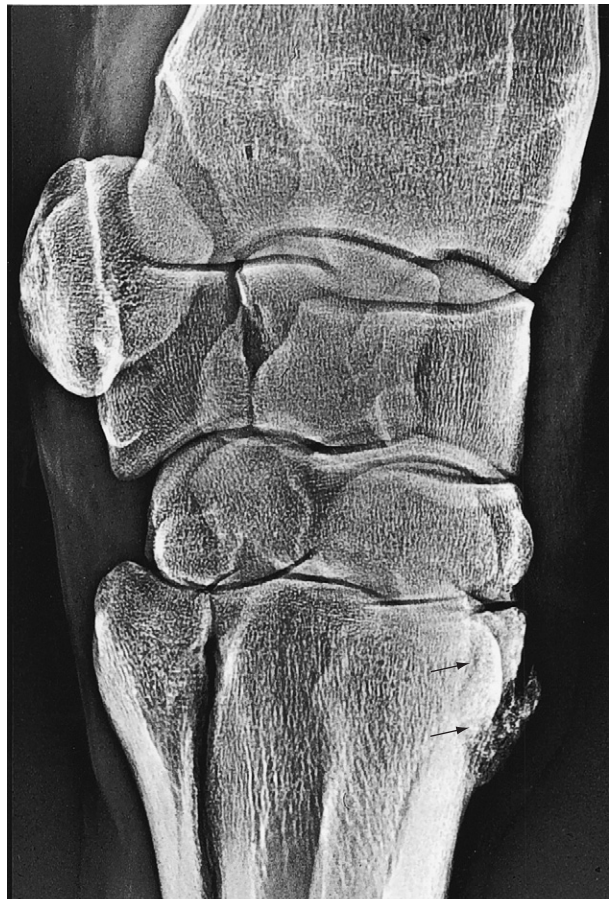

Fig. 38-4 Dorsolateral-palmaromedial oblique xeroradiographic view of the proximal metacarpal region of 3-year-old Standardbred male with a dorsomedial articular fracture of the third metacarpal bone. The wide oblique incomplete fracture line *(arrows)* and presence of proliferative new bone along the dorsomedial aspect of the third metacarpal bone indicate that bone activity in this region preceded the development of acute lameness. (Courtesy Mike Ross, Kennett Square, Pennsylvania.)

Avulsion Fracture of the Third Metacarpal Bone at the Origin of the Suspensory Ligament

Avulsion fracture of McIII at the origin of the SL occurs most commonly in young racehorses (Standardbreds more than Thoroughbreds).[11-14] Some fractures described as avulsions appear to occur immediately distal to the site of attachment of the SL.

Clinical Signs

Onset of lameness usually is acute and lameness is generally moderate to severe and unilateral. In the acute stage the horse generally resents pressure applied over the palmar proximal aspect of McIII. Eliciting pain may be more difficult in horses with chronic lameness. Lameness generally improves but may not resolve by box rest.

Diagnosis

In horses with acute lameness, diagnosis usually can be based on the clinical signs and ultrasonographic or radiographic demonstration of a fracture. In horses with more chronic lameness, local analgesic techniques may be required. Perineural analgesia of the palmar metacarpal (subcarpal) nerves or of the deep branch of the lateral palmar nerve usually improves lameness substantially. Intra-articular analgesia of the middle carpal joint, performed by palmar or dorsal approaches, may also improve the lameness.

An avulsion fracture is best detected radiographically in dorsopalmar or slightly oblique dorsopalmar views and lateromedial, or flexed lateromedial, projections. The fracture may appear as an almost straight or saucer-shaped lucent line (with the base proximal or distal) (Fig. 38-5) or as a punched-out lesion.

An avulsed fragment is usually easiest to detect by ultrasonography in longitudinal images and, if displaced, appears as a discontinuity of the palmar cortex of McIII. An incomplete fracture may be more difficult to detect. Examining the limb while the horse is bearing weight and not bearing weight can be helpful. Slight periosteal callus may be seen in horses with a more chronic fracture. A small focal tear in the dorsal aspect of the SL at the site of the fracture may be visible in transverse and longitudinal images. Nuclear scintigraphy has been used diagnostically.[15]

Treatment

The horse should be restricted to box rest for 6 weeks, followed by box rest and controlled exercise for at least another 6 weeks. The horse should be monitored clinically, radiographically, and ultrasonographically. Lameness caused by avulsion fracture generally takes longer to resolve than in horses with primary proximal suspensory desmitis, sometimes up to 2 months. A fracture may remain detectable radiographically for between 2 and 4 months. Periosteal callus is best seen by ultrasonography and usually is undetectable until 4 to 6 weeks after injury. The total convalescent period is usually between 4 and 6 months. Most Thoroughbreds ultimately make a complete recovery and return to full athletic function without recurrent injury. Occasionally the fracture fragment may sequestrate, or suspensory desmitis may progress, which results in a more guarded prognosis. However, some Standardbreds have long-term lameness that is refractory to treatment.

Osteoarthritis of the Carpometacarpal Joint

Osteoarthritis of the carpometacarpal joint is an unusual cause of forelimb lameness. It usually occurs in mature horses used for any discipline.

Clinical Signs

Lameness may be acute or insidious in onset and is mild to moderate. Usually no localizing clinical signs are present. Carpal flexion is not restricted or resented.

Diagnosis

Lameness may be improved by palmar metacarpal (subcarpal) nerve blocks, probably because of proximal diffusion of the local anesthetic solution. Intra-articular analgesia of the middle carpal joint also improves lameness. Radiographic examination usually reveals that changes are restricted to the medial or lateral side of the joint, with narrowing of the joint space between the carpus and McII or McIV, with subchondral sclerosis (Fig. 38-6) and often periosteal new bone extending along the proximal metaphyseal region of McII or McIV. Lucent zones may appear in the base (head) of McII or McIV.

Treatment

Response to intra-articular medication of the carpometacarpal joint has been poor. Palliative treatment with a non-steroidal anti-inflammatory drug may allow the horse to be maintained in work.

Osseous Cyst-Like Lesions in the Proximal Aspect of the Second Metacarpal Bone

Osseous cyst-like lesions sometimes are identified in the proximal aspect of McII (Fig. 38-7) in association with lameness that is localized to the proximal metacarpal or distal carpal regions. These osseous cyst-like lesions occur most commonly in the presence of a first carpal bone,[6] which also may have radiolucent zones. These lesions often occur bilaterally, although lameness may be unilateral. These lesions are gener-

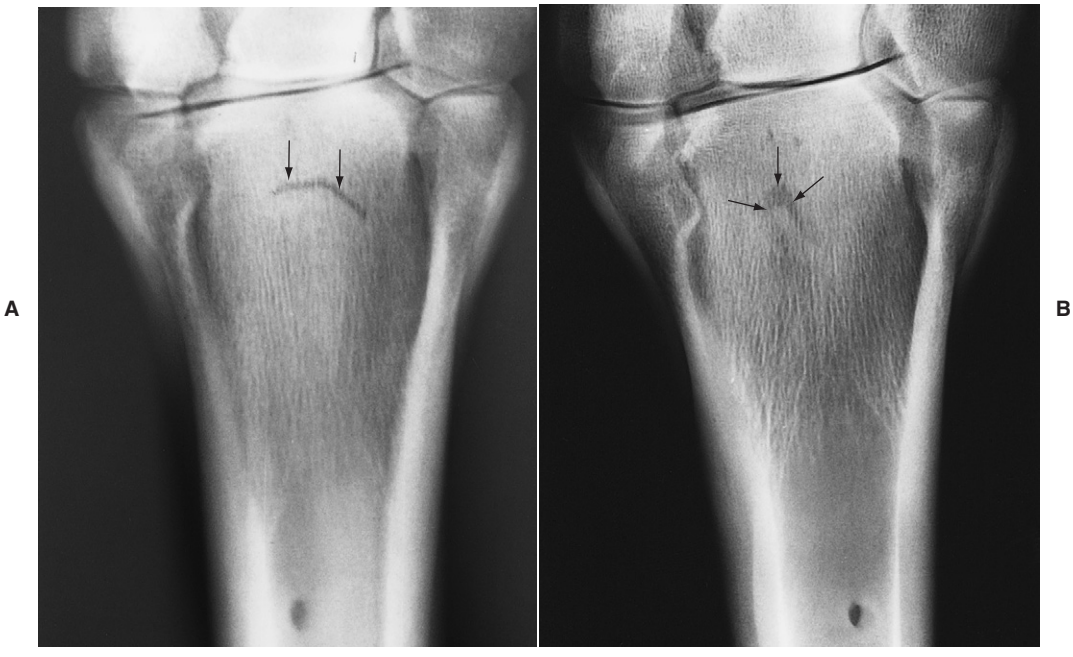

Fig. 38-5 **A,** Dorsopalmar radiographic view of the proximal aspect of the left metacarpal region of 4-year-old Thoroughbred with acute-onset moderate left forelimb lameness. Lameness was alleviated by perineural analgesia of the lateral palmar nerve just distal to the accessory carpal bone. The curved lucent line *(arrows)* in the proximal aspect of the third metacarpal bone represents an avulsion fracture at the insertion of the suspensory ligament. **B,** Dorsopalmar radiographic view of the proximal metacarpal region of 3-year-old Thoroughbred with lameness of 2 weeks' duration. A punched-out opacity distal to a crescent-shaped lucent area *(arrows)* in the third metacarpal bone represents an avulsion fracture at the insertion of the suspensory ligament.

ally considered incidental abnormalities unassociated with pain, and the clinician should search for another potential cause of lameness.

Exostoses of the Second and Fourth Metacarpal Bones (Splints)

Exostoses on McII or McIV (splints) may develop because of direct trauma, resulting in sub-periosteal hemorrhage and lifting of the periosteum, or instability between McII (or McIV) and McIII. However, many splints do not involve the interosseous space between McII (or IV) and McIII and develop without evidence of trauma. Some splints develop without associated pain and lameness, with little evidence of active inflammation, whereas others result in a localized soft tissue inflammatory reaction, pain, and lameness. The reasons for these differences are not known. Splints may occur at any level but most commonly involve the proximal one half of the bone. Lesions involving McII occur most commonly.

Lesions may develop in young, immature horses or, less commonly, in older horses. Horses with bench (offset) carpal conformation seem particularly prone to develop splints involving McII, but often these develop without associated lameness.

Clinical Signs

Lameness may be sudden or insidious in onset and tends to deteriorate with work and be worst on hard ground. There is usually an obvious palpable swelling comprising a bony exostosis, surrounded by an edematous soft tissue reaction, with localized heat and pain on firm palpation. The swelling should be palpated carefully with the limb not bearing weight, because sometimes only focal areas of the swelling appear to be painful.

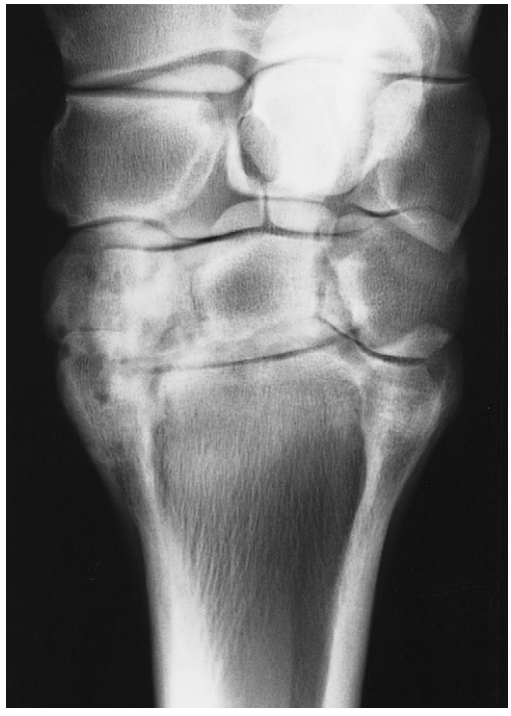

Fig. 38-6 Dorsopalmar radiographic view of the proximal metacarpal region of a 10-year-old gelding. Medial is to the left. Narrowing of the carpometacarpal joint on the medial aspect of the joint and radiolucent areas in the subchondral bone indicate osteoarthritis of the carpometacarpal joint.

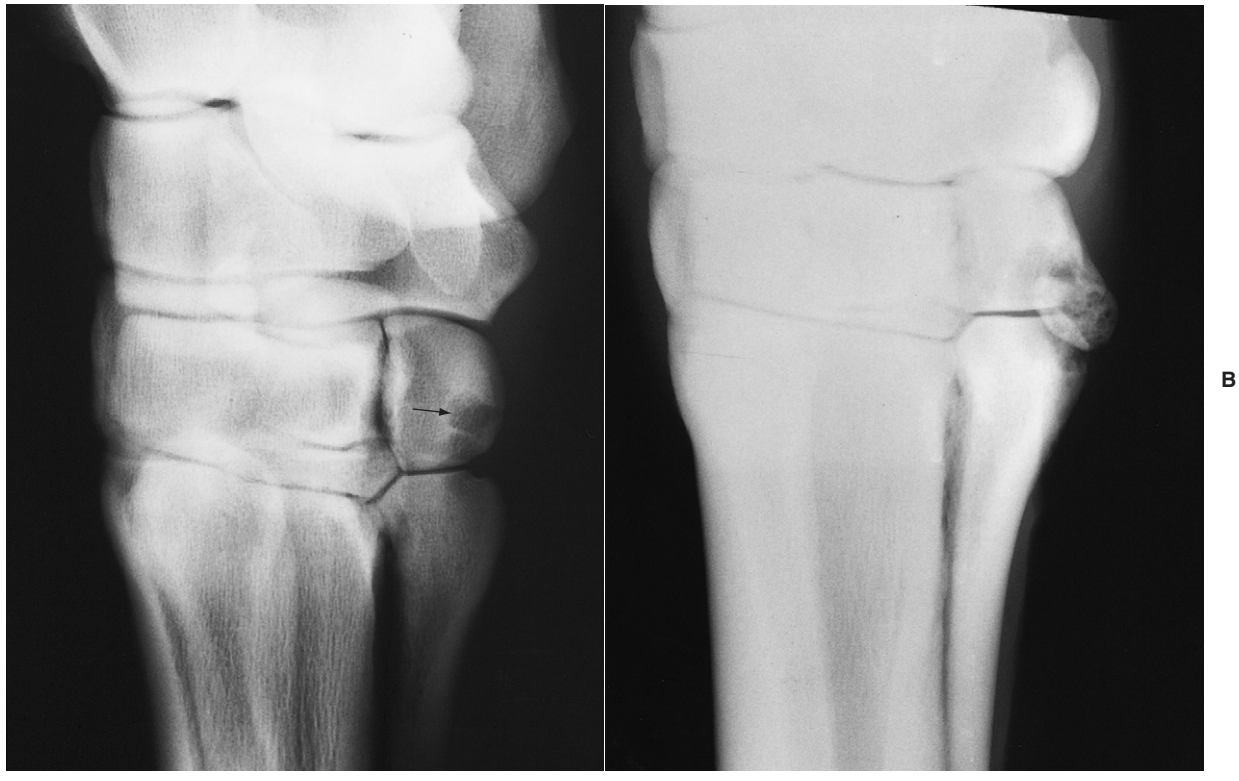

Fig. 38-7 **A,** Dorsomedial-palmarolateral oblique radiographic view of the proximal metacarpal region of 3-year-old Thoroughbred. There is an an osseous cyst-like lesion in the second carpal bone *(arrow)*. **B,** Dorsomedial-palmarolateral oblique view of the proximal metacarpal region of a 6-year-old riding horse. The lucent area in the proximal aspect of the second metacarpal bone is associated with the presence of a first carpal bone. These were incidental radiological findings not related to lameness.

It is important to assess the axial aspect of the exostosis to determine whether there may be impingement on the adjacent SL. Such horses may have had a pre-existing splint that was previously inactive. The horse often has a history of more extensive soft tissue swelling developing with hard work. Lameness may be provoked by hard work but rapidly improves with rest or light work. With the limb not bearing weight it may be difficult to palpate the border of the SL adjacent to the exostosis, and pain may be elicited by firm pressure applied to this localized area of the SL.

Diagnosis

Diagnosis of a straightforward splint often can be based on clinical signs, and further diagnostic procedures may not be necessary. Radiography may be useful to document the size and activity of the exostosis (Fig. 38-8). Several oblique projections using soft exposures are required. Care should be taken not to confuse as a fracture radiolucent lines that are caused by layers of new bone being superimposed, by incorporation of fibrous tissue, or an edge effect created by the parent bone. Radiography cannot properly document the axial extent of any exostosis.

Nerve blocks or local infiltration may be necessary to prove or disprove that an exostosis is the primary cause of pain resulting in lameness and to identify any concurrent problem(s). Diagnostic ultrasonography is essential if an impingement on the SL is suspected. Identification of echogenic tissue axial to the exostosis and contiguous with the SL may be possible (Fig. 38-9). The ipsilateral border of the SL may be irregular and reduced in echogenicity.

Nuclear scintigraphy is generally unnecessary, but it should be noted that even long-standing splints, which appear to be insignificant clinically, often have mild to moderate increased uptake of ^{99m}Tc-MDP compared with the parent bone. Why active bone modeling occurs is not known.

Treatment

Lameness associated with a clinically active exostosis usually resolves with rest, and the surrounding soft tissue swelling resolves. The exostosis remodels and is usually ultimately somewhat smaller. However, sometimes irregular-appearing, palisade-like new bone may persist radiographically. The time taken for the bony reaction to settle is extremely variable, ranging from 2 to 3 weeks to 2 to 3 months. This can be difficult to predict accurately and may reflect how quickly the condition was recognized and box rest instituted. Local infiltration with corticosteroids may facilitate reduction of the soft tissue reaction, but whether this alters the course of the condition is debatable. Topical application of dimethylsulfoxide has a similar effect.

Work should not resume until firm palpation of the exostosis fails to induce pain. A premature return to work is likely to exacerbate the problem. Some degree of fitness may be maintained by swimming exercise.

Some horses seem particularly predisposed to produce large amounts of new bone. The reason for this is unknown. Large exostoses on McII are vulnerable to direct trauma from the contralateral limb, especially if the horse moves closely in front, or dishes. Some protection may be provided by always applying

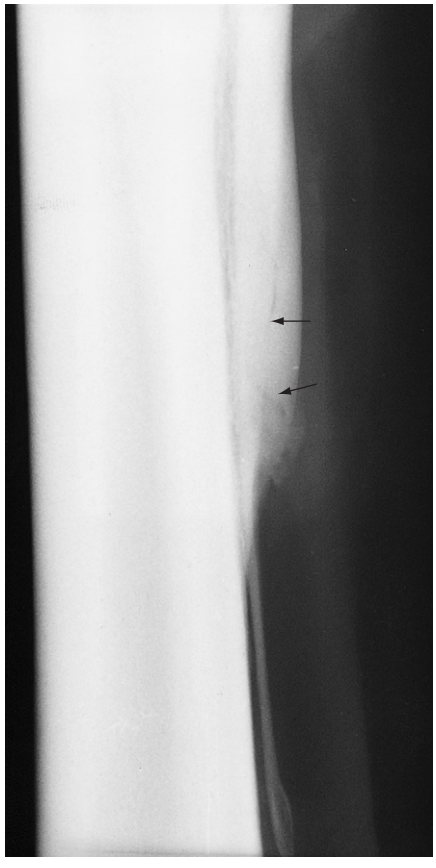

Fig. 38-8 Dorsomedial-palmarolateral oblique view of the metacarpal region. There is smoothly outlined enlargement of the middle of the diaphysis of the second metacarpal bone, with ill-defined, irregularly outlined periosteal new bone at the distal end of the middle third of the bone. The ill-defined lucent lines *(arrows)* within the bone result from new bone formation and should not be confused with fractures.

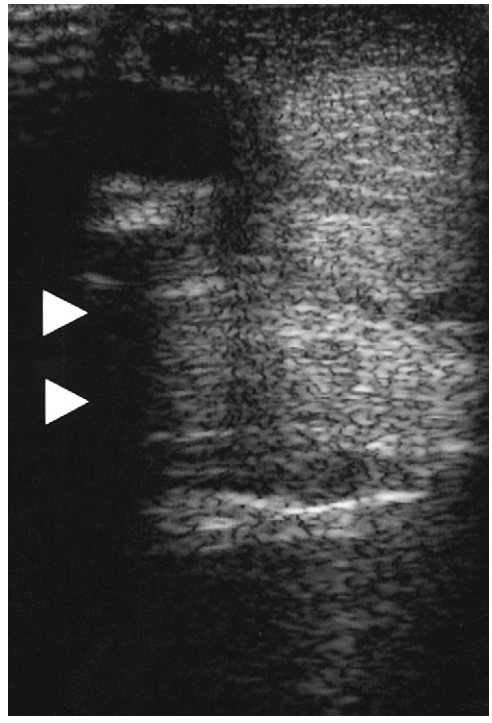

Fig. 38-9 Transverse ultrasonographic image of the palmar metacarpal soft tissues at 14 cm distal to the accessory carpal bone. Medial is to the left. The irregular medial border of the suspensory ligament and the echogenic material *(arrowsheads)* medial to it are apparent. A moderate size exostosis (splint) on the second metacarpal bone extended axially to impinge on the suspensory ligament. Granulomatous-like tissue was interposed between the splint and the suspensory ligament.

protective boots when the horse is worked or turned out. In horses with severe exostoses, surgical amputation of the exostosis and more distal aspect of the metacarpal bone should be considered. Ostectomy of the exostosis and liberal removal of the surrounding periosteum from the splint bone and McIII without removing the distal segment of the splint bone also has been cosmetically and functionally successful in some horses.

In some horses chronic pain associated with a splint persists, despite appropriate conservative management. Pin-firing is suggested if pain persists longer than 6 weeks after lameness was first recognized (see Chapter 89). An additional 6 weeks of walking is required before normal work can be resumed after firing.

If axial impingement of the exostosis on the SL occurs, surgical treatment is required. Amputation of McII or McIV proximal to the exostosis is the treatment of choice, assuming that this leaves the proximal one third of the bone intact, providing stability to the carpus. Often a granulomatous-type reaction occurs between the exostosis and the ipsilateral margin of the SL; this material should also be removed.

Fractures of the Second and Fourth Metacarpal Bones

Fractures of the McII and McIV bones may result from direct external trauma or internal forces, frequently in association with suspensory desmitis.[16-19] The latter is particularly common in horses that race over fences, Standardbreds and, less frequently, event horses.

Fractures caused by internal forces usually occur at the junction between the proximal two thirds and distal one third of the metacarpal bone. The distal ends of McII and McIV are connected by fibrous bands to the abaxial surface of the medial and lateral PSBs. Hyperextension of the fetlock and stretching of these fibrous bands may predispose to fracture. Suspensory desmitis may precede fracture and result in modeling and progressive deviation of the distal part of the bone away from McIII because of pressure or adhesions, thus predisposing to fracture. Fractures at this location unassociated with suspensory desmitis are relatively uncommon, but they do occasionally occur.

Fractures Caused by Internal Trauma

Clinical signs Lameness is acute in onset and moderate. Diffuse edematous soft tissue swelling rapidly develops in the distal half of the metacarpal region, more extensively medially if McII is fractured and laterally if McIV is involved. Occasionally McII and McIV are fractured simultaneously. One or both forelimbs may be affected. Careful palpation reveals enlargement of the body and or branch of the SL. Palpating instability of the distal piece of the fractured bone may be possible, but extensive soft tissue swelling may prevent this.

Diagnosis Radiographic examination should include the metacarpal bone and the ipsilateral PSB, because lesions may occur at both sites concurrently. Pre-existing abnormalities of McII or McIV suggest long-term suspensory desmitis (Fig. 38-10, *A*). The fracture should be evaluated to determine the degree of displacement and the presence of comminution and callus. Ultrasonographic examination is performed to assess the degree of suspensory desmitis, which is the most important prognostic factor.

Treatment Some controversy exists concerning the optimal treatment for horses with fractures of the distal one third of McII and McIV. Some infer that most horses can be managed conservatively, whereas others advocate surgical removal of the fracture fragment. Non-displaced or slightly displaced fractures usually heal satisfactorily within 4 to 6 weeks if the horse is

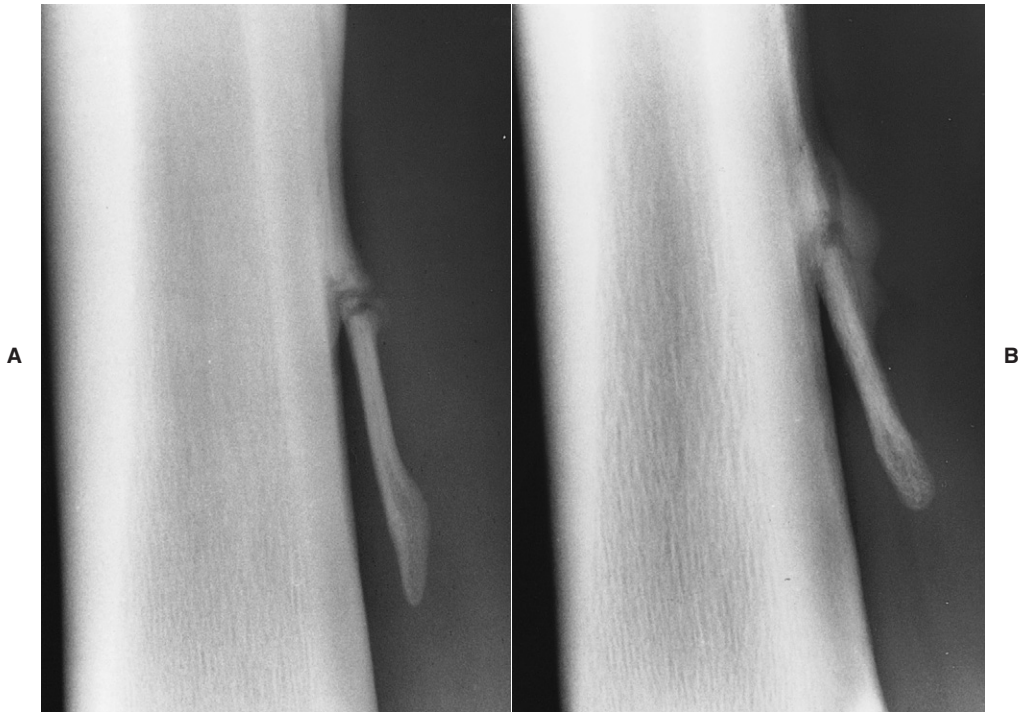

Fig. 38-10 **A,** Dorsomedial-palmarolateral oblique radiographic view of the second metacarpal bone of 7-year-old Thoroughbred steeplechaser. There is a complete, displaced fracture at the junction between the middle and distal one thirds of the bone. The axial deviation of the bone indicates previous distortion of the bone in association with desmitis of the medial branch of the suspensory ligament. Some endosteal and periosteal callus is present. **B,** Dorsomedial-palmarolateral aspect of the second metacarpal bone of an endurance pony. There is a healing fracture of the second metacarpal bone, associated with desmitis of the medial branch of the suspensory ligament.

confined to box rest. Only a small amount of callus develops, which subsequently remodels (Fig. 38-10, *B*). Small radiolucent defects may persist because of incorporation of fibrous tissue. If a fracture is moderately displaced, a larger amount of callus may develop, or a thick layer of fibrous tissue may be laid down, which envelops the entire distal aspect of the metacarpal bone. A large exostosis may impinge on the SL. Surgical removal of these fragments is therefore recommended. The convalescent program is dictated by the degree of suspensory desmitis. Prognosis is better in Standardbreds than in Thoroughbreds.

Fractures Caused by External Trauma

Fractures of McII or McIV caused by external trauma may be simple or comminuted, open or closed.

Clinical signs Lameness is acute in onset and is associated with soft tissue swelling around the fracture site, heat, and pain. An open wound may be present. Thorough debridement, cleaning, and lavage of open wounds are important to minimize the risks of infection.

Diagnosis Radiographic examination is essential to determine the precise position and nature of the fracture (Fig. 38-11). If the fracture is chronic or associated with an open wound, the radiographs should be appraised carefully for evidence of infectious osteitis.

Treatment Fractures involving the proximal one third of the bone may require surgical stabilization of the bone to prevent carpal instability, especially if the fracture involves McII. Insertion of small plates with screws into McII without traversing the interosseous space is the preferred method of management. Alternatively, screws placed between McII and McIII can be used, but fixation may be unstable, and chronic lameness could develop because of synostosis between the two bones or reaction from the implants themselves. When horses have simple displaced or comminuted fractures of McII or McIV, but with infection, all efforts should be made to resolve infection before surgical fixation is attempted. However, if the fracture is comminuted and the carpus appears to be stable, conservative management may be satisfactory. Simple or more complicated fractures in the distal two thirds of the bone can be treated either conservatively or by surgical removal, depending on the size of the pieces and their location.

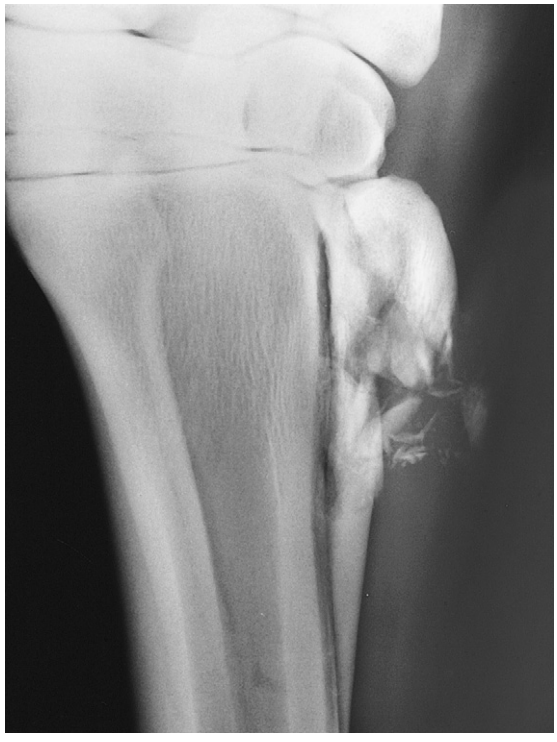

Fig. 38-11 Dorsolateral-palmaromedial oblique radiographic view of the metacarpal region. There is a comminuted, displaced articular fracture of the proximal third of the fourth metacarpal bone. The fracture was repaired surgically using a small plate, but osteoarthritis of the carpometacarpal joint developed subsequently.

Palmar Annular Desmitis

See Chapter 75 for a discussion of palmar annular desmitis (pages 678 to 681).

Avulsion of the Attachment of the Palmar Annular Ligament from a Proximal Sesamoid Bone

See Chapter 73 for a discussion of avulsion of the attachment of the palmar annular ligament from a proximal sesamoid bone (page 666).

Constriction of the Digital Flexor Tendon Sheath by the Palmar Annular Ligament

See Chapter 75 for a discussion of constriction of the digital flexor tendon sheath by the palmar annular ligament (pages 678 to 681).

Tenosynovitis of the Digital Flexor Tendon Sheath: Primary and Secondary

See Chapter 75 for a discussion of primary and secondary tenosynovitis of the digital flexor tendon sheath (pages 676 to 678).

Proximal Suspensory Desmitis

See Chapter 73 for a discussion of proximal suspensory desmitis (pages 654 to 662).

Desmitis of the Body of the Suspensory Ligament

See Chapter 73 for a discussion of desmitis of the body of the SL (pages 662 snd 663).

Desmitis of the Medial or Lateral Branch of the Suspensory Ligament

See Chapter 73 for a discussion of desmitis of the medial or lateral branch of the SL (pages 663 to 666).

Desmitis of the Accessory Ligament of the Deep Digital Flexor Tendon

See Chapter 72 for a discussion of desmitis of the ALDDFT.

Deep Digital Flexor Tendonitis

See Chapter 71 for a discussion of deep digital flexor tendonitis.

Superficial Digital Flexor Tendonitis

See Chapter 70 for a discussion of superficial digital flexor tendonitis.

Traumatic Lacerations of the Superficial Digital Flexor Tendon and the Deep Digital Flexor Tendon

See Chapter 83 for a discussion of traumatic lacerations of the superficial digital flexor and deep digital flexor tendons.

Distention of the Carpal Sheath

See Chapter 76 for a discussion of distention of the carpal sheath.

DIFFERENTIAL DIAGNOSIS OF DIFFUSE FILLING IN THE METACARPAL REGION

Diffuse filling in the metacarpal region, with or without stiffness or lameness, is common. The clinician makes a diagnosis of the cause through review of the history of the horse and careful clinical examination. (Box 38-1).

The clinician should assess the following points:
- Is the swelling symmetrical, or does it predominantly involve only one side of the limb?
- Does swelling extend the entire length of the metacarpal region, or only part of it?
- Has any filling occurred in the pastern region, or the carpus and proximally?
- Does the palmar aspect of the limb have a straight contour?
- Is the swelling edematous?
- What is the intensity of the digital pulses?
- Is the limb hot or cold?
- Is the horse febrile?
- Is the horse bright and alert, or depressed?
- Is more than one limb involved?
- Are any skin lesions apparent?
- What color is the limb?
- Is the horse sound, stiff or lame?
- What is the intensity of lameness?
- Is each anatomical structure palpable?
- Is each structure intact and of normal size and shape?
- Does palpation of a specific structure elicit pain?

Hemorrhage

Hemorrhage in the proximal metacarpal region is an occasional cause of acute-onset, severe lameness during work. Extensive soft tissue swelling rapidly develops, mimicking a severe tear of the ALDDFT. The swelling makes it difficult to palpate accurately specific structures. However, ultrasonographic examination reveals no detectable structural abnormality of the flexor tendons and the ALDDFT. Application of a large support bandage and treatment with non-steroidal anti-inflammatory drugs brings rapid relief. The bandage should be maintained and changed as needed for approximately 7 to 10 days. When the bandage is removed, the limb is usually of normal contour. The limb should be re-examined by ultrasonography to confirm that no structural abnormalities exist before allowing the horse to resume normal work. The cause of this condition is unknown and the likelihood of recurrence is small.

Thrombosis

Thrombosis of the medial palmar vein is a rare cause of lameness in the horse. Partial thrombosis may result in periodic diffuse filling of the distal limb associated with lameness. Complete occlusion may result in severe lameness and distal limb swelling soon after starting any exercise. Diagnosis is based on careful palpation of the vasculature combined with ultrasonography, including Doppler. Treatment requires vascular surgery.

Infection of Tendons

Infection of the SDFT or DDFT, without any evidence of a penetrating wound, is an unusual cause of lameness, but unless infection is recognized early and treated aggressively, it can have catastrophic consequences.[20]

Clinical Signs Sudden-onset, severe unilateral lameness involves a forelimb or a hindlimb. Frequently this follows within 24 hours of strenuous exercise. The horse is usually reluctant to bear weight on the limb, and effective control of pain can be difficult. Considerable peritendonous edema develops rapidly, precluding accurate palpation of the flexor tendons. Palpation reveals localized heat and exquisite pain. The skin may become tight, and if the infection is unrecognized and untreated, within several days pus may exude through defects in the skin.

Diagnosis Ultrasonographic evaluation reveals a central anechoic defect within the infected tendon (Fig. 38-12) that progresses rapidly and may within a few days extend the length of the metacarpal or metatarsal region. In the early stages lameness may be disproportionately severe relative to the perceived amount of tendon damage. Bacterial culture has frequently yielded a coagulase-positive *Staphylococcus*.

Treatment Aggressive systemic antimicrobial therapy with crystalline penicillin, gentamicin, and metronidazole has usually proved inadequate, with progression of tendon destruction despite therapy. Drainage of pus and lavage combined with antimicrobial therapy has successfully controlled infection in some, but not all, horses. However, the prognosis for return to athletic function is extremely guarded.

Box • 38-1

Differential Diagnosis of Diffuse Filling in the Metacarpal Region

- So-called cold edema
- Cellulitis
- Edema and inflammatory reaction associated with a sub-solar abscess
- Scabby skin lesions on the palmar aspect of the fetlock
- Mud fever
- Lymphangitis
- Purpura hemorrhagica
- Hemorrhage
- Filling caused by direct trauma
- Tendonous or ligamentous injury
- Thrombosis
- Fracture
- Equine viral arteritis
- Infection of a tendon
- Infection of the digital flexor tendon sheath
- Photosensitization
- Hypertrophic osteopathy

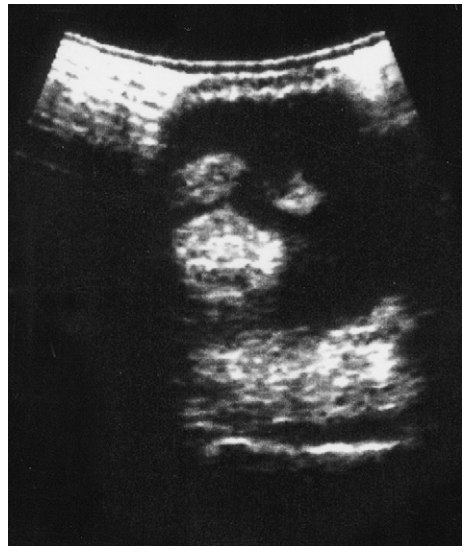

Fig. 38-12 Transverse ultrasonographic image of the palmar metacarpal region. There is extensive subcutaneous edema. The superficial digital flexor tendon has a large anechoic region extending through the dorsal and palmar borders that progressed rapidly. The horse had extensive peritendonous soft tissue swelling and severe lameness associated with the infected tendon.

Cellulitis, Skin Necrosis, and Necrosis of the Superficial Digital Flexor Tendon after Topical Applications

Cellulitis, skin necrosis, and subsequent necrosis of the SDFT are poorly understood conditions. The syndrome has been recognized in National Hunt racehorses.[21] After racing, many horses receive a topical application of a clay-like substance to the metacarpal regions, with or without overlying bandages.

Clinical Signs Clinical signs are apparent within 24 to 72 hours and include peritendonous edema and serum ooze progressing to skin slough and exposure of the SDFT. The tendon may also be affected, without evidence of pre-existing strain-type injury. Lameness varies from moderate to severe. The condition may be unilateral or bilateral. The horse may have had the same topical application previously, but not necessarily so. Frequently other horses have been treated with the same batch of the proprietary clay-like substance with no adverse effects. A variety of different proprietary products have been incriminated. Curiously, to my knowledge, the condition has not been seen in event horses, although many receive similar treatments after competing.

Treatment The condition usually progresses to a huge area of skin loss on the palmar aspect of the metacarpal region, with or without major damage to the underlying SDFT. No therapy has successfully halted this progression. Healing is by granulation tissue and fibrosis. If the horse survives, it has a massively thickened limb, and most horses have not been returned to racing.

Cellulitis Associated with Superficial Digital Flexor Tendonitis

Cellulitis sometimes occurs with superficial digital flexor tendonitis. Clinical signs are usually recognized within 24 to 48 hours after competition. The horse has diffuse filling in the metacarpal region(s), and within several days pinpoint pricks appear in the skin through which serum oozes. The horse may be slightly lame or have a stiff gait. Symptomatic therapy usually resolves the majority of filling and lameness, and without careful inspection a primary tendon injury may go unrecognized. Careful ultrasonographic evaluation is mandatory, because almost invariably primary tendonitis is present. Care should be taken in the presence of significant subcutaneous edema, because acoustic enhancement may mask a subtle tendon lesion. Such horses should be re-evaluated after resolution of the edema.

Enostosis-like Lesions

Enostosis-like lesions are focal areas of new bone formation within the medullary cavity of a bone, occurring on the endosteal surface or close to a nutrient foramen. They occasionally occur in McIII and may be single or multiple. They can occur as incidental radiographic abnormalities or in association with lameness. Nuclear scintigraphy may reveal increased uptake of ^{99m}Tc-MDP at the site of these lesions, but this does not necessarily imply that associated pain exists. Clinical significance can only be ascribed by eliminating all other sources of pain.

Hypertrophic Osteopathy

Hypertrophic osteopathy, or Marie's disease, frequently involves the metacarpal region, resulting in localized or diffuse soft tissue swelling that overlies new bone formation.[22] Hypertrophic osteopathy has been associated with thoracic disease, a variety of vascular lesions, granulomatous enteritis, and some tumors in the thorax and abdomen. Identification of the primary lesion and successful treatment may result in resolution of the bony lesions and associated clinical signs.

Clinical Signs

The acute stage of hypertrophic osteopathy produces heat and edematous swelling overlying areas of new bone formation. Palpation of the affected bones produces pain. All the limbs should be inspected carefully to establish the extent of the lesions. Depending on the limbs affected, the horse may show overt lameness or generalized stiffness.

Although careful clinical appraisal of the entire horse may reveal clinical signs of an underlying disease process, often no clues are present. Clinical evaluation should include palpation of all lymph nodes, auscultation of the heart and lungs, and rectal examination.

Diagnosis

Radiographic examination, using soft exposures, reveals palisading periosteal new bone in the diaphyseal and metaphyseal regions of McIII and other affected bones but usually excluding the joints (Fig. 38-13). The palisading new bone, perpendicular to the cortices, is pathognomonic.

Every effort should be made to try to define the primary causal lesion. Additional diagnostic tests should include thoracic radiography and echocardiography, examination of a tracheal aspirate and peritoneal fluid, and routine hematological and clinical biochemistry testing. If weight loss is evident, an oral glucose tolerance test should be performed to assess small intestinal absorption of disaccharide sugars. A labeled white blood cell nuclear scintigraphic examination of the thorax and abdomen may be helpful. Skin biopsies may reveal evidence of vasculitis.

Treatment

If the primary cause of the condition can be identified and successfully treated, then the bony lesions will resolve spontaneously. The new bone becomes progressively more radiopaque and corticalized and gradually models to restore a more normal contour. Occasionally lesions have resolved spontaneously after treatment with phenylbutazone.

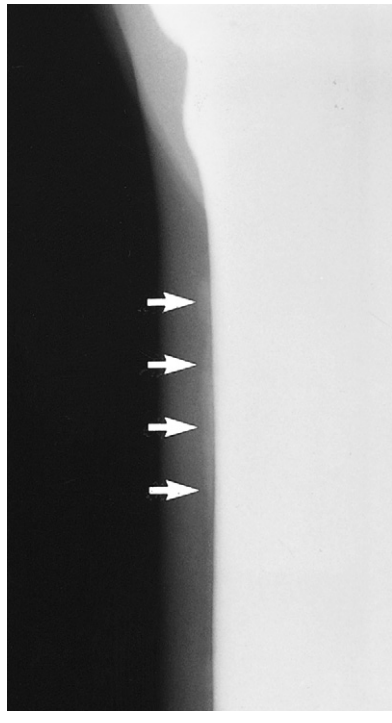

Fig. 38-13 Dorsolateral-palmaromedial oblique radiographic view of the proximal metacarpal region, using soft exposure factors to show palisading new bone *(arrows)* on the dorsal aspect of the third metacarpal bone typical of hypertrophic osteopathy. The 7-year-old breeding stallion had avian tuberculosis and was successfully treated, with the bony lesions resolving.

Osteitis or Osteomyelitis

Infectious osteitis or osteomyelitis occurs commonly in the metacarpal region because of the relative lack of soft tissue coverage of the bones and is usually a sequel to known trauma, with or without an open wound.

Clinical Signs

There is localized soft tissue swelling and mild to moderate lameness, and usually a draining tract is apparent.

Diagnosis

Ultrasonographic evaluation reveals an anechogenic region adjacent to the bone in the acute stage, and the accumulation of pus, before radiographic changes are evident. Radiographic abnormalities may include a radiolucent region within the cortex of the bone, periosteal new bone, and in horses with advanced infection, a sequestrum and involucrum (Fig. 38-14).

Treatment

Aggressive antimicrobial therapy sometimes successfully resolves the infection, but in horses with advanced sequestration, surgical debridement is indicated.

USE OF BOOTS AND BANDAGES TO PREVENT INJURY

The use of stable bandages, protective boots and bandages for exercise, and proprietary clays and cooling agents after exercise is common among horse owners in their efforts to protect horses from injury and to help to manage pre-existing injuries. Bandages that are applied too tightly have tremendous potential to cause local damage, not only to the underlying skin on the dorsal and palmar aspects of the metacarpal region, but also to the underlying tendons. The mid-metacarpal region appears to be particularly vulnerable. Transient (a few hours') application of an overly tight bandage results in a "bandage bow," localized subcutaneous edema overlying the SDFT. More prolonged application of a bandage that is too tight results in hair loss and skin necrosis. Hair that regrows is white. Excessive sustained pressure causes tendon necrosis.

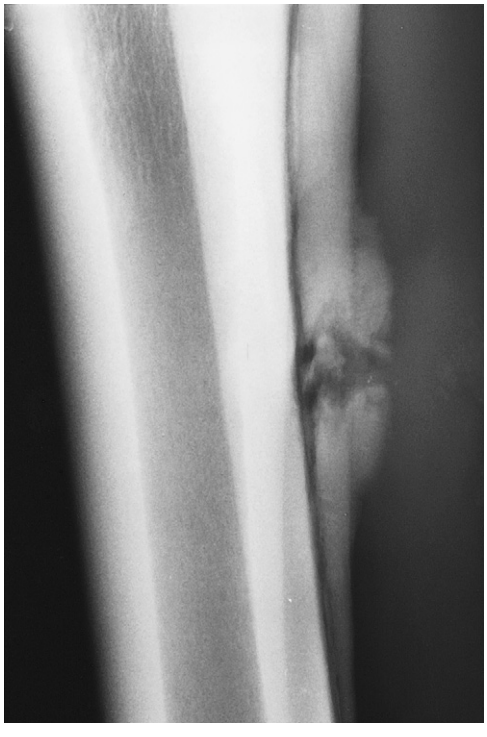

Fig. 38-14 Dorsolateral-palmaromedial oblique view of the metacarpal region. A lucent zone (an involucrum) surrounds a separate radiopaque fragment within the fourth metacarpal bone (a sequestrum). Periosteal new bone appears on the diaphysis of the fourth metacarpal bone, through which is a lucent canal (a cloaca).

Persistent bandaging at appropriate pressures does not cause damage but leaves permanent rings in the hair; if these rings are visible at a prepurchase examination, they should alert the veterinarian that the horse usually stands in bandages. Properly applied stable bandages can be used to control filled legs and in horses prone to distention of the fetlock joint capsule or the digital flexor tendon sheath.

The use of bandages or boots during exercise is somewhat controversial. A properly applied bandage should aim to permit a normal range of motion, but protect against movement in an abnormal range. Little evidence exists that a bandage applied circumferentially around the limb reduces loads on the flexor tendons and thereby reduces the risk of over-stretch injury. Studies on cadaver models have shown that distal limb bandages may increase the amount of energy a limb can absorb during repeated loading.[23,24] Strain on the proximal aspect of the SL may be decreased in a standing or walking horse.[25] In exercising horses, fetlock extension was reduced.[26] However, shock attenuation was unaffected.[27] The effectiveness of stabilizing support bandages in people decreases rapidly with exercise, and in healthy athletes restriction in motion may have an adverse effect on performance. In exercising horses the core temperature within the SDFT increases substantially, and although the role of temperature in tendon injury remains uncertain, bandaging has the potential to promote heating. Therefore little current rationale exists for use of bandages or boots to prevent tendon strain injuries.

Boots and bandages have the potential to attenuate forces from direct trauma to the limb, for example, as the result of a horse hitting a fixed fence. However, without reinforcement, none properly protects against strike injuries from the hindlimb of the same horse or from strike injuries from adjacent horses. Such strike injuries can result in severe tendon injuries. The introduction of a more resilient bullet-proof type of material into part of a boot, or applied as a palmar reinforcement under a bandage, can provide some protection against these devastating injuries, but the effect of such rigid material on normal movement of the flexor tendons is not known.

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CHAPTER • 39

Carpus

Mike W. Ross

ANATOMY

The carpus comprises the antebrachiocarpal, middle carpal, and carpometacarpal joints. The antebrachiocarpal and middle carpal joints are considered gingylmi, but they are not typical of hinge joints; the carpometacarpal joint is arthrodial.[1] Arthrodial joints also exist between carpal bones in each respective row. Effective movement of the carpus originates from the antebrachiocarpal and middle carpal joints. The carpometacarpal joint does not open, but it is subject to shear stress. The antebrachiocarpal joint lies between the distal radius and proximal row of carpal bones. The distal, dorsal aspect of the radius has deep grooves in which run the tendons of the extensor carpi radialis and common digital extensor muscles. In flexion the tendons compress the dorsal aspect of the antebrachiocarpal joint, limiting visibility when performing arthroscopic examination. The proximal row includes the accessory carpal bone that articulates with the distal radius and the ulnar carpal bone. The accessory carpal bone forms the lateral border of the carpal canal. From lateral to medial, the ulnar carpal, the intermediate carpal, and the radial carpal bones complete the proximal row.

The middle carpal joint lies between the proximal and distal row of carpal bones. The number of bones in the distal row varies but always includes from medial to lateral, the second, third, and fourth carpal bones. A first carpal bone is present unilaterally or bilaterally in approximately 50% of horses[1] and should not be mistaken on radiographs for an osteochondral fragment. The first carpal bone articulates with the second metacarpal bone (McII) and the second carpal bone, and its presence is often associated with radiolucent areas in McII. A fifth carpal bone is rare, but if present is small, articulates with the fourth carpal bone and the proximal aspect of the fourth metacarpal bone (McIV), and can be confused with an osteochondral fragment. The second, third , and fourth carpal bones articulate with McII, the third metacarpal

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CHAPTER • 39

Carpus

Mike W. Ross

ANATOMY

The carpus comprises the antebrachiocarpal, middle carpal, and carpometacarpal joints. The antebrachiocarpal and middle carpal joints are considered gingylmi, but they are not typical of hinge joints; the carpometacarpal joint is arthrodial.[1] Arthrodial joints also exist between carpal bones in each respective row. Effective movement of the carpus originates from the antebrachiocarpal and middle carpal joints. The carpometacarpal joint does not open, but it is subject to shear stress. The antebrachiocarpal joint lies between the distal radius and proximal row of carpal bones. The distal, dorsal aspect of the radius has deep grooves in which run the tendons of the extensor carpi radialis and common digital extensor muscles. In flexion the tendons compress the dorsal aspect of the antebrachiocarpal joint, limiting visibility when performing arthroscopic examination. The proximal row includes the accessory carpal bone that articulates with the distal radius and the ulnar carpal bone. The accessory carpal bone forms the lateral border of the carpal canal. From lateral to medial, the ulnar carpal, the intermediate carpal, and the radial carpal bones complete the proximal row.

The middle carpal joint lies between the proximal and distal row of carpal bones. The number of bones in the distal row varies but always includes from medial to lateral, the second, third, and fourth carpal bones. A first carpal bone is present unilaterally or bilaterally in approximately 50% of horses[1] and should not be mistaken on radiographs for an osteochondral fragment. The first carpal bone articulates with the second metacarpal bone (McII) and the second carpal bone, and its presence is often associated with radiolucent areas in McII. A fifth carpal bone is rare, but if present is small, articulates with the fourth carpal bone and the proximal aspect of the fourth metacarpal bone (McIV), and can be confused with an osteochondral fragment. The second, third , and fourth carpal bones articulate with McII, the third metacarpal

bone (McIII), and fourth metacarpal bone (McIV), respectively. The articulation of the second carpal bone and McII is broader than is that of the fourth carpal bone and McIV, and hence McII receives greater load, an important fact to consider with fractures of McII and McIV. The third carpal bone, the largest bone in the distal row, has two fossae separated by a distinct ridge, the intermediate (lateral) and radial (medial). The radial fossa is largest, receives greater load, and is more commonly injured. The third carpal bone is L-shaped and has a large, dense palmar portion that is rarely injured.

The carpal bones are held together by intercarpal ligaments: the dense palmar carpal ligament from which the accessory ligament of the deep digital flexor tendon arises, the long and short medial and lateral collateral ligaments that originate on the radius and attach to the proximal aspects of McII and IV, and the abaxial surface of the carpal bones, respectively. The collateral ligaments provide the major resistance to dorsal displacement of the proximal row of carpal bones. The strong intercarpal ligaments play a major role in stability, and the palmar intercarpal ligaments were shown to provide more resistance to extension of the carpus than does the palmar carpal ligament.[2] When large medial and lateral corner osteochondral fragments of the third carpal bone are removed, the intercarpal ligaments and capsular attachments must be incised. These dense attachments provide stability that can be advantageous when repairing slab fractures. The dorsomedial intercarpal ligament courses between the medial aspect of the second carpal bone and the dorsomedial aspect of the radial carpal bone,[3] but during arthroscopic examination it appears to blend with the joint capsule. A theory was proposed that the dorsomedial intercarpal ligament became hypertrophied and impinged on the articular surface of the radial carpal bone, causing secondary modeling in young racehorses and lameness.[4] Recent studies of normal carpi found that the dorsomedial intercarpal ligament was neither hypertrophied nor impinging on the radial carpal bone. A definite relationship exists between the development of pathological conditions on the distal radial carpal bone and the attachment of the dorsomedial intercarpal ligament, but I have not observed hypertrophy or impingement. The majority of radial carpal bone osteochondral fragments occur within or just lateral to the attachment site of the dorsomedial intercarpal ligament. Because the dorsomedial intercarpal ligament resists dorsomedial displacement of the radial carpal bone,[3] this site is prone to develop osteochondral fragments. In abnormal carpi, hypertrophy of the dorsomedial intercarpal ligament was apparent, but no correlation existed between hypertrophy and cartilage or subchondral bone damage.[5]

The medial and lateral palmar intercarpal ligaments resist displacement and dissipate axial forces by allowing abaxial translation of carpal bones.[6,7] Whereas the collateral ligaments provided the majority of resistance to dorsal displacement of the carpus during experimental loading, the small but important palmar intercarpal ligaments contributed 23% resistance.[2] The lateral palmar intercarpal ligament mostly attaches proximally on the ulnar carpal bone and distally on the third carpal bone and may be divided,[3] findings different from those previously reported that the distal attachment was mostly on the fourth carpal bone.[8] The medial palmar intercarpal ligament has four bundles that vary in size, and it courses between the radial carpal bone proximally and the palmaromedial surface of the third carpal bone and palmarolateral surface of the second carpal bone distally.[3] Tearing of the medial palmar intercarpal ligament and to a lesser extent the lateral palmar intercarpal ligament was observed in horses with carpal disease and was recently proposed to be associated with cartilage and subchondral bone damage (see the following discussion).[8,9]

The carpus has a dense joint capsule dorsally that blends with the overlying fascia and retinaculum. Synovium in young horses is often thickened or folded dorsally in the middle carpal joint and can interfere with visibility during arthroscopic surgery. This fold appears to smooth as horses age or as osteoarthritis develops. The antebrachial fascia blends with the retinaculum that functions to restrain extensor tendons. Retinaculum thickens and forms the medial and palmar borders of the carpal canal. The palmar retinaculum is sometimes severed in horses with carpal tenosynovitis and tendonitis (see Carpal Canal, Chapter 76). Anatomical considerations and flexor and extensor tendons injuries are discussed elsewhere (see Chapters 70 and 78). The sheathed extensor carpi radialis and common digital extensor tendons, located dorsal and dorsolaterally, respectively, limit carpal palpation and restrict access. Cul-de-sacs of distended antebrachiocarpal and middle carpal joint capsules can be palpated medial to the extensor carpi radialis tendon or between the extensor carpi radialis and common digital extensor tendons in the standing horse. Athrocentesis and arthroscopic examination require careful placement of needles and instruments in these portals to avoid injury to tendons and sheaths. These portals can be easily felt as distinct depressions when the carpus is flexed. The sheathed lateral digital extensor tendon, located on the lateral aspect, should be avoided during arthrocentesis of the palmarolateral pouches. The sheathed extensor carpi obliquus tendon is small and passes obliquely over the antebrachiocarpal joint from lateral to medial to attach to McII. This tendon can readily be seen medially during arthroscopic examination of the antebrachiocarpal joint. Extensor tenosynovitis must be differentiated from middle carpal and antebrachiocarpal joint effusion and hygroma.

Knowledge of the communications and boundaries of the carpal joints is important in understanding the extent of disease processes and the results of diagnostic analgesia (see Chapter 10).The antebrachiocarpal joint is considered solitary, although in a single specimen in a cadaver study the joint communicated with the middle carpal and carpometacarpal joints.[10] In some horses a communication appears between the antebrachiocarpal joint and the carpal sheath. The middle carpal and carpometacarpal joints always communicate. Communication between the middle carpal and carpometacarpal joints and the carpal sheath is rare (see Fig. 10-4). The carpometacarpal joint has distinct distopalmar outpouchings located axial to McII and McIV that have secondary pouches interdigitating within the proximal aspect of the suspensory ligament (SL). These outpouchings explain inadvertent analgesia of the carpometacarpal and middle carpal joint during performance of high palmar analgesia and possibly why lameness abates during middle carpal analgesia in horses with avulsion fractures of McIII or proximal suspensory desmitis.[11]

CONFORMATION

Racehorses, especially Thoroughbreds (TBs), with offset-knee (bench-knee) and back-at-the-knee (calf-knee) conformation are predisposed to develop carpal lameness. Mild in-at-the-knee (carpus valgus) deformity is common and of little concern, but if the deformity is severe, it can predispose to carpal lameness similar to that in horses with out-at-the-knee (carpus varus) conformation (see Chapter 4).

CLINICAL CHARACTERISTICS AND DIAGNOSIS OF CARPAL LAMENESS

Carpal lameness is a common finding in many sport horses, but it is most common in racehorses. Former racehorses used in other disciplines may suffer from chronic osteoarthritis or have recurrence of osteochondral fragmentation. Primary carpal

lameness in non-racehorses occurs from trauma such as from falls, kick wounds, and hitting fences; hyperextension injury resulting in fractures of the accessory carpal bone; and occasionally primary osteoarthritis. Old horses appear prone to develop inexplicable chronic, often severe osteoarthritis of the carpometacarpal joint. Few historical facts are pathognomonic for carpal lameness except if severe swelling and lameness develop acutely or a trauma is observed. TB racehorses may lug (bear) in, lug out, or fail to change leads. A Standardbred (STB) racehorse may be on a line. Horses may be racing poorly, particularly those with bilaterally symmetrical lameness. In racehorses with right forelimb carpal lameness, signs may be worse on the turns. Although most STBs are on the ipsilateral line, rarely a horse with right forelimb carpal lameness will be on the left line, presumably because the horse is bearing away from medially located pain or has a shortened stride in the right forelimb. Non-racehorses may have poor performance, fail to change leads, and hit or refuse fences.

Degree of lameness varies with the type and severity of carpal injury. Horses with early or mild, chronic osteoarthritis have mild lameness, whereas those with acute osteochondral fragments, slab fractures, or other more serious injuries have more severe lameness. Horses with infectious arthritis and comminuted carpal or other severe fractures may not bear weight at the walk. Lameness may be intermittent in horses with early and incomplete osteochondral fragments and may be apparent only after training or racing. Racehorses with bilaterally symmetrical lameness may not show overt lameness but have a wide, short gait bilaterally. Advancing and placing the affected limb wide while walking or trotting (pacing) is typical of carpal lameness (see Chapter 7). Horses with severe osteoarthritis and natural carpal ankylosis or surgical arthrodesis swing (abduct) the limb because the carpus cannot flex. Advancement and placement of the limb in a lateral (abducted) position is not pathognomonic for carpal lameness, and horses with proximal palmar metacarpal pain or those with pain originating laterally in the digit may manifest similar signs. However, carpal tenosynovitis does not result in this typical carpal gait. Horses with carpal lameness have a shortened cranial phase of the stride. Lameness can be worse with the limb on the inside or outside of the circle depending on whether the location of pain is medial or lateral, but in general lameness in most horses with carpal lameness is worse with the limb on the *outside* of the circle.

Increased temperature (heat) over the dorsal surface of the carpus is a reliable indicator of carpal disease, but false positive and negative findings occur. If horses have been clipped for painting or blistering, or effects of topical counterirritation are still present, the area can be warm and sensitive without carpal lameness. The dorsal surface of each carpus should be evaluated and compared, but differences are difficult to detect if lameness is bilateral. Effusion is usually a reliable indicator for carpal synovitis, but it is not pathognomonic for carpal lameness. Horses with subchondral bone injury without overlying cartilage damage or incomplete osteochondral fragments often have carpal lameness without effusion. Effusion is suppressed in horses that have recently had corticosteroid injections. Effusion occurs commonly in young horses with early carpitis and in horses with advanced cartilage damage, osteochondral fragments, and infectious arthritis. With the horse's limb in a weight-bearing position, the clinician's fingers are used to ballot fluid between extensor tendons. Older horses with chronic carpal changes often have mild or moderate effusion but can perform satisfactorily.

Horses with acute carpal lameness, especially those with synovitis, often show a marked response to static flexion of the carpus. Horses with carpal tenosynovitis or other palmar carpal lameness also respond. Horses with pain from the proximal limb, such as myositis in the proximal antebrachium or elbow region pain, respond to carpal flexion (false-positive response). Static and dynamic flexion can be negative in horses with carpal lameness, especially in those with subchondral bone pain. Degree of flexion is usually decreased in horses with chronic osteoarthritis because of joint capsule fibrosis. Careful palpation of all bony and soft tissue structures of the carpal region should be performed with the limb in standing and flexed positions, and responses should be compared with the contralateral limb. Swellings are best felt with the horse's limb in a standing position. Swellings of nearby sheaths should be differentiated from authentic carpal effusion by location and ballottement. Horses with chronic osteoarthritis often have firm, fibrous thickening at joint capsule attachments. Dorsomedial bony swelling of the radial and third carpal bones is observed and palpated in horses with chronic severe osteoarthritis. The proximal aspect of McII and McIV should be palpated for bony and soft tissue swelling associated with fracture or exostoses. The proximal palmar metacarpal region should be palpated with the horse in the standing and flexed positions to differentiate pain in this region from carpal pain.

In my experience, the carpal flexion test is the most specific of any flexion test used, and if the test result is positive, carpal pain is highly probable. A negative test result does not rule out carpal pain. Horses with subchondral bone pain, usually young racehorses with sclerosis of the third carpal bone, often have a negative or equivocal response to flexion. Horses with palmar metacarpal or elbow pain can respond positively.

Diagnostic Analgesia

In many horses, clinical signs and characteristic gait may allow a tentative diagnosis of carpal lameness, but in most horses diagnostic analgesia should be performed. Analgesia of the middle carpal and antebrachiocarpal joints should be performed independently and sequentially. Dorsal intra-articular techniques are most common, but in horses with scurf from previous counterirritant application or dorsal wounds, the palmarolateral pouches are used. Careful selective perineural and intra-articular analgesic techniques should be performed to differentiate between proximal palmar metacarpal pain and authentic carpal pain (see Chapter 10). Intra-articular analgesic techniques are highly specific, but false-negative results may occur.[12] Subchondral bone pain may not always be eliminated by intrasynovial deposition of local anesthetic solution, because nerve fibers may be located in bone or travel to the site by another extrasynovial route. The median and ulnar nerve block, although lacking specificity, is useful in horses with suspected carpal region pain that is not abolished by intra-articular techniques.

Laboratory analysis of synovial fluid is reserved for horses in which acute inflammation or infectious arthritis is suspected, but color and viscosity should be evaluated, and abnormalities may help to convince an owner or trainer of a carpal problem. In a normal flexed carpus, fluid does not readily drain from a small-gauge needle, and compression of the joint capsule at a distant site is usually necessary. Horses with effusion have thin synovial fluid that drips spontaneously without compression of the nearby capsule. Horses with true serosanguineous (as opposed to contamination by capsular or synovial vessels) fluid likely have cartilage damage with exposed subchondral bone or an osteochondral fragment. Hemarthrosis can be caused by trauma or bleeding from a torn intercarpal ligament.

Imaging

Radiography

A minimum of six well-exposed and positioned radiographic views are necessary for comprehensive examination of the carpus, including the dorsopalmar (DPa), lateromedial (LM),

dorsal 45° lateral-palmaromedial oblique (DL-PaMO), dorsal 45° medial-palmarolateral oblique (DM-PaLO), and flexed lateromedial views and the dorsoproximal-dorsodistal (tangential, skyline) view of the distal row of carpal bones. The skyline view is most important for assessing subtle radiographic changes of the third carpal bone, but well-positioned views are often difficult to obtain. Evaluation of the radial fossa requires flexion of the limb in the sagittal plane with the metacarpal region beneath the antebrachium (Fig. 39-1). Lateral positioning of the distal part of the limb results in overlap of the radial fossa of the third carpal bone and the radial carpal bone. The skyline view underestimates the amount of sclerosis of the third carpal bone and magnifies normal anatomy and lesions approximately twofold.[13] The skyline view is not a true proximal to distal view of the second, third, and fourth carpal bones, and therefore lesions located palmar to the dorsal edge of the radial carpal bone cannot be seen. The skyline view cannot be used to evaluate fracture lines located more than 8 to 10 mm from the dorsal edge of the third carpal bone or to differentiate large osteochondral fragments from frontal slab fractures of the third carpal bone. Additional views, such as the tangential view of the proximal row of carpal bones (used to identify osteochondral fragments and unusually located frontal or sagittal slab fractures), flexed oblique views (e.g., DL-PaMO view with the limb held in flexion to evaluate the articular surfaces of the third and radial carpal bones), and weight-bearing, oblique views of different obliquity (e.g., off DPa views, used to identify sagittal slab fractures of the third carpal bone) are sometimes useful. Considerable confusion arises in description of oblique views, with the use of terms *lateral* and *medial oblique*, instead of naming the views according to the direction of the radiographic beam. To most clinicians, the lateral oblique is equivalent to a DL-PaMO view, but to others it is just the opposite. Follow-up radiographic examination is recommended in 10 to 14 days if fracture is suspected but initial radiographic findings are negative or equivocal.

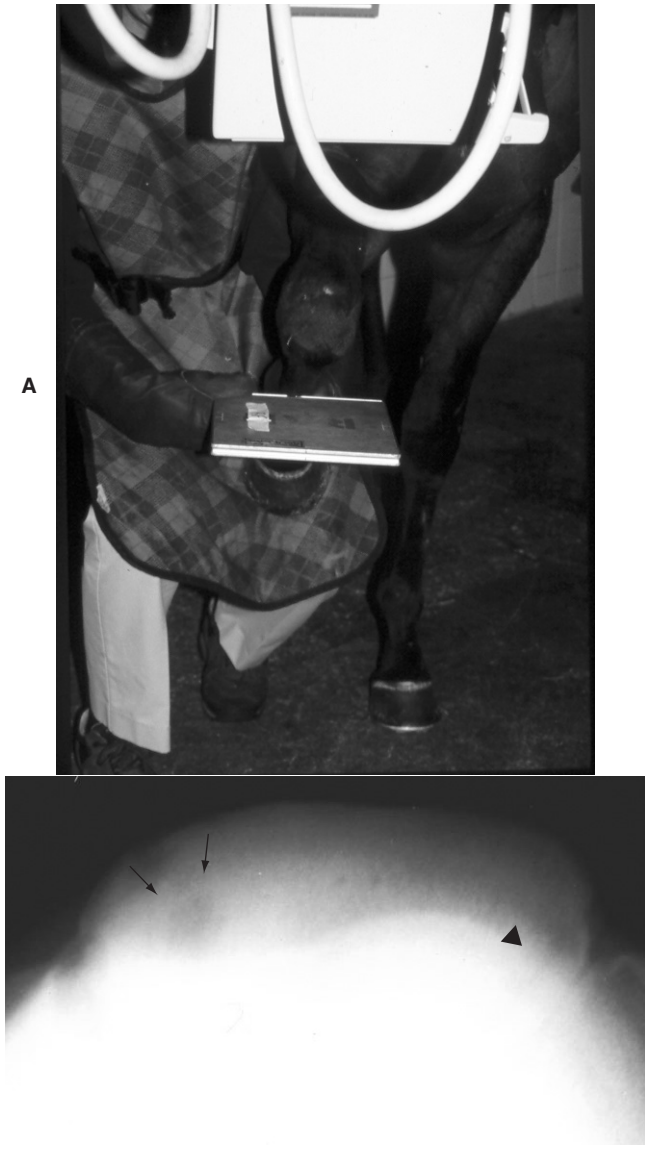

Fig. 39-1 **A,** A well-positioned skyline radiographic view of the distal row of carpal bones requires that the third metacarpal bone be aligned directly under the radius. This makes the radial facet visible. **B,** Resulting radiographic projection shows radial and intermediate (right) facets of the third carpal bone. Sometimes overlap of the normal articulation between the third and fourth carpal bones appears as a linear radiolucent defect *(arrowhead)* in the lateral aspect of the third carpal bone, a finding confused with sagittal fracture of that bone. There is a subchondral lucency of the third carpal bone (round radiolucent defect deep within the radial fossa; *arrows*) and a marginal defect of the bone.

Normal radiographic anatomy of the carpus is difficult because carpal bones overlap considerably, bones shift during flexion, and normal radiolucent defects and aberrant carpal bones can be difficult to interpret. In the skyline view of the distal row, the normal articulation between the third and fourth carpal bones and can be superimposed on the lateral aspect of the third carpal bone and confused with a sagittal fracture (Fig. 39-1, B). On a DM-PaLO view the normal articulation between the second and third carpal bones should not be confused with a sagittal slab fracture, but this view is essential to diagnose sagittal fracture of the third carpal bone correctly, which runs parallel to this articulation. Radiolucent defects or osseous cyst-like lesions are often seen in the ulnar carpal bone and are considered incidental findings, but when they appear in other bones, they can cause lameness regardless of whether communication with a joint exists. In LM and oblique views, the first and fifth carpal bones can be confused with osteochondral fragments. Radiolucent defects in McII and McIV often occur in the presence of the first and fifth carpal bones but are normal (Fig. 39-2). In the flexed LM view the radial carpal bone moves distally relative to the intermediate carpal bone. This normal finding is quite useful in determining the exact positioning of osteochondral fragments or other lesions on the proximal or distal surfaces of the radial and intermediate carpal bones. Xeroradiography and computed radiography are available at some institutions and practices and can be useful for diagnosing centrally located carpal pathological conditions, although xeroradiographs may not depict marginal changes as well as plain films because of edge enhancement. Computed tomography and magnetic resonance imaging (MRI) would be most useful to determine precise diagnosis and location of bone and soft tissue injuries, but availability is currently limited. Ultrasonographic examination of the carpus can be useful to determine the extent of soft tissue damage, if wounds or fistulous tracts communicate with carpal joints, and for diagnosis of extensor and flexor tendon injury, carpal tenosynovitis, and desmitis.

Scintigraphy

Scintigraphy is especially useful to diagnose early stress-related subchondral bone injury and differentiate carpal lesions from those of the proximal metacarpal region. A common finding in young racehorses is carpal lameness localized by clinical signs and diagnostic analgesia with negative or equivocal radiological abnormalities. Focal areas of increased

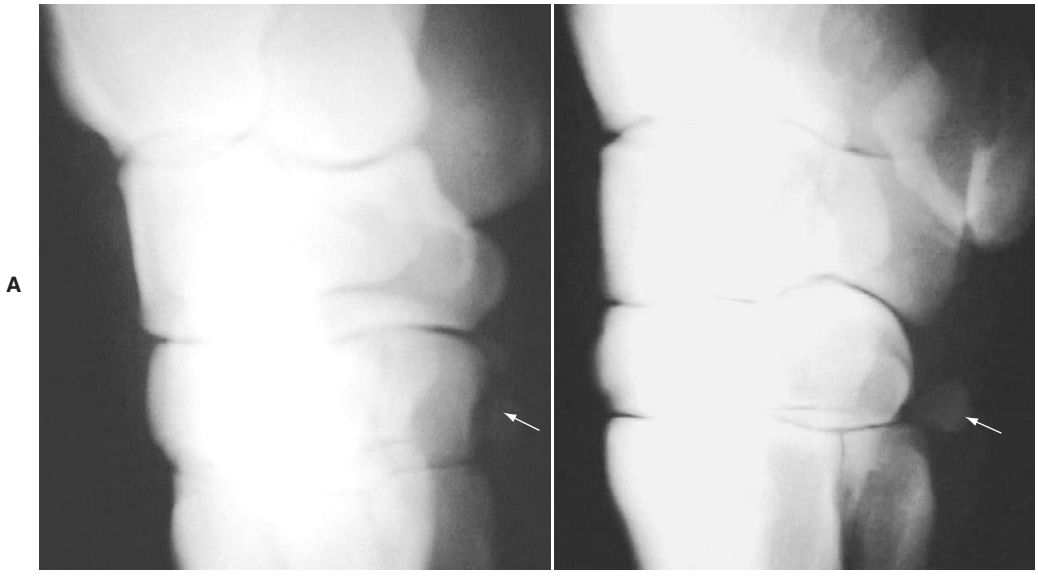

Fig. 39-2 **A,** Dorsomedial-palmarolateral oblique radiographic view of a carpus. A first carpal bone is present *(arrow)*. Note also the lucent zones in the second carpal bone and in the proximopalmar aspect of the second metacarpal bone. Such lucent areas are often seen as incidental findings in the presence of a first carpal bone. **B,** Dorsolateral-palmaromedial oblique radiographic view of a carpus. A fifth carpal bone is present *(arrow)*. Note the lucent area in the proximal aspect of the fourth metacarpal bone. The first and fifth carpal bones should not be confused with osteochondral fragments.

radiopharmaceutical uptake (IRU) are often found unilaterally or bilaterally, most commonly in the third carpal bone (see Fig. 19-16). Scintigraphy can be used to verify or refute the importance of sclerosis in the third carpal bone. Scintigraphy is useful in diagnosing unusual fractures of the palmar aspect of the third carpal bone, corner fractures or table surface collapse of the third carpal bone or other carpal bones, and lesions that are not apparent or are located in obscure areas not depicted radiographically. Focal areas of IRU occur with many carpal injuries, and although sensitivity is high, the specificity of scintigraphic images is low and differentiation of specific types of injuries is difficult. Scintigraphy is most useful in localizing the site of injury, based on which additional radiographic views are obtained, or rest is recommended, followed by repeated radiographic examination.

In general, scintigraphic examination is used when lameness is localized but a specific diagnosis cannot be made. In racehorses referred for evaluation of poor performance and obscure high-speed lameness, comprehensive scintigraphic examination of all limbs often reveals focal areas of IRU in the carpus, even when evidence of overt lameness is lacking. Areas of IRU are in locations typical for horses to develop osteochondral fragments and signs of osteoarthritis. Whether these areas of IRU represent sources of pain causing high-speed or subtle lameness is unknown, but often radiographic evidence of bone modeling (sclerosis, marginal osteophytes) or incomplete osteochondral fragments exists.

Diagnostic Arthroscopy

Specific diagnosis is usually made before surgery, and in most horses arthroscopy is interventional rather than diagnostic. When results of thorough clinical, radiographic, and scintigraphic examinations are combined, a specific site of injury is usually identified. In racehorses, lameness of the carpus without scintigraphic or radiographic abnormalities is unusual. Lack of subchondral bone involvement leads the veterinarian to suspect soft tissue diseases such as synovitis and intercarpal ligament tearing. In these horses, careful examination of the proximal palmar metacarpal region and carpal canal should be performed to avoid inadvertent misdiagnosis. Arthroscopic examination then is used to eliminate primary cartilage damage or intercarpal ligament tearing, but often arthroscopic findings can be unrewarding. Without overt cartilage damage the prognosis is favorable, so information gained by arthroscopic examination is useful, even if a primary diagnosis cannot be made.

In horses with scintigraphic evidence of IRU and those with osteoarthritis but without radiographic confirmation of osteochondral fragmentation, arthroscopic examination usually reveals cartilage damage, the extent of which can be graded. Prognosis is inversely related to degree of cartilage damage (see Figs. 23-2 and 23-3). Occult osteochondral fragments, most commonly involving the third and radial carpal bones, and intercarpal ligament tearing are found frequently in these horses.

SPECIFIC CONDITIONS OF THE CARPUS

Osteoarthritis

Osteoarthritis is the most common carpal problem, but clear differentiation of osteoarthritis from osteochondral fragmentation is difficult, because both problems are intertwined. Horses with osteochondral fragments often develop osteoarthritis, and horses with early osteoarthritis, and some with

chronic osteoarthritis, develop osteochondral fragments. Pathogenesis of osteoarthritis and osteochondral fragmentation appears similar if not identical in some horses, but osteoarthritis of the equine carpus has two forms. The most common form is seen in racehorses or ex-racehorses that initially develop stress-related subchondral bone injury of the middle carpal and antebrachiocarpal joints that leads to, or accompanies, overlying cartilage damage and osteochondral fragmentation. A second form of osteoarthritis develops in non-racehorses and is less common. Horses are usually middle aged or older, but occasionally it occurs in younger horses. Typical clinical and radiographic evidence of osteoarthritis exists, but osteochondral fragments are unusual (see Chapters 63 and 85).

Osteoarthritis in racehorses develops from a continuum of stress-related subchondral bone injury and cartilage damage, resulting from impact loading of the carpal bones during training and racing. This process has been studied most thoroughly in the third carpal bone but also occurs in other bones of the middle carpal and antebrachiocarpal joints. Sclerosis of the dorsal aspect of the third carpal bone is an adaptive response in racehorses.[14,15] With continued loading the third carpal bone becomes densely sclerotic, and in this stage the response becomes non-adaptive and pathological. Subchondral changes precede those in overlying cartilage, a finding seen experimentally[16] and clinically during arthroscopic examination in horses with primary stress-related subchondral bone injury. Sclerotic subchondral bone may induce overlying cartilage damage from abnormal shear forces existing between normal and sclerotic areas.[17] In most horses sclerosis leads to areas of resorption and necrosis, which then lead to osteochondral fragmentation, and eventually to more advanced osteoarthritis.[6,14,18,19] Because many of the changes in early osteoarthritis in racehorses are mechanically induced, factors such as faulty conformation, intense exercise programs, and differences between racing breeds alter rate of development and severity of osteoarthritis. The pathological process continues, and some horses develop osteoarthritis without osteochondral fragments, whereas others develop osteochondral fragments initially and then osteoarthritis secondarily.

In many racehorses extensive osteoarthritis and osteochondral fragmentation lead to retirement, but some are able to compete in other sporting events. Progressive osteoarthritis can then develop later in life. In middle-aged to old non-racehorses primary osteoarthritis develops without stress-related subchondral bone injury, high-impact loading, or development of osteochondral fragmentation. This condition can be seen in Western performance horses, other sports horses, or even in horses and ponies used for pleasure riding. Often severe radiographic evidence of osteoarthritis is seen on initial examination when lameness is subtle. Faulty conformation such as carpus valgus, back at the knee, or bench knee is seen in some horses, but in others neither mechanical nor training-related factors are present. Osteoarthritis in these horses can involve the antebrachiocarpal and middle carpal joints together or separately, but when disease involves the carpometacarpal joint, chronic and severe lameness develops (see Fig. 3-2).

Clinical Signs and Diagnosis

Clinical signs of osteoarthritis vary depending on age and use of horse, but they are similar to those of other carpal diseases. Classic signs of osteoarthritis, such as obvious lameness, typical carpal gait, effusion, and a painful response to static and dynamic flexion, may be present, particularly in horses with advanced osteoarthritis and in old horses with severe changes, but clinical signs can be subtle in young racehorses. In racehorses with early osteoarthritis, historical information such as lugging in or out, being on a line, or poor performance may be present. Effusion varies and lack of this clinical sign does not preclude the carpus as the source of pain. Racehorses in early training are prone to develop effusion primarily of the middle carpal joint, but the antebrachiocarpal joint can also be involved. Effusion may be most evident after work, but lameness usually is not present. Effusion usually results from strain of soft tissues, such as intercarpal ligaments or joint capsule. Clinical signs resolve after a brief period of rest or reduction in training. Most commonly, racehorses with early osteoarthritis manifest clinical signs later in training as a 2-year-old or when racing begins.

Diagnosis should be confirmed using diagnostic analgesia of the involved joint(s). In horses with severe osteoarthritis, complete resolution of lameness may not occur until median and ulnar blocks are performed. After pain in the primary limb is abolished, lameness may be seen in the contralateral limb, indicating bilateral carpal lameness.

Radiographic evidence of osteoarthritis in young horses is often lacking, but sclerosis of the third carpal bone may be seen in a skyline view. Early radiographic changes include mild enthesophyte formation, most common on the radial carpal bone, and subtle marginal osteophytes on the carpal bones and distal radius. In horses with advanced osteoarthritis, marginal osteophytes and enthesophytes become numerous and large, sometimes causing obvious visible bony swelling (Fig. 39-3). Loss of joint space occurs late in osteoarthritis, but it is not an obvious radiographic sign. Osteochondral fragments may be present and can be numerous and may have occurred earlier during a racing career or can develop when marginal osteophytes break. Radiographs of the contralateral carpus should also be obtained.

Scintigraphic examination is an excellent tool for diagnosis of early osteoarthritis in racehorses. Initially, focal areas of IRU are located on one side of a joint, such as in the third carpal bone, but later, subchondral IRU can be seen diffusely in one or more joints. Arthroscopic examination may be useful to establish prognosis and to evaluate possible soft tissue injury, such as intercarpal ligament tearing, and may be useful therapeutically.

Management

Management of osteoarthritis is discussed in Chapter 85. Because pathogenesis involves stress-related subchondral bone injury, an important part of management of osteoarthritis in racehorses involves stress relief by enforced rest or a reduction in training or racing intensity. Racehorses (STBs) without obvious radiographic changes are given 2 to 3 weeks of hand walking or walking in the jog cart, non-steroidal anti-inflammatory drugs (NSAIDs), and local therapy such as cold water hosing or icing. Topical counterirritation is still a popular management technique administered to young racehorses early in training. Intra-articular administration of hyaluronan, with or without a short-acting corticosteroid, may help in horses with synovitis, but it is of limited value if the primary source of pain is subchondral bone with intact overlying cartilage. In racehorses with advanced sclerosis of the third carpal bone and early enthesophyte and marginal osteophyte formation, extended rest of 3 to 4 months is recommended. However, economic factors may dictate that these horses remain in training; thus work intensity often is decreased and horses receive a single or a series of intra-articular injections with hyaluronan or polysulfated glycosaminoglycans (PSGAGs). Horses with radiographically apparent osteochondral fragments are surgical candidates, but post-operative progression of osteoarthritis and recurrence of fragmentation are common. As osteoarthritis progresses in horses with or without osteochondral fragments, numerous intra-articular injections, often including methylprednisolone acetate, may be necessary to reduce inflammation, but if training and racing continue, osteoarthritis progresses in accelerated fashion.

In ex-racehorses or non-racehorse sport horses with primary osteoarthritis, radiographic changes may be advanced

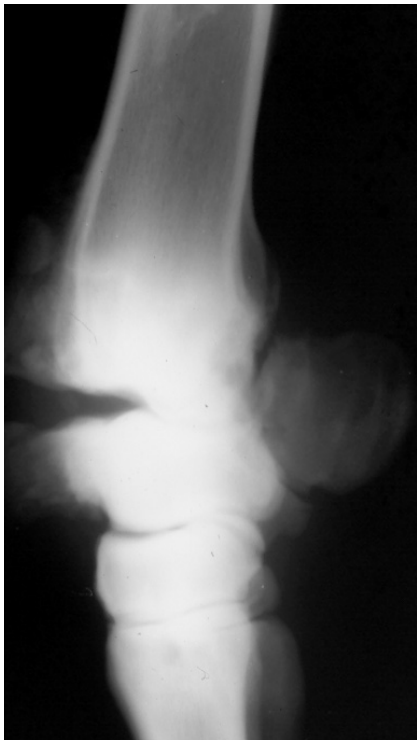

Fig. 39-3 Dorsolateral-palmaromedial oblique radiographic view of a horse with chronic, severe osteoarthritis of the antebrachiocarpal joint showing extensive marginal osteophyte formation and osteochondral fragmentation.

when lameness is first recognized, because osteoarthritis is chronic and well tolerated. Conservative management is recommended and includes rest, NSAID therapy, and intra-articular injections. Horses with radiographic evidence of large intact or broken osteophytes or old osteochondral fragments may be considered candidates for arthroscopic surgery, but although arthroscopic examination may be important to establish prognosis, surgical removal of osteophytes and old osteochondral fragments does not often result in long-term clinical improvement and may accelerate radiographic progression of osteoarthritis (see Fig. 39-3). Involvement of the carpometacarpal joint warrants a guarded prognosis.

Prognosis depends on the number of joints and limbs affected, rate of progression of clinical signs, severity of cartilage damage, presence of osteochondral fragments, and level of competition. Most racehorses with early osteoarthritis and even those with radiographic evidence of osteophytes and enthesophytes can race at some level, if horses have raced before diagnosis. Horses with advanced changes before racing begins, particularly those with faulty conformation, are unlikely to race but can be sound enough to perform other sporting activities. Valuable horses with severe osteoarthritis, such as broodmares or breeding stallions, may be candidates for partial (intercarpal) or pan-carpal arthrodesis.

Osteoarthritis of the Carpometacarpal Joint

Primary osteoarthritis of the carpometacarpal joint is unusual to rare but occurs primarily in old horses and is insidious and progressive (see Fig. 3-2 and Chapter 38).

Osteochondral Fragmentation

Small Fragments

Osteochondral fragmentation is a disease primarily of racehorses or ex-racehorses, and pathogenesis is identical to that described for osteoarthritis. I prefer to use the terms *carpal osteochondral fragments* (osteochondral fragments) and *osteochondral fragmentation* instead of *carpal chip fractures* to emphasize the importance of pathogenesis. The term *chip fracture* implies a single event traumatic injury. Although trauma plays a role, osteochondral fragments are not single event injuries but are the end result of stress-related subchondral bone injury caused by repetitive loading, initially an adaptive response, but later becoming non-adaptive remodeling and pathological.[14,16,18,19] Sites located dorsally are prone to develop osteochondral fragments. Osteochondral fragments differ in size and number of joints affected. Small osteochondral fragments are those that involve one joint surface, such as either the middle carpal or the antebrachiocarpal joints. Large osteochondral fragments refer to slab fractures that involve two joint surfaces. Often advanced changes occur in subchondral bone, such as sclerosis of the third carpal bone or other evidence of modeling or remodeling that preceded development of lameness and osteochondral fragments, findings that support the concept that osteochondral fragments are not single event injuries. The antebrachiocarpal joint is more susceptible to injury from supra-physiological loads or acute overload injury,[6] particularly in fatigued horses, but fractures still occur associated with pathological conditions of the bone. Palmar carpal osteochondral fragments, such as those involving the accessory carpal bone, occur as single event injuries such as falls, carpal hyperextension, or recovery from general anesthesia.

In my experience, faulty conformation such as back-at-the-knee predisposes TB and STB racehorses to develop osteochondral fragments, and trotters are more at risk than pacers. However, in a study attempting to evaluate the role of back-at-the-knee conformation in the development of osteochondral fragments in 21 horses, no differences in carpal angle were found compared with 10 horses without osteochondral fragments.[20]

Carpal osteochondral fragments occur in defined locations in all racehorses, but distribution varies depending on the type of racehorse. In the STB, osteochondral fragments occur almost exclusively in the medial aspect of the middle carpal joint and rarely in the antebrachiocarpal joint.[21] Although rare, osteochondral fragments in the STB antebrachiocarpal joint are most common in trotters and usually involve the distal radius. In the middle carpal joint we found a nearly equal distribution of osteochondral fragments between the radial and third carpal bones, but in another study those of the third carpal bone outnumbered osteochondral fragments of the radial carpal bone by 2:1.[21,22] The preponderance of osteochondral fragments in the medial aspect of the middle carpal joint in the STB is interesting, because training involves clockwise and counterclockwise exercise, whereas racing is counterclockwise. The assumption is that counterclockwise direction of training and racing places asymmetrical and uneven load distribution on each forelimb and may predispose the lateral left forelimb and medial right forelimb to compression injury. In two STB studies a nearly equal distribution of osteochondral fragments was noted between left and right middle carpal joints,[21,22] but in another study osteochondral fragments of the third carpal bone occurred more commonly in the right carpus.[19] Trotters were more likely to have right osteochondral fragments and had significantly more osteochondral fragments of the third carpal bone than the radial carpal bone compared with pacers.[21]

In TBs in North America, third carpal bone fractures occur more commonly in the right carpus, but considering all small osteochondral fragments, the distribution between left and right is similar.[18,19,22] In TB and Quarter Horse (QH) racehorses, osteochondral fragments are commonly seen in the middle carpal and antebrachiocarpal joints.[22-24] These breeds in North America have a predilection for development of osteochondral fragments in the lateral left antebrachiocarpal

joint and the medial right middle carpal joint, a distribution supporting the concept that osteochondral fragments develop on the compression side during counterclockwise training and racing. Overall, in TB and QH racehorses the most common sites for osteochondral fragments are the proximal aspect of the third and distal aspect of the radial carpal bones,[22,24] followed by the proximal aspect of the intermediate carpal bone and the distal lateral aspect of the radius. Osteochondral fragments often develop on apposing surfaces, supporting the concept that certain sites are biomechanically at risk. This may be evident radiographically, but in some horses apposing fragmentation is only identified at arthroscopic surgery and both limbs should be examined radiographically routinely, even if clinical signs are absent. Osteochondral fragments can be found at numerous sites in one or both carpi. Differences between racing breeds in distribution of osteochondral fragments may be explained by gait and sites predisposed to stress-related subchondral bone injury. Almost all STB osteochondral fragments occur medially, equally in both middle carpal joints. Most forelimb scintigraphic changes in STBs occur medially, indicating that the medial aspect in both forelimbs is at risk of stress-related subchondral bone injury. Classic training programs include many miles each day of jogging (trotting or pacing) clockwise (the wrong way of the track), and training one to two times each week, counterclockwise (the right way). Although all speed is performed in one direction, STBs jog many more miles in the other direction, and the number of loading cycles may be more important than speed. Direction of training and racing is not the only factor influencing fracture location because QHs and TBs have a similar distribution yet race differently. The two-beat trot and pace and a more caudal center of balance with a cart and driver result in reduced carpal loads in STBs at speed compared with galloping racing breeds. The low occurrence of osteochondral fragments in the antebrachiocarpal joint of STBs may reflect absence of supraphysiological loads. Racing speeds in the STB are lower than the TB and substantially lower than in the QH. Fatigue rather than speed of racing may be a factor.

Clinical signs. Lameness in horses with small osteochondral fragments varies from subtle to severe. In most horses, prominent to severe lameness is seen immediately after the fracture occurs, but subtle prodromal clinical signs are often present. Historically, horses may have been treated for suspected carpal lameness, may be on a line (STB), lug in or out, or fail to take a lead or change leads. Degree of lameness depends on location and number of osteochondral fragments and whether osteochondral fragments are present bilaterally. Horses with third carpal bone osteochondral fragments, and in particular incomplete fractures, show more pronounced lameness than those with osteochondral fragments elsewhere. Lameness is increased when the affected limb is on the outside of the circle in horses with osteochondral fragments in the middle carpal joint, but this response varies with those with osteochondral fragments in the antebrachiocarpal joint. TB and QH racehorses with osteochondral fragments of the distal, lateral radius or proximal intermediate carpal bone may only show mild signs of lameness. Horses with bilateral osteochondral fragments may show minimal overt lameness. Horses with lameness inappropriately severe for the location, number, or size of fragment(s) present may have substantial cartilage damage.

There is generally effusion and heat over the carpus, but in horses with incomplete fractures, clinical signs can be subtle. Palpation may reveal a focal painful response over the site of fragmentation, and occasionally fragments can be palpated directly. Degree of flexion varies, but usually the response to a carpal flexion test is positive. Signs of osteoarthritis including joint capsule fibrosis and enthesophyte production are common, particularly in horses with chronic lameness.

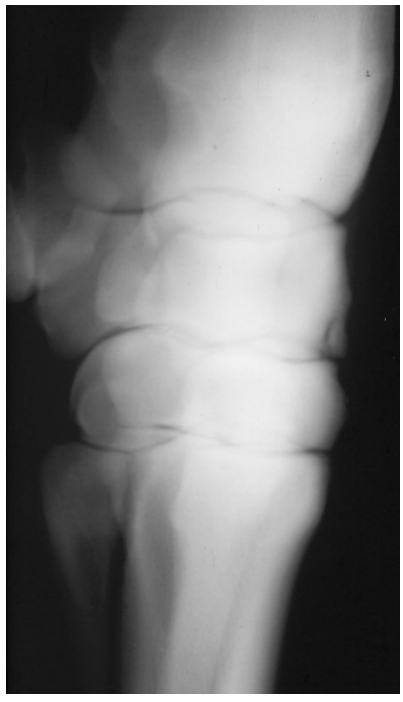

Fig. 39-4 Dorsolateral-palmaromedial oblique radiographic projection of a Thoroughbred racehorse with a typical distal radial carpal bone chip fracture, the most common small osteochondral fragment.

Arthrocentesis usually reveals serosanguineous fluid, particularly if lameness is acute. Intra-articular analgesia usually, but not always, abolishes signs of pain.

Radiographic examination is usually diagnostic, and all views should be obtained. In STBs abnormalities are usually only detected in DL-PaMO and LM views and a skyline view of the distal row of carpal bones. All common osteochondral fragment locations should be carefully evaluated. Fragments involving the distal aspect of the radial carpal bone are most visible on a DL-PaMO view and can vary in size, can be displaced or non-displaced, and may extend to the level of the joint capsule attachment (Fig. 39-4). Osteochondral fragments involving the third carpal bone most commonly affect the radial fossa and can be seen on the DL-PaMO, LM, and skyline views (Fig. 39-5). Small osteochondral fragments involve only the middle carpal joint surface and usually break out dorsally, near the joint capsule attachment on the third carpal bone. It is important to differentiate small osteochondral fragments from frontal or sagittal slab fractures. Third carpal bone osteochondral fragments can be singular or numerous, involve only the radial or intermediate (rare) fossa or both, can be complete or incomplete, or involve the medial (most common) or lateral corners of the bone. Medial corner fragments of the third carpal bone may resemble subchondral lucency and sagittal slab fracture, so other views must be carefully interpreted. Authentic sagittal slab fractures must be confirmed using a DM-PaLO view. Osteochondral fragments of the distal, lateral radius are the largest of the small osteochondral fragments and are often bipartite. The large dorsal fragment is separated from the parent radius by a small separate fragment in the interposed trough (Fig. 39-6). The large dorsal fragment can extend proximal to the joint capsule attachment on the distal lateral radius. Concomitant osteochondral fragments frequently involve the proximal medial aspect of the intermediate carpal bone. Osteochondral fragments of the proximal aspect of the intermediate carpal bone may occur alone or in combination with osteochondral fragments of the distal lateral radius

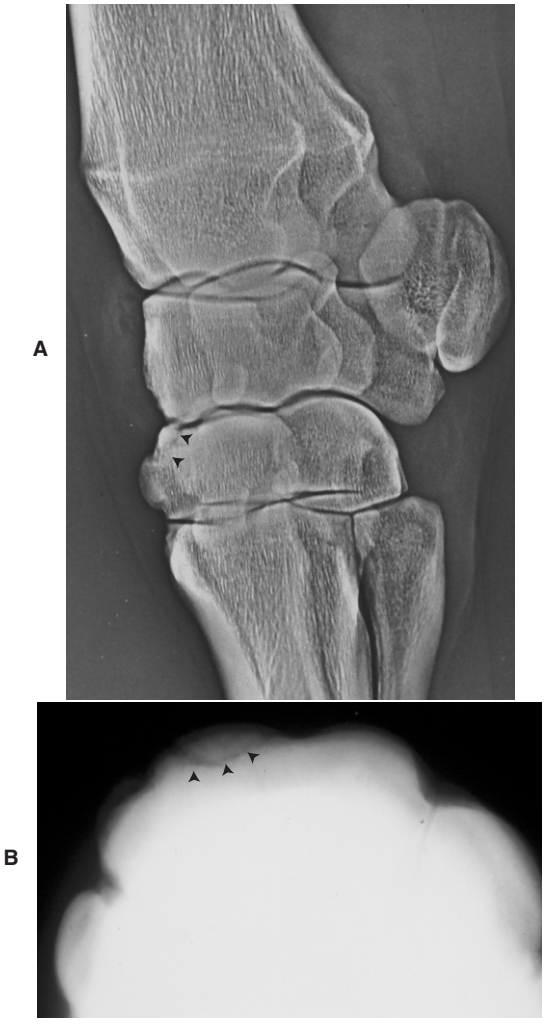

Fig. 39-5 **A,** Dorsolateral-palmaromedial oblique xeroradiographic and **B,** skyline radiographic views of a horse with a third carpal bone small osteochondral fragment. The osteochondral fragment *(arrowheads)* breaks out of the dorsal aspect of the third carpal bone in the dorsolateral-palmaromedial oblique view, confirmation that it does not span both articular surfaces of the third carpal bone.

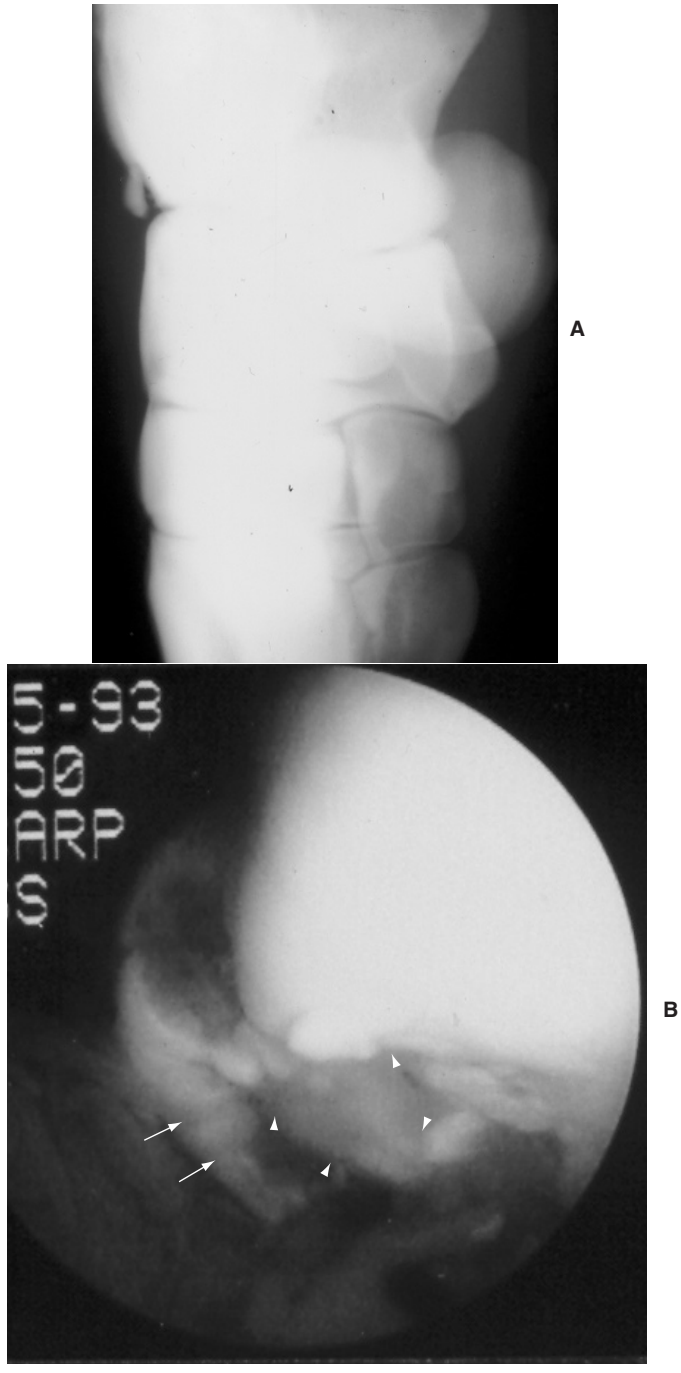

Fig. 39-6 **A,** Dorsomedial-palmarolateral oblique radiographic view showing typical distal lateral radius small osteochondral fragment in a Thoroughbred. **B,** Intra-operative photograph (dorsal is to the left; proximal is up) shows large dorsal fragment *(arrows)* separated from the parent radius by a small, interposed wedge-shaped fragment *(arrowheads)*.

(Fig. 39-7). Other, less common osteochondral fragments do occur alone but usually in combination with other osteochondral fragments. Evidence of osteoarthritis such as enthesophytes and marginal osteophytes may be present. Sclerosis of the third carpal bone is common in horses with osteochondral fragments of the third and radial carpal bones. Degree of sclerotic change is important, because in my experience a positive correlation exists between the degree of the third carpal bone sclerosis and the extent of cartilage damage on the third and radial carpal bones. However, a recent radiographic study showed no significant relationship between degree of sclerosis and prognosis.[25]

Scintigraphic examination is an excellent method to diagnose incomplete or occult osteochondral fragments and to evaluate other sites of stress-related subchondral bone injury in the involved or contralateral carpus. Focal IRU on one side of a joint seen in several scintigraphic views, including a flexed dorsal view, can help pinpoint exact location of osteochondral fragments.

Management The ideal treatment is surgical removal of osteochondral fragments, because unstable surfaces and fragment movement predispose to additional synovitis and development of osteoarthritis. Fragments left in place to heal in displaced fashion cause uneven joint surfaces and are prone to re-fracture. Arthroscopic examination also allows evaluation and grading of cartilage damage and intercarpal ligament integrity and identification of occult fragments. However, factors such as economic value, racing class, and time of year relative to upcoming races and location of osteochondral fragments are relevant. Horses not worthy of arthroscopic surgery are managed with short-term rest and intra-articular injections of hyaluronan and corticosteroids, or they are given 3 to

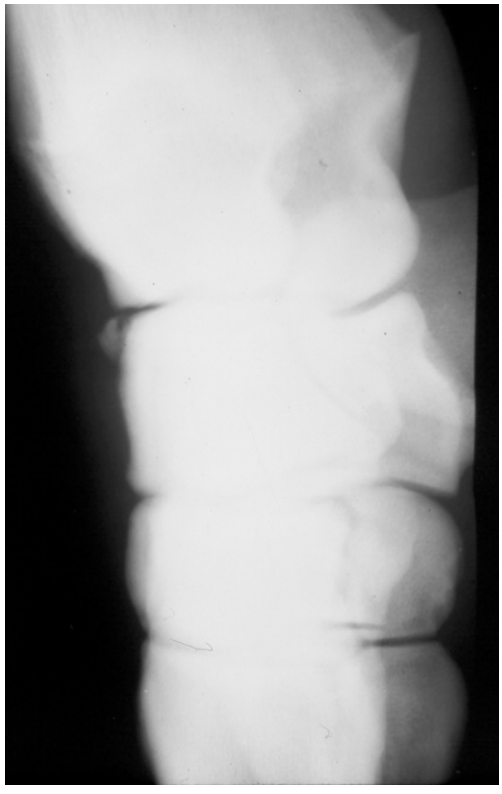

Fig. 39-7 Dorsomedial-palmarolateral oblique radiographic view of a typical osteochondral fragment of the proximal intermediate carpal bone. These fragments often appear in combination with osteochondral fragments of the distal lateral radius and vary in size, but they generally occur slightly more medial than those of the distal, lateral radius.

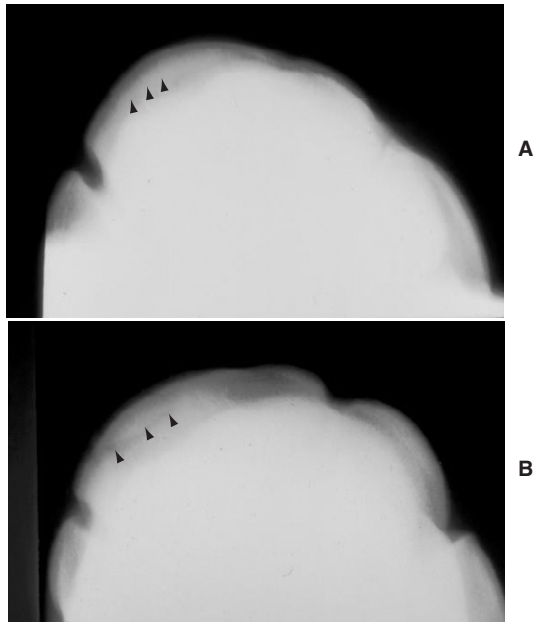

Fig. 39-8 **A,** Skyline radiographic view of 2-year-old Standardbred colt with incomplete osteochondral fragment of the third carpal bone. In this view there is predominantly dense sclerosis of the radial fossa and the faint fracture is difficult to identify *(arrowheads)*. **B,** Seven months later lameness and fracture recurred when the horse was 3 years old. The horse was given inadequate rest to allow the fracture to heal completely.

6 months of rest. When conservative management procedures are used, fractures develop fibrous unions and become stable, but they are usually displaced and may resemble marginal osteophytes when healed. Many horses that are managed conservatively, especially those with fractures in the antebrachiocarpal joint, return to racing successfully, but recurrence of osteochondral fragments and development of osteoarthritis is likely. It is difficult to convince owners and trainers to give horses long-term (>6 months) rest. However, rest, with or without arthroscopic surgery, is critical not only for healing of incomplete fractures, or if osteochondral fragments are removed, the fracture site, but also for healing of the surrounding cartilage and subchondral bone. Horses with incomplete osteochondral fragments are candidates for conservative management, but if horses are not given adequate time for fracture healing, recurrence is likely (Fig. 39-8). Alternatively, I have recommended arthroscopic surgery and fragment removal in horses with incomplete fractures if I know the client will not opt for long-term rest without surgical intervention. Although carpal lameness may recur, at least the original osteochondral fragment will not re-fracture. During arthroscopic surgery, cartilage damage is graded as mild, moderate, severe, or global and prognosis is inversely proportional to the extent of damage, a clinical finding substantiated in studies in STB, TB, and QH racehorses[21,24] (see Figs. 23-2 and 23-3). During arthroscopic surgery, the osteochondral fragment(s) are removed and the surrounding cartilage and bone are curetted, depending on the amount of damage seen. Damage on apposing surfaces is common, and occult osteochondral fragments are often found there or at distant locations. Bilateral carpal arthroscopic surgery is commonly performed in all racing breeds and in the TB and QH it may be necessary to evaluate all major joints.

After arthroscopic surgery horses are given a progressively increasing exercise program, the length of which depends on severity of damage and location of osteochondral fragments. For instance, in TB racehorses with osteochondral fragments of the distal, lateral radius and proximal intermediate carpal bone, I recommend 2 weeks of stall rest, followed by 4 weeks of stall rest with hand walking (or walking in a caged walker), followed by 2 to 4 weeks of turnout or swimming physiotherapy. Lameness is less pronounced initially, and horses appear able to return to work earlier when osteochondral fragments involve the antebrachiocarpal joint rather than the middle carpal joint. In horses with routine distal radial carpal bone and proximal third carpal bone fragments, I recommend 4 weeks of stall rest, followed by 4 weeks of stall rest with hand walking, followed by 8 weeks of turn out or swimming physiotherapy. In horses with numerous fragments and severe or global cartilage damage, I recommend 4 to 6 months of total rest.

After arthroscopic surgery I recommend the use of intra-articular injections of hyaluronan at 14 and 28 days in horses with mild or moderate cartilage damage, and a series of intra-articular PSGAGs injections at 3, 5, and 7 weeks after surgery. I recommend intra-muscular administration of PSGAGs once weekly for 8 weeks, beginning 14 days after surgery. Little concrete evidence shows that any form or combination of intra-articular therapy is of benefit for cartilage healing after surgery, although anti-inflammatory effects are likely mildly beneficial.

Cartilage resurfacing techniques have been used experimentally and in a limited number of horses with osteochondral fragments and osteoarthritis (see Chapter 23). Cell-based techniques such as injection of cloned chondrocytes in autogenous fibrin loaded with growth factors appear promising. Microfracture of calcified cartilage appears promising in

experimental trials, but seeing horses with partial-thickness cartilage damage is unusual. Subchondral bone is usually already exposed, obviating the need to use this.

Prognosis depends on several factors, including type and age of horse, racing class, limb(s) affected, number and location of osteochondral fragment(s), and amount of cartilage damage. Intuitively, a better prognosis would be expected in the STB than the TB and QH, because load is better shared by the hindlimbs and gait allows compensation by a lateral or diagonal limb. However, in STBs osteochondral fragments develop later in the non-adaptive remodeling process, when osteoarthritis is already well beyond that in TBs with comparable lesions. In most racehorses any damage of the third carpal bone is a major limiting factor in prognosis, and because osteochondral fragments of the third carpal bone are common in STBs, overall prognosis for STBs might be expected to be lower than for TBs and QHs. The prognosis for lesions in the antebrachiocarpal joint is better than for the middle carpal joint, and because osteochondral fragments occur with similar frequency in both joints in TBs and QHs, the overall prognosis is better. The prognosis for return to racing is good to excellent, but the likelihood of racing at the pre-injury level is inversely proportional to the degree of cartilage damage. Seventy-four percent of STBs with osteochondral fragments returned to racing after arthroscopic surgery, whereas only 61% raced at or above the pre-injury level. Pacers were more likely than trotters to start a race and to have five starts before and after injury.[21] Kinematic studies show that the pace may slow forelimb fatigue and reduce forelimb load.[26] Median earnings per start decreased significantly after injury and arthroscopic surgery, but horses went significantly faster after surgery.[21]

Of 445 TB and QH racehorses, 303 (68%) raced at a level equal to or better than the pre-injury level, but when grouped according to cartilage damage, only 53% of horses with the most severe cartilage damage raced at these levels. Eleven percent of horses had decreased performance and had carpal lameness, 6% developed additional osteochondral fragments, and 2% developed collapsing slab fractures while racing.[24] Prognosis was worse for horses with osteochondral fragments of the third carpal bone.

Unusual Articular Osteochondral Fragments

Osteochondral fragments do occur in the palmar aspect of the carpus, but they result from a single event injury. Trauma may result from falling, hitting a fence, or landing with subsequent hyperextension of the carpus. Osteochondral fragments usually involve the palmar aspect of the radial and intermediate carpal bones and the articular surface of the accessory carpal bone, can be singular or numerous, and occur medially and laterally. Articular osteochondral fragments of the accessory carpal bone are usually comminuted and involve at least two to three pieces. In horses with known chronic osteochondral fragments in the dorsal aspect of the carpus and those with osteoarthritis, radiographs occasionally reveal what appear to be small fragments or mineralization in the palmarolateral or palmaromedial aspects of the middle carpal joint (Fig. 39-9). One suggestion is that this represents dystrophic mineralization following corticosteroid injections, but in most horses these small fragments migrate from the dorsal to the palmar aspect of the joint and occur when training and racing continues in horses with existing osteochondral fragments, which then become macerated. When the condition is observed, osteoarthritis is usually extensive, and prognosis is guarded.

Lameness is acute in onset, with effusion of the middle carpal or antebrachiocarpal joints. Often the response to static flexion of the carpus is profound. Careful interpretation of radiographs is necessary to confirm the presence of osteochondral fragments, locate precisely the parent bone from which the osteochondral fragment arose, and determine the number of fragments. The clinician should recognize that osteochondral fragments in the palmar aspect of the carpus may represent only one aspect of more global damage to the joint(s). Any evidence of fragmentation dorsally, or active osteoarthritis if trauma occurred at least 10 to 14 days before radiographic examination, is a poor prognostic indicator, because this reflects substantial subchondral bone and cartilage injury dorsally and perhaps soft tissue instability. Osteoarthritis and chronic lameness are inevitable, regardless of management.

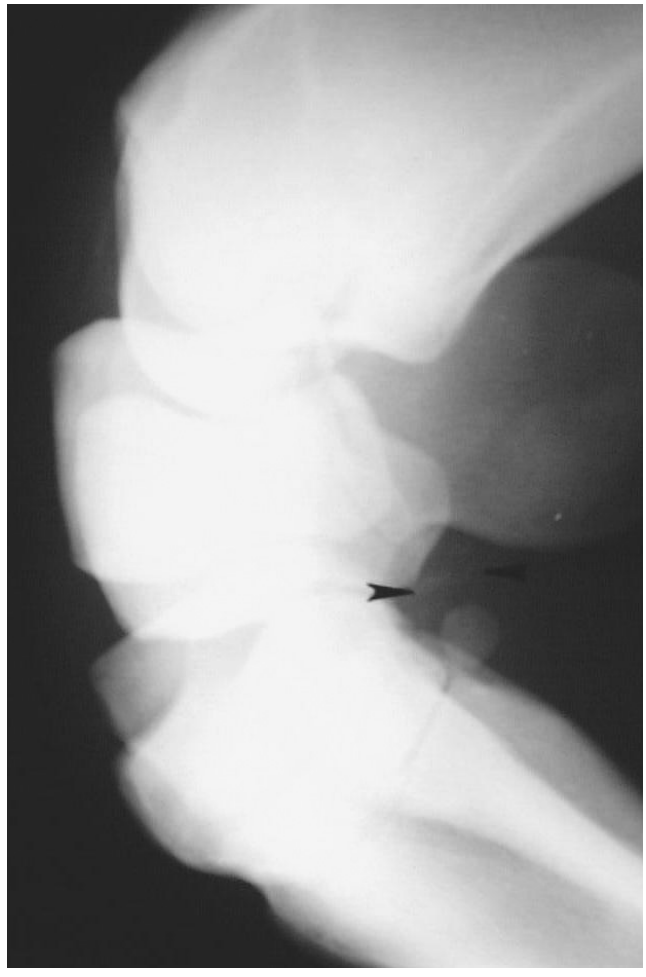

Fig. 39-9 A flexed lateromedial radiographic view. There is debris (*arrowheads*) in the palmar pouch of the middle carpal joint. This Standardbred had a large, loose, macerated third carpal bone chip fracture, and debris migrated to the palmar pouch. A first carpal bone can be seen distal to the loose debris and should not be confused with an authentic osteochondral fracture.

Palmar osteochondral fragments can be removed using arthroscopic surgery or arthrotomy,[27] but access can be difficult. The palmarolateral pouch of the antebrachiocarpal joint is large, and osteochondral fragments involving the accessory and intermediate carpal bones can be removed from this approach. Osteochondral fragments involving the palmaromedial or the palmarolateral aspect of the middle carpal joint can be removed using arthroscopic surgery, but both portals must be made within a small joint pouch, making triangulation difficult. Prognosis for future soundness is fair in nonracehorses, unless osteochondral fragments were associated with substantial instability or bone and cartilage damage at more distant sites. Prognosis is guarded in racehorses.

Large Fragments: Slab Fractures

Slab fractures are defined as fractures that involve a proximal and distal articular surface and thus traverse the entire depth

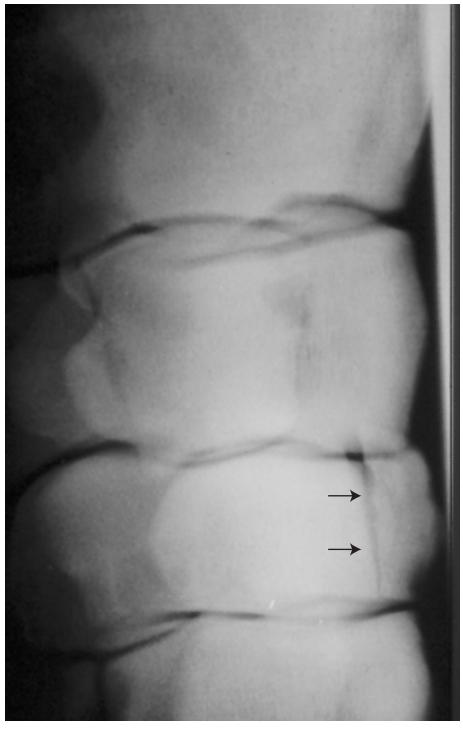

Fig. 39-10 Dorsolateral-palmaromedial oblique radiographic view showing a complete, non-displaced frontal slab fracture of the radial fossa of the third carpal bone *(arrows)*. The fracture line traverses the entire depth of the third carpal bone involving the middle carpal and carpometacarpal joints.

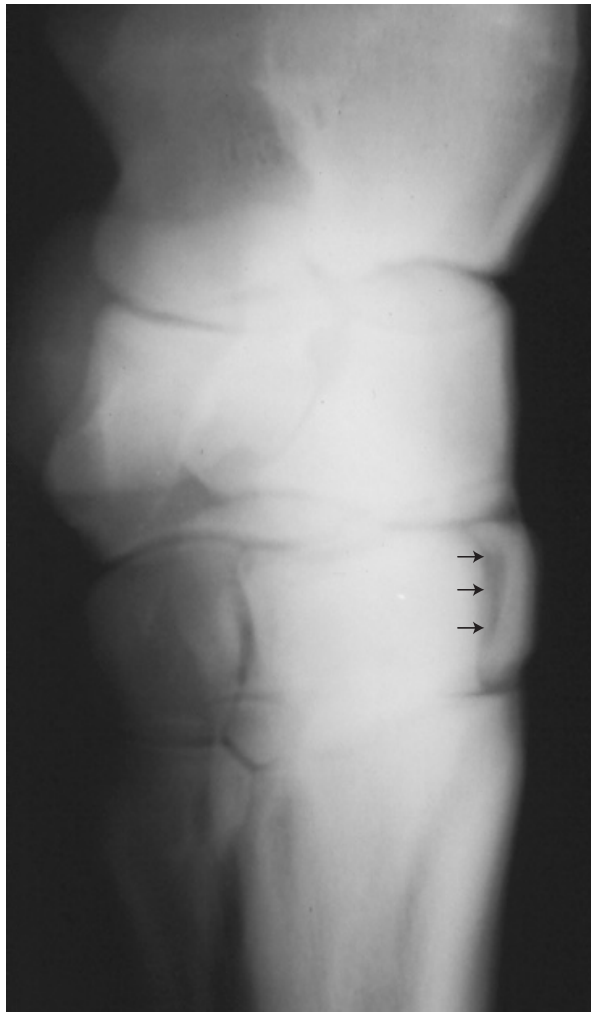

Fig. 39-11 Dorsomedial-palmarolateral oblique radiographic view showing a displaced frontal slab fracture of the intermediate fossa of the third carpal bone *(arrows)*.

(proximal-distal direction) of the bone. Size and involvement of two articular surfaces differentiate these fractures from small osteochondral fragments. Slab fractures of the third carpal bone are by far the most common form of large osteochondral fragments in the carpus, but slab fractures of the radial, fourth, and intermediate carpal bones occasionally occur. A combination of radial and third carpal bone slab fractures occurs in TB racehorses and may lead to instability and carpal collapse. In most instances, slab fractures develop as a terminal event in the cascade of non-adaptive remodeling changes leading to sclerosis, biomechanical weakness, and subsequent fracture. When slab fractures occur in unusual locations, the possibility of single-event injury must be considered. Slab fractures occur almost exclusively in racehorses.

Frontal slab fractures of the third carpal bone. The most common large osteochondral fragment in the carpus is a frontal slab fracture of the third carpal bone. A frontal slab fracture of the third carpal bone usually involves the radial fossa, but fractures of the intermediate fossa alone or in combination with fracture of the radial fossa do occur (Figs. 39-10 and 39-11). Frontal slab fractures of the third carpal bone involving the radial fossa vary in size and can involve the entire medial-to-lateral width of the fossa, or any portion of it, and range from the common size of 8 to 10 mm in the dorsal to palmar direction up to 20 to 25 mm. These latter, large fragments cannot be seen on a skyline radiographic view and extend across the entire medial to lateral width of the third carpal bone involving both fossae and are L-shaped (Fig. 39-12).

Lameness in horses with a frontal slab fracture is acute and severe and is generally worse than those with small osteochondral fragments, but chronic, subtle prodromal lameness may have been present. A previous history of small osteochondral fragments and arthroscopic surgery is common. Lameness may be less than expected in horses recently injected with corticosteroids and in those with bilateral frontal slab fractures of the third carpal bone. Clinical signs such as heat, effusion, and response to static and dynamic flexion are pronounced and horses are usually lame at the walk. Horses with non–weight-bearing lameness may have unstable carpi and should be evaluated carefully for collapse from comminuted fractures. The contralateral carpus should always be evaluated and palpation should be complete to uncover compensatory lameness problems. In TB racehorses in North America the right third carpal bone is affected more commonly than the left,[28] but in STBs the distribution between right and left is similar.[29]

Frontal slab fractures of the third carpal bone are usually obvious radiographically. If they involve the radial fossa, fractures are best seen on the LM, flexed LM, and DL-PaMO views and a skyline view. Because frontal slab fractures of the third carpal bone by definition involves the middle carpal and carpometacarpal joint surfaces, if a fracture line breaks out dorsally before reaching the carpometacarpal joint, the fragment is not a true slab fracture. This is important when determining a management plan. A frontal slab fracture of the third carpal bone can be incomplete, complete but non-displaced, or complete and displaced and may be associated with other small osteochondral fragments. Degree of displacement is worse on a standing LM view, but when the carpus is flexed, the fracture returns to near normal alignment. Flexion is a

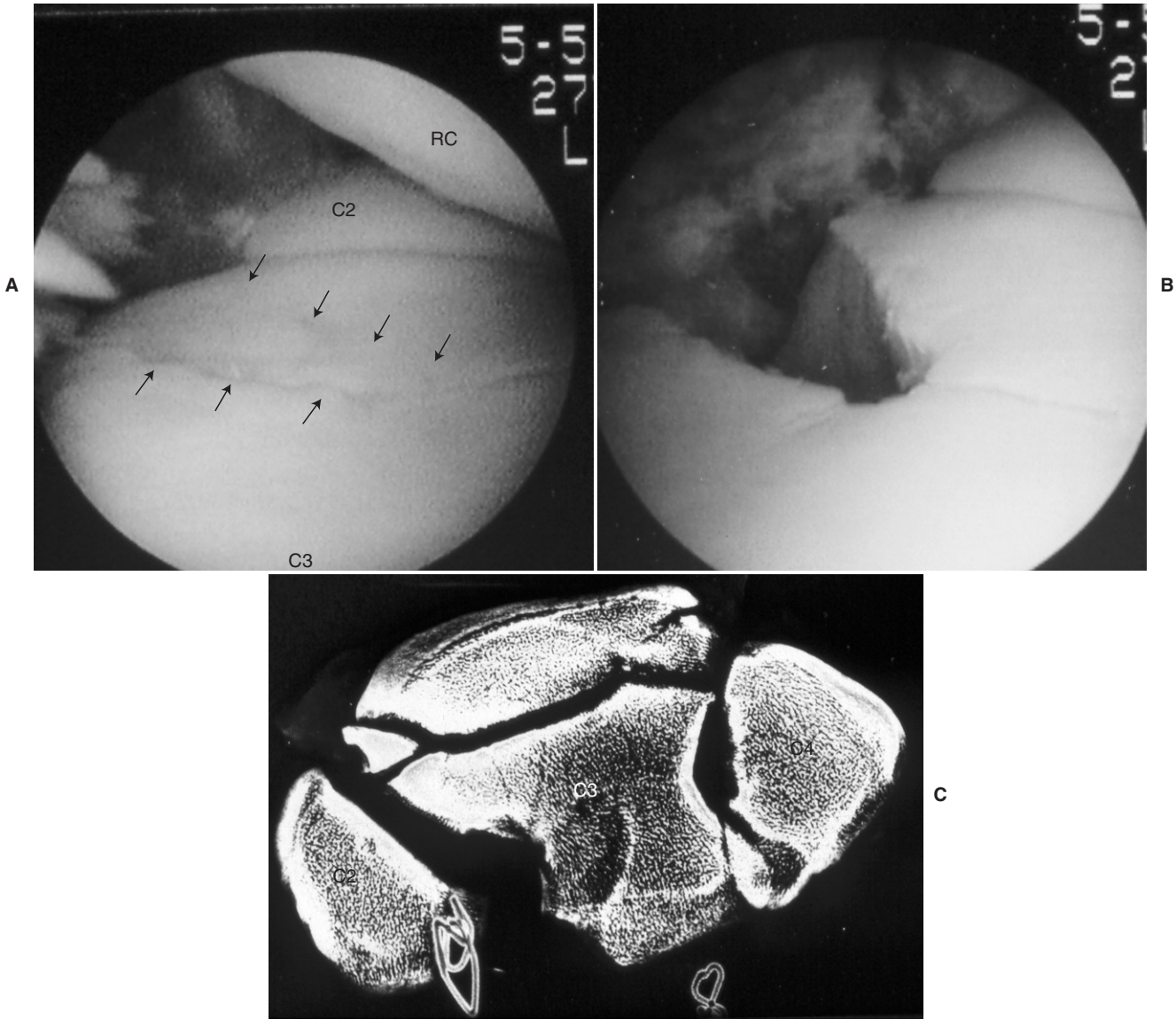

Fig. 39-12 This pacer had an L-shaped fracture of the third carpal bone that involved a sagittal and frontal component. **A,** The initial intra-operative photograph shows the sagittal component *(arrows)* and crushed medial aspect of the third carpal bone. **B,** This area was debrided and the fracture healed without internal fixation. The sagittal component of the fracture appears palmar to the defect. **C,** True proximal to distal radiographic view of a trotter taken at necropsy shows that the large slab fracture of the third carpal bone has frontal and sagittal components. *C2*, Second carpal bone; *C3*, third carpal bone; *RC*, radial carpal bone; *C4*, fourth carpal bone.

useful maneuver during reduction. In most instances a triangular, wedge-shaped fragment appears in the trough between the frontal slab fracture fragment and the parent bone, and this accounts for the appearance of many fracture lines in a skyline view. Careful examination of the overlying radial carpal bone is required in TB racehorses, because concomitant slab fracture of the radial carpal bone can accompany a frontal slab fracture of the third carpal bone.

Management depends on several factors including the horse's value, age, racing class, presence of other lameness problems, and specifically whether the frontal slab fracture of the third carpal bone is incomplete, displaced, or comminuted. Long-term rest (6 months) is successful in horses with incomplete or complete non-displaced fractures. However, surgical fixation can help preserve articular surfaces by preventing displacement. Frontal slab fracture fragments that are thin (<5 mm in the dorsal to palmar direction) can be removed using arthroscopic surgery or conventional arthrotomy techniques or, if the articular surface is intact, can be repaired. Surgical removal of small frontal slab fractures of the third carpal bone can be difficult using arthroscopic techniques and can leave a large defect radiographically but is successful. Horses with large frontal slab fragments that are displaced are usually managed with arthroscopic surgery and internal fixation, using one or two cortical screws placed in lag fashion. Either 3.5- or 4.5-mm screws can be used, but I prefer using one or two 3.5-mm screws because the screw heads are smaller and countersinking in the dorsal aspect of the third carpal bone is not necessary. Use of needles and intra-operative radiographs to position and guide screw insertion accurately is preferred.[30]

A single bone screw provides adequate fixation in horses with most frontal slab fracture of the third carpal bone involving only the radial fossa, because capsular attachments maintain rotational stability. Two or three 3.5-mm screws are necessary to repair large fragments traversing both fossae. The fracture line is debrided, often before screw placement, and the wedge-shaped trough fragment is removed leaving a gap at the fracture line. Loose cartilage is curetted, cartilage damage is graded, and other small osteochondral fragments are removed if present. Alternatively the proximal articular surface of the fragment may be removed, with repair of the distal aspect, leaving the portion of the fragment with capsular attachments intact. Occasionally, additional slab fractures of the third or radial carpal bones are found and repaired. In horses with more than one slab fracture a Robert Jones bandage or cast is used for recovery from general anesthesia. In TB racehorses with a frontal slab fracture of the third carpal bone, an ominous defect in the articular cartilage of the radial carpal bone is often seen, caused by the incongruent apposing surface of the third carpal bone, but in most horses subchondral bone fracture is not present. Horses are typically given stall rest for 4 weeks, followed by stall rest with hand walking for 8 weeks, followed by turnout in a small paddock for 2 to 3 months before beginning race training. Recommendations for intra-articular therapy are similar to those in horses with small osteochondral fragments.

In a large retrospective study of TB and STB racehorses with a frontal slab fracture of the third carpal bone, the radial fossa was involved in 87% of horses, and females of both breeds were less likely to race after injury and surgery, but treatment and fracture characteristics had no effect on outcome.[29] Fracture characteristics such as size and degree of displacement did influence treatment selection, so thin frontal slab fractures of the third carpal bone were removed, displaced fractures were repaired, and horses with incomplete or non-displaced frontal slab fracture of the third carpal bone were managed conservatively.[29] All STBs that raced before, raced after surgery and overall 77% raced after surgery. Sixty-five percent of TBs with frontal slab fractures of the third carpal bone raced after surgery, and although earnings per start decreased in both breeds, the decrease was more pronounced in TBs.[29] In another study, 67% of TBs with a frontal slab fracture of the third carpal bone raced at least once after surgery, but the mean claiming value in horses decreased significantly.[28]

Sagittal slab fractures of the third carpal bone Sagittal slab fractures of the third carpal bone are much less common than frontal slab fractures, usually involve the medial aspect of the radial fossa (Fig. 39-13, *A*), and occur in a direction parallel to the articulation between the second and third carpal bones. However, sagittal slab fractures of the third carpal bone can occur in the intermediate fossa, and fractures can be bilateral. Authentic sagittal slab fractures of the third carpal bone involve both articular surfaces of the bone and can be best seen radiographically in skyline and DM-PaLO views. Sagittal slab fractures of the third carpal bone must be differentiated from corner fractures, subchondral lucency, and other crushing type of injuries that occur in the radial fossa. Clinical signs associated with sagittal slab fractures of the third carpal bone are similar to those of other osteochondral fragments but are usually less severe than a frontal slab fracture of the bone. Lameness is usually prominent, but in horses with bilateral fracture or substantial osteoarthritis or osteochondral fragments in the contralateral carpus, diagnostic analgesia may be required. Often horses have a prominent history of chronic carpal lameness before acute lameness develops. Of 28 horses with sagittal slab fractures of the third carpal bone, 17 were TBs, 9 were STBs, and 2 were Arabian racehorses, and fractures were found in the right forelimb (17), left forelimb (10), and bilaterally in one horse.[31]

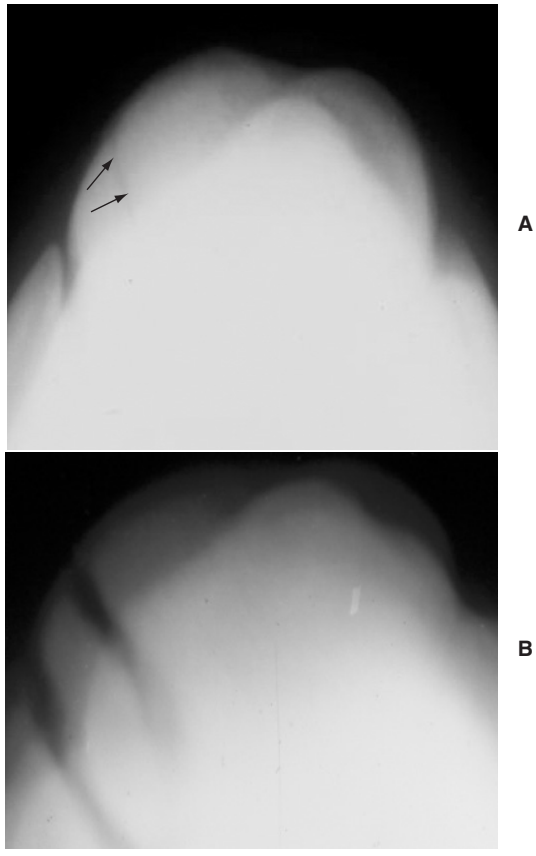

Fig. 39-13 **A,** Initial and **B,** 8-month follow-up skyline radiographic views of the third carpal bone showing a sagittal slab fracture *(arrows)* that failed to heal and became a non-union **(B).** This Standardbred trained and raced with the non-union fracture but dropped substantially in class. Results such as this have prompted me to manage sagittal slab fractures of the third carpal bone surgically.

Conservative management can be successful, but only 7 of 12 horses with sagittal slab fracture of the third carpal bone managed conservatively raced.[32] Fracture healing takes extensive time, and a chronic non-union often develops (Fig. 39-13, *B*). Horses can be sound enough to race with chronic non-unions, but this is undesirable. I have seen one STB and one TB racehorse with bilateral lateral, non-union sagittal slab fractures of the third carpal bone racing successfully, but with conservative management comes a risk of chronic high-speed lameness, worse if fracture involves the right forelimb, and horses drop substantially in racing class. If any doubt exists about the nature of a lesion affecting the medial aspect of the third carpal bone, arthroscopic examination can be performed to formulate a surgical plan. If an authentic sagittal slab fracture of the third carpal bone does not exist, but medial corner osteochondral fragments, necrotic subchondral bone, or other crush type of injuries are found, a combination of fragment removal and curettage is performed. I currently recommend surgical management for most horses with sagittal slab fractures of the third carpal bone. I prefer a direct view provided by arthrotomy, because positioning a screw perpendicular to the fracture line using arthroscopic surgery and stereotactic techniques is difficult and because the fracture is closely associated with the second carpal bone. A single 3.5-mm screw is placed in lag fashion. Management after surgery is similar to that described for a frontal slab fracture of the third carpal bone. Results using screw fixation appear superior to those with conservative management.[31]

Slab fractures of other carpal bones Slab fractures of carpal bones other than the third are unusual. Whenever slab fractures of the radial carpal or other unusual slab fractures are discovered, all radiographic views should be evaluated carefully. Slab fractures of the radial carpal bone can occur independent of or in combination with frontal slab fracture of the third carpal bone. When frontal slab fractures of the medial and third carpal bones occur simultaneously and carpal instability results, the fracture is called a *comminuted carpal fracture*. Repairing both bones by placing numerous screws using arthroscopic surgery may be possible. A sleeve or full-limb fiberglass cast is placed, and an assisted recovery from general anesthesia is recommended.

Slab fractures of the intermediate, fourth, and ulnar carpal bones occur rarely. In five horses with intermediate and fourth carpal bone slab fractures, outcome was poor because of delay in diagnosis, and four horses were only pasture sound.[33] Surgical repair should proceed as early as possible, but prognosis is limited by cartilage damage and other osteochondral fragments.

Subchondral Lucency of the Third Carpal Bone

Subchondral lucency of the third carpal bone is an unusual condition occurring almost exclusively in STB racehorses and seen radiographically as single or multiple central areas of bone loss in the radial fossa of the third carpal bone (see Fig. 39-1, *B*). Lesions can be seen in a DL-PaMO view in horses with advanced subchondral lucency of the third carpal bone and in xeroradiographs. Mild to severe sclerosis may surround radiolucent defects,[34] but in a recent study no relationship was found between radiolucency of the third carpal bone and sclerosis.[25] The lesion was more common in the right carpus, and although pacers predominated, distribution was similar to the racing population in the United States.[34] Pathogenesis is identical to that described for osteoarthritis, osteochondral fragments, and slab fractures of the third carpal bone, but in horses with subchondral lucency of the third carpal bone, necrosis of subchondral bone causes table surface collapse, rather than osteochondral fragments or slab fractures involving the dorsal margin. Subchondral lucency of the third carpal bone is not a disease of young STBs, because the mean age was 4.1 years (range, 3 to 7 years) and chronic stress-related subchondral bone injury is required. The condition is rarely recognized in TBs probably because of gait differences and the fact that often osteoarthritis is more advanced in the STB before overt signs of lameness are seen.

Prodromal clinical signs of mild carpal lameness and a history of numerous intra-articular injections are present before onset of acute lameness, usually graded between 2 to 4 on a 5-point scale. Heat, effusion, and a positive response to flexion may be absent, and diagnostic analgesia is usually required to confirm diagnosis. The results of initial radiographic examination may be equivocal, and follow-up radiographic and scintigraphic examinations can help pinpoint subchondral lucency of the third carpal bone.

Surgical debridement is the treatment of choice. During arthroscopic examination, soft crumbly necrotic subchondral bone and damaged overlying cartilage are found. In some horses only a small full-thickness cartilage defect can be probed, but deep extensive subchondral bone softening is later found, whereas in others, fibrin-filled, full-thickness defects resembling subchondral bone collapse or table surface fracture are found. The damaged tissue is curetted, and if the resulting lesion is <5 mm palmar to the dorsal margin of the third carpal bone, this lip is removed, creating a defect that resembles removal of an osteochondral fragment. If subchondral lucency of the third carpal bone lesions are deep within the third carpal bone, the dorsal margin is not removed. Cartilage resurfacing could be considered, but it is not necessary for a successful outcome.

Prognosis is excellent for return to racing, particularly in pacers, but horses drop in racing class. Prognosis is guarded in 3- or 4-year-old trotters to return to racing the next year, but some make useful older racehorses. Of nine horses, eight returned to racing, but only six raced at the previous level.[34]

Comminuted Fractures

Comminuted fractures are unusual, but they occur occasionally in TB and QH racehorses and other horses that suffer injury while turned out in a field. Comminuted in this sense refers to numerous fractures in more than one carpal bone. In STB racehorses large, comminuted fractures of the third carpal bone can occur, but I have not seen a STB with a large osteochondral fracture involving more than one bone. The most common combination of fractures in TB and QH racehorses involves the third carpal bone and either the radial or intermediate carpal bone, or both. Carpal instability, most commonly carpus varus, is usually readily apparent if horses bear weight, but they are usually non–weight bearing. These injuries occur almost exclusively in racehorses, and often horses have chronic carpal lameness before injury, but fracture can occur in other types of horses without previous lameness during turnout exercise. Effusion and edema of surrounding soft tissues occurs. Diagnosis is confirmed radiographically (Fig. 39-14). If fractures involve only the distal row of carpal bones, instability is minimal and prognosis is better than if the middle carpal and antebrachiocarpal joints are involved. Consideration should be given for euthanasia if the horse is a gelding or intact male with limited breeding potential. Conservative management using sleeve or full-limb casts, or a combination of splints with a Robert Jones bandage, may give support and provide comfort, but usually instability is pronounced, horses develop extensive cast sores, and they are at risk to develop laminitis in the contralateral limb. Severe osteoarthritis, collapse, and angular limb deformity generally result in an unacceptable outcome in horses managed conservatively. Surgical management using carpal arthrodesis should be considered.

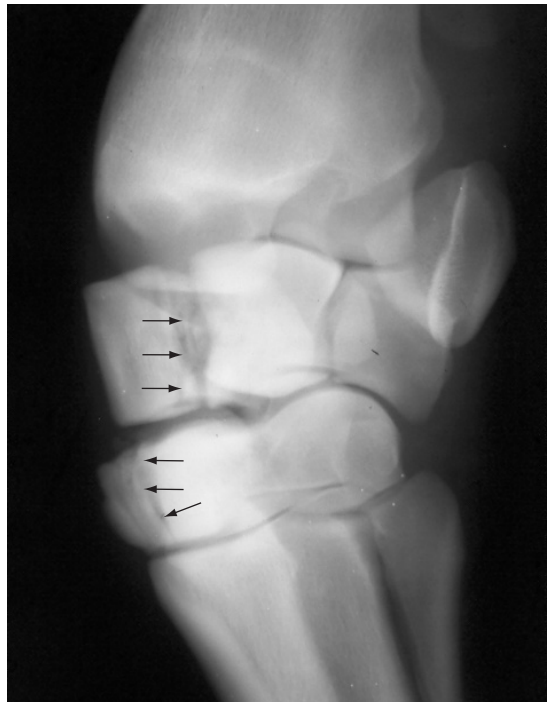

Fig. 39-14 Non–weight-bearing dorsolateral-palmaromedial oblique radiographic view of a Thoroughbred with a comminuted carpal fracture. The radial carpal and third carpal bones have frontal slab fractures *(arrows)*.

Carpal Arthrodesis

Arthrodesis is indicated when horses have comminuted carpal fractures and instability, severe lameness and osteoarthritis of one or more carpal joints, or when osteoarthritis is severe with collapse and angular deformity of the carpus. Partial (intercarpal) carpal arthrodesis involves fusing the middle carpal and carpometacarpal joints using bone plates applied to the proximal row of carpal bones and McIII. Pan-carpal arthrodesis involves bridging the entire carpus with bone plates applied to the distal radius, both rows of carpal bones, and McIII, fusing all three joints. Two dynamic compression plates are used for either technique, and in one study allografts were used in some horses to provide axial support for horses with collapsed carpi undergoing pan-carpal arthrodesis. In one horse, I successfully used acrylic to create an artifical fourth carpal bone to maintain axial stability, in addition to two bone plates and interfragmentary repair of fractures. Prognosis for salvage was good in horses with partial carpal arthrodesis[35] and fair to good in horses requiring pan-carpal arthrodesis.[35,36]

Accessory Carpal Bone Fractures

Fracture of the accessory carpal bone results from trauma, such as a fall while jumping, a hyperextension injury when landing, or an accident while turned out. Acute, severe lameness and swelling involve the palmar aspect of the carpus. Horses have severe pain during carpal flexion. Carpal tenosynovitis may occur acutely, but it is difficult to differentiate from diffuse swelling. Pronounced lameness lasts for several days, but horses are rarely non–weight bearing, and swelling resolves in 2 to 3 weeks. Horses with a chronic fracture may have little to no swelling, and diagnosis may be difficult, unless carpal tenosynovitis is present, because the lameness is not typical of carpal pain.

The most common fracture of the accessory carpal bone is a vertical slab fracture that involves the palmar aspect of the bone in the frontal plane (Fig. 39-15). Most fragments are simple, but small fragments at the proximal aspect of the fracture may occur. Fractures can be incomplete, but most are complete with mild displacement. Gross displacement and large comminuted fragments are unusual. Articular fractures were discussed previously.

Conservative management of non-articular fractures of the accessory carpal bone is recommended. The forces on the accessory carpal bone are substantial and result in failure of screw fixation, chronic instability, implant loosening, and osteitis (Fig. 39-15). Surgical fixation using the tension band principle may be successful, but I have not attempted repair because prognosis is favorable with conservative management. Horses are given NSAIDs, and a heavy full-limb cotton bandage is applied. Horses are given stall rest for 8 weeks, followed by stall rest with hand walking for 8 weeks, followed by walking with a rider up for 8 weeks. Radiographs most often reveal bony proliferative changes and fibrous rather than bony union of the fracture. Even with fibrous union, horses are usually serviceably sound. Prognosis for TB racehorses appears to be worse than for field hunters, jumpers, and other horses that may be able to perform with a mild gait restriction from chronic fibrosis. Carpal canal syndrome and carpal tenosynovitis may result (see Chapter 76).

Osteochondrosis

Osteochondrosis of the carpus is rare. In experimental studies extensive cartilage lesions histologically similar to those in naturally occurring osteochondritis dissecans have been produced. Rarely, radiographs of yearlings before or after public sales reveal rounded osteochondral fragments, usually involving the distal medial radius or the distal medial aspect of the radial carpal bone or proximal aspect of the third carpal bone. These fragments appear as solitary osteochondral fragments and do not have associated marginal osteophytes or enthesophytes, unlike osteochondral fragments caused by trauma at a young age. At the time of surgery these unusual fragments appear intercalated in dense joint capsule attachments, are rounded, and often have a smooth defect in the distal radius or third carpal bone, apparently the origin of the fragment. Whether these fragments represent a form of osteochondritis dissecans or old trauma is not known, but they appear similar to forms of osteochondritis dissecans found elsewhere.

Osseous Cyst-like Lesions

Osseus cyst-like lesions do occur in the carpus. Those of the ulnar carpal bone are common, appear to be incidental radiographic findings, are usually non-articular, and have sclerotic borders. Incidental osseous cyst-like lesions may also be seen in the second carpal bone, often in the presence of the first carpal bone, and also in the base (head) of McII. Osseous cyst-like lesions of the radial carpal bone and distal radius can cause lameness.[37] Effusion, heat, and a positive response to flexion are inconsistent and diagnostic analgesia is usually necessary. Radiographic signs may be obvious, but in some horses they are subtle, and scintigraphic examination is useful to pinpoint the area of modeling. In horses with an osseous cyst-like lesion that communicates with an articular surface, I have performed debridement and curettage, but results have been unfavorable to fair at best.

Osteochondromatosis

Osteochondromatosis is rare but has been seen in the carpometacarpal joint.[38] Progressive enlargement of the carpometacarpal joint and numerous unusual radiopacities were seen radiographically.[39] Arthroscopic examination of the carpometacarpal joint was possible, and osteochondral fragments were removed[38] (see Chapter 68).

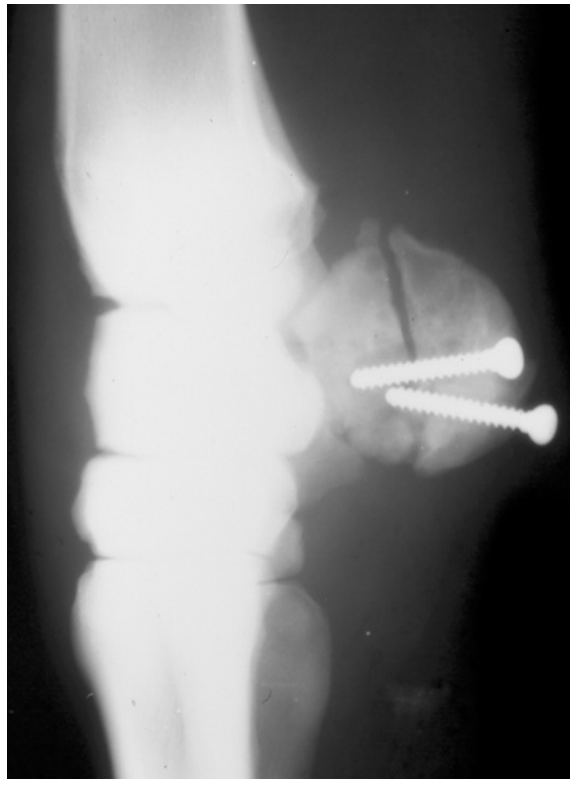

Fig. 39-15 A lateromedial radiographic view of a horse with a failed attempt at surgical management of a frontal slab fracture of the accessory carpal bone. These fractures are best managed conservatively without attempting repairs such as this one.

Infectious Arthritis

See Chapter 66 for a discussion of infectious arthritis.

Other Fractures Involving the Carpus

Avulsion Fracture of the Third Metacarpal Bone Associated with the Origin of the Suspensory Ligament

See Chapter 38 for a discussion of avulsion fracture of the third metacarpal bone associated with the origin of the suspensory ligament (page 367).

Incomplete, Longitudinal Fracture of the Proximal Palmar Cortex of the Third Metacarpal Bone

See Chapter 38 for a discussion of incomplete, longitudinal fracture of the proximal palmar cortex of the third carpal bone (page 364).

Dorsomedial Articular Fracture of the Proximal Aspect of the Third Metacarpal Bone

See Chapter 38 for a discussion of dorsomedial articular fracture of the proximal aspect of the third metacarpal bone (page 367).

Fracture of the Proximal Aspect of the Second and Fourth Metacarpal Bones

See Chapter 38 for a discussion of the proximal aspect of the second and fourth metacarpal bones (page 371).

Articular Fracture of the Distal Radius

Fractures of the distal radius rarely involve the antebrachiocarpal joint. Complete or incomplete, displaced or nondisplaced fractures of the radius in the sagittal plane and resembling condylar fractures of McIII/MtIII can occur.[39] Horses should be managed conservatively using full-limb bandages, NSAIDs, and absolute stall rest if fractures are nondisplaced. Some consideration should be given to cross-tying the horse for 3 to 6 weeks. Comminuted fractures of the radius, or those involving the distal radial physis and epiphysis that involve the antebrachiocarpal joint, should be repaired.

Soft Tissue Injuries of the Carpus and Carpal Region

Intercarpal Ligament

Various degrees of tearing of the medial palmar intercarpal ligament have been observed during arthroscopic examination of horses with osteochondral fragments or osteoarthritis and in some horses with occult carpal lameness. The medial palmar intercarpal ligament has recently been the source of considerable study and supposition, but whether tearing of this ligament observed in horses with carpal disease is causative or simply results from other pathological conditions is not yet known. The anatomy of the medial palmar intercarpal ligament and the relationship between osteochondral fragments and the dorsomedial intercarpal ligament were previously discussed (see page 377).

Because tearing of the medial palmar intercarpal ligament is an observation during arthroscopic examination of the middle carpal joint and a diagnosis impossible currently to make before surgery, specific clinical signs cannot be described. Once MRI becomes more widely available, diagnosis in horses with occult carpal lameness suspected to originate from the medial palmar intercarpal ligament may be possible before arthroscopic examination. Tearing of the medial palmar intercarpal ligament was observed in 27 joints in 20 horses with carpal lameness.[5] No correlation was found between tearing and severity of clinical signs. Joints with more medial palmar intercarpal ligament tearing had significantly *less* cartilage and subchondral bone damage, and an inverse relationship existed between the size and number of osteochondral fragments and ligament damage.[5] Therefore, although tearing of the medial palmar intercarpal ligament occurred commonly in horses with middle carpal joint damage, tearing was not seen with severe subchondral bone damage, and what role damage plays in the development of joint disease remains questionable.

In my experience, tearing of the medial palmar intercarpal ligament is unusual without at least mild cartilage damage or osteochondral fragmentation, but I have seen a small number of horses in which the only identifiable lesion was tearing and hemorrhage of the frayed ends, resulting in hemarthrosis. In these horses examining the palmar aspect of the middle carpal joint from the routine dorsal approach was possible. Rupture of the medial palmar intercarpal ligament has been seen in a small number of non-racehorse competition horses with pain localized to the middle carpal joint.[40] No other lesion was identified during arthroscopic examination. I suspect tearing of the medial palmar intercarpal ligament in horses with confirmed carpal lameness, but in which scintigraphic and radiographic examination findings are negative or equivocal, and in those with unexplained hemarthrosis of the middle carpal joint. Because horses that fall into this category are rare, prognosis is difficult to estimate.

Collateral Ligament

Injury of the dense collateral ligaments of the carpus is rare (see Chapter 68). Usually fracture of the distal radius, carpal bones, or the proximal aspect of McII and McIV occurs instead of collateral ligament damage. In foals, collateral ligament laxity is seen commonly and is self-limiting but can contribute to the development of angular limb deformity.

Carpal Tenosynovitis and Flexor Tendonitis

See Chapters 70 and 76 for a discussion of carpal tenosynovitis and flexor tendonitis (pages 629, 637, and 686).

Extensor Tendon

See Chapter 78 for a discussion of extensor tendon injury (pages 693 to 695).

Proximal Suspensory Desmitis

See Chapter 73 for a discussion of proximal suspensory desmitis (pages 654 to 662).

Synovial Ganglion

See Chapter 68 for a discussion of synovial ganglion injury (page 611).

Carpal Hygroma

Hygroma refers to a sac, bursa, or cavity filled with fluid. Carpal hygroma usually results from direct and blunt trauma to the dorsal aspect of the joint, including the capsule and overlying extensor tendons and sheaths. Carpal hygroma is an occasional complication after arthroscopic surgery or arthrotomy of the carpus. During arthrotomy, a distinct subcutaneous space over the dorsal aspect of the carpus is encountered, but in most instances this is a potential rather than a real space. When traumatized, this space fills with fluid, resulting in a large sac. Initially, diffuse edema may be present, and horses may be lame, particularly after flexion, but lameness soon abates. A large, fluid filled, non-painful swelling results that must be differentiated from extensor tenosynovitis (longitudinal swellings) and herniation of the middle carpal and antebrachiocarpal joint capsules (horizontal swellings). Lameness is usually not observed in horses with chronic hygroma unless a large swelling prohibits carpal flexion. Communication with an extensor sheath or joint capsule is rare, unless previous arthroscopic surgery has been performed and a portal inadvertently was made through a tendon sheath. In naturally occurring carpal hygroma a secretory lining is present, but it must develop from cell differentiation because communication with a source of synovial cells is lacking. Diagnosis is based on clinical signs. Positive contrast radiography and ultrasonographic examination are used to determine extent and communication with nearby structures.

The swelling is usually persistent, but it often does not compromise gait, although swelling does result in a cosmetic blemish. I have heard of resolution after injection with oxytetracycline, atropine, or contrast material. Drainage, with or without corticosteroid injection, and chronic bandaging are

usually unsuccessful in permanently resolving swelling. Surgical management in the form of en bloc resection is the treatment of choice for cosmetic results. Simply draining the cavity and inserting through-and-through or closed-suction drains may temporarily resolve swelling, but recurrence is likely. I attempt resection en bloc without entering the hygroma cavity, but invariably the lining is penetrated. The lining is dissected from all overlying subcutaneous tissues and underlying capsule and tendon sheaths. Once removed, Penrose drains are placed, a light sterile bandage is applied, and horses recover with a sleeve cast-bandage that is left in place a minimum of 7 to 10 days. Drains are removed between 7 and 10 days when cast material is cut, and then the cast material can be used as a splint over a heavy padded bandage. Horses are given absolute stall confinement for 3 weeks. This treatment is successful but expensive.

Neoplasia

A rare cause of carpal region lameness is neoplasia. Lameness and progressive swelling were seen in a 20-year-old horse with chondrosarcoma of the distal radius that caused bony proliferation and swelling of the antebrachiocarpal joint.[41] I have seen an undifferentiated sarcoma of the distal antebrachium causing progressive swelling, mild lameness, and a reduction in carpal flexion in a TB broodmare.

Hypertrophic Osteopathy

Lameness associated with hypertrophic osteopathy (see Chapters 15 and 38) is usually mild, and the condition does not directly affect the carpus, but fibrous and bony swelling of the distal radius may cause enlargement of the antebrachiocarpal joint region, prompting radiographic examination.

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CHAPTER • 40

Antebrachium

Lance H. Bassage and Mike W. Ross

ANATOMY

The antebrachium lies between the elbow and carpus and comprises principally the radius and small vestigial portion of the ulna and the flexor and extensor muscles. The tendons of the superficial and deep digital flexor muscles, the accessory ligament of the superficial digital flexor tendon and the carpal sheath are discussed elsewhere (see Chapters 70, 71, and 76). The medial aspect of the antebrachium is relatively devoid of soft tissue coverage and this is important when considering fractures of the radius. Major neurovascular structures include the median artery (continuation of the brachial artery in the proximal antebrachium), vein, and nerve; the radial, ulnar, and cutaneous antebrachial nerves; and the accessory cephalic and cephalic veins.

CLINICAL DIAGNOSIS AND IMAGING CONSIDERATIONS

Lameness associated with the antebrachial region is relatively unusual. Clinical signs are obvious in horses with unstable radial fractures or in those with marked soft tissue swelling. In others diagnosis can be challenging and ruling out other causes of forelimb lameness and then using diagnostic imaging to reach a definite diagnosis may be necessary. Perineural analgesia of the median and ulnar nerves (see Chapter 10) is performed to rule out more distal sources of pain.

Definitive diagnosis of most lameness problems of the antebrachium can be made using conventional radiography and ultrasonography, but nuclear scintigraphy is useful for diagnosing incomplete and stress fractures of the radius, enostosis-like lesions, and enthesopathy at the origin of the accessory ligament of the superficial digital flexor tendon.

Osteochondroma of the Distal Radius

See Chapter 76 for a discussion of osteochondroma of the distal radus.

Physeal Dysplasia of the Distal Radius (Physitis)

See Chapter 59 for a discussion of physitis.

Traumatic Physitis and Closure of the Distal Radial Physis

A syndrome of vague forelimb lameness believed to be associated with inflammation or pain originating from the distal radial physis has been recognized in young racehorses in early training. Anecdotally the condition appears to be more prevalent in 2-year-old colts. Distal radial physeal closure measured radiographically occurred earlier in fillies (701 days) than in colts (748 days).[1] Presumably the condition results from repetitive trauma to an open physis. The term *open knees* is commonly used to describe the state of skeletal immaturity. This condition is distinct from physeal dysplasia (physitis), because the condition is not a developmental abnormality, is not associated with clinically apparent enlargement of the metaphyseal region, and occurs in 2-year-old horses in active training.

Mild to moderate forelimb lameness is vague, without indications of a problem elsewhere in the limb. The condition is usually bilateral, but horses can show unilateral lameness, because one limb is more painful than the other. Horses often have a choppy stride, with the legs carried wide. Focal heat may be present, but swelling is usually absent or minimal and pain may be difficult to detect. Presumptive diagnosis is made on the basis of history, clinical signs, and ruling out other causes of lameness. A positive response to median and ulnar nerve blocks can be used to confirm that the distal antebrachium is the source of pain, but this can also abolish subchondral bone pain in the carpus. Definitive radiographic abnormalities are rarely present, but a radiographically open physis supports the diagnosis. Nuclear scintigraphy is generally not helpful, because all horses of this age have moderate to intense increased radiopharmaceutical uptake at the physis. However, scintigraphy is useful for identifying or ruling out other potential causes of lameness, and asymmetrical radiopharmaceutical uptake (greater in the affected physis of the more severely affected limb) may support the diagnosis.

Treatment consists primarily of rest or a reduction in exercise intensity and systemic non-steroidal anti-inflammatory drugs (NSAIDs). Duration of rest varies with the skeletal maturity of the horse and severity of the condition. Local injection of corticosteroids or other drugs, such as homeopathic remedies, and systemic treatment with anabolic steroids

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Treatment consists primarily of rest or a reduction in exercise intensity and systemic non-steroidal anti-inflammatory drugs (NSAIDs). Duration of rest varies with the skeletal maturity of the horse and severity of the condition. Local injection of corticosteroids or other drugs, such as homeopathic remedies, and systemic treatment with anabolic steroids

have been used but are of dubious value. For some horses slow jogging for 4 to 6 weeks may be all that is required, and for others stall confinement with hand walking exercise progressing to paddock turnout for several months may be necessary. Follow-up radiographs can be used to monitor physeal closure, which is often used to determine the appropriate time to resume harder training.

The issue of distal radial physeal closure, the role of radiographs in making this determination, and how to determine the point when training should commence are controversial. Many trainers and veterinarians customarily radiograph the distal radius of 2-year-olds, and those with open knees (radiographic evidence that bony union at the physis is incomplete) are withheld from hard training until the physes have closed. Radiographic closure of the distal radial physis generally occurs by 20 to 24 months of age[2] or slightly later.[1] Scintigraphic activity of the distal radial physis persists well after radiographic evidence of closure is observed.[3] However, endochondral ossification ceases (biological closure) before fusion is evident radiographically, and in our experience horses with a thin or faintly visible physeal remnant visible radiographically are at low risk of traumatic physitis.

Because the diagnosis is difficult to substantiate and pain may originate from an undetermined source, giving an accurate prognosis is difficult. If other more common conditions have been ruled out and the diagnosis of traumatic physitis is accurate, the prognosis is excellent. Many horses remaining in training develop signs of carpal lameness, and traumatic physitis may simply represent a prodromal phase of early osteoarthritis and bone pain. Finally, no correlation between age or month of closure of the distal radial physes and money won, races won, fastest mile, or fastest win mile during the 2-year-old year was found in Standardbreds.[4]

Radial Fractures

Radial fractures almost always result from external trauma, often a kick from another horse in adults, or from being stepped on or kicked by a mare in foals. Stress fractures of the radius also occur,[5,6] but in our experience true stress fractures of the radius are rare, and the description of those reported by others is similar to what we have termed *enostosis-like lesions* (see the following discussion).

Clinical signs depend on the severity and location of the fracture. Horses with complete fractures (which are nearly always displaced) are severely lame (non–weight bearing, grade 5) and have marked soft tissue swelling associated with the fracture itself or the site of the original wound. The limb may have an unusual angle and crepitus is usually audible and palpable. Often an associated wound results from the initial injury or is caused by fragment penetration, especially on the medial aspect of the antebrachium. Horses with incomplete or non-displaced fractures have moderate to severe lameness (grade 3 to 5) shortly after the injury, but within 12 to 72 hours they are often fully weight bearing and walking with minimal lameness. However, resumption of exercise or turn out often results in the fracture becoming displaced within 1 to 2 days. Horses with true stress fractures have mild to moderate lameness (grade 1 to 3) at a trot.

For horses with complete, displaced (unstable) fractures of the radius the diagnosis is straightforward. Radiography is only needed to define fracture configuration, and to determine if repair is possible. Radiographs are *essential* in the initial evaluation of *any* horse with a wound in the antebrachium or over the proximal aspect of the carpus that has a history of acute, moderate to severe lameness associated with the injury. Lameness associated with incomplete or hairline fractures of the radius may be transient, but radiographs often reveal obvious or suspicious fracture lines (Fig. 40-1, *A*). Any radiographic evidence of bone injury, often a localized cortical fragmentation or compression fracture, warrants high suspicion of an incomplete fracture, and a full series of radiographs should be obtained. Any horse that has persistent lameness after antebrachial trauma in which original radiographs were negative should be re-evaluated within 7 to 10 days, when a fracture may be evident. The horse should be confined to box rest in the interim. Diagnosis in horses with incomplete fractures or stress fractures can sometimes be difficult. Scintigraphic examination is important to differentiate fracture from other problems of the radius, such as enostosis-like lesions.

Emergency management of horses with long-bone fractures has been well described[7] (see Chapter 87). Horses with unstable fractures should be sedated, wounds should be treated, and a full-limb Robert Jones dressing with splints should be applied. A caudal splint extending from the ground to the point of the elbow (olecranon process) and a lateral splint extending to the withers are attached with tape. The bandage should have a flat surface to allow the lateral splint to be *in contact with* the skin of the upper limb and torso to prevent distal limb abduction. An oversized bandage reduces the effectiveness of the splints. Properly applying external coaptation is time consuming and difficult, but appropriate stabilization is essential to reduce the risks of further fracture displacement and skin penetration. NSAIDs and broad-spectrum antimicrobial therapy should be administered. Horses should travel facing backward.

Confinement to a stall is *mandatory* for any horse with any radiographic abnormality, including focal cortical defects, or if there is known trauma but with no detectable lesions. Catastrophic failure of the radius often results when small cortical defects or incomplete fractures become displaced (Fig. 40-1, *B*). We recommend conservative management in adult horses with incomplete or non-displaced radial fractures. If possible, the horse should be transported to a surgical facility even if conservative management is chosen, because the fracture could become displaced and immediate surgical repair may be necessary. However, transport also involves risks that must be weighed against economic considerations and

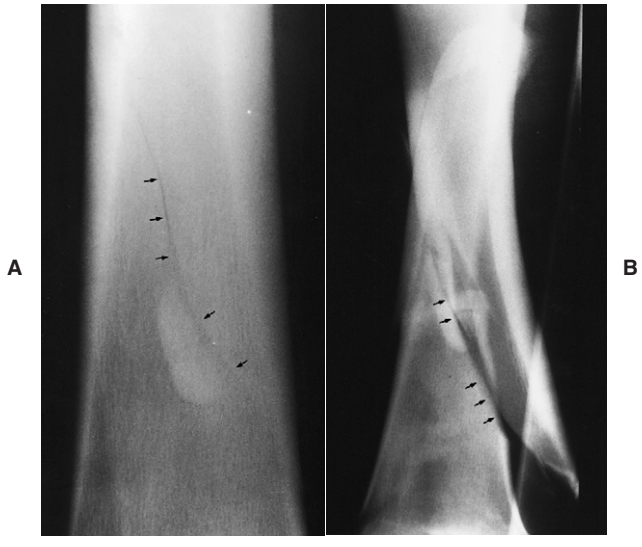

Fig. 40-1 **A,** Craniocaudal radiographic view of the distal radius of a horse revealing a non-displaced fracture (*arrows*). The horse was found acutely severely lame with a small wound over the distal cranial aspect of the antebrachium earlier that day. **B,** Craniocaudal radiographic view 1 day later. Catastrophic fracture occurred even though the horse was confined to a box stall. The original fracture line (*arrows*) and the edge of a plastic fence post used as a splint are visible.

other factors affecting prognosis. Horses with incomplete fractures should be strictly confined to a stall for 8 weeks. External coaptation is applied as described previously for 4 to 8 weeks but can be difficult to maintain. Cross-tying is generally unnecessary, because horses tend not to lie down with bulky external coaptation in place. If radiographs reveal acceptable progression of healing after 8 weeks, the horse is restricted to box stall rest with hand-walking exercise for another 2 months. The majority of fractures are clinically healed in 4 months, although complete radiographic healing may take up to 6 months or more in adult horses. Paddock turnout is generally allowed after 4 months, and horses return to work 5 to 6 months after injury.

NSAIDs are administered as needed to provide comfort and minimize the potential for contralateral limb laminitis. NSAID administration is usually only necessary for 7 to 10 days, and if pain is not adequately controlled, the horse should be reassessed carefully for fracture displacement or progression or infection associated with any wounds. Antibiotic therapy should be given to horses with wounds or deep pressure sores associated with the bandage and splints.

Complete or displaced radial fractures require open reduction and internal fixation, but the prognosis in adult horses is poor to grave. The current method of choice is the application of two dynamic compression plates, one on the cranial surface and one on the lateral or medial surface, depending on fracture configuration.[8,9] In adult horses use of the dynamic condylar screw plate should be considered. Repair of fractures located at the most proximal or distal aspect of the radius, or those with severe comminution is considerably more difficult, and these horses have a grave prognosis. In foals surgical repair is more successful, and a mid-shaft transverse or short oblique fracture with minor comminution is most common. Although infection is still a concern, implant or bone failure, seen commonly in adult horses, is less frequent. Physeal fractures result in premature closure of the physis, even if repaired surgically. Plate removal is necessary for foals intended to be used for racing.

Management of horses with rare radial stress fractures consists of rest for 4 months. Horses are given 2 months of box stall rest and 2 months of individual turnout in a small paddock before begin returned to training. Follow-up radiographic and scintigraphic examinations are recommended.

Prognosis for horses with incomplete or non-displaced fractures of the radius is good, but clients should be warned of the possible complications of fracture propagation and contralateral laminitis, if initial lameness is severe. Those that do not develop complications, and in which fractures heal uneventfully, have an excellent prognosis for return to full athletic function. Prognosis for surgical repair of radial fractures in adult horses is poor but depends greatly on fracture configuration and whether the fracture is open. Horses with open fractures have a worse prognosis, because infection is an important and frequent complication. Adult horses with long spiral or oblique fractures are the best surgical candidates, whereas those with comminution or fractures involving the proximal and distal aspects of the radius, in particular those involving the antebrachiocarpal or elbow joints, have a poor to grave prognosis. Overall the success rate of surgical management of an adult horse with a complete radial fracture is no better than 10% and is expensive. If severe comminution exists, the fracture is open, or it involves a joint, euthanasia should be recommended. In foals, prognosis is considerably better, with a good prognosis for survival and a fair to good prognosis for future athletic use.[8,9] Foals with radial fractures involving the distal physis will likely develop angular limb deformity, and prognosis for athletic use is worse than in those with mid-diaphyseal fractures. The prognosis for horses with rare radial stress fractures is excellent.

Enostosis-like Lesions of the Radius

Enostosis-like lesions are an unusual condition affecting many long bones, including the radius. Enostosis is defined as "bone within a bone," and because the cause has yet to be determined, the term *enostosis-like lesion* is used. Enostosis-like lesions are focal or multifocal intramedullary mild to dense radiopacities. Scintigraphically, enostosis-like lesions are associated with mild to intense increased radiopharmaceutical uptake (IRU) on delayed (bone phase) images (Fig. 40-2, *A*). Lesions may occur in one or more bones simultaneously but are not necessarily associated with lameness.[10,11] Previously, enostosis-like lesions have been described as bone infarcts or bone islands. Histological examination of specimens harvested from an enostosis-like lesion in a humerus revealed changes compatible with ischemia of cancellous bone and bone marrow.[12] Histological changes were similar to those in specimens examined from patients with medullary infarcts or those in bone adjacent to diaphyseal fractures and cortical stress fractures. However, the findings were not pathognomonic or exclusively characteristic of either of these entities.[12] One of us (L.H.B.) believes enostosis-like lesions may be caused by primary disruption of medullary vasculature and secondary development of bone sclerosis. Enostosis-like lesions are possibly an atypical form of bone infarct. Enostosis-like lesions are frequently found close to a nutrient foramen.[10] Because enostosis-like lesions occur in adult horses of all ages and performance categories, they are not likely to be stress fractures.

Although enostosis-like lesions can be an incidental radiographic or scintigraphic finding of the radius, or most long bones, lameness apparently attributable to the condition occurs in approximately 50% of affected horses. In horses with clinically relevant enostosis-like lesions, lameness is usually mild but may be severe.[13] Although enostosis-like lesions can affect many bones simultaneously, lameness is usually restricted to a single limb.

Because completely abolishing pain originating from the radius may be difficult using diagnostic analgesia, in most horses with enostosis-like lesions diagnosis is made by ruling out other potential causes of lameness. Enostosis-like lesions must be differentiated from true stress fractures. It is imperative that two perpendicular scintigraphic views are obtained to allow differentiation of cortical and medullary IRU. Although treatment of horses with enostosis-like lesions or stress fractures is similar, cause and recurrence are different. Enostosis-like lesions are associated with mild to intense IRU in the medulla. In our experience enostosis-like lesions causing lameness are usually moderately to intensely active. Radiographs reveal corresponding single or multiple, focal or multifocal sclerotic lesions within the medullary cavity (Fig. 40-2, *B*). The lesions are frequently in contact with the endosteal surface and are most often in close proximity to the nutrient foramen. In the radius, follow-up scintigraphic and radiographic evaluation between 4 and 9 months after initial diagnosis often reveals resolution of the enostosis-like lesions, although resolution of radiographic changes lags behind that visible scintigraphically.

Treatment of horses with enostosis-like lesions thought to be causing lameness is nearly identical to that for those with stress fractures. NSAIDs (phenylbutazone, 2.2 mg/kg, bid) are administered for 5 to 10 days or longer depending on degree of lameness. Horses are restricted to stall rest with hand-walking exercise for 2 months, followed by a minimum of 2 months in an individual small paddock. Follow-up clinical, scintigraphic, and radiographic evaluation is recommended. Prognosis for most horses with enostosis-like lesions is excellent, and recurrent lameness is rare, although occasionally longer (6 to 9 months) convalescence is required.

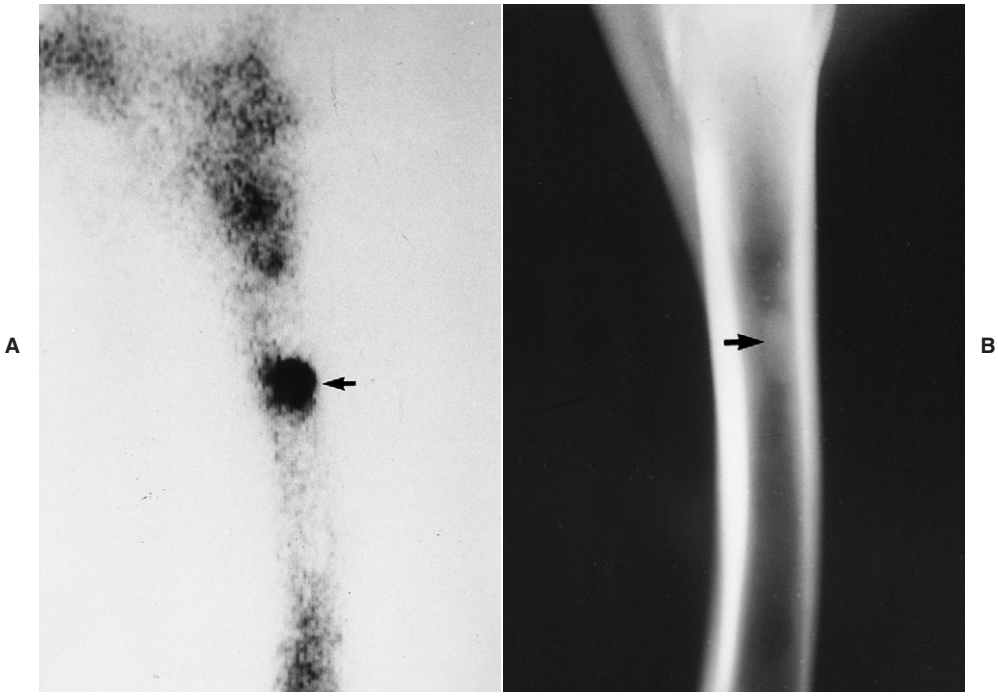

Fig. 40-2 **A,** Lateral or delayed (bone) phase scintigraphic view showing focal, intense increased radiopharmaceutical uptake (*arrow*) in the medullary cavity consistent with an enostosis-like lesion. Two scintigraphic views were compared and increased radiopharmaceutical uptake determined to involve the medullary cavity rather than the cortex. **B,** Lateromedial radiographic view reveals intramedullary sclerosis (*arrow*) on the endosteal surface and adjacent to the nutrient foramen, radiographic signs typical in horses with enostosis-like lesions.

Osseous Cyst-Like Lesions of the Distal Radius

Osseous cyst-like lesions occur in immature[14] and adult horses[15] and may result from osteochondrosis or trauma. Occasionally, osseus cyst-like lesions occur secondary to severe osteoarthritis of the antebrachiocarpal joint. Lameness ranges from mild to severe (grade 1 to 4) and may be sudden or insidious in onset.[14,15] Often no localizing clinical signs are present, unless osseus cyst-like lesions are secondary to osteoarthritis of the antebrachiocarpal joint, when effusion may be present.

Lameness is localized to the antebrachiocarpal joint and distal radius by intra-articular analgesia or by using median and ulnar nerve blocks. Response to intra-articular analgesia of the antebrachiocarpal joint is inconsistent; most horses show partial improvement. Scintigraphy is useful in horses in which diagnostic analgesia fails to localize lameness. Radiographs reveal a well-defined radiolucent defect in the subchondral bone, often with a sclerotic margin (Fig. 40-3), with or without communication with the antebrachiocarpal joint.

Management remains controversial, and a universally accepted method is not currently available. Successful surgical management using an extra-articular approach, debridement, and cancellous bone grafting has been described.[15] Osseus cyst-like lesions that enter the antebrachiocarpal joint can be debrided using an intra-articular approach, whereas those without communication are best managed using an extra-articular approach. Non-surgical management was successful and consisted of restricted exercise, with or without intra-articular administration of hyaluronan.[14] We treat horses conservatively. Duration of restricted exercise and number and type of intra-articular injections are determined on a case-by-case basis. Horses are given a minimum of 6 months of rest and longer if gradual improvement in clinical signs is seen. Conservative management is the first choice in immature

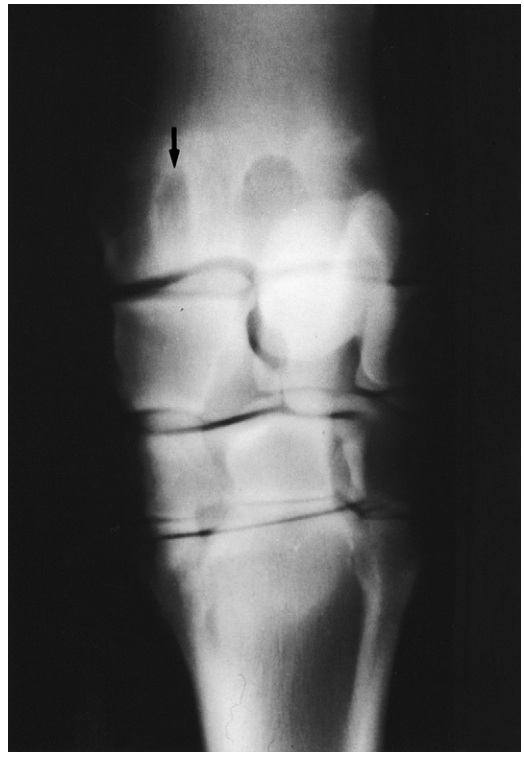

Fig. 40-3 Craniocaudal radiographic view of the distal radius showing an osseous cyst-like lesion (*arrow*).

horses (<2 years of age). Extra-articular corticosteroid injection using a small drill bit has been successful in other sites and may be applicable for distal radius lesions. If little improvement is seen after 6 months, then surgical debridement (with or without grafting) should be considered. Limited data are available on which to base prognosis, which may be better in immature horses (<2 years of age).

Desmitis of the Accessory Ligament of the Superficial Digital Flexor Tendon

See Chapter 76 for a discussion of desmitis of the accessory ligament of the superficial digital flexor tendon.

Acute Caudal Antebrachial Myositis

Myositis and traumatic injury of the muscles in the caudal antebrachium are relatively rare. Horses that compete over jumps at speed such as Three-Day Event, timber, and steeplechase horses appear to be at risk to traumatize these muscles, presumably as a result of hyperextension of the metacarpophalangeal and carpal joints during landing. Occasionally, horses develop inexplicable myositis or injury of these muscle unassociated with a known traumatic event. Infectious myositis can result from puncture or kick wounds (see the following discussion).

Clinical signs include acute, mild to severe soft tissue swelling of the caudal antebrachium, along with a variable degree of lameness. Lameness may be relatively severe immediately after the injury (grade 4, lameness evident at a walk), but with rest and anti-inflammatory treatment for 12 to 72 hours the lameness improves to grade 1 to 3. Lameness is usually proportional to the degree of swelling but in some horses may be inappropriately severe. Even mild swelling of muscles located deep to the dense antebrachial fascia can cause profound lameness, presumably from compartment syndrome (swelling within a rigid tissue compartment). Horses commonly stand with the carpus held in partial flexion. Carpal flexion is usually severely restricted initially because of extensive swelling and pain. It is important to differentiate traumatic injury from infectious myositis or cellulitis. Horses with diffuse swelling caused by infection often have fever, severe lameness, and pain on palpation.

Presumptive diagnosis can usually be made on the basis of history and clinical signs. The antebrachium should be carefully evaluated for even small puncture wounds. A recent but seemingly unrelated area of injury or small wound in the elbow or axillary region may be an important part of the history or physical examination. The clinician should measure rectal temperature. Pyrexia indicates infection. Radiography should be performed to rule-out bony injury, such as an incomplete fracture of the radius. Ultrasonographic examination reveals heterogeneous echogenicity within the bellies and between the fascia of the affected muscles, compatible with fiber disruption and hematoma formation.

Treatment consists of systemic NSAID administration (7 to 10 days) and hydrotherapy. Cold water hosing is administered for 15 to 20 minutes twice a day for 5 days and then warm water hosing is initiated for 5 days. Horses are confined to a stall for 6 weeks and hand-walking exercise is initiated, beginning at 5 minutes twice a day and increasing by 5 minutes each week. Passive flexion and extension of the carpus (30 to 50 repetitions daily) helps to improve range of motion and possibly fiber alignment. Clinical and ultrasonographic evaluation is recommended after 6 weeks. Work should not commence until carpal flexion is normal and the horse is sound. The prognosis for return to full athletic function is good.

Infectious Myositis and Cellulitis

Small puncture wounds in the antebrachium, elbow, and pectoral region can result in infections that cause tremendous antebrachial swelling associated with subcutaneous or deeper tissues. Small wounds may seal over quickly but result in inoculation of bacteria into deeper tissues, causing diffuse cellulitis or infectious myositis. Abrasions from equipment such as hobbles in a Standardbred racehorse can lead to deep infections of the proximal antebrachium. Usually swelling is also present in the distal limb and the carpal sheath may show sympathetic effusion. Infectious myositis may lead to deep abscess formation. Lameness is usually moderate to severe. The horse may be depressed and is usually pyrexic. White blood cell count and fibrinogen concentration are raised. Skin sloughing may develop with aggressive infections caused by *Streptococcus* or *Staphylococcus* species. Radiographs should be obtained, because a radial or ulnar fracture may occur concurrent with infection, but are usually negative unless osteitis of the radius or ulna exists.

Treatment involves administration of broad-spectrum antimicrobial drugs and NSAIDs and the application of topical warm water hydrotherapy. Full-limb bandaging is recommended to reduce swelling in the antebrachium and distal limb. Horses with diffuse cellulitis usually respond quickly (within 3 to 5 days), whereas those with deeper infections require prolonged therapy and an abscess needs surgical drainage.

Swelling of the Antebrachium Associated with Other Conditions

Horses with infectious arthritis of the elbow joint, fractures of the proximal radius or ulna, or distal humeral fractures often have swelling of the antebrachium. These conditions should be kept in mind if diagnosis of a primary problem in the antebrachium cannot be made.

Hypertrophic Osteopathy

Hypertrophic osteopathy is an unusual disorder involving bilaterally symmetrical proliferation of fibrous tissue and periosteal bone in the appendicular and, less frequently, the axial skeleton, involving the metaphyseal and diaphyseal regions of several bones of all limbs.[16,17] Hypertrophic osteopathy may be associated with disease in the thorax or abdomen or with vascular abnormalities. Cause is unknown but may be neurogenic, mediated by the vagus nerve, or hormonal, leading to changes in regional blood flow. Clinical signs include soft tissue swelling and stiffness or lameness, often associated with elevated fibrinogen levels. Radiography of affected bones reveals palisading periosteal new bone. Identification of the primary lesion is important, because bony lesions may resolve with successful treatment of the underlying disease.[16-19] NSAIDs may ameliorate clinical signs.

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CHAPTER • 41

The Elbow, Brachium, and Shoulder

Sue J. Dyson

ANATOMICAL CONSIDERATIONS

The elbow joint comprises the humerus, radius, and ulna. The distal humerus develops from three ossification centers: the diaphysis, the distal epiphysis, and the epiphysis of the medial condyle. These close radiographically between 11 and 24 months of age. The radius and ulna have a single proximal epiphysis. The radial physis closes radiographically between 11 and 24 months of age, but the ulnar physis does not close until 24 to 36 months of age. Physeal closure occurs later in non-Thoroughbred breeds.

The cranial aspect of the olecranon of the ulna has articular and non-articular components. The anconeal process and trochlear notch articulate with the humerus. At the distal part of the trochlear notch is a distinct ridge. Distal to this is a large, non–weight-bearing synovial fossa.

The elbow is a ginglymus joint, supported medially and laterally by collateral ligaments. The medial collateral ligament comprises a long superficial part and a deeper short part. The medial collateral ligament arises from an eminence on the medial epicondyle of the humerus. The deep part inserts on the radial tuberosity; the superficial part inserts on a more distal prominence, just distal to the interosseous space between the radius and ulna. The lateral collateral ligament arises from a depression in the lateral epicondyle of the humerus and inserts on the lateral tuberosity of the radius, just distal to the joint margin. The joint capsule is extremely thin caudally, where it forms a pouch in the olecranon fossa. Cranially the joint capsule is strengthened by oblique fibers and blends with the collateral ligaments medially and laterally.

The most readily palpable landmarks are the olecranon of the ulna and the lateral collateral ligament of the humeroradial joint. Minimal soft tissue covers the lateral aspect of the elbow, making it vulnerable to the effects of direct trauma and penetrating wounds.

The humerus is surrounded by muscles, which largely protect it from the effects of direct trauma. The deltoid tuberosity on the craniolateral aspect is usually readily palpable and is potentially vulnerable to the effects of direct trauma. The deltoid tuberosity is the most useful landmark for identifying the point for needle insertion for synoviocentesis of the intertubercular (bicipital) bursa. The proximal humerus has several centers of ossification for the humeral head and the greater and lesser tubercles, which gradually fuse from 3 to 5 months of age. Radiographic closure of the proximal humeral physis occurs between 24 and 36 months of age.

The scapula has four centers of ossification: the scapular cartilage, body of the scapula, cranial part of the glenoid cavity of the scapula, and supraglenoid tubercle and coracoid process. The ossification center for the cranial part of the glenoid cavity fuses directly with the body of the scapula, and this is complete radiographically by 5 months of age. The physis between the supraglenoid tubercle and coracoid process and the body of the scapula closes radiographically between 12 and 24 months of age, being earlier in Thoroughbred and Thoroughbred crossbreeds than in ponies. These physeal lines remain weak links and it is through these that fractures of the supraglenoid tubercle and cranial part of the glenoid cavity of the scapula tend to occur.

The scapula is attached by the serratus ventralis muscles to the axial skeleton. Other muscles involved in attachment of the thoracic limb to the trunk and neck are the four pectoral muscles, the braciocephalicus, and omotransversarius.

The brachial plexus lies on the axial aspect of the scapula and is derived from the sixth, seventh, and eighth cervical nerve roots and the first thoracic nerve. These nerve roots are potentially vulnerable to trauma where they exit the cervical

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The brachial plexus lies on the axial aspect of the scapula and is derived from the sixth, seventh, and eighth cervical nerve roots and the first thoracic nerve. These nerve roots are potentially vulnerable to trauma where they exit the cervical

and cranial thoracic vertebrae, and these are relatively common sites for neuroma formation after trauma. The nerves of the brachial plexus are responsible for innervation of many of the principal muscles of the shoulder region.

The scapulohumeral or shoulder joint is unusual because of the absence of collateral ligaments. Stability therefore depends on muscular support on the medial and lateral aspects by the subscapularis, infraspinatus, supraspinatus, and teres minor. Cranial support is provided by biceps brachii and supraspinatus, and caudal support is rendered by the long head of triceps brachii. Overlying muscles make it impossible in mature horses to appreciate distention of the joint capsule by palpation. The joint capsule attaches closely to the margins of the scapulohumeral joint, and this, together with the surrounding muscles, restricts arthroscopic evaluation within the joint. The suprascapular nerve wraps around the cranial margin of the scapula proximal to the supraglenoid tubercle and provides innervation to the infraspinatus and supraspinatus muscles.

Considering its embryological development and function the so-called intertubercular (bicipital) bursa would be more appropriately called a tendon sheath.[1] The bursa surrounds the tendon of biceps brachii, which originates from the supraglenoid tubercle of the scapula. The craniodistal pull of biceps brachii results in cranial and distal displacement of fractures of the supraglenoid tubercle. Communication was identified between the scapulohumeral joint capsule and the intertubercular bursa using contrast arthrography in 3 of 18 limbs (17%)[2]; thus in some horses intra-articular analgesia of the scapulohumeral joint has the potential to cause improvement in lameness caused by pain arising from the intertubercular bursa. The tendon of biceps brachii is partially cartilaginous proximally and passes over the smooth intertuberal groove of the humerus and then becomes predominantly muscular with a tendonous core.

The most important palpable landmarks in the shoulder region are the cranial and caudal eminences of the greater tubercle of the humerus. The notch between these eminences provides the portal for arthrocentesis of the scapulohumeral joint. The scapular spine is usually readily palpable except in exceptionally fat horses or heavily muscled individuals and becomes more prominent if the supraspinatus or infraspinatus muscles show atrophy.

DIAGNOSIS

Clinical Signs

In the adult horse lameness associated with the elbow or shoulder regions is comparatively rare, except after direct trauma caused by a fall, collision with a solid object such as a gatepost, or collision with another horse. In immature athletic horses, stress fractures of the scapula, humerus, and radius; osseous cyst-like lesions; and osteochondrosis are quite common.

Lameness associated with the shoulder or elbow regions is usually sudden in onset and generally moderate to severe. After trauma to the shoulder or elbow, or in association with severe lameness, the horse tends to stand with its weight inclined toward the contralateral limb, not fully load bearing on the lame limb. The horse may resent turning on the limb. Muscle atrophy in the shoulder region is not specific for lameness associated with the proximal limb but is often more severe than if the pain arises farther distally. Rapid loss of the bulk of supraspinatus and infraspinatus muscles alone is likely to reflect damage to the suprascapular nerve, whereas involvement of additional muscles is more likely to reflect a brachial plexus injury. Swelling in the elbow or shoulder regions usually reflects direct trauma but may be seen with subluxation or luxation of the shoulder or elbow joints. Patchy sweating is sometimes seen with a lesion of the brachial plexus. Pain elicited by deep palpation is relatively unusual and is generally associated with direct trauma. Care should always be taken to compare the reaction to palpation of the contralateral limb. The reaction to manipulation of the proximal limb joints should also be interpreted with care because many normal horses resent extreme flexion, extension, or abduction.

Many normal horses show some resentment of firm palpation of the braciocephalicus muscles at the base of the neck. These muscles often become sore with a more distal source of pain causing lameness. Primary muscle pain causing lameness does sometimes occur and is associated with a more marked pain reaction on palpation and muscle spasm.

If lameness is mild, then the character of the lameness is non-specific, but if lameness is moderate to severe, it is often characterized by a shortened cranial phase to the stride, a reduced height of the arc of foot flight, and a marked head lift and nod. These gait characteristics are evident at the walk and the trot. Observation of the moving horse from the front and the side is particularly useful. The horse may pivot on the lame limb when turning. Lameness is frequently accentuated with the lame limb on the outside of a circle. In horses with proximal limb lameness, especially associated with muscle fibrosis, lameness may only be evident when the horse is ridden, performing specific movements. Manipulative tests of the proximal limb joints are rather non-specific and frequently unrewarding.

Lameness may fluctuate in degree under different circumstances and within an examination period; therefore it is important to observe the horse for a sufficient length of time before proceeding with local analgesic techniques. If lameness is only apparent when the horse is ridden, the horse may be sensitive to the diagonal on which the rider sits.

Lameness caused by trauma resulting in only bruising usually improves rapidly, within a few days. Persistence of lameness merits further investigation.

Local Analgesia

It is important to recognize the potential effect of a median nerve block, performed in the proximal antebrachium, on elbow pain. Elbow lameness may be substantially improved, presumably because of local diffusion of the local anesthetic solution. Techniques for intra-articular analgesia of the elbow and shoulder and intra-thecal analgesia of the intertubercular bursa are described in detail in Chapter 10. Intra-articular analgesia of the elbow and shoulder joints usually improves but rarely eliminates pain associated with either joint. The elbow and shoulder joints are relatively large, therefore at least 10 ml of local anesthetic solution (mepivacaine, 2%) is recommended for the elbow joint and 20 ml for the shoulder joint. Retrieving synovial fluid from each joint is usually possible. Absence of resistance to injection is not a guarantee that the needle is in an intra-articular location. Walking the horse after the block facilitates circulation of the local anesthetic solution throughout the joint. Although improvement in lameness may be seen rapidly, within 10 to 15 minutes after injection, at least 1 hour should elapse after the block before the result is considered negative. The block generally is effective for up to 2 hours. The block usually has no influence over pain associated with peri-articular structures.

When performing intra-articular analgesia of the shoulder, it is important to recognize that there is communication with the intertubercular bursa in some horses. In some horses instability of shoulder, so-called shoulder slip, appears transiently (for up to 2 hours) after injection of local anesthetic solution. This prohibits interpretation of the nerve block. The cause is presumably diffusion of local anesthetic solution to nerves

innervating the muscles responsible for maintaining stability of the shoulder. Positive-contrast arthrography showed that injection of volumes greater than 20 ml posed a danger of pooling at the site of injection or leakage from the joint capsule.[1]

Intra-thecal analgesia of the intertubercular bursa usually improves lameness associated with lesions of the tendon of biceps brachii, the bursa itself, or the humeral tubercles. The bursa is large, and 20 ml of local anesthetic solution is recommended.

Many other potential sources of pain in the proximal limb cannot be desensitized by local analgesic techniques. If the response to distal limb analgesia and the blocks described previously is negative, nuclear scintigraphic evaluation is warranted.

Assessment of Muscles

Electrical stimulation of muscles may help identify muscle-related pain (see page 728).

IMAGING

Radiography

Radiographic examination of the elbow requires mediolateral and craniocaudal views.[3] The limb should be pulled forward sufficiently to avoid the pectoral muscle mass to evaluate properly the distal humerus. Routine radiographic examination of the scapulohumeral joint should include mediolateral and craniomedial-caudolateral oblique views. In some horses cranioproximal-craniodistal oblique views of the humeral tubercles are useful. Examination of the entire length of the humerus can be difficult, because often when such examination is indicated, the horse has pain and is reluctant to allow the limb to be adequately protracted. Accurate evaluation of the entire scapula is also not easy because of superimposition over the thoracic vertebrae and the contralateral limb.

Fast-speed, rare earth screens and appropriate film are essential. Use of a grid will greatly enhance image quality, especially in the shoulder region. High exposure factors are required (e.g., 100 kV, 100 mAs, for a mediolateral view of the shoulder). Underexposure will result in lesions being missed.

It is important to recognize that the cranial articular margin of the proximal radius has several lips that should not be confused with osteophytes (Fig. 41-1). The cranial tuberosity of the proximal radius may appear roughened in slightly oblique mediolateral views.

In the scapulohumeral joint a small circular radiolucent region is sometimes seen in the subchondral bone in the middle of the glenoid cavity of the scapula (Fig. 41-2). A radiolucent edge effect is often seen in the proximal humerus, the result of superimposition of the lateral rim of the glenoid cavity of the scapula.

Ultrasonography

Diagnostic ultrasonography is invaluable for assessing muscle structure in the shoulder region. Normal muscle has a homogeneous echogenicity. Identification of hyperechogenic regions indicative of muscle necrosis, fibrosis, or mineralization is usually associated with lameness.

Evaluation of the elbow joint itself is limited in the weight-bearing position because of the difficulty in getting access medially. Examination of the lateral collateral ligament of the humeroradial joint is straightforward. To evaluate the medial aspect, the limb should be pulled forward, but unless the medial collateral ligament is under tension, its echogenicity may lack homogeneity.

In the shoulder region ultrasonography is important for assessing the intertubercular bursa, humeral tubercles, tendon of biceps brachii (Fig. 41-3, *A* and *B*), tendons of insertion of supraspinatus and infraspinatus muscles (Fig. 41-3, C and *D*) and infraspinatus bursa (Fig. 41-3, *B*).[4-7] Care should be taken to ensure that the horse is fully load bearing on the limb, because hypoechogenic artifacts can be created, especially in the tendon of biceps brachii, unless the musculature is under tension. The medial and lateral lobes of the tendon of biceps brachii and the isthmus between them should each be evaluated individually, because getting the entire structure into focus simultaneously is difficult.

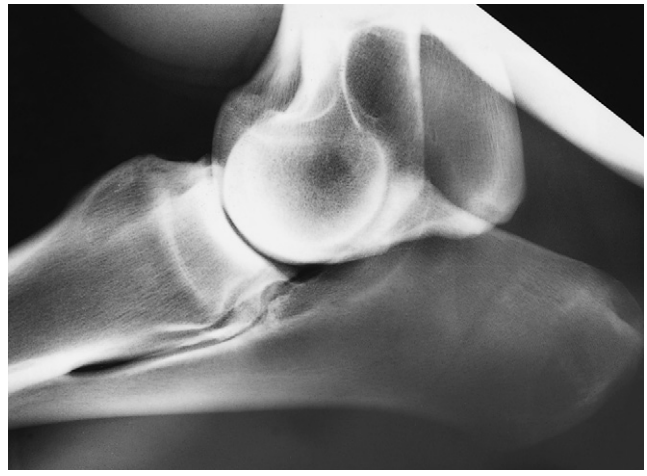

Fig. 41-1 Mediolateral radiographic view of the elbow joint of a normal adult horse. The lips on the cranioproximal aspect of the radius are a normal radiographic feature.

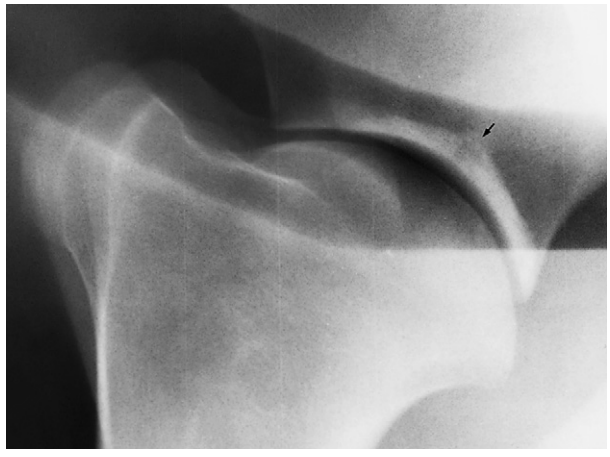

Fig. 41-2 Mediolateral radiographic view of a scapulohumeral joint of a normal adult horse. A small lucent zone appears in the subchondral bone in the middle of the glenoid cavity of the scapula (*arrow*). Note also the lucent band crossing the humeral head and the edge effect caused by superimposition of the articular margins of the scapula.

Nuclear Scintigraphy

Nuclear scintigraphic evaluation of the proximal forelimbs is indicated if the response is negative to median and ulnar nerve blocks, intra-articular analgesia of the elbow and shoulder joints, and intrathecal analgesia of the intertubercular bursa. Scintigraphy is also indicated if history and clinical signs suggest a stress fracture. Scintigraphy has also been useful for identifying enostosis-like lesions (bone islands) in the humerus

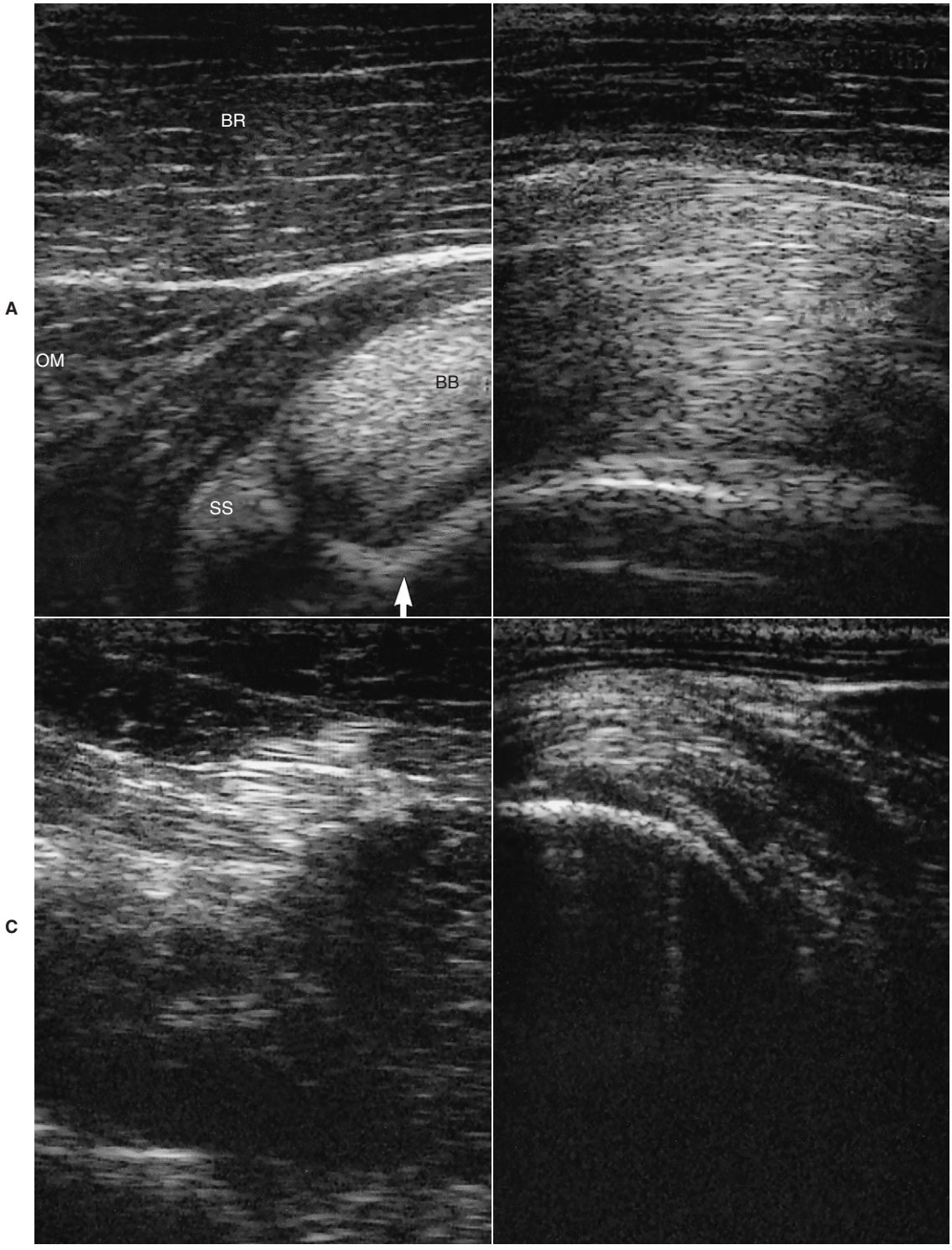

Fig. 41-3 **A,** Transverse ultrasonographic image of the cranial aspect of the proximal humerus of a normal horse. Medial is left. A small amount of anechoic fluid appears within the intertubercular bursa, and the contour of the humeral tubercle is smooth (*arrow*). **B,** Longitudinal ultrasonographic image of the tendon of biceps brachii and the overlying braciocephalicus muscle. Proximal is left. **C,** Transverse image of the tendon of infraspinatus within the infraspinatus muscle. **D,** Transverse ultrasonographic image of the insertion of infraspinatus on the proximal humerus. The anechoic space, the infraspinatus bursa, appears between the tendon of insertion and the bone. *OM*, Omotransversarius; *BR*, braciocephalicus; *BB*, medial lobe of the tendon of biceps brachii; *SS*, medial tendon of insertion of supraspinatus.

and fractures of the deltoid tuberosity of the humerus. Care should be taken to localize any region of increased radiopharmaceutical uptake as precisely as possible to identify the likely underlying pathological condition. Examination of the scintigraphic images should include careful assessment of the soft tissues, because abnormal uptake of the radiopharmaceutical in muscle can sometimes be identified in bone-phase images.

DIFFERENTIAL DIAGNOSIS

Elbow

Osteoarthritis

Osteoarthritis of the elbow is relatively unusual and tends to be seen in older athletic horses. Often the horse has a history of trauma. The horse may resent manipulation of the elbow, but appreciating joint effusion is usually not possible. Lameness often varies in degree, within and between examinations, and is usually worst on a hard surface, especially on a circle.

Intra-articular analgesia improves lameness. Peri-articular osteophyte formation, alterations in subchondral bone opacity, and narrowing of joint space width may be seen radiographically (Fig. 41-4). Care should be taken not to misinterpret normal bony lips as osteophytes on the dorsoproximal aspect of the radius.

The repeated use of intra-articular polysulfated glycosaminoglycans has been most effective in resolving lameness, but the long-term prognosis for return to full athletic function is guarded.

Osseous Cyst-like Lesions

Osseous cyst-like lesions occur most commonly medially in the proximal radial epiphysis (Fig. 41-5) and usually result in acute onset, relatively severe lameness in immature athletic horses.

Lameness may be vary greatly in degree, within and between examinations, and is usually substantially improved by intra-articular analgesia. In addition to a well-defined osseous cyst-like lesion, there is often periosteal new bone on the proximal medial metaphyseal region of the radius. Intra-articular medication with hyaluronan or corticosteroids (methylprednisolone acetate) has resulted in successful resolution of lameness.[8,9] In some horses the osseous cyst-like lesions have resolved radiographically. Surgical treatment by curettage of the cyst using an extra-articular approach has been successful in some horses, but fracture through the cyst has been a recognized complication.[10] Recurrent lameness after surgical treatment is also possible.[11]

Less commonly, large, less well-defined osseous cyst-like lesions have been identified in the distal humerus in young Thoroughbreds being prepared for the yearling sales or just after entering training.[9] Lameness is acute in onset, persists despite box rest, and is generally not influenced by intra-articular analgesia of the elbow. Nuclear scintigraphic examination reveals a region of increased radiopharmaceutical uptake in the distal humerus, more centrally located than that associated with a stress fracture (see page 405). Careful radiographic examination reveals a less well-defined osseous cyst-like lesion. Conservative management has resulted in persistent lameness, and the results of surgical treatment have been disappointing. Young horses with smaller osseous cyst-like lesions in the distal medial humerus have been identified and have responded to conservative management.[8]

Osteochondrosis

The elbow is a relatively rare location for osteochondrosis, but occasionally lameness is identified in young Thoroughbreds and Standardbreds in training associated with osteochondritic lesions of the distal humerus or proximal radius. Lameness is improved by intra-articular analgesia. A variety of radiological changes have been identified. Results of medical and surgical management have been disappointing. Osteochondritic lesions of the anconeal process of the ulna also occur rarely,[12] but they should not be confused with a separate ossification center in young foals.[13]

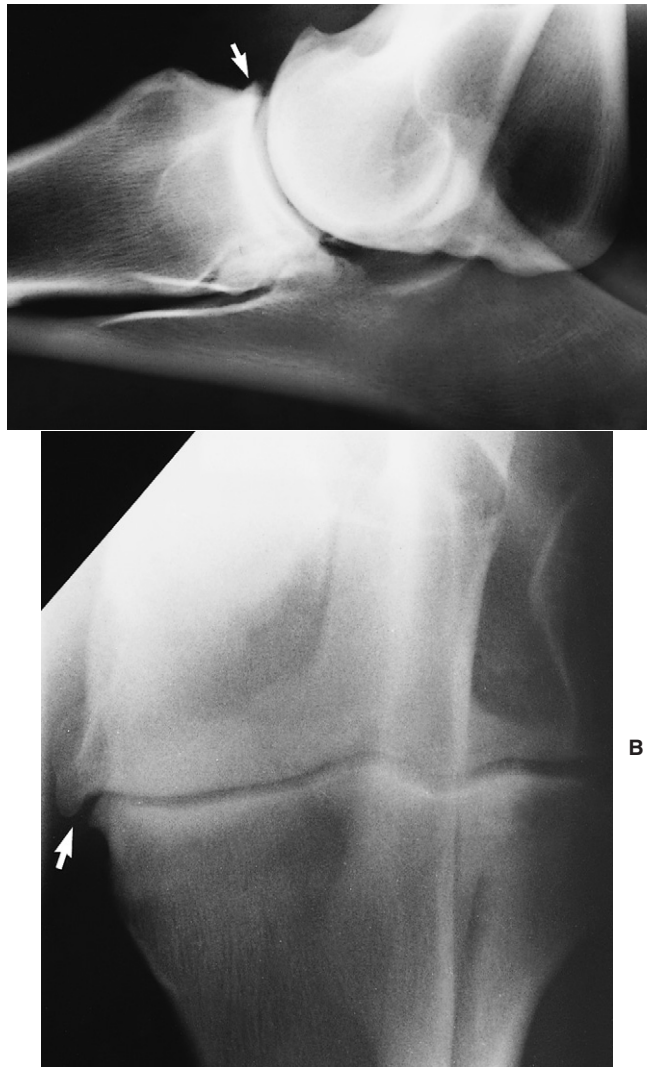

Fig. 41-4 **A,** Mediolateral and **B,** craniocaudal (medial is to the left) radiographic views of the elbow joint of a 9-year-old event horse with left forelimb lameness alleviated by intra-articular analgesia of the elbow. The periarticular osteophytes (*arrows*) and subtle narrowing of the joint space medially indicate osteoarthritis. The horse failed to respond to intra-articular medication.

Stress Reactions in Subchondral Bone

Intermittent lameness has been identified in a small number of event horses associated with an assumed stress reaction in the subchondral bone of the distal humerus.[9] Lameness is induced by jumping but tends to resolve if the horse is not jumped. Lameness may be improved by intra-articular analgesia of the elbow (curious). No bony abnormalities have been identified radiographically. Nuclear scintigraphic examination reveals a region of increased radiopharmaceutical uptake in the subchondral bone of the distal humerus. Box rest and controlled walking exercise for 3 months has resulted in resolution of lameness and a normal distribution of the radiopharmaceutical. Horses have been able to return to full athletic function without recurrent injury.

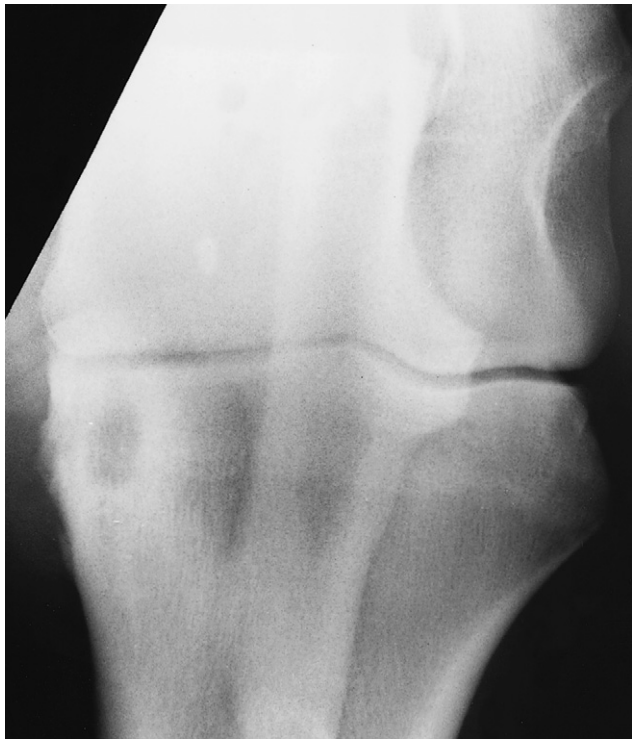

Fig. 41-5 Craniocaudal radiographic view of the proximal radius of 3-year-old Thoroughbred racehorse with left forelimb lameness, which substantially improved with intra-articular analgesia of the elbow. There is a well-defined osseous cyst-like lesion in the proximomedial aspect of the radius. Note also the periosteal new bone on the proximomedial aspect of the radius. Intra-articular medication with hyaluronan resolved lameness, and the horse raced successfully.

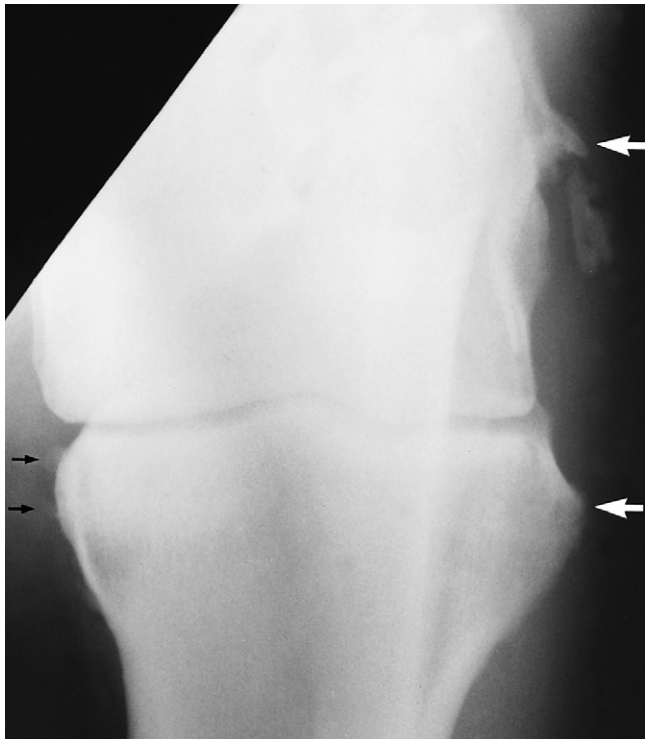

Fig. 41-6 Craniocaudal radiographic view of the elbow of 15-year-old pony that had chronic left forelimb lameness after a fall. There is entheseous new bone at the attachments of the lateral collateral ligament of the humeroradial joint (*white arrows*) with mineralization within the ligament. There is also new bone on the proximomedial aspect of the radius (*black arrows*). Periarticular osteophyte formation was also visible in a mediolateral view. Ultrasonographic examination revealed poor fiber pattern within the lateral collateral ligament. Post-mortem examination confirmed desmitis of the lateral collateral ligament and osteoarthritis of the elbow joint.

Collateral Ligament Injury

Injury to the collateral ligaments of the elbow is not common and usually results from a traumatic injury such as a fall. Damage to the lateral collateral ligament has been identified most frequently. Severe injuries may also be associated with injury to the joint capsule, and osteoarthritis may ensue. Lameness is acute in onset. Subtle soft tissue swelling may be appreciated in the elbow region, and manipulation of the elbow may induce pain. Lameness may be partially improved by intra-articular analgesia of the elbow. If damage is restricted to extra-articular structures, then the response may be negative. Nuclear scintigraphic examination may be helpful in these horses. Increased radiopharmaceutical uptake occurs at the sites of ligament attachment on the distal humerus and proximal radius.

Definitive diagnosis requires ultrasonographic examination and identification of disruption of the normally linear pattern of echoes within the ligament. Sometimes periosteal new bone or avulsion fractures can be identified at the region of ligamentous attachment.[14] Radiographic examination should also be performed to identify any concurrent pathological condition of the bone (Fig. 41-6).

Minor lesions have responded well to a period of box rest and controlled walking exercise, but lesions that have been associated with periosteal new bone formation have often been associated with persistent lameness and the development of osteoarthritis.

Luxation

Luxation of the elbow joint is usually seen with a fracture of the olecranon or proximal radius or separation of the radius and ulna, although luxation occasionally occurs alone. Lameness is acute in onset and severe and is associated with considerable swelling in the elbow region. Comprehensive radiographic examination is essential to determine if one or more concurrent fractures are present. Surgical repair can be considered, but the prognosis for athletic function is poor.

Enthesopathy of Biceps Brachii

Tearing of the attachment of biceps brachii from the craniproximal aspect of the radius may be associated with a traumatic injury such as a fall, but frequently history does not suggest the cause. Lameness is sudden in onset and often not associated with any localizing signs, except pain on manipulation of the elbow in horses with acute lameness. The lameness has no particular characteristics. The response to local analgesic techniques is negative. Radiographic examination may reveal periosteal new bone on the cranioproximal aspect of the humerus in horses with chronic lameness[15] (Fig. 41-7). Sometimes an adjacent mineralized fragment is present, an avulsion fracture or dystrophic mineralization. Some new bone formation on the cranioproximal aspect of the radius can be seen in normal horses, reflecting previous injury, thus care should be taken in interpreting its current clinical significance. Nuclear scintigraphic examination is helpful for acute lameness before periosteal new bone develops and in horses with chronic lameness. Ultrasonographic evaluation has not been helpful. Treatment is by rest. The prognosis is guarded to fair.

Fracture of the Olecranon

Fracture of the olecranon of the ulna usually results from a kick or fall, resulting in acute onset, severe lameness. The horse may stand with the elbow dropped because of lack of an effective

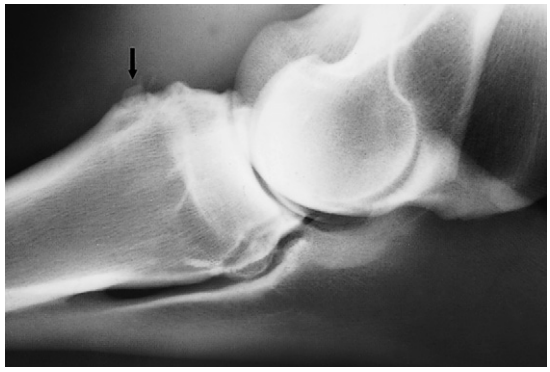

Fig. 41-7 Mediolateral radiographic view of the right elbow of 9-year-old show jumper with chronic right forelimb lameness that no local analgesic technique influenced. There is new bone on the cranial tuberosity of the radius at the site of insertion of biceps brachii (*arrow*). Nuclear scintigraphy gave evidence of increased bone modeling at this site. The horse had persistent lameness despite prolonged rest.

attachment of triceps, the principle extensor of the elbow. Soft tissue swelling may or may not be palpable. Traction on the summit of the olecranon elicits pain. Although many fractures are comminuted, detecting crepitus is unusual. Radiographic examination should include mediolateral and craniocaudal views to assess the configuration of the fracture and to identify any other bony lesions (see Fig. 11-4). Fractures have been classified into five types[16]:

- Type 1. Fractures of immature horses that involve the growth plate and the metaphysis: These are subdivided into type 1a, a non-articular fracture involving only the growth plate, and type 1b, a Salter-Harris type II articular fracture involving the anconeal process and proximal part of the trochlear notch.
- Type 2. A simple articular fracture involving the middle of the trochlear notch
- Type 3. A non-articular fracture involving the proximal metaphyseal region
- Type 4. Comminuted articular fractures
- Type 5. A fracture involving the distal olecranon/ulnar shaft, extending proximally and entering the distal, non-articular part of the trochlear notch

A fracture that enters the trochlear notch should be examined carefully to determine whether it involves the articular or non-articular portion. Treatment by internal fixation usually warrants a fair to good prognosis,[17,18] provided that the fracture is identified early and unless comminution is excessive. Conservative management has also been successful if the fracture is not displaced; the best results are achieved with type 5 fractures.[19] If the horse is treated conservatively, healing should be monitored radiographically to ensure that no displacement has occurred. Potential complications of conservative management include non-union, osteoarthritis, development of a flexural deformity or contralateral laminitis, or development of an angular limb deformity in the contralateral limb.

Olecranon Bursitis

See Chapter 80 for a discussion of olecranon bursitis.

Humerus

Fractures of the Deltoid Tuberosity

Fracture of the deltoid tuberosity of the humerus occurs occasionally.[3,20] Often no cause is identifiable, although in some horses trauma has been recognized. Lameness is acute in onset and moderate to severe, depending on the configuration of the fracture. Generally no soft tissue swelling is detectable, although deep palpation in the region may cause pain in acute, but not chronic, injuries. Turning on the limb induces pain. In the absence of localizing signs, nuclear scintigraphic examination may be the best indicator of the injury (Fig. 41-8, *A*).

Diagnosis is by radiographic examination. A craniomedial-caudolateral oblique view is essential (Fig. 41-8, *B*), because abnormalities may not be detectable in mediolateral projections. Two configurations of fracture have been identified: a longitudinal fracture through the tuberosity itself and an incomplete oblique fracture extending proximocaudally through the humeral diaphysis and metaphysis. Conservative management results in a favorable outcome, unless an oblique fracture is displaced, for which internal fixation is recommended. The prognosis is good.

Stress Fractures

Stress fractures of the humerus are relatively common in young Thoroughbreds in training. The most common locations are the proximocaudal aspect of the humerus and the distal cranial and caudal aspects.[21-23] Unilateral lameness is usually acute in onset and relatively severe. This lameness often improves relatively rapidly with box rest. Despite preexisting callus formation indicative of previous ongoing bony reaction, previous lameness has often not been recognized. Usually no localizing signs are apparent, and the response to distal limb nerve blocks is negative. Nuclear scintigraphic examination is the most sensitive means of detecting a fracture and should be performed if a stress fracture is suspected on clinical grounds, because premature return to work may potentially result in a more catastrophic fracture. A focal region of increased radiopharmaceutical uptake is identified in the proximocaudal aspect of the humerus or the distal cranial or caudal aspects (Fig. 41-9). Fractures are usually medial. Radiographic examination is less sensitive, although in some horses new bone formation can be identified on the caudal aspect of the proximal epiphysis, sometimes with increased opacity of the subchondral bone or new bone on the cranial aspect of the distal metaphysis and physis. Identification of new bone associated with a distal caudal fracture is rare.[23] With treatment of box rest and controlled walking exercise for a minimum of 3 months, followed by a graduated return to work, the prognosis is good.

Fractures of the Greater or Lesser Tubercles

A fracture of the greater or lesser tubercle of the humerus is an occasional cause of unilateral forelimb lameness.[5,24-26] The history is often unknown and usually no localizing clinical signs are apparent. Lameness varies in degree. Intrathecal analgesia of the intertubercular bursa may improve the lameness. Although some fractures may be suspected on a mediolateral radiographic view, craniomedial-caudolateral oblique and cranioproximal-craniodistal (skyline) oblique radiographic views are often necessary. Fracture fragments may also be accurately localized using diagnostic ultrasonography[26] and in some horses are only detectable ultrasonographically (Fig. 41-10). The overlying intertubercular bursa and tendon of biceps brachii should be inspected carefully. Small fractures may be removed surgically. Endoscopy of the intertubercular bursa permits reasonable access to the greater tubercle but not the lesser tubercle.[24]

Diaphyseal Fractures

Diaphyseal fractures of the humerus usually result from a fall or other traumatic incident and may be oblique, spiral, or severely comminuted. Lameness is acute in onset and severe, associated with considerable swelling, pain, and crepitus. The horse may stand with the elbow dropped, slight carpal flexion, and the weight only on the toe. Radial nerve paralysis may occur concurrently (see pages 132 and 414). The diagnosis is usually obvious, and the prognosis is poor, so humane destruction is justified.[27] Successful repair may be achieved in selected horses younger than 3 years of age.[28] Conservative

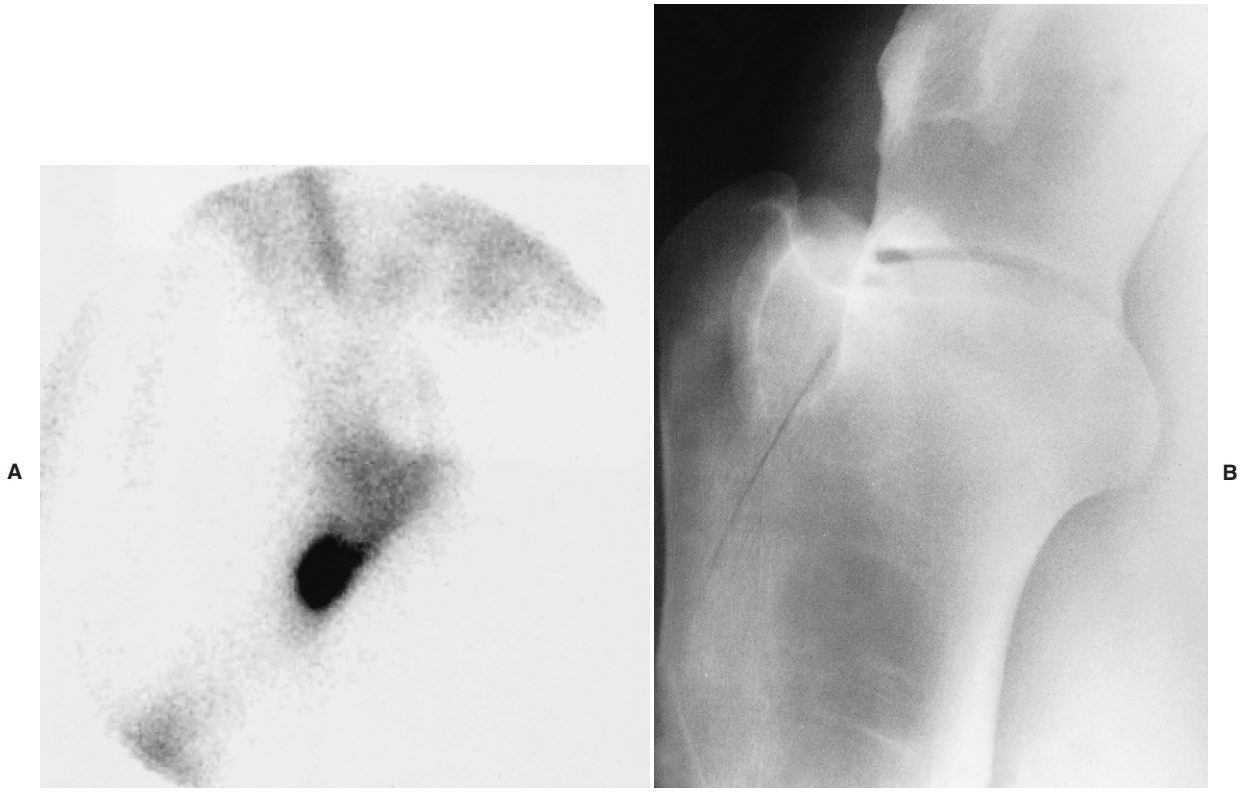

Fig. 41-8 **A,** Lateral scintigraphic image of the right shoulder of 7-year-old dressage horse with acute-onset, moderately severe lameness of 2 weeks' duration with no associated localizing clinical signs. There is marked focal increased radiopharmaceutical uptake in the region of the deltoid tuberosity of the humerus. Radiographic examination revealed an incomplete fracture line through the base of the tuberosity and slight periosteal callus. **B,** Craniomedial-caudolateral oblique radiographic view of the proximal humerus of 3-year-old Thoroughbred broodmare with acute-onset, severe right forelimb lameness. There is an incomplete fracture of the deltoid tuberosity of the humerus. The mare made a complete functional recovery, but a radiographically visible osseous cyst-like lesion developed distal to the greater tubercle of the humerus.

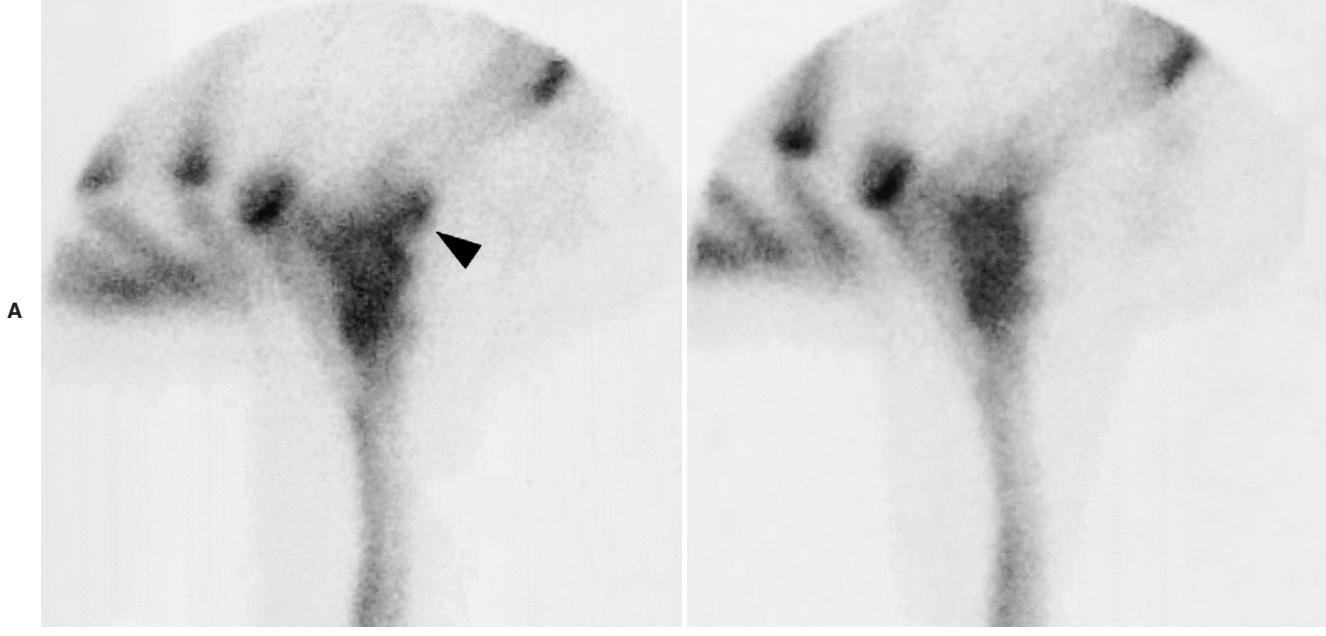

Fig. 41-9 Lateral scintigraphic images of the left **(A)** and right **(B)** elbows of 2-year-old Thoroughbred racehorse with acute onset, severe left forelimb lameness that improved rapidly with rest. The image of the left elbow is reversed to facilitate comparison with the right. There is moderate, focal increased radiopharmaceutical uptake in the craniodistal aspect of the right humerus (*arrow*), compatible with a stress fracture. Radiographic examination revealed slight periosteal and endosteal callus on the craniodistal aspect of the humerus.

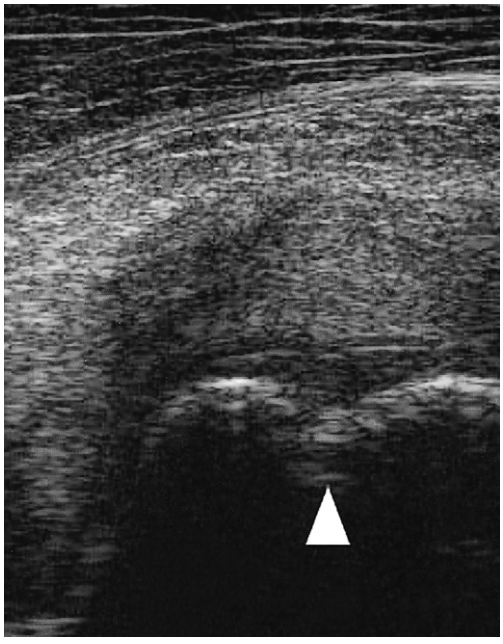

Fig. 41-10 Transverse ultrasonographic image of the humeral tubercles and tendon of biceps brachii of 2-year-old Thoroughbred filly with severe lameness at the walk. Lateral is to the right. There was slight soft tissue swelling in the shoulder region, but no focus of pain could be identified. Nuclear scintigraphic examination revealed marked focal increased radiopharmaceutical uptake in the humeral tubercles. Radiographic examination was negative. Cortical disruption (*arrow*) is apparent.

management in foals with diaphyseal fractures is a reasonable choice, but breakdown of soft tissues and angular limb deformities of the contralateral limb are potential complications.

Proximal Physeal Injuries

Injuries of the proximal humeral physis are rare, but Salter-Harris type I and II fractures have been seen in horses younger than 2 years of age.[29] The prognosis with conservative management is poor.

Enostosis-like Lesions (Bone Islands)

Enostosis-like lesions, or solitary bone islands, have only been recognized since the advent of nuclear scintigraphy. An *enostosis* is defined as bone developing within the medullary cavity or on the endosteum, resulting in a relatively sclerotic region. In the horse, enostosis-like lesions have been described as focal or multifocal intramedullary sclerosis in the diaphyseal region of long bones, near the nutrient foramen, often developing on the endosteal surface of the bone. The cause of these lesions is unknown. Such endosteal reactions must be differentiated from endosteal callus secondary to a fatigue fracture. The presence of such sclerotic lesions radiographically is not always associated with lameness.

In the humerus, enostosis-like lesions occasionally cause acute-onset, moderate to severe lameness in young Thoroughbreds, but they may be present asymptomatically. Usually no localizing clinical features are present. Lameness resolves relatively rapidly with rest, thus mimicking the behavior of a stress fracture and therefore warranting nuclear scintigraphic examination. A region of intense increased radiopharmaceutical uptake occurs in the distal caudal aspect of the humerus, distinguishable from a stress fracture because of its slightly more proximal and medullary location (Fig. 41-11, *A*). Radiographic examination reveals a relatively large, oval-shaped opacity within the medulla, adjacent to the nutrient foramen (Fig. 41-11, *B*). Because lesions may be present asymptomatically, it is important to rule out other potential causes of lameness.

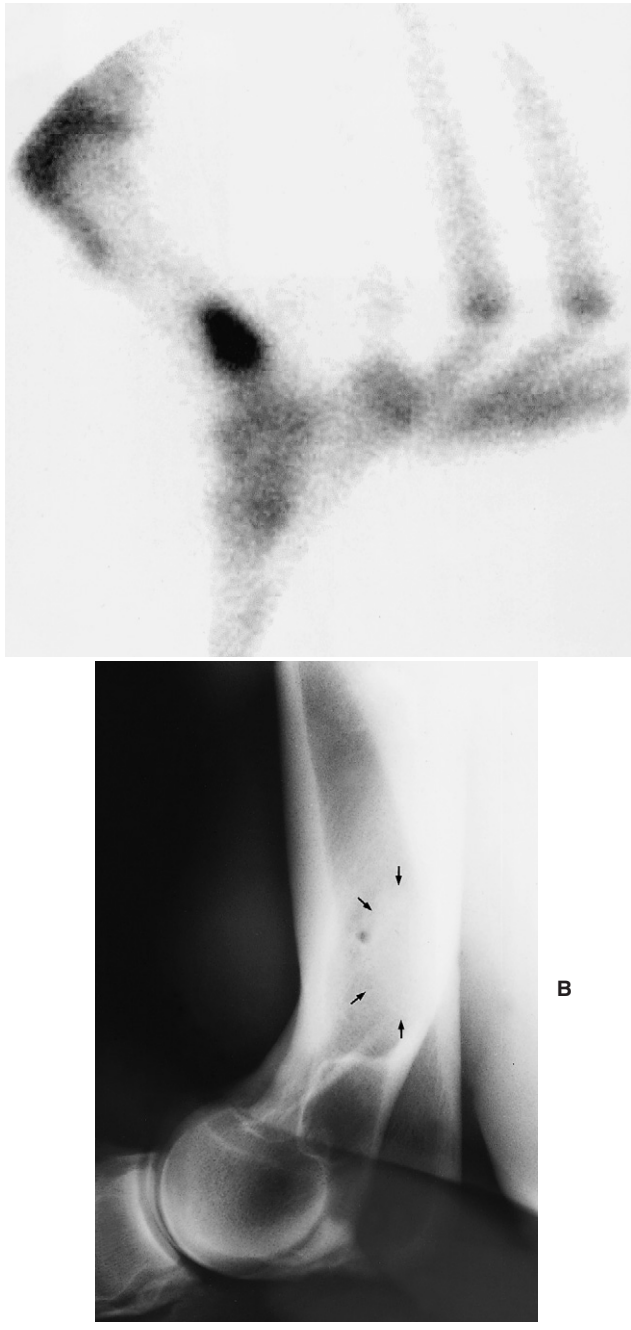

Fig. 41-11 **A,** Lateral scintigraphic image and **B,** mediolateral radiographic view of the left humerus of 3-year-old Thoroughbred filly in race training with right forelimb lameness of 3 weeks' duration. The lameness was not altered by local analgesic techniques. **A,** The nuclear scintigraphic image shows a region of intense focal increased radiopharmaceutical uptake in the distal diaphyseal region of the humerus, proximal to the usual site of stress fractures. **B,** Radiographically a well-circumscribed area of increased opacity (*arrows*) appears adjacent to the principal nutrient foramen. This is an enostosis-like lesion. Lameness resolved after 2 months of box rest, and the lesion was scintigraphically silent, although there was no change in radiographic appearance. The horse remained sound when returned to training.

Treatment is similar to that for a stress fracture: box rest and controlled walking exercise for approximately 3 months. Although a relatively sclerotic region may persist radiographically, follow-up scintigraphic evaluation reveals normal radiopharmaceutical uptake. The prognosis is good.

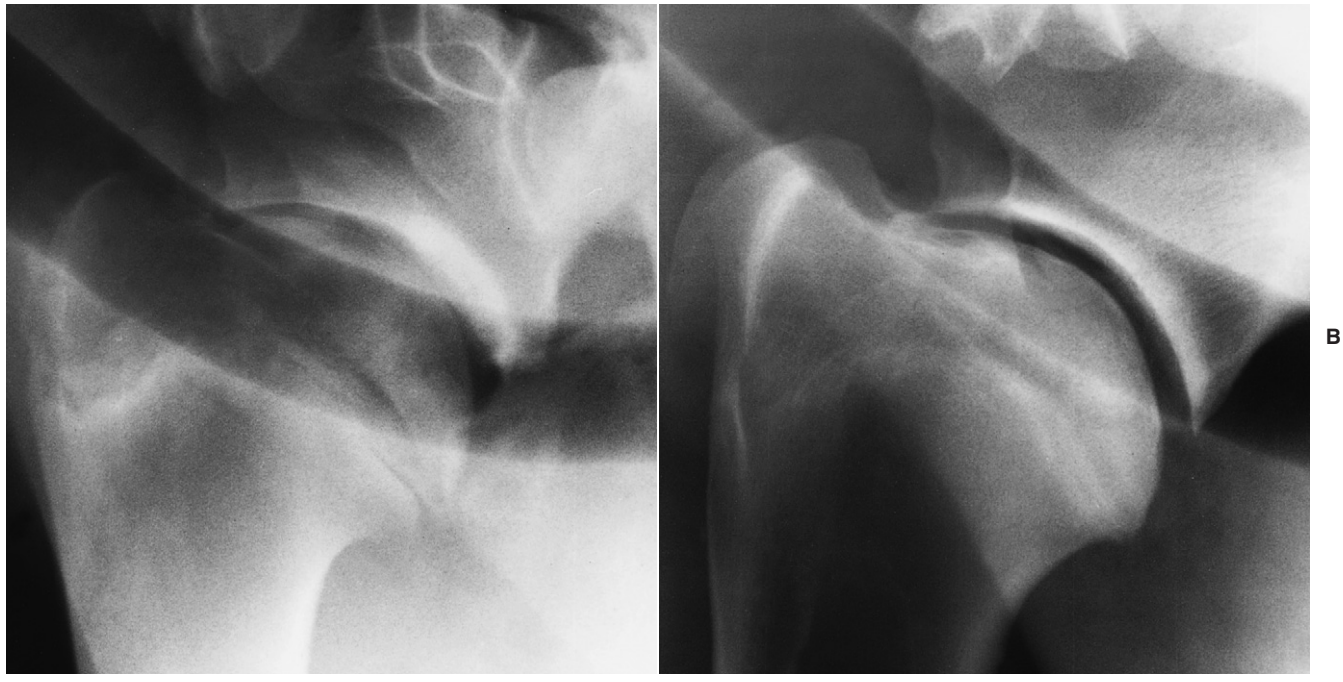

Fig. 41-12 **A,** Mediolateral radiographic view of the scapulohumeral joint of an 8-month-old Thoroughbred colt with severe left forelimb lameness. Lucent areas in the subchondral bone of the distal scapula and sclerosis and modeling of the glenoid cavity and ventral angle of the scapula are compatible with osteochondrosis. The humeral head also has variable opacity. **B,** The same horse 15 months after surgical debridement. The horse was sound. Significant modeling of the glenoid cavity of the scapula has occurred. The horse trained and raced.

Scapulohumeral Joint

Osteochondrosis

Osteochondrosis of the scapulohumeral joint is often clinically evident in the first year of life, but in some horses it is not manifested until the horse is between 2 and 5 years of age, although periods of unexplained lameness may occur. Delayed-onset lameness is often associated with relatively subtle radiological changes, which are more localized compared to those in horses that have lameness evident earlier. Lameness is often acute in onset and moderate to severe and is generally characteristic of proximal limb lameness. Often the foot of the most severely affected limb rapidly becomes more upright and boxy. In young, immature athletic horses radiographic changes are often present bilaterally, although clinical signs may only be evident unilaterally. Intra-articular analgesia usually results in improvement in lameness rather than complete alleviation; therefore the clinical significance of lesions in the contralateral limb can be difficult to interpret.

Radiographic abnormalities involve the glenoid cavity of the scapula, the humeral head, or both and include flattening of the articular surfaces, resulting in loss of congruity of the articular surfaces, irregular lucent zones in the subchondral bone, modeling of the ventral angle of the scapula, and blurring of the normally sharp outline of the glenoid cavity, because of modeling of the articular margins (Figs. 41-12 and 41-13). Generally a good correlation exists between the degree of lameness and the severity of clinical signs.

If lameness is recognized in a horse younger than 18 months of age, conservative management usually results in persistent lameness. Surgical treatment by radical arthroscopic debridement has resulted in some young horses being able to withstand race training, although their longer-term future has been poorly documented. Forty-five percent of treated horses were able to return to athletic activity.[30] In horses treated early, the bone shows a remarkable capacity to remodel (Fig. 41-12). Most horses show significant clinical improvement, even if lameness persists. However, surgical treatment in older horses is much less rewarding. In horses with minor radiographic abnormalities, identified when in race training, periodic intra-articular medication with sodium hyaluronan and triamcinolone has permitted the horses to remain in training and race, although lameness has tended to recur. In weanlings and yearlings with moderate to severe lesions, conservative management, including stall rest, intra-articular injections of hyaluronan, and periodic radiographic assessment of healing, has been successful.[31]

Subchondral Bone Cysts and Other Osseous Cyst-like Lesions

True subchondral bone cysts occur in the middle of the glenoid cavity of the scapula (Fig. 41-14). Other osseous cyst-like lesions have been identified less frequently in the humeral head. Subchondral bone cysts in the distal scapula and other osseous cyst-like lesions most commonly cause lameness in immature athletic horses, between 1 and 3 years of age, but subchondral bone cysts in the distal scapula have also been identified in much older horses, with no history of lameness. Trauma may be an inciting cause. Lameness is usually acute in onset and moderate to severe. In some horses the lameness varies extremely, within and between examination periods, from barely detectable at any gait to obvious at the walk. Lameness is usually significantly improved by intra-articular analgesia of the scapulohumeral joint. However, interpretation can be extremely difficult in those horses that show significant, spontaneous variations in lameness.

Small radiolucent zones are occasionally seen in the middle of the opaque band of subchondral bone of the glenoid cavity of the scapula in clinically normal horses and in the contralateral limb of horses with lameness associated with a large subchondral bone cyst in the distal scapula. Lameness that is improved by intra-articular analgesia associated with such

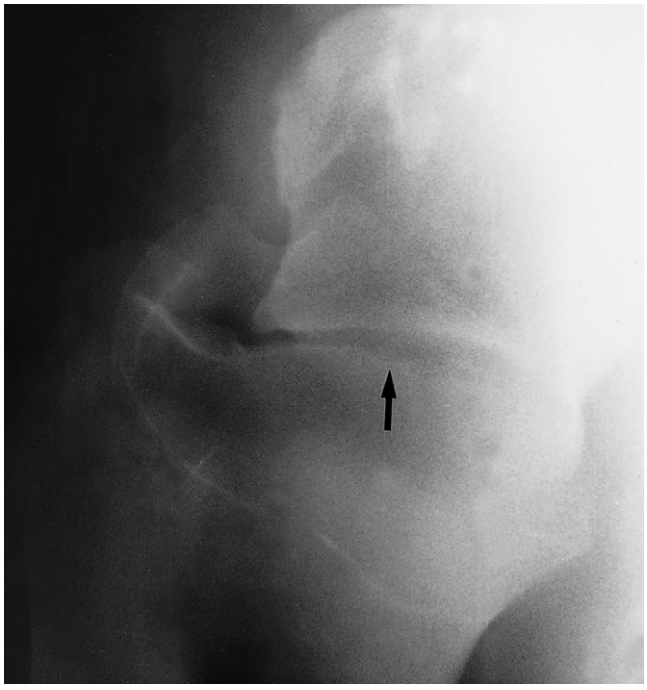

Fig. 41-13 Craniomedial-caudolateral oblique view of the scapulohumeral joint of 3-year-old Thoroughbred with lameness alleviated by intra-articular analgesia of the scapulohumeral joint. A well-defined depression in the proximal articular surface of the humerus is compatible with mild osteochondrosis (*arrow*). This was the only detectable radiographic abnormality. With repeated intra-articular medication the mare raced successfully, winning several more Group 1 races.

lesions but no other radiological change is seen infrequently.[2,9,32] This has been correlated with defects in the articular cartilage extending into the subchondral bone.[32] More commonly a large circular or dome-shaped radiolucent area is seen, surrounded by a narrow rim of sclerosis. In some older horses in which such subchondral bone cysts have been identified, the cysts have been less well defined because they are less radiolucent. In skeletally mature horses similar lesions have occasionally been identified bilaterally associated with apparently unilateral lameness.

In skeletally immature horses subchondral bone cysts have been seen to enlarge progressively, and some appear to move proximally in the bone. Those identified in skeletally mature horses seem to persist unchanged. Osseous cyst-like lesions in the proximal humerus occur as circular radiolucent regions in the middle to caudal aspect. Some of these have been seen to fill in radiographically with time. Subchondral bone cysts in the distal scapula are generally not associated with any other detectable radiological change, whereas other osseous cyst-like lesions are sometimes seen together with remodeling of the ventral angle of the scapula.

Nuclear scintigraphic examination may be helpful in horses in which lameness varies extremely in degree within an examination period. Subchondral bone cysts are associated with active bone modeling; therefore an intense focal region of increased radiopharmaceutical uptake in the bone is seen.

Intra-articular medication of the scapulohumeral joint using hyaluronan and methylprednisolone acetate has been successful in resolving lameness in immature athletic horses, but results have been poorer in older horses.

Surgical treatment of subtle, small lucent zones has been successful,[32] but effective debridement of large lesions is impractical.

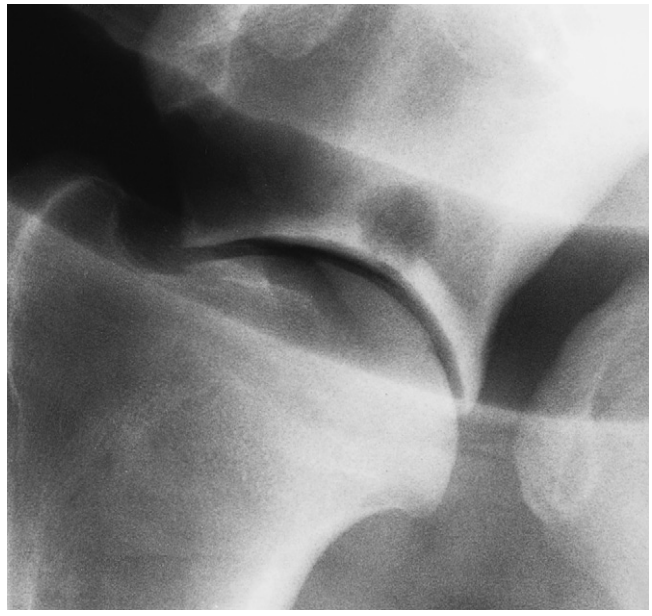

Fig. 41-14 Mediolateral radiographic view of the scapulohumeral joint of 9-year-old hunter with episodic severe right forelimb lameness. There is a large, well-defined subchondral bone cyst in the middle of the distal scapula. The lameness responded to intra-articular medication with corticosteroids (triamcinolone).

Osteoarthritis in Miniature Breeds

Sudden onset, severe lameness is sometimes seen in Shetland and Falabella ponies and Miniature horses.[33] The cause of this condition is not known, although dysplasia of the joint predisposing to instability may be a predisposing factor in some.[33,34] Usually no known history of previous lameness or trauma exists. Lameness is typical of proximal limb pain. Manipulation of the shoulder may cause pain. Lameness is improved but rarely alleviated by intra-articular analgesia. Radiographic examination sometimes reveals evidence of advanced osteoarthritis, including periarticular osteophyte formation, and modeling of the ventral angle of the scapula (Fig. 41-15). Occasionally, fragmentation of the ventral angle of the scapula occurs. Often new bone forms on the distal caudal border of the scapula and the proximal caudal aspect of the humerus in the regions of attachment of the joint capsule. In some ponies the contour of the glenoid cavity of the scapula appears flattened (see "Dysplasia," page 410), resulting in loss of congruity between the distal scapula and the humeral head. In ponies with less severe abnormalities, radiographic abnormalities may only be detectable in a craniomedial-caudolateral oblique projection. In those ponies with minor radiological change at the time of onset of clinical signs, changes tend to have been rapidly progressive.

Arthroscopic evaluation of the joint reveals extensive softening of the articular cartilage, and surgical debridement has resulted in some clinical improvement, although lameness has persisted. The prognosis for athletic function is guarded.

Osteoarthritis in Non-Miniature Breeds

Osteoarthritis in the scapulohumeral joint is relatively uncommon,[29] except as a sequel to osteochondrosis, an intra-articular fracture, or a tear of the scapulohumeral joint capsule. Lameness is mild to moderate and is improved by intra-articular analgesia. Radiographic abnormalities include loss of congruity between the glenoid cavity of the scapula and the humeral head, because of flattening of the humeral head or modeling of the ventral angle of the scapula; periarticular osteophyte formation, most easily seen on the cranial articular

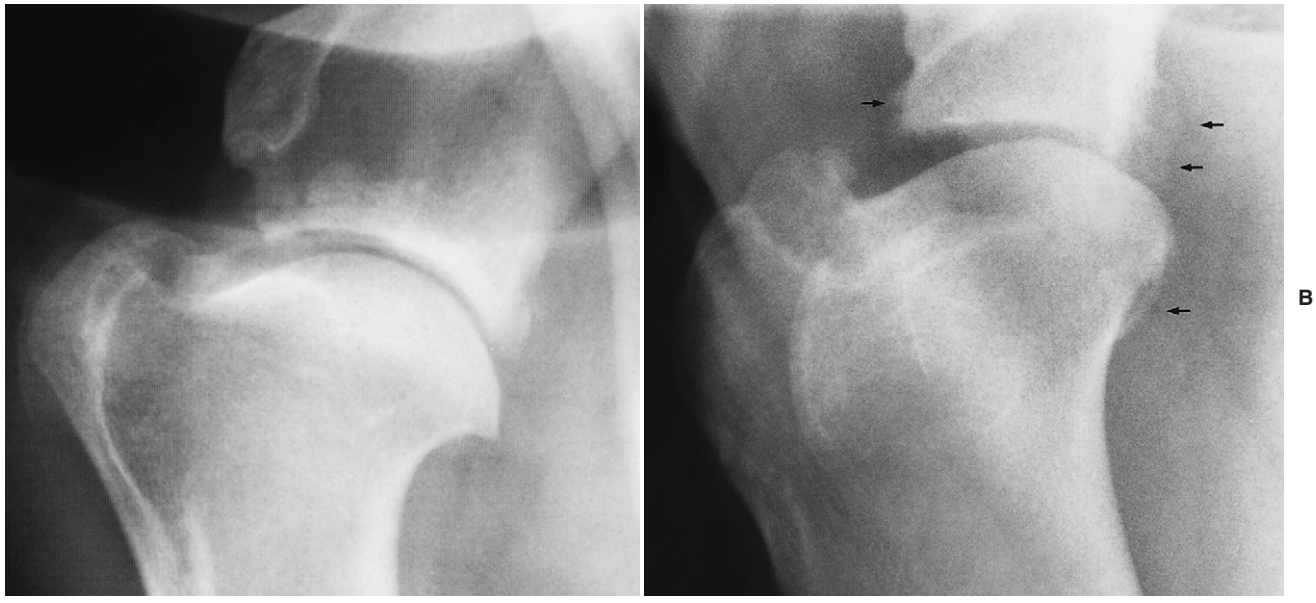

Fig. 41-15 **A,** Mediolateral radiographic view of the scapulohumeral joint of a 4-year-old Shetland pony with acute onset, severe lameness that improved with intra-articular analgesia. There is marked modeling of the ventral angle of the scapula, periosteal new bone formation, and generalized increased opacity of the distal scapula caused by extensive new bone. **B,** Craniomedial-caudolateral oblique radiographic view of a scapulohumeral joint of 5-year-old Miniature horse. There is poor congruity between the glenoid cavity of the scapula and the humeral head. Note also the periarticular periosteal new bone (*arrows*).

margins of the scapula; and lucent areas in the subchondral bone. The response to intra-articular medication is usually poor, and the prognosis for athletic function is guarded.

Care should be taken when evaluating the scapulohumeral joint arthroscopically, because widespread aging changes occur in the articular cartilage, especially of the distal scapula.[2] These include softening of the articular cartilage and extensive fissure formation (Fig. 41-16). Modeling changes of the articular margins of the glenoid cavity of the scapula may also occur.

Tearing of the Scapulohumeral Joint Capsule

Localized tearing of the scapulohumeral joint capsule is an unusual cause of lameness, which is improved by intra-articular analgesia. In the acute stage no detectable radiographic abnormalities may be apparent, but with more chronic lameness entheseous new bone may be seen in the region of damage. Nuclear scintigraphic examination may reveal focal or more generalized radiopharmaceutical uptake in the region of the scapulohumeral joint. Although ultrasonographic examination may be helpful if the lesion is lateral, medial lesions cannot be seen. Definitive diagnosis is based on diagnostic arthroscopy of the scapulohumeral joint. The prognosis for recovery for athletic function is guarded.

Dysplasia

Dysplasia of the scapulohumeral joint has been identified in Shetland ponies[34] and Miniature horses,[9] sometimes together with subluxation of the joint or with osteoarthritis (see the previous discussion, "Osteoarthritis in Miniature Breeds," page 409). Flattening of the radius of curvature of the glenoid cavity of the scapula occurs.

Luxation

Luxation of the scapulohumeral joint has been seen in ponies[2] more frequently than in horses, and results in acute onset, severe lameness associated with extensive swelling in the shoulder region (Fig. 41-17, *A*). If the humerus has luxated laterally, the scapular spine is less easy to palpate than usual. The horse is reluctant to bear full weight on the limb at rest and is usually non–weight bearing at the walk. Diagnosis is confirmed by radiographic examination (Fig. 41-17, *B*). Mediolateral and craniomedial-caudolateral oblique views should be obtained to determine whether a concurrent fracture is present and whether the luxation is medial or lateral. The humerus can luxate cranioproximally or caudoproximally.

A horse with simple acute luxation, without any concurrent fracture, can be treated by manual reduction of the luxa-

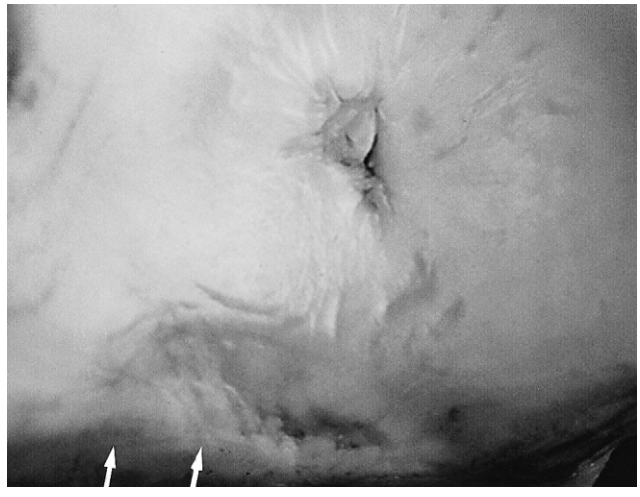

Fig. 41-16 Post-mortem appearance of the glenoid cavity of the scapula of 8-year-old Thoroughbred with no history of lameness. Modeling of the articular margin is apparent *(arrows)*, and the articular cartilage appears irregular. This was bilaterally symmetrical and is a typical finding in mature horses.

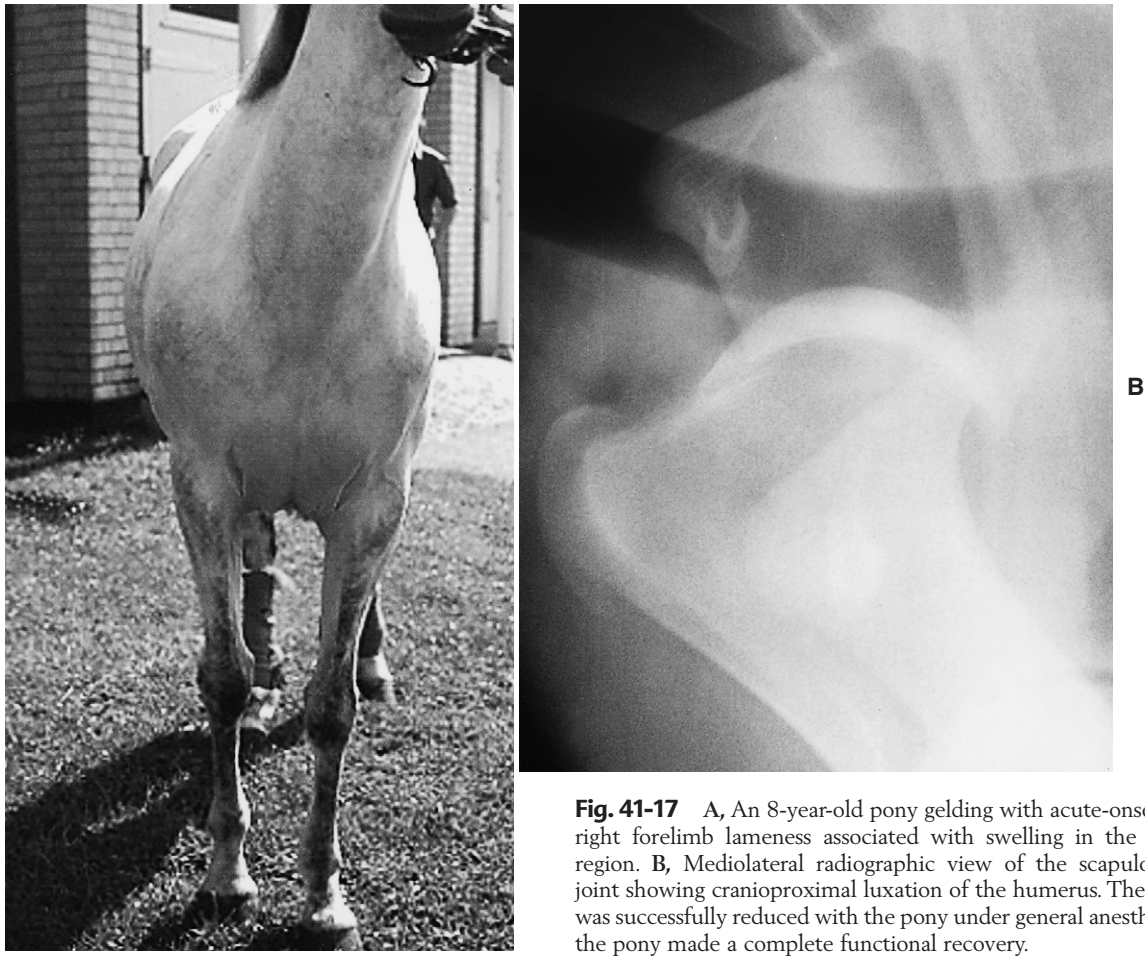

Fig. 41-17 **A,** An 8-year-old pony gelding with acute-onset, severe right forelimb lameness associated with swelling in the shoulder region. **B,** Mediolateral radiographic view of the scapulohumeral joint showing cranioproximal luxation of the humerus. The luxation was successfully reduced with the pony under general anesthesia, and the pony made a complete functional recovery.

tion with the horse under general anesthesia in dorsal recumbency. A hobble is placed around the distal aspect of the affected limb, and the limb is maximally extended vertically. Pressure is applied to the shoulder region to reduce the luxation. An assisted recovery from general anesthesia is recommended, followed by cross-tying to prevent the horse from lying down, minimize the risks of re-injury, and allow the traumatized joint capsule to heal. Periarticular fibrosis inevitably develops, and treating the horse with non-steroidal anti-inflammatory drugs and a controlled exercise program to restore normal joint mobility may be necessary. A complete functional recovery has been achieved in a small number of ponies. The presence of any concurrent fracture predisposes to recurrent luxation and warrants an extremely guarded prognosis. Persistent luxation results in the rapid development of a deep groove in the humeral head, extending from cranially to caudally; thus the prognosis is poor.[2]

Fracture of the Articular Surface of the Distal Scapula

Fracture of the articular surface of the distal scapula may result from a fall when the horse is jumping, resulting in acute-onset, severe lameness.[35] Manipulation of the joint produces pain, and sometimes crepitus can be elicited. Periarticular soft tissue swelling occurs in the acute stage, but this resolves relatively rapidly. Diagnosis requires high-quality radiographs, because the only detectable radiographic abnormality may be loss of clarity of the articular margins of the distal scapula. Mediolateral and craniomedial-caudolateral oblique views should be obtained. The prognosis for athletic function is hopeless.

Periarticular Trauma

Periarticular trauma—the result of a fall, collision between two horses, or collision between a horse and a solid object—may result in sudden onset, severe, unilateral forelimb lameness because of severe bruising. Extensive periarticular soft tissue swelling may develop rapidly, with pain on deep palpation. In the acute stage determining whether a concurrent fracture is present may be difficult, especially fracture of the supraglenoid tubercle of the scapula or the first rib, because history and clinical signs are so similar.

If damage is restricted to bruising, lameness generally rapidly improves over the following 7 days. However, if concurrent trauma to the suprascapular nerve or its branch to the infraspinatus muscle occurred, significant neurogenic atrophy of the supraspinatus or infraspinatus muscles may occur within 7 days (see page 413). If moderate to severe lameness persists, then radiographic examination is indicated. If damage is restricted to soft tissue structures, then treatment with non-steroidal anti-inflammatory drugs combined with restriction to box rest and controlled exercise usually results in a rapid and complete recovery.

Intertubercular Bursa

Tendonitis of Biceps Brachii

Injury of the tendon of biceps brachii is a relatively uncommon cause of forelimb lameness, with no typical history. Frequently no localizing signs suggest the source of pain. In some horses forced retraction of the limb induces discomfort, but this is an unreliable finding. Lameness varies from mild to

severe, and only if lameness is severe are the gait characteristics typical of proximal limb lameness. Lameness is improved by intrathecal analgesia of the intertubercular bursa, although it may take up to half an hour.

Diagnosis is based on ultrasonographic examination. The entire length of the tendon of biceps brachii should be examined carefully from its origin on the supraglenoid tubercle of the scapula to the musculotendonous junction. The horse should be standing fully load bearing on the limb to avoid hypoechogenic artifacts within the tendon. It is important to be aware of the anatomy of the tendon, which changes in shape from proximally to distally.[6] Examining the medial and lateral lobes independently is often necessary, because maintaining both lobes in focus simultaneously is difficult. The tendon should be examined in transverse and longitudinal planes and careful comparison made with the contralateral limb. Ultrasonographic abnormalities include enlargement of the tendon, loss of definition of one of its margins, hypoechogenic defects within the tendon, and loss of fiber pattern in longitudinal images. The humeral tubercles should be inspected carefully for evidence of any concurrent damage. Concurrent evidence of bursitis characterized by an abnormal amount of fluid within the sheath (bursa) may also be present and results in an increased anechogenic space between the tendon and the humerus. Horses with chronic lameness may show evidence of hyperechogenic foci within the tendon, representing fibrosis or mineralization. Adhesions may develop between the tendon and the wall of the bursa. Occasionally echogenic bodies are seen free within the bursa.

Treatment of horses with tendonitis of biceps brachii consists of box rest and controlled walking exercise.[36] Injection of hyaluronan may be beneficial. The tendon should be monitored by ultrasonography at up to 3-month intervals. At least 6 to 9 months of convalescent time is required for horses with acute lesions, which have a fair prognosis. The prognosis for horses with chronic injuries is poor. Permanent enlargement of the tendon within the confined space between the brachiocephalicus muscle and the humerus and loss of normal gliding function cause persistent pain.

Osteitis of the Humeral Tubercles

Osteitis of the humeral tubercles is a rare cause of forelimb lameness.[26] Horses show moderate to severe lameness and resent manipulation of the shoulder. Abnormalities of the humeral tubercles are seen radiographically as lucent areas or by ultrasonography as a roughened surface to the bone and overlying cartilage. The intertubercular bursa may contain an abnormal amount of fluid and the tendon of biceps brachii may be enlarged, with peritendonous reaction. Surgical exploration may reveal necrotic areas of bone that are readily debrided, resulting in improvement in lameness.

Fracture of the Humeral Tubercles

A discussion of fracture of the humeral tubercles is on page 405 (see Fig. 41-10).

Infection

Infection of the intertubercular bursa occasionally causes acute onset, moderate to severe forelimb lameness.[37-39] Infection may be a sequel to known trauma, a previous injection, and a penetrating wound, but it has been recognized with no history of trauma or evidence of a wound, presumably infection being hematogenous in origin. Infection occurs in immature athletic horses and older horses.

The horse may stand slightly favoring the lame limb. Deep palpation in the region of the bursa may elicit pain. Retraction of the limb is resented. Lameness is often evident at the walk. Synovial fluid withdrawn from the bursa is usually grossly brown discolored and turbid, and therefore intra-thecal analgesia is usually unnecessary. Diagnostic ultrasonography reveals an abnormal amount of fluid within the bursa; sometimes echogenic material is seen within the fluid, and the surface of the tendon of biceps brachii may be less well defined than normal because of fibrin deposition. Diagnosis is confirmed by measuring differential white blood cell count and total protein concentration and by cytological examination of the synovial fluid, with polymorphonuclear leukocytes dominating.

Treatment is by radical surgical debridement and thorough lavage of the bursa by open surgery[37] or endoscopy,[38] combined with long-term, broad-spectrum antimicrobial therapy (e.g., crystalline penicillin and gentamicin) and the use of nonsteroidal anti-inflammatory analgesic drugs. If a penetrating wound is identified, consideration should be given to using metronidazole. When the acute inflammatory response has subsided, controlled exercise is important to try to limit adhesion formation. If recognized and treated early and aggressively, a favorable outcome can be achieved, but prolonged therapy is often required.[37]

Non-Infectious Bursitis

Non-infectious bursitis does occur occasionally alone, together with tendonitis of biceps brachii (see page 411), or with osteitis of the humeral tubercles.[4,40] Lameness varies in degree and is usually substantially improved by intrathecal analgesia. Diagnosis is verified by ultrasonographic examination of the intertubercular bursa. The bursa has an abnormal amount of fluid but no abnormalities of the enclosed tendon or the humeral tubercles. Treatment by intrathecal administration of hyaluronan and triamcinolone, combined with controlled exercise, is usually successful.

Mineralization

Mineralization within the tendon of biceps brachii has been seen alone or as a sequel to a previous fracture of the supraglenoid tubercle. Ectopic mineralization within the bursa has been associated with significant proliferation of the synovial membrane of the intertubercular bursa.[9] No typical history is known. Lameness varies in degree and only if moderate to severe is typical of a proximal limb lameness. Lameness is improved by intrathecal analgesia of the intertubercular bursa. Mineralization may be identified radiographically, but it can be obscured by the humeral tubercles. Radiography is useful to identify any previous fracture. Diagnostic ultrasonography provides more accurate information about the precise site and extent of mineralization and any concurrent pathological condition of the tendon. The prognosis for return to full athletic function is guarded.

Scapula

Fracture of the Supraglenoid Tubercle

The scapula has four centers of ossification: the scapular cartilage, body of the scapula, cranial part of the glenoid cavity of the scapula, and supraglenoid tubercle. The cranial part of the glenoid cavity of the scapula fuses with the body by 5 months of age. The physis of the supraglenoid tubercle closes between 12 and 24 months of age. Fracture of the supraglenoid tubercle of the scapula is a relatively common injury, resulting from a collision with a solid object or fall. Fractures frequently occur through the original physes.[35] The fracture may be simple or comminuted, involving only the supraglenoid tubercle, without an articular component. Alternatively, the fracture may pass through the glenoid notch of the scapula, which may result in one large fracture or separation of both original physes.

Lameness is sudden in onset and moderate to severe. Over the first week the horse may show progressive improvement, but lameness persists. Neurogenic atrophy of the supraspinatus and infraspinatus muscles may develop within 7 days if a concurrent injury of the suprascapular nerve occurred. Disuse atrophy develops more slowly.

A palpable thickening in the region of the supraglenoid tubercle exists, but crepitus is rarely appreciated.

Diagnosis is confirmed radiographically (see Fig. 13-1). The fracture fragments are often displaced craniodistally, because

of the pull of biceps brachii, and if treated conservatively, a non-union develops. The radiographs should be inspected carefully to determine if the fracture has an articular component, which warrants a more guarded prognosis.

The prognosis with conservative treatment is poor for return to athletic function. Surgical treatment by removal of the fracture fragment(s) has been successful in some horses, although the level of work to which horses have returned has not been well documented. Various methods of internal fixation have been attempted, but the large distracting forces of biceps brachii have to be overcome. A small number of horses younger than 2 years of age and less than 400 kg body weight have been successfully treated by complete tenotomy of the biceps brachii and internal fixation using three 5.5-mm cortical bone screws.[41]

Stress Fractures

Stress fractures of the body of the scapula are an occasional cause of acute-onset forelimb lameness in young Thoroughbreds in training.[9] No localizing clinical signs are present, and lameness often frequently improves spontaneously with rest. A premature return to training may result in a catastrophic fracture. Diagnosis relies on nuclear scintigraphic examination. Treatment requires 6 to 8 weeks of box rest and controlled walking exercise, followed by a graduated return to full work.

Fracture of the Body of the Scapula

Fractures of the body of the scapula usually result from the horse falling at a fence when jumping at speed. Complete fractures result in severe lameness and extensive soft tissue swelling and merit destruction on humane grounds. Diagnosis is usually obvious based on clinical signs, and radiographic examination is not necessary. Radiography is required for the diagnosis of incomplete fractures (Fig. 41-18). High-quality radiographs are required, and this may be difficult because the horse may resent protraction of the limb because of pain. Horses with fractures that enter the scapulohumeral joint have a poor prognosis. However, horses with extra-articular, incomplete fractures of the body of the scapula have a good prognosis for return to full athletic function with conservative management.

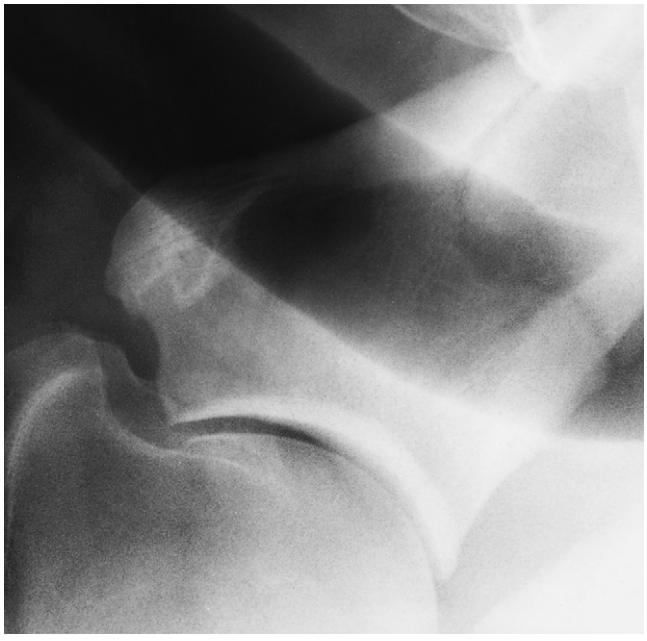

Fig. 41-18 Mediolateral radiographic view of 7-year-old event horse with acute-onset, severe left forelimb lameness after a fall. There is an incomplete fracture of the body of the scapula. The horse was treated conservatively and made a complete recovery.

Fracture of the Scapular Spine

Fractures of the scapular spine result from a fall, collision with a solid object, or occasionally a kick. Acute-onset, moderate lameness occurs with associated localized soft tissue swelling and pain on palpation. Oblique radiographic skyline views of the scapular spine are necessary to identify a fracture. Diagnostic ultrasonography may be more helpful in identifying a fracture. Most fractures heal adequately with conservative management. Occasionally a sequestrum develops, necessitating surgical debridement. The prognosis for return to full athletic function is good.

Muscle

Triceps Myopathy

See Chapter 84 for a discussion on triceps myopathy (page 735).

Muscle Lesions: Braciocephalicus, Biceps Brachii, and Pectorals

Lesions of muscles in the shoulder region have not been well documented and probably occur more commonly than recognized. However, clinical signs are rather non-specific, and diagnosis is difficult. A number of dressage horses have been identified that have shown forelimb lameness only while performing lateral work. Palpation has revealed no detectable abnormality. Local analgesic techniques have not altered the lameness. Ultrasonographic examination has revealed hyperechoic areas of fibrosis or mineralization within braciocephalicus in the lame limb only.

A dressage horse showed lameness only at the walk. Local analgesic techniques were ineffective. Nuclear scintigraphic evaluation revealed increased radiopharmaceutical uptake in the musculature cranioproximal to the elbow. This correlated with a hyperechogenic region within biceps brachii.

Some horses have forelimb lameness associated with palpable soreness of the pectoral muscles, which responds well to physiotherapy. Soreness of the braciocephalicus muscles is often seen with lameness caused by distal limb pain. Lameness is sometimes seen associated with primary braciocephalicus muscle damage in event horses and horses that race over fences. In most horses with acute lameness, palpation reveals muscle soreness and induces muscle spasm. However, in chronic cases pain can be more difficult to identify.

Rupture of Serratus Ventralis

The serratus ventralis muscles arise from the third to seventh cervical vertebrae and the first eight to nine ribs and insert on the medial proximal aspect of each scapula. They sling the thorax between the forelimbs. Rupture of one or both of these muscles is a rare injury, caused by trauma. The proximal border of the scapula moves proximally. If rupture is bilateral, the scapulae become higher than the summits of the dorsal spinous processes in the withers region. Rupture of the serratus ventralis should be differentiated from neurological disease causing loss of function of the muscle.[31]

The horse tends to stand with the forelimbs close together. Palpation of the withers region or manipulation of the forelimbs causes pain. The horse is reluctant to move, takes small steps, and nods the neck stiffly.

Radiographic examination should be performed to rule out a fracture of the scapula or the dorsal spinous processes of the cranial thoracic vertebrae. The prognosis for athletic function is poor.

Nerve

Atrophy of Supraspinatus and Infraspinatus Muscles: Damage to the Suprascapular Nerve

The suprascapular nerve wraps around the cranial aspect of the neck of the scapula and in this position is vulnerable to trauma resulting in perineural edema, stretching, and neuroma formation. Neuroma formation usually results in permanent loss of

function and subsequent neurogenic atrophy of supraspinatus and infraspinatus muscles. This can occur within 7 days of injury. Atrophy results in abnormal prominence of the scapular spine, but damage to the suprascapular nerve alone does not result in any loss of stability of the scapulohumeral joint. Lameness may be due to trauma at the time of the injury but generally resolves rapidly, unless a concurrent fracture is present.

This condition has been called *sweeny*, but this can be confusing because this term has also been used to describe instability of the shoulder or shoulder slip resulting from loss of collateral support of the shoulder musculature. This latter condition is separate and should not be confused (see the following discussion). Experimental transection of the suprascapular nerve in two ponies and one adult horse resulted in rapid atrophy of the supraspinatus and infraspinatus muscles, which persisted over the long term but produced no gait abnormality.[2]

The injury usually results from a fall or collision with another horse or solid object. Permanent muscle atrophy may ensue, but this results only in a cosmetic defect, not a functional deficit. Electromyography can be used to determine whether any other muscles are affected, which would suggest an injury to the brachial plexus or to other nerves in addition to the suprascapular nerve. Patchy sweating indicates a lesion elsewhere.

Surgical treatment has been performed to decompress the suprascapular nerve by cutting out a notch on the craniodistal aspect of the scapula and removing fibrotic material from around the nerve.[42] Surgical treatment has resulted in restoration of more normal muscle mass. Care must be taken not to traumatize the scapula unduly, because this may predispose to fracture.

Instability of Shoulder: Damage to the Brachial Plexus

Lack of collateral support to the scapulohumeral joint by loss of function of the muscles on the medial (subscapularis) and lateral aspects of the joint results in so-called shoulder slip. As the horse starts to bear weight on the limb, the shoulder joint bulges abaxially (Fig. 41-19). Concurrently the heel of the foot tends to rotate outward. This gait abnormality is evaluated most easily viewing the horse walking toward the observer. The gait abnormality may be slight or severe.

Instability of the shoulder was formerly ascribed to atrophy of the supraspinatus and infraspinatus nerves secondary to suprascapular nerve damage, but profound atrophy of these muscles can be present with no detectable gait abnormality.[2,9] Experimental transection of the suprascapular nerve resulted in profound atrophy of supraspinatus and infraspinatus muscles but no detectable gait abnormality.[2] Seven horses with persistent instability of the shoulder that have been humanely destroyed and examined post mortem have had atrophy of at least the supraspinatus, infraspinatus, and subscapularis muscles. All horses had neuroma formation involving nerves of the brachial plexus or the ventral nerve roots of the contributing nerves. The suprascapular nerve appeared normal as it passed over the cranial aspect of the scapula.

Instability of the shoulder invariably results from trauma, usually the horse colliding with a solid object. Pain related lameness may also be present initially, but this generally resolves within 1 to 2 weeks provided that a concurrent fracture does not exist. Similar clinical signs occasionally occur transiently (for 1 to 3 hours) after intra-articular analgesia of the scapulohumeral joint, presumably because of extra-articular leakage of local anesthetic solution. Instability of the shoulder has also been seen in a Miniature horse with dysplasia of the scapulohumeral joint and advanced osteoarthritis.

After trauma, initially no muscle atrophy is detectable, but atrophy of several muscles is usually detectable within 7 to 10 days. Patchy sweating may develop in the caudal neck region or over the scapula, depending on which nerves have been damaged. Electromyography can be used to determine accurately which muscles are affected.

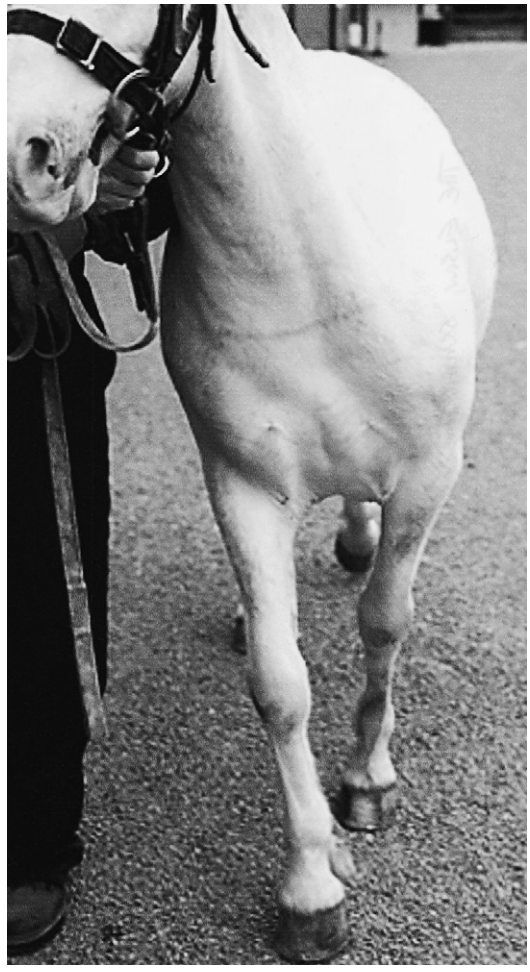

Fig. 41-19 A 7-year-old driving pony with acute onset instability of the left shoulder joint probably caused by a brachial plexus injury. The injury was of 12 days' duration. Note the medial placement of the left front foot. The heel rotated laterally as the foot broke over. There was moderate atrophy of the left infraspinatus, supraspinatus, and triceps muscles. The pony was treated conservatively, progressively improved, and made a complete functional recovery.

Prognosis depends on the nature of the nerve damage and the ability to achieve re-innervation of the affected muscles. Nerve regeneration occurs at approximately 1 mm per day. Progressive improvement in gait is a good prognostic sign. A normal gait may be restored despite persistence of atrophy of the supraspinatus and infraspinatus muscles.[2,43] However, a persistent gait abnormality 6 months after injury warrants a guarded prognosis. Surgical treatment is impractical because of the inaccessibility of the brachial plexus or the ventral nerve roots. Scapular notch resection is not indicated, because nerves in addition to or other than the suprascapular nerve are involved. This procedure also risks secondary fracture of the scapula.

Radial Nerve Paralysis

Radial nerve paralysis is an occasional cause of lameness. The clinical signs depend on the site or sites of damage. Complete radial nerve paralysis is most common, with loss of function of the extensor muscles of the elbow, carpus, and digits, and

results in inability to stand on the limb. The elbow is dropped, and the horse tends to stand with the affected limb forward, with the antebrachium at an angle of approximately 45° to the ground, with the carpus and fetlock semiflexed. Less commonly only the extensor muscles of the elbow are affected, so although the limb may be advanced forward, the elbow will drop during weight bearing. Occasionally only the extensor muscles of the carpus and digit are affected, so that the limb is advanced and placed normally, but then the carpus or fetlock may slightly knuckle forward. The horse has a tendency to stumble.

Lameness is usually sudden in onset. The horse may have a history of trauma. Paralysis occurs most commonly in young horses turned out together. Although damage to the radial nerve itself may be the cause of lameness,[44] many injuries are probably caused by compression of the brachial plexus between the scapula and the ribs.[4] An electromyogram may demonstrate involvement of more than the radial nerve. Equine protozoal myelitis should be considered in older horses, especially if evidence of other lower motor neuron pathological conditions exists (see page 129).

Many horses make a slow progressive recovery. Electrical stimulation of the affected muscles may help to maintain muscle mass. Problems may arise in young animals with overload of the contralateral limb, unless symptoms resolve quickly. An improvement within 2 to 4 weeks warrants an optimistic prognosis. Persistence of clinical signs for more than 6 months warrants a guarded prognosis.

First Rib

Occasionally acute-onset, severe lameness follows trauma from a fall or collision with a solid object associated with a fracture of the first rib. The lameness is typical of a proximal limb injury, but no other localizing signs are present. Atrophy of infraspinatus alone has also been recognized in some horses. Diagnosis is by radiographic examination. Nuclear scintigraphic examination may facilitate diagnosis. The prognosis is good after rest.

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CHAPTER • 42

The Hind Foot and Pastern

Mike W. Ross

The contribution of the hind foot and pastern to hindlimb lameness is considerably less important than that of the digit to forelimb lameness. Extensive information regarding lameness of the foot and pastern in the forelimb is found in Chapters 26 to 36 and 83. This information is directly applicable to the hindlimb. During lameness examination, the hind foot and pastern are easily overlooked because they are in a potentially dangerous place to examine. Other regions of the hindlimb have been presumed to be more important and, historically, little has been taught about the distal part of the limb. Without a commitment to performing diagnostic analgesic techniques in the distal hindlimb, there is little way to discover whether the digit is the authentic source of pain unless the problem is severe or obvious. Subtle primary or compensatory lameness problems of this region are likely to go unnoticed daily. However, in certain sport horses, such as the draft horse, lameness of the hind foot and pastern is so common that this region cannot be overlooked.

ANATOMY AND INNERVATION OF THE HIND FOOT AND PASTERN

The bones are essentially the same as in the forelimb. The hind distal phalanx is narrower compared with the forelimb and has a steeper dorsal angle. The plantar surface is more concave, and the plantar processes are closer together.[1] The external appearance of the hind foot usually is more upright than the fore foot, but abnormal wear and shoeing practices can produce a common pathological condition of low, under-run heels (see Chapter 6). The hind middle phalanx is narrower and longer, and the hind proximal phalanx is slightly shorter than the corresponding bones in the forelimb.[1] The ligaments, tendons, digital flexor tendon sheath (DFTS), and distal interphalangeal and proximal interphalangeal joints are the same as in the forelimb. Innervation of the foot is derived primarily from the medial and lateral plantar nerves that originate from the tibial nerve (see Chapter 10). The medial and lateral plantar metatarsal nerves, which originate from the deep branch of the lateral plantar nerve, become superficial just distal to the "bell" of the second and fourth metatarsal bones. Unlike the palmar metacarpal nerves, the hindlimb medial and lateral plantar metatarsal nerves supply sensation to the pastern region and coronary band, a fact that can complicate interpretation of perineural analgesia in the hind digit.[2]

EXAMINATION, CLINICAL SIGNS, AND DIAGNOSIS

The clinical examination of the hind foot and pastern was described in Chapter 6, and a detailed description of the clinical investigation of the foot and shoeing is found in Chapters 26 to 28. A conscientious effort must be made during every lameness examination to evaluate the hind foot with the limb in both the standing and flexed positions. The type of shoeing and shoe wear are important in understanding potential lameness conditions of the foot, but perhaps more important, study of hind foot balance and shoeing can provide important clues in the diagnosis of lameness located more proximally in the limb. Dramatic abnormalities in balance and shoe additives, such as calks, grabs, trailers, and bars, can place added forces of shear and torsion on hindlimb bones and joints. Therapeutic recommendations cannot be made unless the clinician is aware of the type of shoes that are currently in place. Hoof imbalance has not been studied extensively in the hindlimb, but dorsal to plantar and medial to lateral hoof imbalance is a likely contributor to lameness in the proximal interphalangeal and metatarsophalangeal joints. Palpation for signs of inflammation, such as increased digital pulse amplitude, heat, swelling, and pain, must be performed but can be difficult in fractious horses. Palpation is difficult in the hindlimb because of the stay apparatus, which causes the digit to flex involuntarily. In Draft horses, signs of inflammation can be obscure (Fig. 42-1). Examination with hoof testers is important, but normally horses show considerable sensitivity across the heels (see Chapter 6). Because the foot is incorporated in the rigid hoof capsule and swelling may not be apparent, even severe lameness conditions, such as abscesses or fractures, can go unnoticed. Commonly lameness of the hind foot is misinterpreted as high up in the limb, because clinical signs are not prominent and easy to overlook.

Degree of lameness varies greatly. Most recognized problems of the hind foot cause obvious, often severe lameness, but occasionally subtle or insidious problems occur. Horses

Fig. 42-1 The defect in the coronary band of this draft horse was not detected until the long hair (or feathers) was shaved. Hoof tester examination yielded negative findings, but investigation of the foot was prompted by a positive response to perineural analgesia.

with the most common problems, such as hoof abscess and penetrating wounds, may not bear weight. If they are bearing weight, horses usually are severely lame at the trot. Horses with severe hind foot lameness, particularly of the toe region, may walk with a shortened caudal phase of the stride, similar to horses with pain in the coxofemoral region (see Chapter 7). The horse may bear weight only on the toe. Turning may accentuate pain. At the trot, horses with hind foot and pastern lameness have a shortened cranial phase of the stride and travel like horses with pain that originates anywhere distal to the distal crus. When the horse is viewed from behind, limb flight is characterized by moving the foot straight ahead or slightly medial to the line of expected limb flight and then stabbing laterally during the later portion of the stride, just before impact.

Many lameness conditions in this region worsen with a lower limb flexion test. Horses with osteoarthritis of the distal interphalangeal and proximal interphalangeal joints or tenosynovitis and tendonitis of the deep digital flexor tendon respond prominently. In general, hindlimb flexion tests have low specificity, and in horses that respond markedly to lower limb flexion, a source of pain located more proximal in the limb cannot be dismissed without using diagnostic analgesia.

Lameness must be localized using perineural or intra-articular analgesic techniques. Differentiation of sources of pain in the hind foot and pastern can be problematic because the same difficulties in interpretation of plantar digital and distal interphalangeal analgesic techniques occur as in the forelimb. When the plantar digital block is done with the limb elevated, it is difficult to inject the solution just above the cartilages of the foot, so this block is usually performed in a more proximal location than in the forelimb. It may be difficult to remove all pain in horses with severe lameness. Pain associated with the proximal aspect of the proximal phalanx, such as from mid-sagittal fractures, can easily be removed inadvertently during performance of plantar digital analgesia.[3] Careful interpretation of analgesic techniques is necessary, particularly in racehorses, in which metatarsophalangeal joint region lameness is common.

Imaging considerations are similar to those described in the forelimb. It may be preferable to obtain plantarodorsal rather than dorsoplantar radiographic views of the foot. Many horses are reluctant to stand on a block with the foot bearing weight for lateromedial views. Satisfactory images can readily be obtained with the foot bearing weight only on the toe provided that the foot is not rotated.[4]

SPECIFIC LAMENESS CONDITIONS

Overall the most common conditions of the hind foot and pastern involve hoof abscesses and bruises, penetrating wounds into the hoof capsule and soft tissues of the pastern region, and cellulitis. Scratches can cause horses to appear lame at a walk and when severe, lame also at the trot. Management of these conditions is well described elsewhere.

Distal Phalanx Fractures

Fractures of the distal phalanx occur in any type of sport horse as the result of direct trauma from kicking a wall or trailer. Occasionally horses step on a hard object while turned out or during competition. In racehorses, fractures of the distal phalanx can occur as a result of non-adaptive remodeling, but this is much less common than in the forelimb. Because many fractures are the result of direct trauma, the distribution varies. Articular plantar process fractures are most common, but incomplete (stress) fractures and mid-sagittal fractures also occur. Lameness is variable in degree, and it is important to recognize that lameness may be episodic in horses with a non-articular plantar process fracture. Fractures often can be seen in lateromedial radiographic views, but special oblique views may be required. Comminuted fractures are rare but develop as the result of direct trauma. Conservative management in most horses with fracture of the distal phalanx is quite successful. The decision to perform neurectomy is often made earlier than for injuries in a forelimb.

Laminitis

In horses with enterocolitis, pleuritis, metritis, or other systemic diseases, laminitis can develop in all four limbs, but rarely is the condition seen in only the hindlimbs. Even when hind feet are involved, clinical signs usually are most prominent in the front feet. Laminitis restricted to the hind feet only has been seen in overweight cob-type horses and ponies.[5] Traumatic laminitis can occur from intense exercise on hard ground in unshod horses, but it most commonly develops in the contralateral limb of horses with severe unilateral hindlimb lameness. In the latter form, called *orthopedic laminitis*, distal displacement or rotation of the distal phalanx generally takes considerable time to develop and can go unrecognized for several weeks. Often the first clinical sign that a problem is developing in the contralateral (laminitic) limb is that the lameness is observed to be inexplicably considerably improved in the original limb. Rarely, I have observed hindlimb laminitis as an apparently incidental finding in horses referred for scintigraphic examination for primary forelimb lameness or when whole-body scintigraphic examinations are performed. Marked increased radiopharmaceutical uptake (IRU) involving the distal or dorsal aspect of the distal phalanx and radiographic evidence of rotation were seen. Primary lameness is found elsewhere, but laminitis in these horses may represent an unusual form of compensatory lameness.

I have examined two pleasure horses with primary bilateral hindlimb laminitis (without forelimb involvement) in which an unusual hindlimb gait was observed. At the walk horses had a goose-stepping gait like that seen in horses with fibrotic myopathy. The horses appeared to be attempting to land with an exaggerated heel-to-toe hoof strike. Both horses had a marked shortening of the caudal phase of the stride. At the trot horses had a short, choppy gait, but because lameness was bilateral, overt unilateral signs could not be seen.

Keratoma

Keratoma involving the hind feet is rare. An unusual radiolucent defect thought to be a keratoma that caused lameness and poor racing performance, and associated with IRU was seen in the distal phalanx of a Thoroughbred racehorse (Fig. 42-2).

Navicular Syndrome

Navicular disease, osseous cyst-like lesions, and fractures of the navicular bone are unusual in the hindlimb but do occur in all types of horses and ponies (see Chapters 30 through 32). Lameness is variable in degree, usually unilateral, and improved by perineural analgesia of the plantar digital nerves. Lesions are usually readily detectable radiographically (Fig. 42-3). However, it is more difficult to obtain a skyline view of the navicular bone in a hindlimb than in a forelimb. Scintigraphy can be helpful in some horses.

Distal Interphalangeal Joint

Primary osteoarthritis of the hind distal interphalangeal joint is rare. Osteophytes involving the extensor process of the distal phalanx are commonly seen but are usually incidental radiographic findings. Osteoarthritis occurs as a result of instability or incongruity caused by articular fractures of the distal phalanx. Occasionally subchondral bone damage of the articular surface of the distal phalanx occurs and is recognized as radiolucent defects and secondary osteoarthritic changes;

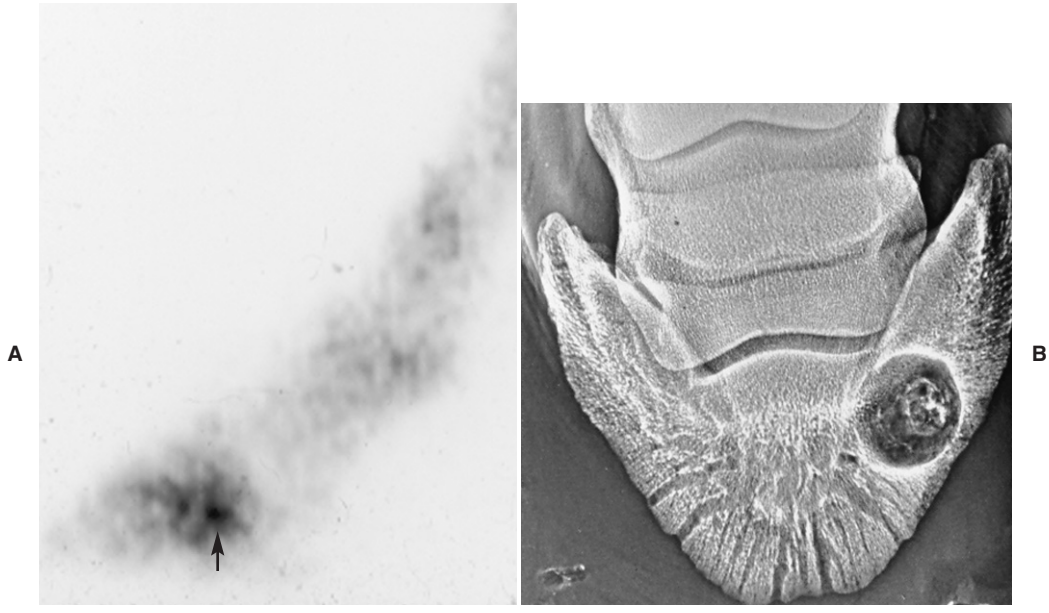

Fig. 42-2 **A,** Lateral delayed (bone) phase scintigraphic image. There is focal increased radiopharmaceutical uptake in the distal phalanx *(arrow)* of this Thoroughbred with undiagnosed right hindlimb lameness. **B,** Dorsoproximal-plantarodistal xeroradiographic view of a nearly circular radiolucent defect of the distal phalanx with either fragmentation or dystrophic mineralization within it. A tentative diagnosis of keratoma was made, but surgery was not performed.

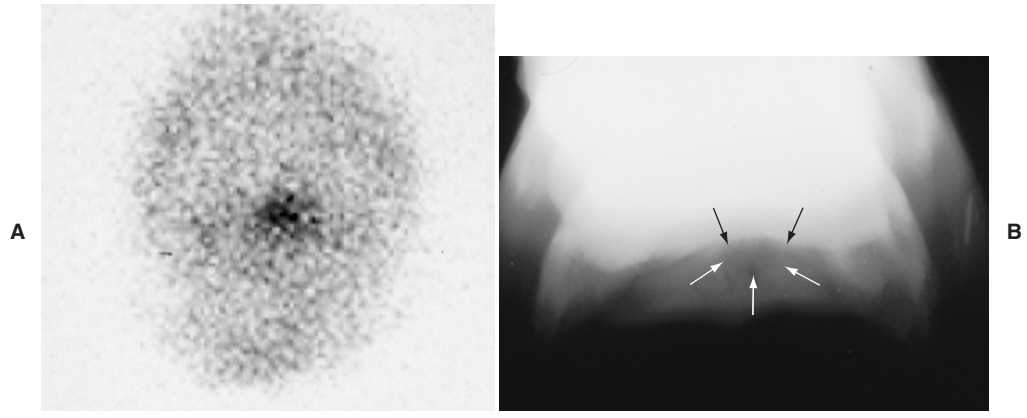

Fig. 42-3 **A,** Delayed (bone) phase scintigraphic image showing focal, moderately increased radiopharmaceutical uptake of the navicular bone (dorsal is to the top). **B,** Corresponding plantaroproximal-plantarodistal (skyline) radiographic view showing a distinct radiolucency *(arrows)* of the navicular bone in a 4-year-old Standardbred racehorse with left hindlimb lameness.

this damage usually is seen in older non-racehorse sport horses. Chronic, undiagnosed hindlimb lameness is followed by acute, prominent lameness. Diagnosis may be difficult, thus prompting scintigraphic examination. Focal areas of IRU in the subchondral bone of the distal phalanx should be differentiated from those in horses with incomplete fracture.

Extensor process fragments or fractures are rare. Osseous cyst-like lesions as a result of osteochondrosis of the distal phalanx are seen occasionally. Some small lesions restricted to the subchondral bone of the distal phalanx are difficult to identify radiographically, but usually result in intense, focal IRU scintigraphically. Definitive diagnosis may be possible only with magnetic resonance imaging or at post-mortem examination.[5] Other fractures involving the distal aspect of the middle phalanx and comminuted fractures of this bone occur. Involvement of the distal interphalangeal joint in horses with comminuted fractures of the middle phalanx is a negative prognostic finding.

The Hind Pastern

Lameness of the hind pastern region includes osteoarthritis of the proximal interphalangeal joint, osseous cyst-like lesions of the distal aspect of the proximal phalanx, osteochondral fragmentation of the proximal interphalangeal joint, fractures of the middle and proximal phalanges, subluxation of the proximal interphalangeal joint, tenosynovitis of the DFTS, tendonitis of the deep digital flexor tendon and the branches of the superficial digital flexor tendon, distal sesamoidean ligament desmitis, penetrating wounds, and cellulitis (see Chapters 36, 71, 75, and 83).

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CHAPTER • 43

The Metatarsophalangeal Joint

Mike W. Ross

Lameness of the metatarsophalangeal joint is similar to that of the metacarpophalangeal joint, but historically it has largely been ignored and is mentioned only twice in a leading lameness textbook.[1] With fastidious use of both intra-articular and peri-neural analgesia, the metatarsophalangeal joint is now known to contribute substantially to hindlimb lameness.[2]

ANATOMY

The metatarsophalangeal joint is nearly identical to the metacarpophalangeal joint and is composed of the distal articular surface of the third metatarsal bone (MtIII), its sagittal ridge, and medial and lateral condyles; the medial and lateral proximal sesamoid bones (PSBs); and the proximal articular surface of the proximal phalanx, which has a prominent axially located sagittal groove (see Chapter 37). Minor differences exist in the shape and length of the proximal phalanx between the forelimbs and hindlimbs but are not clinically relevant. The metatarsophalangeal joint normally is more upright than the metacarpophalangeal joint and can achieve a greater degree of flexion. The lateral to medial width of the lateral condyle of the MtIII is less than that of the medial condyle. In racehorses, stress-related bone injury occurs predominantly in the lateral aspect of the hindlimb when based on scintigraphic examination. When combined with a smaller surface area, the lateral condyle may be prone to injury. The joint capsule, intersesamoidean and collateral sesamoidean ligaments, suspensory ligament (SL) attachments, and the flexor tendons all function to move and support the metatarsophalangeal joint. The dense collateral ligament has short, deep, and long superficial components. In the hindlimb the lateral digital extensor tendon joins with the long digital extensor tendon in the proximal dorsal metatarsal region. Therefore only the long digital extensor tendon is encountered during arthrocentesis or surgical procedures performed in the dorsal aspect of the metatarsophalangeal joint.

CONFORMATION

Fetlock valgus and varus deformities affect the metatarsophalangeal joint in foals, but fetlock varus deformity is of most concern and needs to be corrected early (see Chapter 60). Most normal horses are slightly toed out in the hindlimbs, but in some toed-out conformation may play a role in uneven load distribution and affect hindlimb gait. Horses with toed-out conformation tend to travel close behind and stab laterally during limb advancement, a gait that may cause excessive lateral shoe wear and hoof imbalance. Such gait and hoof imbalance may predispose the lateral aspect of the metatarsophalangeal joint to stress-related bone injury. Horses that are excessively straight behind have an abnormal degree of extension (or dorsiflexion) of the metatarsophalangeal joint. This conformation is undesirable because it places abnormal load on the SL and predisposes to suspensory desmitis, stifle joint lameness, and secondary osteoarthritis of the metatarsophalangeal joint.

CLINICAL CHARACTERISTICS AND DIAGNOSIS OF METATARSOPHALANGEAL JOINT LAMENESS

Lameness of the metatarsophalangeal joint occurs in most sport horses and is common in the racehorse. Both the Standardbred (STB) and Thoroughbred (TB) racehorse are prone to injury of this joint, but metatarsophalangeal joint lameness is more common in the STB, because gait and load distribution predispose to hindlimb lameness in this breed (see Chapter 2). Curiously, metatarsophalangeal joint lameness is apparently uncommon in Western performance horses despite the predominant use of the hindlimbs for many maneuvers and thus tremendous forces applied to the metatarsophalangeal joint (see Chapter 121). There is no pathognomonic historical information that incriminates the metatarsophalangeal joint more than other sources of hindlimb lameness, but racehorses are usually worse in turns. In horses with bilateral metatarsophalangeal joint lameness, poor performance may be the only historical finding and overt signs of lameness may not be present, but a short, choppy gait or intermittent, shifting hindlimb lameness is seen. A gait typical of bilateral stress-related bone injury of the distal aspect of the MtIII in TB racehorses being trotted in hand is described as an "exaggerated pelvic excursion" in a dorsal ventral direction (see Chapter 109).

Clinical signs of metatarsophalangeal joint lameness vary from subtle to overt and depend on the nature and severity of injury. Signs of inflammation may be absent in horses with stress-related bone injury but severe in horses with displaced or comminuted fractures. At the trot limb flight is similar to lameness originating from the metatarsal region and hock. As the limb moves forward, it deviates medially and is then stabbed laterally at the end of the cranial phase of the stride.

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Clinical signs of metatarsophalangeal joint lameness vary from subtle to overt and depend on the nature and severity of injury. Signs of inflammation may be absent in horses with stress-related bone injury but severe in horses with displaced or comminuted fractures. At the trot limb flight is similar to lameness originating from the metatarsal region and hock. As the limb moves forward, it deviates medially and is then stabbed laterally at the end of the cranial phase of the stride.

The cranial phase is shortened commensurate with the degree of pain. Lameness is more pronounced with the affected limb on the inside of a circle. I find it useful to characterize clinical signs into three categories: severe, unrelenting lameness; intermittent, severe lameness; and chronic, low-grade lameness.

Severe, Unrelenting Lameness

This form of metatarsophalangeal joint lameness is associated with intra-articular fractures or severe, end-stage osteoarthritis. Horses are obviously lame at the walk, cannot be trotted, and may be non–weight bearing. Deformity of the metatarsophalangeal joint may be obvious, as in horses with comminuted fractures of proximal phalanx. Effusion is obvious and diffuse, periarticular soft tissue swelling may be present. Horses with acute, displaced condylar fractures of the MtIII may have acute, progressive edema of the diaphyseal region, and those with comminuted fractures of the proximal phalanx often have severe soft tissue swelling. Palpation and flexion elicit severe pain and, in horses with comminuted or displaced fractures, crepitus. Radiographs are usually diagnostic. This category includes severe, complete, or comminuted fractures (e.g., spiral fractures of the MtIII), comminuted or complete fractures of the proximal phalanx, mid-body fractures of the PSBs, luxation or subluxation, and end-stage osteoarthritis. Horses with acute tendonitis of the deep digital flexor tendon (DDFT) with tenosynovitis can be severely lame, and intrathecal analgesia of the digital flexor tendon sheath (DFTS) may only partially remove pain. Ultrasonographic examination is required.

Intermittent, Severe Lameness

Horses with intermittent, severe lameness of the metatarsophalangeal joint may be able to train and perform at some level but develop severe lameness afterward. When exhibiting clinical signs, horses are lame at the walk, even toe-touching lame, and are obviously lame at the trot (grade 3 or 4 of 5), but after resting and receiving non-steroidal anti-inflammatory drugs (NSAIDs), horses are often able to gallop, jog, or train within 1 to 5 days. In some instances horses may be able to race, only to become lame once again. When walking in hand, horses often show marked lameness while turning, even if they are sound while walking in a straight line. Obvious signs of inflammation usually are not present, but horses generally respond positively to the lower limb flexion test and may show a painful response to deep palpation. Horses with incomplete mid-sagittal or dorsal frontal fractures of the proximal phalanx often manifest a painful response when firm digital pressure is placed on the proximal, dorsal aspect. Effusion may be present, but it is often absent or minimal even in horses with incomplete fractures. Because clinical signs may be difficult to detect, diagnostic analgesia is often necessary. Radiographs are usually diagnostic, but if findings are equivocal or the radiographs are obtained before radiographic changes develop, scintigraphic examination or follow-up radiographic examination in 10 to 14 days is required. Xeroradiographs and computed radiographs can be helpful. Conditions such as incomplete fractures of the MtIII, the proximal phalanx, and the PSBs and moderate osteoarthritis are in this category.

Chronic, Low-Grade Lameness

Diagnosis of chronic, low-grade metatarsophalangeal joint lameness is difficult. Specific signs that localize lameness to the metatarsophalangeal joint are lacking and lameness may only be subtle to mild (grade 1 of 5) at the trot in hand. Horses examined at the track and carrying a rider may only show mild lameness. Effusion and heat generally are absent and the response to lower limb flexion varies, but it usually is negative. Palpation may reveal mildly suspicious areas (e.g., pain over the abaxial aspect of the PSBs) in horses with sesamoiditis, but in most horses no abnormalities are noted. Concomitant lameness of the metatarsophalangeal joint and stifle region can occur in racehorses and clinical signs may not abate until both sites are treated. The association between the stifle and metatarsophalangeal joint is difficult to explain. In young horses with "loose stifles," knuckling of the metatarsophalangeal joint occurs when horses, particularly STBs, are jogged or worked slowly. Perhaps stretching of metatarsophalangeal joint capsule attachments or early subchondral bone trauma occurs during knuckling. Horses with "loose stifles" are thought to have patellar ligament and muscular instability and laxity and may benefit from counterirritant injection and simultaneous management of the metatarsophalangeal joint problem.

Diagnostic analgesia is required to localize pain causing lameness to the metatarsophalangeal joint, but in many horses lameness is difficult to accurately assess with the horse at a trot in hand. Often radiographic examination findings are normal and horses are referred for scintigraphic examination, which often reveals stress-related bone injury. Special radiographic projections may be needed to evaluate the distal aspect of the MtIII or for accurate identification of osteochondral fragments located in the dorsal and plantar aspects of the joint. In this category are conditions such as stress-related bone injury of the MtIII, early osteoarthritis, osteochondral fragments that occur traumatically or as the result of osteochondrosis, and sesamoiditis.

Diagnostic Analgesia

Articular pain originating from the metatarsophalangeal joint can usually be abolished or at least partially alleviated by intra-articular analgesia. Because only one injection is required, intra-articular analgesia is easier and safer to perform than perineural techniques, but pain originating from subchondral bone may not abate or may be only partially alleviated by intra-articular analgesia. Therefore the low plantar peri-neural technique or a variation must be used, because it is more effective in alleviating pain from all sources (see Chapter 10).

A variation of the low plantar block, the lateral plantar metatarsal block, can be performed in horses with stress-related bone injury of the distal plantarolateral aspect of the MtIII (see Chapter 109).[3] This block is particularly valuable in horses with bilateral lameness, because after one limb is blocked, the horse becomes obviously lame in the other.

In horses with chronic, low-grade lameness it may be necessary to perform analgesia and watch the horse train. Resolution of subtle signs, such as bearing in or out, not feeling right behind, performing dressage maneuvers, or in STB racehorses, being on a shaft or a line, may be the only sign of a positive response to diagnostic analgesia. Although peri-neural analgesia may result in slight loss of proprioception, it is generally preferable, because intra-articular analgesia may result in a false-negative response. If pain is elicited by firm palpation of the dorsoproximal aspect of the proximal phalanx, suspect a mid-sagittal fracture. Nerve blocks are contraindicated because of the risk of creating a complete or comminuted fracture.

The clinician should be aware that it is possible to inadvertently block pain associated with subchondral bone of the metatarsophalangeal joint when performing plantar digital or plantar peri-neural analgesia. This occurs most frequently in racehorses with mid-sagittal fracture of the proximal phalanx but can occur in horses with osteoarthritis or other conditions (see Chapters 10 and 42).

IMAGING CONSIDERATIONS

Radiographic Examination

Examination should include dorsoplantar (DPl), lateromedial (LM), dorsolateral-plantaromedial oblique (DL-PlMO), and dorsomedial-plantarolateral oblique (DM-PlLO) views. A

flexed LM view is useful to evaluate the sagittal ridge for the presence of osteochondrosis lesions and to see dorsal frontal fractures of the proximal phalanx. Vacuum phenomenon can occur because the metatarsophalangeal joint can be placed in extreme flexion. Sudden decompression of the joint during stress flexion is believed to cause what appears to be an air artifact in the distal plantar aspect of the MtIII.[4]

Horizontal oblique views are useful to evaluate the proximal-dorsal aspect of the proximal phalanx for the presence of osteochondral fragments, but overlap between the base of the PSBs and the proximal aspect of the proximal phalanx can hide lesions associated with the distal-plantar aspect of the joint. The x-ray beam should be angled down 15° to 20° to separate the PSBs and proximal phalanx (Fig. 43-1).

Using a horizontal x-ray beam in a DPl view, the PSBs are superimposed over the distal aspect of the MtIII and metatarsophalangeal joint. To better evaluate these areas, dorsal 15° proximal-plantarodistal oblique (D15° Pr-PlDiO), flexed DPl, and standing 125° DPl views should be used. Proximolateral (medial)-distolateral (medial) and proximoplantar-distoplantar tangential views of the PSBs are occasionally used to evaluate the abaxial and plantar surfaces of the PSBs, respectively.

Approximately 5% to 10% of normal horses have a small unilateral or bilateral radiolucent notch (1 mm in length) in the sagittal grove of the proximal phalanx that is seen in a DPl view and should not be confused with a mid-sagittal fracture. If clinical signs are consistent with mid-sagittal fracture, scintigraphic examination is recommended. Flattening of the plantar aspect of the distal MtIII condyles is not as common as in the metacarpophalangeal joint, but sclerosis of the plantar aspect of the MtIII can be seen in horses with stress-related bone injury and early osteoarthritis if well-exposed LM and flexed LM views are obtained. I question the significance of flattening of the condyle of MtIII in most horses. Xeroradiographs and computed radiographs are useful in the evaluation of horses with stress-related bone injury and incomplete fractures. Computed tomography and magnetic resonance imaging are also useful, but availability is limited.

Scintigraphic Examination

Scintigraphic examination is the best way to establish a diagnosis in many horses with intermittent, severe, or chronic low-grade lameness of the metatarsophalangeal joint. The most common scintigraphic findings in the metatarsophalangeal joint of STB and TB racehorses are focal areas of increased radiopharmaceutical uptake (IRU) that involve the distal plantarolateral aspect of the MtIII (Fig. 43-2).[5] This IRU is a form of stress-related bone injury and early osteoarthritis that is found in many racehorses with chronic, low-grade lameness; bilateral hindlimb lameness; and poor performance. Special radiographic views may then reveal sclerosis or radiolucent defects (Fig. 43-3). Similar scintigraphic findings are seen in horses with lateral MtIII condylar fractures. Focal areas of IRU are seen in horses with mid-sagittal or dorsal frontal fractures of the proximal phalanx (Fig. 43-4), sesamoiditis (Fig. 43-5), and osteochondrosis of the plantar process of the proximal phalanx (Fig. 43-6). Often areas of IRU are found in unusual sites, such as those associated with the medial PSB (osteochondral fragments of the abaxial border and sesamoiditis), intersesamoidean ligament injury with radiolucent defects in one or both PSBs, incomplete fractures of the PSBs, and medial condylar fractures of the MtIII. Any focal area of IRU located medially in the metatarsophalangeal joint should be investigated, because incidental findings are unusual in this location.

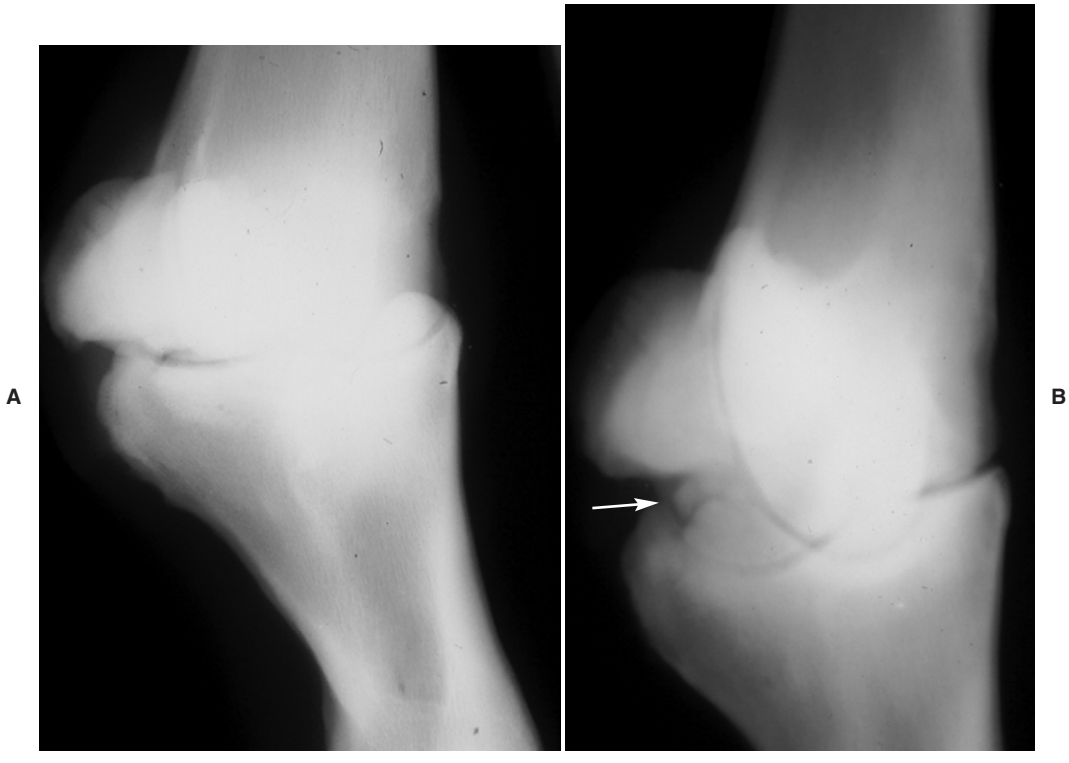

Fig. 43-1 **A,** Dorsomedial-plantarolateral oblique radiographic view obtained using a horizontal x-ray beam. Note the overlap of the distal aspect of the proximal sesamoid bones and the proximal aspect of the proximal phalanx. **B,** The proximal plantar aspect of the proximal phalanx can be clearly evaluated using a dorsomedial proximal-plantarolateral distal oblique view. An axial, intra-articular fragment is present *(arrow)*.

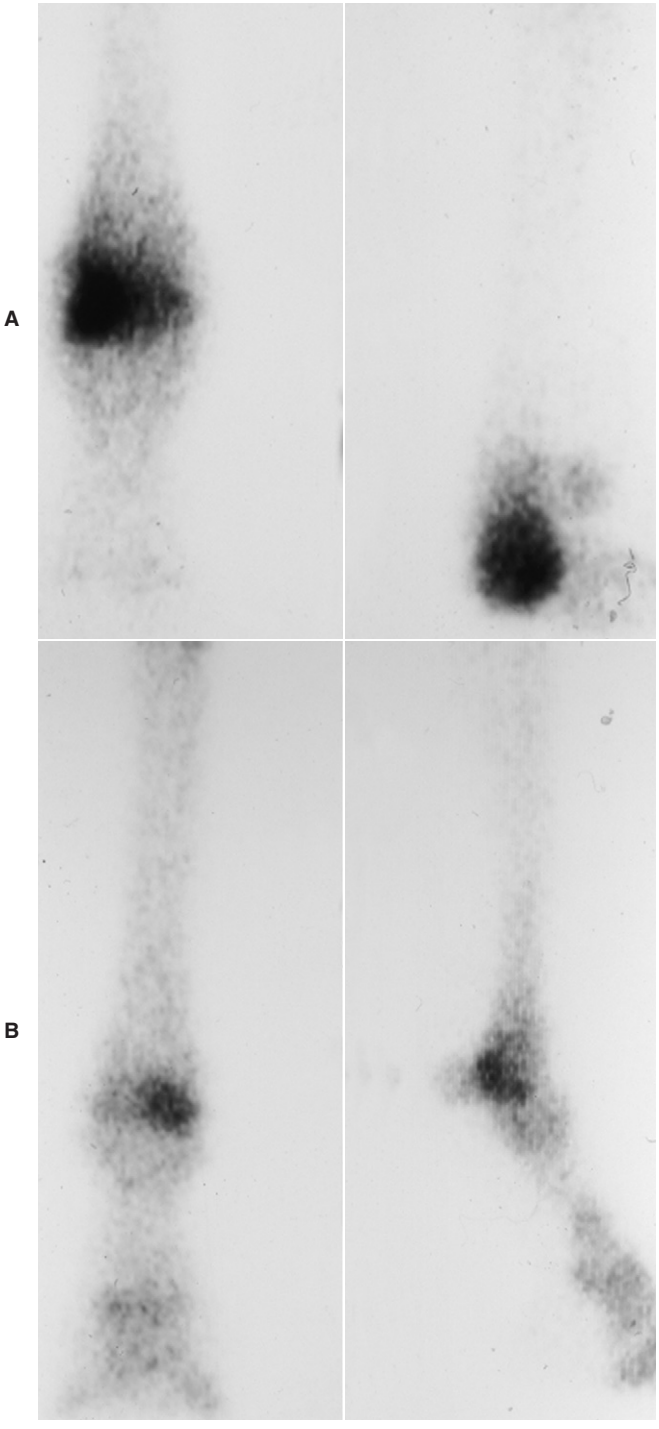

Fig. 43-2 **A,** Plantar (lateral to the left) and flexed lateral (on the right) right hindlimb and **B,** plantar (lateral to the right) and lateral (on the right) left hindlimb delayed (bone) phase scintigraphic images of the metatarsophalangeal joint in two Standardbred racehorses. All images show focal increased radiopharmaceutical uptake of the distal, plantarolateral aspect of the third metatarsal bone, which is the most common scintigraphic finding in the metatarsophalangeal joint of racehorses.

Scintigraphic changes are usually less pronounced in nonracehorses, and negative or equivocal results often occur. The most common scintigraphic findings in jumpers and dressage horses with metatarsophalangeal joint lameness are diffuse mild areas of IRU in all bones or focal mild or moderate IRU in the dorsal aspect of the joint that involves the distal MtIII

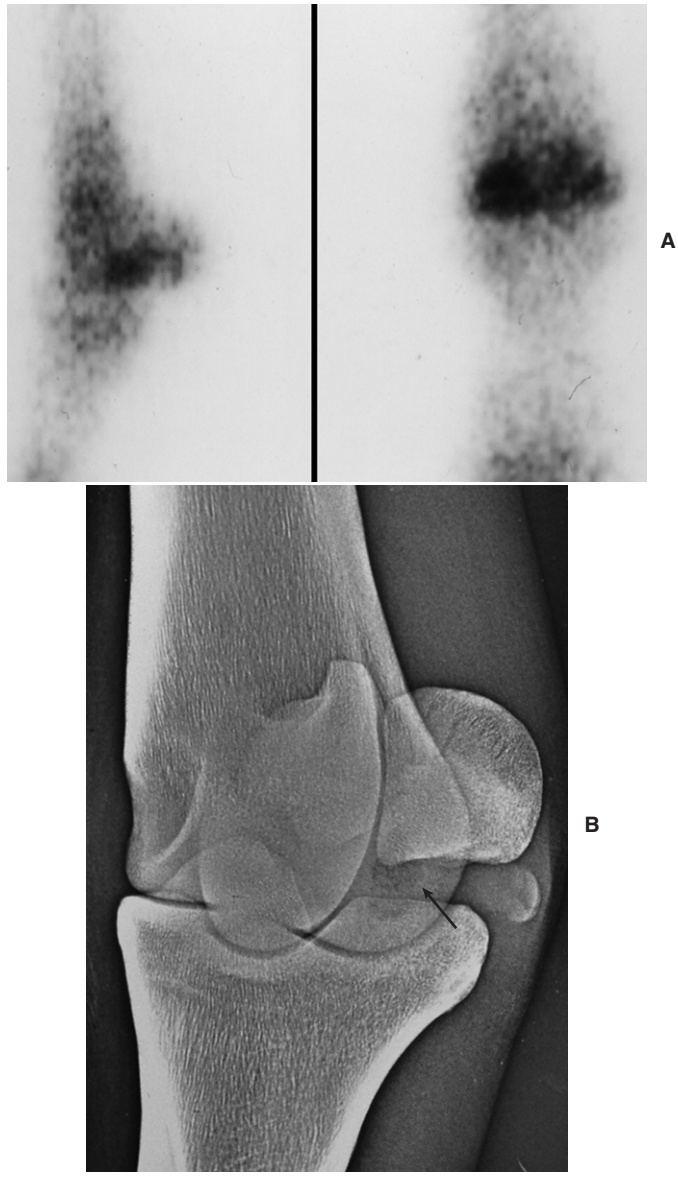

Fig. 43-3 **A,** Lateral (on the left) and plantar (lateral is to the left) bone phase scintigraphic images of a metatarsophalangeal joint. There is increased radiopharmaceutical uptake in the plantar aspect of the lateral condyle of the third metatarsal bone. **B,** Dorsolateral proximal-plantaromedial distal oblique xeroradiographic view showing a radiolucent defect *(arrow)* of the same area. This defect can easily be missed on routinely positioned views, but the increased radiopharmaceutical uptake seen in this area scintigraphically **(A)** prompted further investigation.

and proximal aspect of the proximal phalanx and is associated with marginal osteophytes and enthesophytes. In some horses arthroscopic examination has revealed full-thickness cartilage damage that primarily involves the distal MtIII and is consistent with osteoarthritis. Focal IRU in the proximal aspect of the plantar pouch is seen in horses with severe osteoarthritis (an ominous finding) (Fig. 43-7). I believe this finding is caused by accumulation of subchondral bone fragments in this location or modeling of the distal plantar aspect of the MtIII.

Ultrasonographic Examination

Ultrasonographic examination of the metatarsophalangeal joint region is indicated for suspensory branch desmitis (see Chapter 73). The abaxial aspect of the PSBs should be examined carefully to identify tearing and small avulsion fractures.

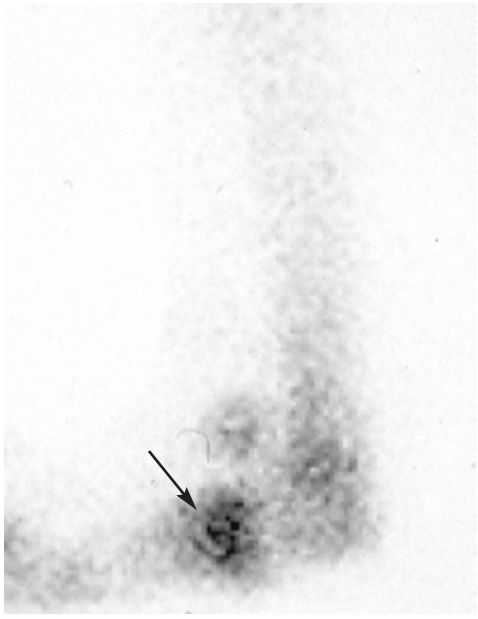

Fig. 43-4 Flexed lateral delayed (bone) phase scintigraphic image of a horse with incomplete mid-sagittal fracture of the proximal phalanx. Focal moderate increased radiopharmaceutical uptake in the proximal phalanx is somewhat more pronounced in the plantar aspect *(arrow)*. In most horses with mid-sagittal fracture of the proximal phalanx, increased radiopharmaceutical uptake is most prominent dorsally.

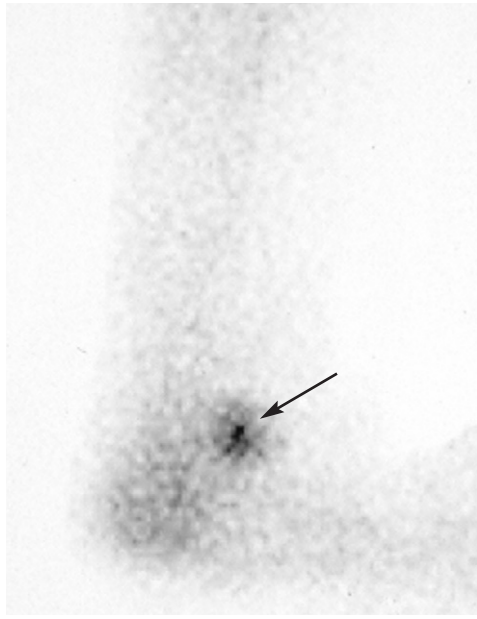

Fig. 43-5 Flexed lateral delayed (bone) phase scintigraphic image of a 2-year-old Standardbred racehorse with lateral sesamoiditis. There is focal moderate increased radiopharmaceutical uptake in the proximal sesamoid bones *(arrow)*. When these findings were combined with those of the plantar view, the increased radiopharmaceutical uptake was localized to the lateral proximal sesamoid bone.

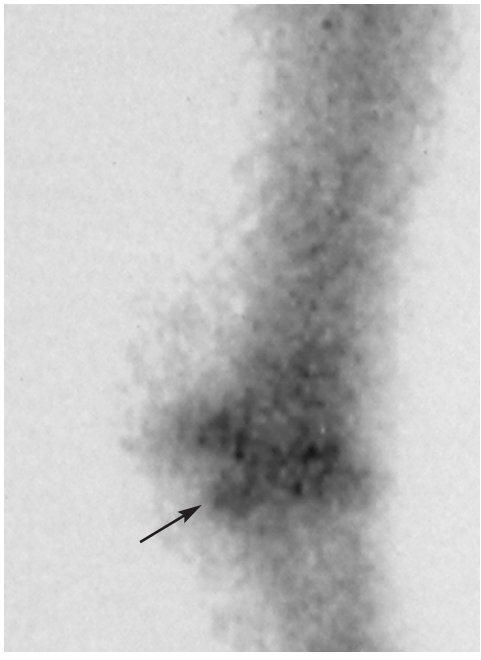

Fig. 43-6 Lateral delayed (bone) phase scintigraphic image. There is focal mild increased radiopharmaceutical uptake involving the proximal plantar process of the proximal phalanx *(arrow)* in a Standardbred racehorse with axial articular fragments of the plantar process.

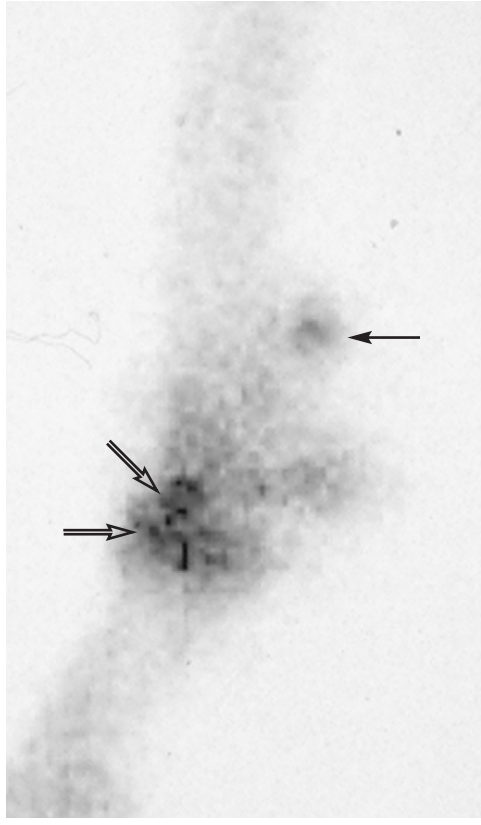

Fig. 43-7 An ominous finding in delayed (bone) phase scintigraphic images is increased radiopharmaceutical uptake in the region of the proximal plantar pouch *(arrow)*, which indicates the presence of severe osteoarthritis. There is increased radiopharmaceutical uptake involving the proximal aspect of the proximal phalanx and distal third metatarsal bone *(open arrows)*.

Proliferative synovitis (villonodular synovitis) is unusual in the metatarsophalangeal joint. The DDFT should be evaluated carefully if tenosynovitis of the DFTS is present[6] (see Chapter 71). Ultrasonographic examination is useful in horses with intersesamoidean and collateral ligament injuries or those with wounds and draining tracts to look for foreign material or communication with the articular surface.

Arthroscopic Examination

Arthroscopic surgery is used frequently for removal of osteochondral fragments and fractures of the PSBs, assistance in fracture reduction and screw placement in horses with condylar fractures of the distal MtIII and fractures of the proximal phalanx, and lavage and debridement in horses with infectious arthritis. Diagnostic arthroscopic examination is indicated if lameness is localized to the metatarsophalangeal joint, but radiographic findings are negative or suggestive of occult osteochondral fragments. Diagnostic arthoscopy is also indicated if scintigraphic images are abnormal, so that cartilage damage or osteochondral fragments are suspected, and to confirm the extent of cartilage damage in horses with osteoarthritis. Occult fragments involving the proximodorsal aspect of the proximal phalanx are occasionally found. Cartilage damage, sometimes full-thickness, is found on the distal-dorsal aspect of the MtIII and proximal aspect of the proximal phalanx in non-racehorses. In racehorses with osteoarthritis cartilage lesions are usually most pronounced in the plantar pouch, with extensive scoring or large areas of full-thickness damage and exposed subchondral bone on the PSBs. Although stress-related bone injury and later overlying cartilage damage is seen on the distal plantarolateral aspect of the MtIII, this area is difficult to evaluate during arthroscopic examination (Fig. 43-8).

SPECIFIC CONDITIONS OF THE METATARSOPHALANGEAL JOINT

Stress-Related Subchondral Bone Injury and Osteoarthritis

The term *osteoarthritis* implies disease of both the supporting bone and the articular surface of the metatarsophalangeal joint. The concept that the earliest lesion in osteoarthritis of the metatarsophalangeal joint in racehorses begins in subchondral bone is essential to understanding clinical signs, successful use of diagnostic analgesia, progression of bone and cartilage loss, and the horse's response (or lack thereof) to therapy. There are two syndromes of metatarsophalangeal joint osteoarthritis; one is seen in racehorses and the other in non-racehorse sport horses.

In racehorses osteoarthritis begins as a non-adaptive stress-related bone injury in subchondral bone. Although the overlying cartilage may be biomechanically and biochemically inferior, obvious clinical signs such as synovitis and a positive response to lower limb flexion are not seen until later. Low-grade unilateral or bilateral lameness is present, which is localized best by low plantar or lateral plantar metatarsal analgesia. Early stress-related bone injury is best substantiated using scintigraphic examination. Focal areas of IRU are present in the MtIII; the PSBs and the proximal aspect of the proximal phalanx also can be affected (see Fig. 43-2). Initial radiographic findings are either negative or reveal subtle sclerosis, but later radiolucent defects develop in subchondral bone, which is best revealed in a DPrL-PlDiMO view (see Fig. 43-3). Sclerosis is most obvious in the LM, flexed LM, and DPrL-PlDiMO views.

Later, overlying full-thickness cartilage damage develops (see Figs. 43-8 and 43-9). Stress-related bone injury can continue to the point where severe osteoarthritis or fracture of the MtIII develops; this type of severe injury is most common in racehorses between 4 and 6 years old. However, in some STBs the process causes lameness when horses are 2- or early 3-year-olds and then subsides or stabilizes. The condition is rarely seen in STBs until training speeds faster than 2 minutes, 20 seconds for a mile are achieved. In TB racehorses, this remodeling process is common in later 2- and 3-year-olds but can also be seen in older horses. Older horses given time off for undiagnosed lameness or other conditions are at risk to develop stress-related bone injury in the metatarsophalangeal joint 6 to 8 weeks after returning to training, when signs of poor gait or lameness develop and a focal area of IRU can be identified. In my experience, subchondral lucency occurs later in the TB than in the STB and is uncommon. Differences between the racing breeds related to gait, speed, and load distribution may account for this difference in progression of osteoarthritis. End-stage osteoarthritis develops in STB racehorses that have continued racing for many years and can be progressive in broodmares and breeding stallions.

In non-racehorse sport horses osteoarthritis is an insidious process without marked subchondral bone involvement. Bone and cartilage gradually deteriorate. Some horses affected may be ex-racehorses, but most are not. I have observed osteo-

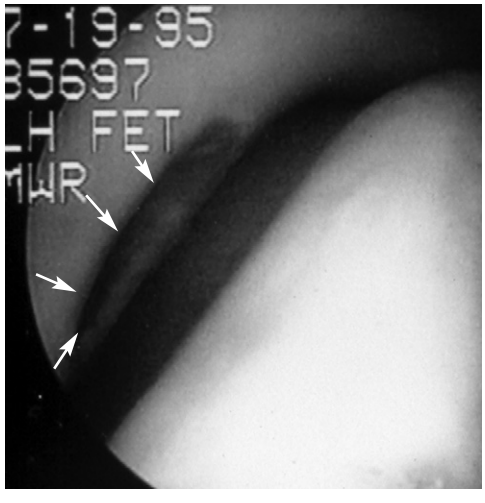

Fig. 43-8 Intraoperative photograph of a 5-year-old Standardbred trotter with severe stress-related bone injury of the distal plantarolateral aspect of the third metatarsal bone *(arrows)*. This area was debrided and the horse raced, but it dropped substantially in race class and was retired.

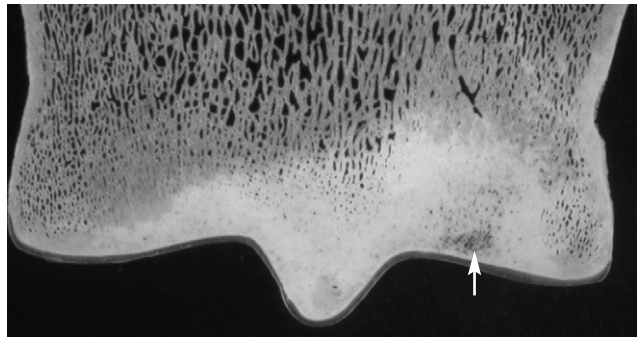

Fig. 43-9 Frontal plane microradiograph (100 μm) of the distal aspect of the third metatarsal bone (lateral is to the right) showing dense subchondral sclerosis and an area of intense resorption or necrosis of bone *(arrow)*. Cartilage overlying this area is intact. Severe subchondral modeling and remodeling and later osteoarthritis develop in the metatarsophalangeal joints of racehorses.

arthritis of the metatarsophalangeal joint mostly in upper-level jumpers and dressage horses. In these horses lameness is often chronic and low grade (grade 1 or 2 of 5), but it can progress to severe or intermittently severe. Effusion and a positive response to lower limb flexion are common findings. Concomitant tenosynovitis of the DFTS and previous lameness caused by suspensory desmitis and distal hock joint pain are common. Lameness is more consistently abolished using intra-articular analgesia in jumpers and dressage horses than in racehorses, but in some horses the low plantar block is required. Marginal osteophytes and entheseophytes may be detectable radiographically. Scintigraphy may reveal focal IRU in the dorsal aspect of the joint, diffuse IRU, or equivocal findings. Occasionally acute-onset lameness associated with focal IRU of the distal MtIII occurs that is similar to that found in racehorses. This lameness may indicate acute subchondral overload trauma from landing wrong after jumping a fence, stepping on uneven ground, or sustaining a fall or other accident in a paddock. In non-racehorse sport horses, arthroscopic examination often reveals cartilage damage most extensive in the dorsal aspect of the joint, marginal osteophytes on the apices of the PSBs, and occasionally osteochondral fragments of the proximal dorsal aspect of the proximal phalanx.

Shoeing and hoof balance may play a role in development or expression of clinical signs of osteoarthritis in all horses but may be more important in racehorses. In the STB racehorse clinical signs often develop within 2 to 3 weeks of a shoeing change, usually with the application of aluminum shoes with a low toe grab. Aluminum shoes are light and generally applied to pacers that are close to qualifying speed, but they are often used in trotters as well. Shear stress associated with these shoes may exacerbate existing disease or cause further trauma to subchondral bone. Aluminum shoeing is common in TB racehorses in North America, where these shoes are associated with other fetlock joint lameness and catastrophic breakdown. Abnormal hoof wear caused by gait, the presence of concomitant distal hock joint or metatarsal region pain, or hoof imbalance may cause asymmetrical loading of the metatarsophalangeal joint and add to subchondral bone trauma.

Management of Stress-Related Bone Injury and Osteoarthritis

Reducing bone stress is key to initial management in racehorses with early osteoarthritis characterized primarily by stress-related bone injury, because high-impact loading or high-strain cyclic fatigue of subchondral bone is the primary lesion. In STBs I previously recommended 4 to 6 weeks of light jogging (<2 miles per day), but results were poor even if recommendations were followed. I reassessed several horses after 6 weeks of light jogging, and although scintigraphic changes subsided, the horses were still as lame or slightly worse. I now recommend 3 weeks of walking and light jogging followed by re-evaluation. Although 3 weeks' reduction in training may allow healing or remodeling of damaged subchondral bone and microfractures, bone and muscle undergo detraining and horses need several (i.e., 3 to 6) weeks of slow return to normal exercise intensity. If lameness resolves, horses are returned to training, but if lameness persists I recommend 3 to 4 months of rest. In TBs, recurrence is quite high if horses are given only 6 to 8 weeks of rest. I recommend either 3 to 4 months of turn out or a program of 3 weeks of hand walking, followed by 3 weeks of walking with a rider up, followed by 3 weeks of trotting either with a rider up or using an exercise pony. A gradual return to normal exercise intensity is then recommended if horses are sound and moving well.

Hoof balance and shoeing characteristics should be evaluated and, in most instances, changed. A simple change in shoeing to an easier shoe, such as a flat steel or aluminum shoe, may reduce shear and torsion of the metatarsophalangeal joint. Toe grabs and any other shoe additives must be removed. If medial to lateral hoof imbalance is poor, it should be corrected.

Because the initial lesion is in subchondral bone, intra-articular injections appear to have limited therapeutic benefit but may make theoretical sense. Active treatment may help in persuading trainers to follow the recommended work program. A series of three intra-articular injections of polysulfated glycosaminoglycans (PSGAGs) are given every other week. Alternatively, if horses are currently racing but have mild unilateral or bilateral lameness or scintigraphic evidence of stress-related bone injury and poor performance, I recommend intra-articular injection with hyaluronan and methylprednisolone acetate (80 mg). Intramuscular administration of PSGAGs (once weekly for 8 weeks) is recommended, but therapeutic benefit is difficult to assess. NSAIDs such as phenylbutazone (2.2 to 4.4 mg/kg bid) are recommended. NSAIDs may help horses in modified exercise programs "get over the hump" when lameness persists after scintigraphic evidence of stress-related bone injury subsides. Because pathogenesis involves sclerotic subchondral bone, the roles of ischemia and increased intraosseous pressure in causing bone pain or necrosis and subsequent collapse of weakened areas of subchondral bone have been questioned. I used to recommend the administration of isoxsuprine (400 mg bid orally) in an attempt to improve peripheral blood flow. However, research evidence supports neither the achievement of adequate blood levels nor increased blood flow by use of the drug, and I do not currently recommend its use. Other drugs, such as aspirin (17 mg/kg bid orally), may be useful in improving blood flow, but half-life is short and therapeutic benefit has yet to be established. If increased intraosseous pressure and early ischemia lead to pain and subchondral bone damage, a procedure such as subchondral bone drilling, which is used to manage stress-related bone injury in cortical bone of the McIII, may be beneficial, but is currently only experimental. Topical counterirritation has historically been used and makes theoretical sense in horses with stress-related bone injury if increased blood flow occurs. The time-honored treatment of blistering and turning out may be the best method of management. We have used focused shock-wave therapy in some STB and TB racehorses with stress-related bone injury by aiming the shock waves at the plantarolateral aspect of MtIII (single treatment, 2000 shocks). Early results appear promising, but horses are given concurrent modified exercise programs and other therapy.

In racehorses with more advanced osteoarthritis the therapeutic value of intra-articular injections becomes greater, because overlying cartilage damage and synovitis become prominent. However, horses generally drop substantially in race class and eventually are retired. In TB racehorses, intra-articular injections have little benefit in horses in training but may have limited value in horses actively racing. The disease process appears to be self-limiting in TBs, because horses with continued chronic, low-grade lameness are often retired before the disease process advances or horses develop compensatory lameness in the forelimbs. In the TB racehorse subchondral radiolucency is unusual in my experience, but others have recognized progression of osteoarthritis and the development of changes similar to those seen in STBs (see Chapter 109). Why some horses are able to race with persistence of IRU, radiolucency, and sclerosis, whereas others cannot, is not easy to answer. Prognosis for both racing breeds appears to be guarded to fair at best. Of 19 STBs with lameness and IRU of the plantarolateral aspect of the MtIII, 18 raced, but only 13 remained at the same racing class or improved.[5] When radiolucent areas become prominent, arthroscopic examination and debridement may be an option, but it is difficult to manipulate instruments in the distal plantar pouch, because manipulation must be done with the joint in flexion (see Fig. 43-8). It is almost

impossible to reach these lesions using a dorsal approach, because they are located approximately 8 mm plantar to the middle of the condyle. In horses with end-stage osteoarthritis (i.e., loss of joint space, tilting of the sagittal ridge and groove, and severe proliferative and radiolucent changes), arthrodesis must be considered in broodmare or stallion prospects. Humane destruction may be required.

Non-racehorses often respond favorably to intra-articular injections of PSGAGs, hyaluronan, and short-acting corticosteroids (e.g., triamcinolone acetonide, isoflupredone acetate) or long-acting corticosteroids (e.g., methylprednisolone acetate). Exercise level is reduced for a period of 2 to 3 weeks and horses are given NSAIDs. Topical therapy using cold water hosing, poulticing, and bandaging is recommended. When lameness persists and scintigraphic examination reveals focal IRU in the dorsal aspect of the joint, or in horses with chronic lameness, long-term rest is necessary. Arthroscopic examination in these horses reveals areas of partial- or full-thickness cartilage damage that can be managed by debridement and microfracture into subchondral bone. However, the therapeutic value of arthroscopy is limited. If osteophytes are removed, they generally reform. If surgery is combined with rest, horses usually become sound, but lameness often returns when competition ensues. Prognosis for horses performing at the previous level of competition is guarded to poor. Rehabilitation of upper-level performance horses is quite difficult, because most have at least one compensatory lameness problem in addition to primary osteoarthritis of the metatarsophalangeal joint. Horses may be able to sustain a lower level of competition.

Plantar Process Osteochondral Fragments

Fragmentation of the proximal plantar processes of the proximal phalanx is a common finding in young STBs and Warmbloods but is also seen in TBs, Arabians, and Western performance horses. This condition is more frequently recognized since pre-sale and post-sale radiography has become common.

There are four distinct manifestations of decreasing frequency: axial articular fragments: abaxial, nonarticular fragments; nonarticular fragments originating from the base of the PSBs; and true acute fractures. (I prefer an anatomical description rather than reference to different types.[7]) The condition is much more common in the hindlimbs, except for non-articular fragments from the base of the PSBs, which occur almost exclusively in forelimbs (see Chapter 37). Combinations of axial and abaxial fragments often occur. The condition can be unilateral or bilateral and can be biaxial (involving both sides of the same joint).

Axial Articular Fragments

Axial articular fragments are most important and are most often medial, but can be lateral, biaxial, and bilateral. Of 119 horses with axial articular fragments, 92% were STBs; 95% of fragments were in the hindlimbs, most commonly the medial aspect of the left hindlimb (44%).[8] The incidence of axial articular fragments in 1- and 2-year-old STBs ranges from 5.6% to 28.8% but in TBs has been estimated at 2%.[9] The etiology remains controversial, but because STBs and the hindlimbs are clearly predisposed and fragments are recognized at an early age, osteochondrosis is the most likely explanation. Heritability estimates of axial articular fragments in STBs using a non-linear model were 0.21, and fragments were seen in 11.8% of foals.[10] I feel it is implausible that a large number of STB foals develop traumatic fractures at this site, whereas other breeds do not. I believe hereditary factors are most important. A traumatic etiology was suggested, because portions of the fragments were irregular, entrapped by mature fibrous tissue, and contained spicules of bone not covered by fibrocartilage, which gave the histological appearance of a fracture healed by fibrous union.[9,11,12] Evidence of short distal sesamoidean ligament insertions was not found and fragments were old.[9] Unfortunately, axial articular fragments examined were taken from horses with a mean age of 3.4 years and after horses had trained and raced, rather than from weanlings and yearlings, so degeneration and remodeling of fragments could have occurred. With only capsular attachments, how do alleged avulsion fractures occur? Etiology is most important when dispute or arbitration ensues after horses are sold at public auction (see Chapter 102).

There is chronic low-grade, high-speed metatarsophalangeal joint lameness. Horses often have a previous history of other ipsilateral hindlimb lameness, such as distal hock joint pain. Effusion and a positive response to lower limb flexion are lacking or inconsistent. Lameness is alleviated most consistently with low plantar analgesia, but differentiation from early stress-related bone injury and osteoarthritis can be difficult without scintigraphic examination. Stress-related bone injury or other bony abnormalities, such as mid-sagittal fracture of the proximal phalanx and occasionally condylar fractures of the MtIII, are commonly found simultaneously with axial articular fragments. Many horses with axial articular fragments never develop lameness and fragments are only detected on survey radiographs. If and how axial articular fragments cause lameness is not well understood. Axial articular fragments may vibrate at speed or become interposed in the joint surface during flexion, but synovitis does not play a role. These fragments may affect mechanics of the ipsilateral PSB or impinge on distal sesamoidean ligaments.[7] Nociceptive fibers were found in soft tissue attachments and stretching during full extension may cause pain.[9] In older horses, evidence of full-thickness cartilage damage on the MtIII and the PSBs suggests axial articular fragments may contribute to the development of osteoarthritis.

Radiographic examination reveals one or more fragments seen best in down-angled oblique views (see Fig. 43-1). If surgery is considered, both limbs should be examined radiographically. Scintigraphic examination may reveal mild abnormal bone modeling, but IRU is more commonly seen in horses with abaxial, nonarticular fragments (see Fig. 43-6).

Axial articular fragments should be removed using arthroscopic surgery. In recent years I have advised removal of fragments before horses begin training, because "down time" associated with arthroscopic surgery is less than if surgery is performed in the middle of a racing year. Subjectively it appears that horses that undergo surgery earlier have fewer problems with stress-related bone injury and osteoarthritis. In breeds such as the TB and Warmblood axial articular fragments may complicate sales and can be removed prophylactically. During arthroscopic surgery fragments are carefully trimmed free of capsular attachments using a specially designed intra-articular blade and removed.[13] If arthroscopic surgery is performed before training begins, horses are given 7 days of stall rest, followed by 7 days of stall rest with hand walking, then 7 to 14 days of walking in the jog cart (STBs) or walking and light trotting with an exercise pony (TB). If lameness develops when horses are in advanced stages of training or are racing, I recommend at least 2 to 3 months of rest before training resumes. The prognosis is good for future soundness if the fragment(s) caused the lameness. Sixty-three percent of racehorses and 100% of non-racehorses had performance similar to pre-injury levels after surgery, but the presence of cartilage damage or synovial proliferation was significantly associated with an adverse outcome.[7]

Abaxial, Non-Articular Fragments

Abaxial, non-articular fragments can be single or multiple and may occur in combination with axial articular fragments, either on the same side or opposite side of the joint. Abaxial, non-articular fragments usually are lateral and can be bilateral. Abaxial, non-articular fragments can be large and involve the

entire lateral, plantar process, including a small articular portion, but most are non-articular. These fragments resemble old fractures radiographically, but I believe they are manifestations of osteochondrosis.[11,14] Fragments are often seen in weanlings or yearlings without clinical signs of lameness, and when seen in older horses, lameness is rarely acute. Abaxial, non-articular fragments have been described as ununited proximoplantar tuberosities, and in some horses fragments identified at a young age subsequently reunited with the parent bone.[14] In my experience union of abaxial, non-articular fragments in older horses is rare.

Lameness is mild (grade 1 of 5 with the horse in hand), usually present at high speed, and worse around turns. Pain is abolished by low plantar analgesia. Palpation may reveal mild bony enlargement, but this enlargement is easily missed. Response to lower limb flexion is inconsistent. Occasionally a weanling or yearling develops acute lameness with obvious soft tissue swelling that involves the fetlock joint. Radiographs reveal an old abaxial, non-articular fragment, but generally lameness resolves quickly and is not related to the fragment.

Radiographs reveal one or more abaxial, non-articular fragments, which are occasionally accompanied by axial articular fragments (Fig. 43-10). Fragments are old and rounded, and radiolucent changes of the parent plantar process are common. A clear separation exists between the fragment and proximal phalanx that is filled by dense, fibrous tissue. Scintigraphic examination may reveal mild IRU.

Abaxial, non-articular fragments may or may not cause lameness and clear differentiation between sources of pain can be difficult. The decision of whether to perform surgery is often difficult, because conventional surgery is required to remove these non-articular pieces.[13] After surgery horses are given 4 weeks of stall rest, followed by 4 weeks of stall rest with hand walking, then 4 to 8 weeks of turn out in a small paddock or light jogging or galloping. Prognosis is excellent for future soundness, but within STB racehorses, pacers have a better prognosis than trotters. In yearlings with a combination of axial articular fragments and abaxial, non-articular fragments, I usually recommend arthroscopic surgery to remove axial articular fragments and leave abaxial, non-articular fragments in place unless clinical signs develop at a later date.

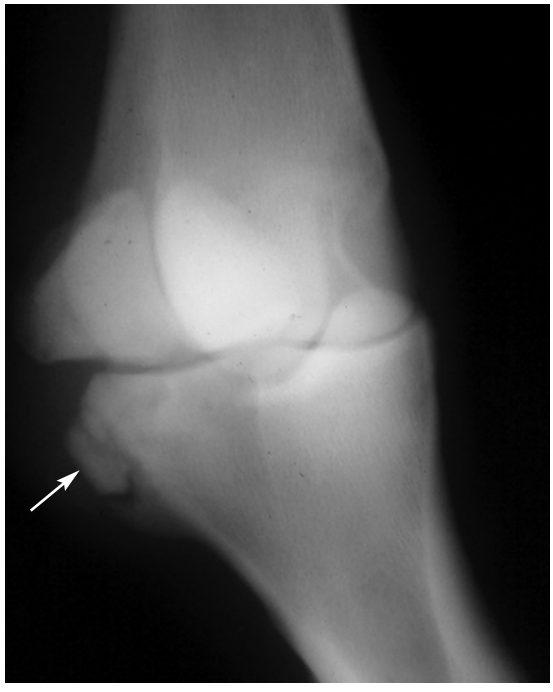

Fig. 43-10 Dorsolateral-plantaromedial radiographic view showing a large abaxial, non-articular fragment *(arrow)* of the proximal plantar process of the proximal phalanx. In this 3-year-old Standardbred racehorse lameness was not evident.

Although I strongly believe that axial articular fragments and abaxial, non-articular fragments cause lameness and I recommend surgery both therapeutically and prophylactically, others question the significance of these fragments. In a study evaluating radiographic findings in STBs before beginning training and subsequent race performance, there was no significant association between the presence of axial articular fragments, abaxial, non-articular fragments, and other forms of osteochondrosis and either race performance or racing longevity.[15]

Acute Fractures of the Proximal Plantar Process

True fractures of the proximal plantar process of the proximal phalanx occur, usually after known or suspected trauma, but are rare. Acute lameness and soft tissue swelling are present. Radiography reveals an acute fracture with a narrow, well-defined fracture line. The margins of the fracture are not rounded, nor are there radiolucent changes in the proximal phalanx. Internal fixation and fiberglass cast application is recommended. Prognosis depends on duration of the fracture before repair and fracture size (see Fig. 36-2). Osteoarthritis is a potential complication.

Osteochondrosis of the Sagittal Ridge of the Third Metatarsal Bone

A discussion of osteochondrosis of the sagittal ridge of the third metatarsal bone is presented in Chapter 58.

Osteochondral Fragments of the Dorsoproximal Aspect of the Proximal Phalanx

Osteochondral fragments of the dorsoproximal aspect of the proximal phalanx are common and occur in two forms. Well-rounded osteochondral fragments often occur in young horses, may be a manifestation of osteochondrosis, and are asymptomatic. Acute osteochondral fragments result in mild lameness or change in performance and are associated with an effusion and a positive response to lower limb flexion. Arthroscopic surgery and removal of these osteochondral fragments is recommended. In older STB and TB racehorses, osteochondral fragments can be large and multiple, and after surgery a minimum of 8 weeks' rest is given (Fig. 43-11). Cartilage damage on the opposing surface of the MtIII is common, and the amount is commensurate with size and duration of osteochondral fragments.

Other Osteochondral Fragments

Osteochondral fragments may be identified dorsal or plantar to the collateral ligaments, usually laterally. These osteochondral fragments are usually traumatically induced and may originate from the distal dorsal aspect of the MtIII, but they are not avulsion fractures. Proliferative changes often develop. Treatment is by surgical removal, but prognosis is guarded in racehorses.

A rare finding in the metatarsophalangeal joint is a loose fragment located in the distal aspect of the plantar pouch. In some horses, small defects in the dorsal sagittal ridge can be seen radiographically. A small fragment may have detached and migrated to the plantar pouch. Large, loose fragments may exist for some time in the plantar pouch without causing lameness.

Fractures of the Proximal Phalanx

The most common major fracture in the metatarsophalangeal joint is mid-sagittal fracture of the proximal phalanx or other fractures with a mid-sagittal component. These fractures occur most often in racehorses but also occur in other sport

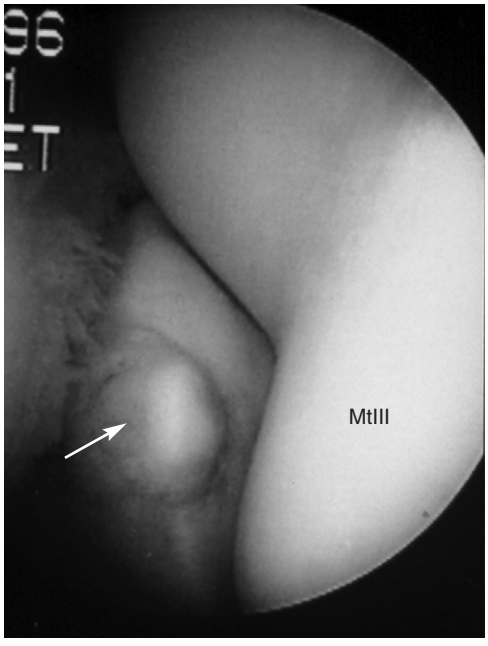

Fig. 43-11 Intraoperative photograph of an osteochondral fragment *(arrow)* of the proximal dorsomedial aspect of the proximal phalanx. *MtIII*, Third metatarsal bone.

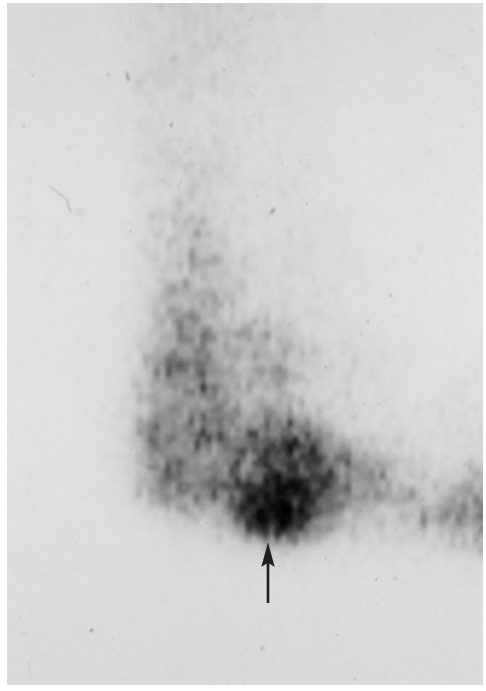

Fig. 43-12 Delayed (bone) phase flexed lateral scintigraphic image of a metatarsophalangeal joint showing focal increased radiopharmaceutical uptake associated with the proximal dorsal aspect of the proximal phalanx *(arrow)* in a Standardbred with dorsal frontal fracture of this bone. Mild increased radiopharmaceutical uptake was seen in the contralateral hindlimb.

horses. In racehorses, mid-sagittal fractures of the proximal phalanx occur in STBs equally in hindlimbs and forelimbs, but in TBs, forelimb fractures are most common. These fractures are discussed in Chapters 36 and 37.

Dorsal Frontal Fractures of the Proximal Phalanx

Dorsal frontal fractures of the proximal phalanx occur exclusively in hindlimbs, although large osteochondral fragments in the forelimb may cause similar clinical signs. They occur in both TB and STB racehorses. Although these fractures are reported to be more common in TBs,[16] in my experience the fracture is more common in STBs and can be unilateral or bilateral. If a dorsal frontal fracture of the proximal phalanx is diagnosed scintigraphically, contralateral IRU of the dorsal proximal aspect of the proximal phalanx is commonly seen (Fig. 43-12). IRU associated with a dorsal frontal fracture of the proximal phalanx must be differentiated from a mid-sagittal fracture. Mid-sagittal fractures are single-event injuries, but a dorsal frontal fracture of the proximal phalanx may be caused by stress-related bone injury, because gradations of IRU are seen in the proximal phalanx and concomitant contralateral IRU occurs. Although bilateral fracture or evidence of stress-related bone injury can be seen bilaterally simultaneously, horses may develop contralateral fracture the next year. TB racehorses can tolerate pain and race with this fracture; there may be chronic proliferative changes and a displaced fracture in a horse that raced within the last 10 days. Displaced fractures do not occur in STBs, and pacers are most likely to have an obvious fracture, whereas fractures in trotters may be scintigraphically active with only subtle radiographic changes.

A dorsal frontal fracture of the proximal phalanx usually results in intermittent, severe lameness, but bilateral fractures may cause poor performance without causing obvious signs of lameness. Effusion is present but may be only mild unless fractures are displaced. The response to lower limb flexion is positive. Intra-articular or low plantar analgesia abolishes lameness in horses with complete or displaced fractures, but the low plantar technique should be used in those with stress-related bone injury or incomplete fractures. Radiographs reveal a single fracture line that is best recognized in LM or flexed LM views. Fractures begin at the articular surface and course in a dorsodistal direction (Fig. 43-13). In TBs with displaced fractures an osteochondral fragment often is interposed between the main fracture fragment and the parent bone.

If fractures are non-displaced, conservative management provides a good to excellent prognosis in both TBs and STBs. Horses are given 4 weeks of stall rest, followed by 4 weeks of stall rest with hand walking, then 4 to 8 weeks of turn out in a small paddock. Two treatment options are available for horses with displaced fractures. In those with large fragments and interposed osteochondral fragments, arthroscopic surgery is recommended to remove small osteochondral fragments and to repair the dorsal frontal fracture using one or two 3.5-mm screws. If the fracture cannot be reduced or is broken or small, the fragment should be removed. Prognosis is good to excellent for future racing, but horses may drop in class, particularly those with compensatory or numerous lameness problems.

Fractures of the Proximal Sesamoid Bones

Overall, fractures of the PSBs are less common than in the metacarpophalangeal joint, but in STB racehorses there is nearly equal distribution of fractures. Apical fractures of the PSBs are more common in the hindlimb of STBs and most often involve the lateral PSB. Fractures of the PSBs can be traumatic or pathological. Young racehorses develop acute lameness and fracture without pre-existing disease of the PSB or SL. However, the PSB undergoes extensive remodeling similar to the MtIII, and it is possible, perhaps even likely, that these fractures represent a form of stress-related bone injury. In older racehorses and in some non-racehorse sport horses there is pre-existing lucent and proliferative sesamoiditis and suspensory branch desmitis, and fractures of the PSB appear to develop as a pathological, secondary, end-stage injury. In some non-racehorse sport horses fracture occurs as a single

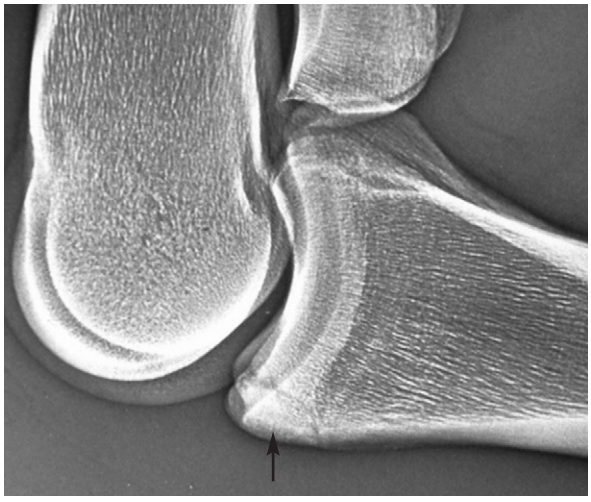

Fig. 43-13 Flexed lateromedial xeroradiograph showing a typical dorsal frontal fracture *(arrow)* in a Standardbred. In the Thoroughbred, fractures are often displaced and have chronic proliferative changes, which indicate that horses have been training or racing with the injury.

event without pre-existing injury. Prognosis depends on the size of the fracture fragment and the degree of suspensory desmitis. In STB racehorses, prognosis is better in pacers than in trotters, and prognosis is poor in horses developing fractures early in training before racing begins (see Chapter 37). The prognosis is good in non-racehorse sports horses without pre-existing injury.

Fractures of the Distal Third Metatarsal Bone

Condylar fractures of the distal MtIII occur less frequently than those of the McIII (see Chapter 37). In a recent study of 135 horses with condylar fractures, 81% and 85% involved the forelimbs and lateral condyles, respectively.[17] Lateral and medial condylar fractures of the MtIII occur, but lateral fractures are most common. Lateral condylar fractures can be short and incomplete, long and incomplete, or long and complete. Bilateral fractures occasionally occur simultaneously. Condylar fractures are primarily racing injuries. In a recent study of TB, STB, and Arabian racehorses, TBs were over-represented and STBs under-represented; TBs had significantly more lateral and forelimb fractures than STBs.[18] Clinical signs are commensurate with the length and degree of displacement. Medial condylar fractures can be short, incomplete, and difficult to diagnose. However, most commonly lameness is severe and radiographs reveal that the fracture is long and incomplete but goes straight proximally to end abruptly in the diaphysis, forms a Y-shaped pattern, or spirals proximally to end near or at the tarsometatarsal joint. Occult fissure lines and spiraling should always be suspected, and it is critical to know the location of the fracture before horses are transported and prognosis is discussed. Horses with medial condylar fractures are at extreme risk for fracture propagation or comminution. Even after surgical repair, horses are at risk during anesthetic recovery and early in the postoperative period. Of 15 horses with medial condylar fractures of the MtIII, 12 were repaired, 2 suffered catastrophic fracture during recovery from general anesthesia, and 3 developed complete fracture within 4 days after surgery.[19] Recovery from anesthesia should be assisted and may best be performed using a pool recovery system.

Horses with short, incomplete lateral and medial condylar fractures can be managed conservatively or undergo surgical repair. Those with longer spiral or medial condylar fractures with a Y shape should undergo surgical repair. This repair can be accomplished by use of a combination of open observation of the fracture line and either screw fixation or a combination of screws and a dynamic compression plate (Fig. 43-14). Prognosis depends on whether fractures are medial or lateral, displaced or non-displaced, and whether a plate or numerous screws are used for repair. If fractures heal and plates (if used) are removed, prognosis for racing is favorable, but horses will drop in class.

Sesamoiditis

In the STB hindlimb sesamoiditis is most common in the lateral PSB but can occur medially and be a source of occult metatarsophalangeal joint lameness. In TBs sesamoiditis is most commonly observed in the forelimb. TBs can have obvious radiolucent lines in the PSBs in hindlimbs that are often asymptomatic. Disparity between limbs, rather than size or number of radiolucent lines, was found to be most important in determining clinical significance in TBs; horses with three or more channels were less likely to race as 2-year-olds and had decreased earnings per start.[20] In the STB racehorse the presence of one to three or more radiolucent defects less than or equal to 1 mm in width was not associated with lameness referable to the PSBs, but horses with wide, abnormally shaped defects developed lameness from sesamoiditis.[21] Conversely, of 753 young STBs that underwent radiographic examination before training, 21 horses had severe sesamoiditis, fractures, or enlarged PSBs, but a correlation between sesamoid pathology and lameness or decreased racing performance and earnings could not be made. In that study hindlimb PSBs were affected in only 6 horses and lameness developed in 14 horses.[22] Although lameness could not be directly attributed to changes in the PSBs, in some horses metatarsophalangeal joint lameness was diagnosed and perhaps compensatory lameness developed as a result of chronic, low-grade or high-speed lameness in these trotters. Seven of 21 horses had concomitant osteochondral fragments of the plantar palmar process of the proximal phalanx, although a clear relationship could not be established.[22]

Radiolucent Defects of the Axial Border of the Proximal Sesamoid Bones

See Chapters 73 and 75 for a discussion of radiolucent defects of the axial border of the proximal sesamoid bones.

Flexor Deformity of the Metatarsophalangeal Joint

Dorsal subluxation of the metatarsophalangeal joint (buckle or knuckle forward) occurs in horses with flexor deformity. Intermittent subluxation occurs in those with mild deformity, but some horses with more severe deformity maintain the metatarsophalangeal joint in partial flexion. Horses with chronic, severe tenosynovitis of the DFTS with extensive adhesions may develop progressive, severe flexor deformity. Desmitis of the accessory ligament of the DDFT may cause flexor deformity. In some horses, application of an extremely elevated heel shoe (e.g., 5 to 8 cm) may help alleviate pain (see Chapter 61).

Dropped fetlock or plantar subluxation (hyperextension) occurs in horses with severe chronic suspensory desmitis or those with severe, acute tearing of the superficial digital flexor tendon (SDFT), the DDFT, or both the SDFT and DDFT.

Soft Tissue Injuries of the Metatarsophalangeal Joint and Fetlock Region

Proliferative Synovitis

Proliferative synovitis (i.e., enlarged synovial pad or villonodular synovitis) occurs in the dorsal aspect of the metatarsophalangeal joint in horses with chronic osteoarthritis. However, it is much less common than in the metacarpophalangeal joint and is a rare primary cause of pain (see Chapter 37).

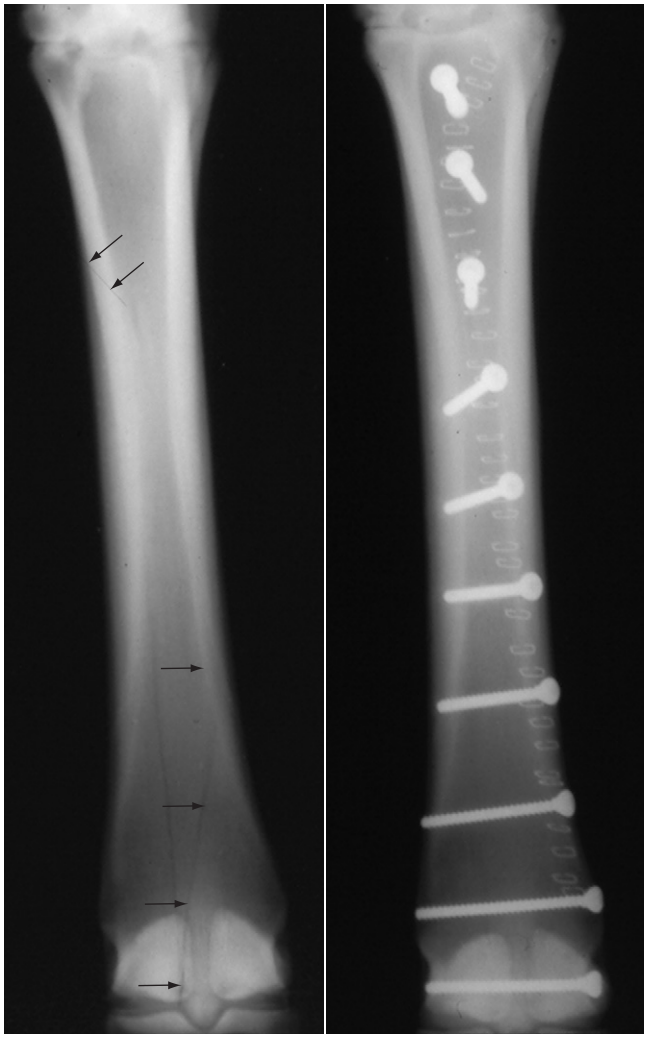

Fig. 43-14 Dorsoplantar radiographic views of the metatarsophalangeal joint and the entire metatarsal region. There is a spiral fracture of the third metatarsal bone *(arrows)* beginning at the medial condyle of the third metatarsal bone (medial is the left) in the metatarsophalangeal joint and extending to near the metatarsophalangeal joint (confirmed at surgery after subperiosteal dissection) before **(A)** and after **(B)** surgery.

Luxation and Subluxation: Tearing of the Collateral Ligament

Complete disruption of a collateral ligament causes luxation and results in severe, non–weight-bearing lameness and usually obvious valgus or varus deformity. This injury is more common in adult horses, because physeal fractures of the distal MtIII and proximal phalanx are more likely in foals. Initially little swelling occurs, but within a few hours soft tissue swelling can be prominent and manipulation of the joint reveals instability. Diagnosis is confirmed radiographically using DPl and stressed DPl views. Radiographs should be examined carefully for the presence of concurrent fractures on the contralateral or plantar aspects of the joint. Ultrasonography is used to identify the injured portion of the collateral ligament and extent of injury. After manual reduction with the horse under general anesthesia a fiberglass cast should be applied. The cast should be changed within 7 to 10 days, because swelling resolves and the cast loosens. Cast coaptation should be maintained for 8 to 10 weeks and then a heavy bandage should be applied. If crushing of the opposite side or plantar aspect of the joint did not occur, prognosis for soundness is guarded to fair. Osteoarthritis and chronic desmitis of the collateral ligament are possible.

Collateral Desmitis

Primary injury of the collateral ligament without luxation is rare in my experience. Clinical signs include firm soft tissue swelling and ultrasonographic evidence of desmitis in horses with lameness that is abolished by low plantar analgesia. Avulsion fractures may occur as a result of trauma.

Suspensory Desmitis

See Chapter 73 for a discussion of suspensory desmitis.

Tenosynovitis of the Digital Flexor Tendon Sheath

See Chapter 75 for a discussion of tenosynovitis of the digital flexor tendon sheath.

Deep Digital Flexor Tendonitis

See Chapters 71 and 75 for a discussion of deep digital flexor tendonitis.

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CHAPTER • 44

The Metatarsal Region

Mike W. Ross

The metatarsal region, like most of the distal hindlimb, has received little attention in previous lameness textbooks. However, it is a common source of lameness problems and should not be overlooked, especially in view of the high incidence of suspensory desmitis in sport horses.

ANATOMY

The metatarsal region is bordered by the tarsometatarsal joint proximally and the metatarsophalangeal joint distally. The large third metatarsal bone (MtIII) provides all structural support and weight bearing and articulates predominantly with the third tarsal bone proximally and the proximal phalanx and proximal sesamoid bones (PSBs) distally. A prominent nutrient foramen in the MtIII should not be mistaken for a fracture and is usually located slightly higher than that of the third metacarpal bone (McIII).[1]

The second metatarsal bone (MtII) and fourth metatarsal bone (MtIV) are commonly referred to as the medial and lateral splint bones, respectively. The MtII articulates with the combined first and second tarsal bones proximally and ends distally with an enlarged "bell." The MtIV articulates with the fourth tarsal bone proximally but transmits less load than the MtII. The dorsal metatarsal artery runs obliquely, in a disto-plantar direction, in the proximal lateral aspect of the metatarsal region and then parallel and close to the dorsal aspect of the MtIV as it courses distally in the mid-metatarsal region. The dorsal metatarsal artery then courses deep to the MtIV and must be avoided during distal splint ostectomy. In one horse the dorsal metatarsal artery coursed through a bony and fibrous ring-like foramen in the distal MtIV and was inadvertently severed during ostectomy of the MtIV. Because the dorsal metatarsal artery is superficial, it can be lacerated from wounds in the lateral metatarsal region. Bleeding can be profound and the ends of a lacerated dorsal metatarsal artery should be ligated unless anastomosis is performed with the horse under general anesthesia. Collateral circulation develops if ligation is necessary. The long plantar ligament attaches to the proximal aspect of the MtIV.

The orientation of the metatarsal bones is clinically important. The proximal aspect of the MtIV is large and is located in a more plantar location than its counterpart, the fourth metacarpal bone (McIV). This orientation makes it impossible to palpate the normal proximal and mid-body portions of the suspensory ligament (SL). Therefore unless it is grossly enlarged, the SL cannot be palpated and conditions such as mild to moderate suspensory desmitis can easily be missed, particularly if lameness is chronic and signs of acute inflammation are not present.

Dense metatarsal fascia attaches to the abaxial margins of the MtII and IV and encircles the SL, deep digital flexor tendon (DDFT), and superficial digital flexor tendon (SDFT). Swelling of the SL within this dense fascial compartment can cause pain by compression of the adjacent metatarsal nerves.[2] The SL has a broad origin on the proximal, plantar surface of the MtIII and a small attachment to the distal tarsal bones. The hind SL is slightly longer and thinner than the front SL, and muscle content may be higher in hindlimbs compared with forelimbs, which can complicate ultrasonographic examination.[3] It was suggested that the higher incidence of suspensory desmitis in Standardbreds (STBs) than in Thoroughbreds (TBs) was related to higher muscle content, but this is unproven.[3]

The plantar surface of the MtIII and origin of SL are oblique and not parallel to the dorsal surface of MtIII. During ultrasonographic examination, care should be taken to account for obliquity and position the transducer perpendicular to SL fibers (see Chapter 73). The SL is bordered by the MtII and MtIV and is thus trapped within a bony and dense soft tissue encasement. In the mid-metatarsal region the body of the SL can be palpated as it emerges from this bony encasement and courses distally to divide into the medial and lateral branches that attach to the abaxial border of each PSB. The accessory ligament of the DDFT (ALDDFT) is thin and inconsistent. Although the ALDDFT is anatomically present in most horses, desmitis is rare (see Chapter 72). Desmotomy

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ANATOMY

The metatarsal region is bordered by the tarsometatarsal joint proximally and the metatarsophalangeal joint distally. The large third metatarsal bone (MtIII) provides all structural support and weight bearing and articulates predominantly with the third tarsal bone proximally and the proximal phalanx and proximal sesamoid bones (PSBs) distally. A prominent nutrient foramen in the MtIII should not be mistaken for a fracture and is usually located slightly higher than that of the third metacarpal bone (McIII).[1]

The second metatarsal bone (MtII) and fourth metatarsal bone (MtIV) are commonly referred to as the medial and lateral splint bones, respectively. The MtII articulates with the combined first and second tarsal bones proximally and ends distally with an enlarged "bell." The MtIV articulates with the fourth tarsal bone proximally but transmits less load than the MtII. The dorsal metatarsal artery runs obliquely, in a disto-plantar direction, in the proximal lateral aspect of the metatarsal region and then parallel and close to the dorsal aspect of the MtIV as it courses distally in the mid-metatarsal region. The dorsal metatarsal artery then courses deep to the MtIV and must be avoided during distal splint ostectomy. In one horse the dorsal metatarsal artery coursed through a bony and fibrous ring-like foramen in the distal MtIV and was inadvertently severed during ostectomy of the MtIV. Because the dorsal metatarsal artery is superficial, it can be lacerated from wounds in the lateral metatarsal region. Bleeding can be profound and the ends of a lacerated dorsal metatarsal artery should be ligated unless anastomosis is performed with the horse under general anesthesia. Collateral circulation develops if ligation is necessary. The long plantar ligament attaches to the proximal aspect of the MtIV.

The orientation of the metatarsal bones is clinically important. The proximal aspect of the MtIV is large and is located in a more plantar location than its counterpart, the fourth metacarpal bone (McIV). This orientation makes it impossible to palpate the normal proximal and mid-body portions of the suspensory ligament (SL). Therefore unless it is grossly enlarged, the SL cannot be palpated and conditions such as mild to moderate suspensory desmitis can easily be missed, particularly if lameness is chronic and signs of acute inflammation are not present.

Dense metatarsal fascia attaches to the abaxial margins of the MtII and IV and encircles the SL, deep digital flexor tendon (DDFT), and superficial digital flexor tendon (SDFT). Swelling of the SL within this dense fascial compartment can cause pain by compression of the adjacent metatarsal nerves.[2] The SL has a broad origin on the proximal, plantar surface of the MtIII and a small attachment to the distal tarsal bones. The hind SL is slightly longer and thinner than the front SL, and muscle content may be higher in hindlimbs compared with forelimbs, which can complicate ultrasonographic examination.[3] It was suggested that the higher incidence of suspensory desmitis in Standardbreds (STBs) than in Thoroughbreds (TBs) was related to higher muscle content, but this is unproven.[3]

The plantar surface of the MtIII and origin of SL are oblique and not parallel to the dorsal surface of MtIII. During ultrasonographic examination, care should be taken to account for obliquity and position the transducer perpendicular to SL fibers (see Chapter 73). The SL is bordered by the MtII and MtIV and is thus trapped within a bony and dense soft tissue encasement. In the mid-metatarsal region the body of the SL can be palpated as it emerges from this bony encasement and courses distally to divide into the medial and lateral branches that attach to the abaxial border of each PSB. The accessory ligament of the DDFT (ALDDFT) is thin and inconsistent. Although the ALDDFT is anatomically present in most horses, desmitis is rare (see Chapter 72). Desmotomy

to manage flexor deformity is not performed and would be ineffective because of the small size of the ALDDFT. The DDFT is located plantar to the SL for most of the metatarsal region, but in the proximal metatarsal region it is positioned plantaromedial. The SDFT is located plantar to both the SL and DDFT. Because the tarsal sheath ends in the proximal metatarsal region, distention can sometimes be palpated. The digital flexor tendon sheath (DFTS) begins in the plantar, distal third of the metatarsal region and encompasses the DDFT and SDFT (see Chapter 75).

In the proximal third of the metatarsal region the lateral digital extensor tendon joins with the long digital extensor tendon and the combined tendons course distally. The fibularis tertius and cranialis tibialis attach to the proximal, dorsal, and medial aspects of the MtIII. Proliferation and fracture can occur in these locations.

Distal continuation of the superficial fibular and saphenous nerves provides dorsal skin sensation. The tibial nerve divides to form the medial and lateral plantar nerves in the plantar tarsal region. The lateral plantar nerve, which courses plantar to the origin of the SL, gives off a deep branch that innervates the proximal aspect of the SL. Lateral plantar neurectomy performed just above this branch provides analgesia of the origin of the SL. The lateral plantar nerve gives off the medial and lateral plantar metatarsal nerves that run axial to each respective splint bone. The lateral plantar nerve and its counterpart, the medial plantar nerve, continue distally between the SL and DDFT.

Conformation

Sickle-hock, but most importantly straight hindlimb conformation predispose to metatarsal region lameness (see Chapter 4).

CLINICAL CHARACTERISTICS AND DIAGNOSIS OF LAMENESS IN THE METATARSAL REGION

There are no pathognomonic historical findings related to metatarsal region lameness. Palpation of the metatarsal region should be done with the horse in standing and non–weight-bearing positions. The metatarsal region cannot be adequately palpated when the horse is standing, because soft tissue structures in the proximal metatarsal region are not readily palpable in this position. Careful palpation for signs of inflammation and bony swelling should be performed with the limb in both positions. Even when the limb is flexed, it is still difficult to define the SL and DDFT proximally. However, it is possible to apply pressure over the proximal SL to check for a painful response. In some horses with proximal suspensory desmitis (PSD), edema may be present or the area feels full or slightly thickened, but dense fascia covering the SL and DDFT prevents expansion and swelling may go undetected. A painful response to deep palpation over the proximal medial splint bone and distal tarsus, referred to as the Churchill test, may indicate the presence of referred pain and primary distal hock joint lameness, but in my experience this is a non-specific test. Many horses with hindlimb lameness originating from sites other than the distal hock joints manifest a positive response to compression of both the medial and lateral splint bones. Palpation of the dorsal cortex of the MtIII may reveal pain in trotters or other horses that interfere and may be an important finding, but palpation may not necessarily localize the primary source of pain that is causing lameness. Careful palpation of the abaxial and axial aspects of both splint bones is necessary to uncover hidden splint exostoses. Even subtle enlargement of the SL body, as it emerges from the bony encasement of the splint bones, that is accompanied by a painful response can indicate early suspensory desmitis. Both branches of the SL and attachments to the PSBs should be carefully palpated. Horses with a history of curb may develop progressive SDF tendonitis in the proximal metatarsal region, but this can easily be overlooked unless the area is carefully palpated (see Chapters 6, 70, and 79).

Diagnostic Analgesia

The high plantar perineural block (see Chapter 10) should be used to localize pain to the metatarsal region. Plantar metatarsal nerves are blocked just distal (approximately 1.5 cm) to the tarsometatarsal joint (see Chapter 10).

Imaging Considerations

Routine radiographic examination includes the dorsoplantar (DPl), lateromedial (LM), dorsolateral-plantaromedial oblique (DL-PlMO), and dorsomedial-plantarolateral oblique (DM-PlLO) views. In a DPl view of the proximal MtIII, there is a normal area of mild sclerosis that should not be interpreted as modeling that is associated with stress reaction of the origin of the SL (Fig. 44-1). Accurate assessment of the proximal aspect of the MtIII for the presence of sclerosis or avulsion fracture requires that radiographic views be centered at this level.

Pool and delayed (bone) phase scintigraphic examination is quite useful to differentiate bone from soft tissue injury in horses with proximal plantar metatarsal pain and osteoarthritis. It is also useful to differentiate other conditions of the tarsometatarsal joint from those involving the proximal aspect of the MtIII. Focal increased radiopharmaceutical uptake (IRU)

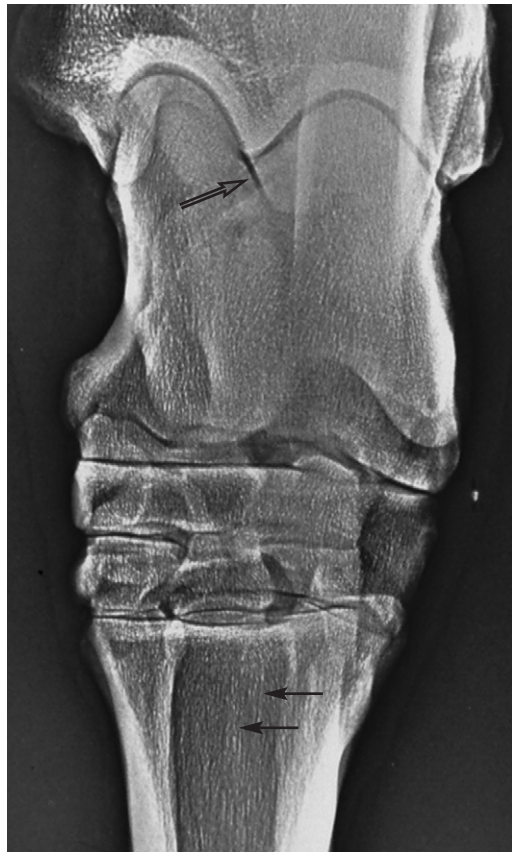

Fig. 44-1 Dorsoplantar radiographic view showing the normal coarse trabecular pattern *(arrows)* seen in the proximal aspect of the third metatarsal bone, which can be mistaken for sclerosis associated with the suspensory attachment. This Standardbred racehorse had a sagittal fracture of the talus. Note the radiolucency of the talus *(open arrow)*.

seen in delayed (bone) phase images involving the proximal plantar aspect of the MtIII is the most important scintigraphic finding in the metatarsal region. Ultrasonographic evaluation should be performed to evaluate the plantar soft tissue structures (see Chapters 16, 73, and 79).

SPECIFIC CONDITIONS OF THE METATARSAL REGION

Bucked and Sore Shins: Dorsal Cortical Fractures of the Third Metatarsal Bone

Modeling and remodeling of the dorsal cortex of MtIII occurs similarly to that of the McIII, but differences in load distribution between forelimbs and hindlimbs account for the relative lack of clinical signs associated with this process in the hindlimbs. Scintigraphic examination often reveals mild, diffuse IRU of the dorsal cortex of MtIII in TB racehorses, but clinical signs of bucked shins are rare. In trotters common findings are pain on palpation, wounds and abrasions, and in some horses bony swelling that is associated with interference injury in the dorsal or dorsal, medial metatarsal region. Although these areas are painful to palpation, most sites are a sign rather than the cause of a high-speed gait deficit. Finding evidence of interference injury, however, is quite important because interference is often a sign of ipsilateral forelimb lameness and can be a cause of horses making breaks (going off stride). Dorsal cortical fractures are rare but can occur in TB and STB racehorses.

Medial and Lateral Condylar and Spiral Fractures of the Third Metatarsal Bone

Medial and lateral condylar and spiral fractures of MtIII are discussed in Chapter 43.

Mid-Shaft, Simple or Comminuted Fractures of the Third Metatarsal Bone

Diaphyseal fractures of the MtIII can result from propagation and displacement of medial condylar fractures. Direct trauma is the most common cause, often from kicks by other horses. Complete fracture of the MtIII occurs in foals and adult horses. Critical prognostic factors are degree of comminution; proximity of the fracture to the metatarsophalangeal joint and tarsometatarsal joints; integrity of the vascular supply; whether the fracture is open or closed; degree of contamination if open; and the horse's age, value, performance level, and intended use. Prognosis for adult horses with open, comminuted fractures of the MtIII is poor to grave, but in those with closed, mildly comminuted or oblique fractures, repair is possible and prognosis is guarded to fair. Horses with open fractures with gross contamination have a grave prognosis, regardless of age and degree of comminution, but those with small areas of skin loss and minor contamination can be successfully managed. Repair in horses with comminuted fractures involving the tarsometatarsal joint is difficult, but consideration of transfixation pin casts by using the distal tibia and distal aspect of MtIII could be given. In foals, vascular supply of the limb distal to fracture is a concern and should be assessed clinically or by using angiography or Doppler ultrasonography. Prognosis in foals with simple or mildly comminuted mid-shaft MtIII fractures repaired using one or two dynamic compression plates is fair to good (Fig. 44-2). In foals, external coaptation often causes profound flexor tendon laxity and should be avoided. External skeletal fixation by using pins and sidebars of casting material or acrylic is possible in the foal. In adult horses, methods of stabilization and repair include internal fixation using two dynamic compression plates, transfixation pin casts in combination with internal fixation, and casts alone.

In a recent study of 25 horses with MtIII or McIII fractures that were managed with internal fixation, external coaptation, or both, age, sex, weight, and the limb affected were not related to outcome, but affected horses were younger than the general hospital population. Seventeen horses had open fractures, and infection was the most common complication after surgery.[4] Non-union in an infected fracture was the most common reason for failure (seven horses). Of 24 horses in which outcome was determined, 16 (67%) had healed fractures and 12 (50%) horses were sound for the intended use.[4] Intended use was not defined; therefore prognosis in foals for future racing could not be determined.

Physeal Fractures of the Distal Third Metatarsal Bone

The most common fracture of the distal MtIII physis is a Salter-Harris type II fracture, but various other fractures can occur. These fractures are usually quite stable, but perfect reduction is difficult to achieve. Fracture reduction and external coaptation are usually successful, but insertion of one or two 3.5- or 4.5-mm bone screws in the metaphyseal component may help stabilize the fracture and reduce the time necessary for external coaptation. Even short periods of cast immobilization in foals can cause rapid onset of flexor tendon

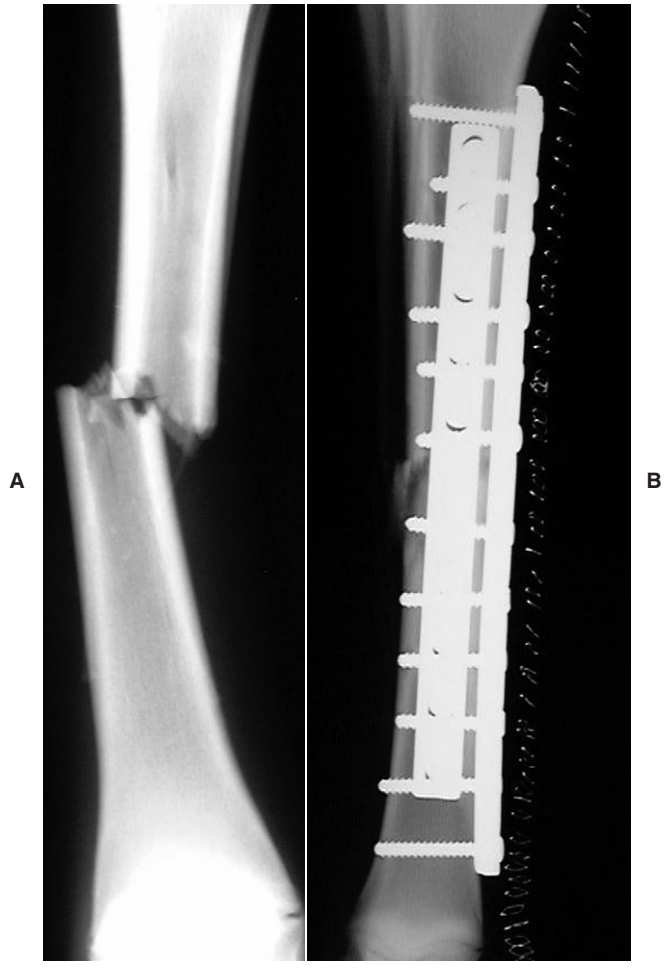

Fig. 44-2 This open (mildly contaminated) mid-shaft fracture of the third metatarsal bone in a 4-month-old Thoroughbred colt was successfully managed using two dynamic compression plates and a half limb bandage. **A,** The preoperative and **B,** postoperative appearance of the third metatarsal bone are shown. The plates were removed 4 months later.

laxity. Cast-bandage and bandage-splint combinations are preferred in young foals (i.e., several weeks old; see Chapters 38 and 73).

Incomplete Longitudinal Fractures of the Plantar Third Metatarsal Bone and Stress Reactions

Incomplete longitudinal fractures of the plantar cortex of MtIII occur considerably less frequently than those of the McIII. Stress reactions of the MtIII, defined as focal areas of IRU without radiographic confirmation of fracture, occur as part of a continuum of stress-related bone injury at the origin of the SL. Combined MtIII injury and PSD worsens prognosis, and it is important to establish whether injury involves bone, soft tissue, or both (see Chapter 38).

Transverse Stress Fractures of the Distal Aspect of the Third Metatarsal Bone

Transverse stress fractures of the distal aspect of the MtIII are described in Chapter 38. I have not recognized this specific fracture type in the MtIII.

Avulsion Fractures of the Third Metatarsal Bone Associated with the Origin of the Suspensory Ligament

Avulsion fractures of the MtIII that are associated with the origin of the SL are frequently seen and can be solitary injuries or associated with PSD. Horses can have acute-onset or chronic mild to moderate hindlimb lameness, depending on size and duration of fracture. Fractures occur most commonly in dressage horses, jumpers, and STB racehorses. Local signs of swelling are usually absent and diagnostic analgesia is essential for localization of pain. Focal areas of IRU are seen that are roughly triangular in shape and involve the proximal plantar aspect of MtIII (Fig. 44-3). It is important to differentiate IRU from that seen in the tarsometatarsal joint, but concomitant IRU in both areas is not unusual. In the MtIII it is unlikely to see a well-defined fracture radiographically, and usually only suspicious areas of radiolucency and sclerosis are seen. Bilateral avulsion fractures or stress reactions occur. Conservative management using a progressive increase in exercise without turn out is usually successful. Recurrence is common and most likely in horses that have associated proximal suspensory desmitis (see Chapter 38.)

Articular Fracture of the Dorsoproximolateral Aspect of the Third Metatarsal Bone

Articular fracture of the dorsoproximolateral aspect of the MtIII occurs primarily in STB and TB racehorses.[5,6] Acute onset of lameness is common, but a history of either chronic, undiagnosed hindlimb lameness or lameness referable to the distal hock joints is usually uncovered. Acute soft tissue swelling is rare. A small bony enlargement is often palpable but is easy to miss. Horses exhibit a positive response to upper limb flexion and to focal, deep pressure over the dorsolateral aspect of the MtIII. Lameness partially resolves after intra-articular analgesia of the tarsometatarsal joint or perineural analgesia of the fibular and tibial nerves. Initial radiographic examination reveals the presence of bony proliferation indicating that bone modeling preceded acute fracture (Fig. 44-4). Incomplete fractures are most common, but complete and mildly displaced fractures also occur. Scintigraphic examination is useful in differentiating this fracture from other conditions involving the tarsometatarsal joint and those involving

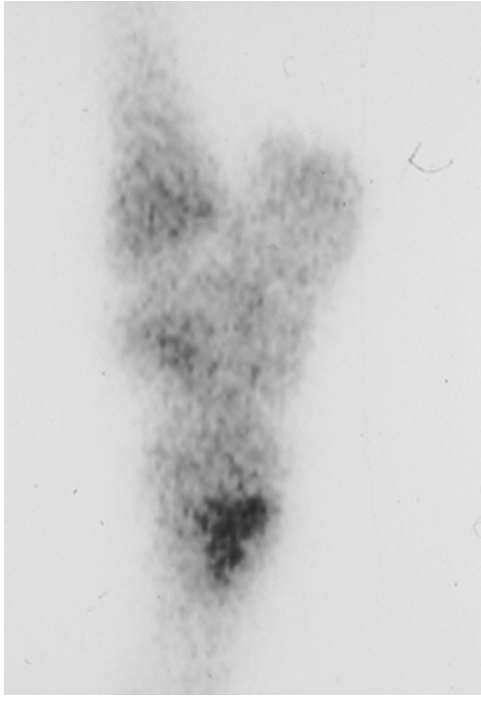

Fig. 44-3 Delayed (bone) phase, lateral scintigraphic image showing focal, triangular-shaped increased radiopharmaceutical uptake typical of an avulsion fracture of the third metatarsal bone that is associated with the origin of the suspensory ligament.

Fig. 44-4 Dorsomedial-plantarolateral xeroradiographic view of the hock and proximal metatarsal region of a Standardbred racehorse. There is an articular fracture of the dorsoproximolateral aspect of the third metatarsal bone *(arrows)*. Note the bone modeling of the proximal aspects of the MtIII and the third tarsal bone that preceded the fracture.

the proximal plantar aspect of the MtIII. Radiographs and scintigraphic images of the contralateral hindlimb often reveal similar but less pronounced changes.

Because proliferative changes precede fracture, it is presumed that the cause involves chronic fatigue and stress-related bone injury. Attachment of the tendons of the fibularis tertius and cranialis tibialis muscles likely contributes substantially to bone stress and may play a role. Horses with sickle-hock conformation appear predisposed to such injury. Concomitant radiographic evidence of osteoarthritis involving the dorsal aspect of the tarsometatarsal and centrodistal joints and dorsoproximolateral fracture of the MtIII is common.

Conservative management is advised in horses with incomplete fractures, but in those with displaced fractures, internal fixation using one or two 3.5-mm bone screws placed in lag fashion is advised. If horses have raced before fracture, the prognosis for future racing is good but only fair to guarded for returning to or sustaining racing in the same class. Horses that develop this fracture before actual racing begins have a poor prognosis. When horses return to training or racing, progressive osteoarthritis of the tarsometatarsal and centrodistal joints appears to be a limiting factor even when the fracture heals and proliferative changes associated with MtIII smooth.

Exostoses of the Second, Third, and Fourth Metatarsal Bones

Exostoses of the metatarsal bones (splint exostoses, splints) occur considerably less commonly than those involving the metacarpal bones, but they can cause lameness, or in racehorses, high-speed soreness. However, it is common to find large exostoses involving the proximal lateral aspect of MtIII or MtIV as an incidental finding that is never associated with lameness, although the lesion is scintigraphically active. Splint exostoses can be caused by direct trauma or instability between the metatarsal bones. There is a common misconception that splints arise from tearing of the interosseous ligaments between the MtII or IV and MtIII, but many splints do not involve the space between bones, and it is difficult to believe that instability and primary desmitis adequately explain the cause of splints. Many splints in the hindlimb involve the MtIII alone (and not MtII or MtIV), although the MtII and MtIV can be affected. Axially located exostoses (blind splints) do occur and are most important proximally, where bony proliferation could crowd or impinge on the proximal aspect of the SL and lateral and medial plantar metatarsal nerves. Direct trauma from interference injury is the most likely cause of most medially located splints. Faulty conformation does not appear to play a prominent role. Mature distal splint exostoses may predispose the MtII and MtIV to fracture in horses with progressive enlargement of the SL.

Lameness associated with splint exostoses is usually mild (grade 1 to 1½ of 5). Direct palpation elicits a painful withdrawal response and lameness is exacerbated. Infiltration of local anesthetic solution alleviates pain and a majority of observed lameness. Radiographs should be obtained when a fracture is suspected but otherwise yield little useful information unless there is drainage associated with bony proliferation.

Management of splint exostoses includes local cold therapy, including cold water hosing and icing, the application of a poultice and bandaging, and the administration of nonsteroidal anti-inflammatory drugs (NSAIDs). Injections of methylprednisolone acetate (80 mg) and Sarapin, an extract of the pitcher plant, (6 ml) subcutaneously and axially if needed for each exostosis reduces inflammation and pain. Repeat injections are often necessary. Cryotherapy is popular in racehorses, but clinicians should be aware that superficial skin pain persists for several weeks afterward, which leaves few ways to monitor improvement other than to monitor performance.

Fractures of the Second and Fourth Metatarsal Bones

Fractures of the MtII and MtIV occur primarily as a result of direct trauma from either a kick from another horse or kicking into or through a stationary object. The MtIV is injured most commonly. Simple, comminuted and displaced fractures occur and often wounds extend directly into the fracture site. The MtIV bears little load and appears to have remarkable recuperative ability. Diagnosis is usually straightforward, particularly if a wound is involved, but in some horses the presence of chronic lameness or drainage several weeks after injury prompts later radiographic examination, and only then is fracture diagnosed. Radiographs often reveal extensive comminution, but in many horses, basic axial alignment of fragments is maintained. When fractures of either the MtIV or MtII involve the tarsometatarsal joint, and particularly if fractures are open, infectious arthritis and osteoarthritis are possible but may be amenable to treatment.

The majority of MtIV fractures heal with conservative management, despite comminution and infectious osteitis, with appropriate antimicrobial administration and wound care. Loose subcutaneously located fragments can be removed with ease and deep tissue cultures should be taken. Although internal fixation[7] or total ostectomy of the MtIV[8] have been described, I have never found it necessary to contemplate surgery. Horses may need 4 to 6 months of rest. Impingement on SL function by callus from fracture healing is a possible complication but is unusual. Fractures of the mid and distal aspects of the MtII and MtIV are caused by chronic suspensory desmitis involving the body or branches. Suspensory desmitis is primary and MtII and MtIV fractures are secondary, resulting from a bowstring effect of the enlarging SL. Occasionally axially located callus from an old splint bone fracture may cause mild lameness from local irritation of the nearby SL. Local injections of anti-inflammatory agents resolve pain in most instances, but occasionally exostoses and the distal aspect of the splint bone are removed.

Ostectomy of distal fragments of the MtII and MtIV can be performed in combination with SL splitting and, in some horses, with ostectomy of apical or abaxial fracture fragments of the PSBs. A triad of clinical problems involving the *S*L, *s*plint bone, and PSBs (called the *three S's*) is often seen, and all three structures should be evaluated before management and prognostic recommendations are made.

Enostosis-like Lesions of the Third Metatarsal Bone

During scintigraphic examination, single or multifocal areas of IRU within the medullary cavity of the MtIII are occasionally seen, and intensity can range from mild to intense. Subsequent radiographic examination reveals one or more round to irregularly shaped radiopacities within the medullary cavity. Lameness in horses with enostosis-like lesions of the MtIII is unusual, particularly if there are small, focal areas of IRU involving only the area around the nutrient foramen. Of 17 enostosis-like lesions (in 10 horses), 4 lesions were identified in the MtIII, but in only 1 horse was lameness localized to the metatarsal region.[9] Lameness, if present, should abate with a high plantar block and resolves with rest and the administration of NSAIDs.

Hypertrophic Osteopathy

Hypertrophic osteopathy is discussed in Chapter 38.

Suspensory Desmitis

Suspensory desmitis, including proximal suspensory desmitis, suspensory body desmitis, and suspensory branch desmitis and associated bony injury, is the most important cause of lameness in the metatarsal region (see Chapter 73).

I have managed a small number of horses with confirmed proximal suspensory and body desmitis by using autogenous

bone marrow injection and proximal metatarsal fasciotomy.[10] Horses are placed under general anesthesia and a medial approach is used to gain access to the proximal aspect of the SL (Fig. 44-5). Thirty to 60 ml of liquid bone marrow should be harvested from the sternum and injected directly into the proximal SL and body as determined by ultrasonographic evaluation. The dense, overlying medial metatarsal fascia is transected from the level of just proximal to the tarsometatarsal joint to the mid-body region. The subcutaneous tissues and skin are closed. Horses should be given 4 to 6 months of progressive increase in exercise, including 4 weeks of stall rest, followed by 4 weeks of stall rest with hand walking, 4 weeks of walking with a rider up or in the jog cart, and then 4 weeks of walking and light trotting. Turn out exercise is forbidden. Of 15 horses with moderate to severe hindlimb proximal suspensory and body desmitis, 8 returned to full competition. However, lameness persisted or recurred in four horses, and three are still recovering. Whether improvement in some horses may result from reparative processes accelerated by the transfer of stem cells, the injection of growth factor–rich substrate into damaged SLs (see Chapter 74), fasciotomy and relief of compartment syndrome, or a combination of factors is unknown. We are currently evaluating the concentration of growth factors in bone marrow and platelet-rich plasma to evaluate possible future injections using this approach and others to augment healing of the SL.[11]

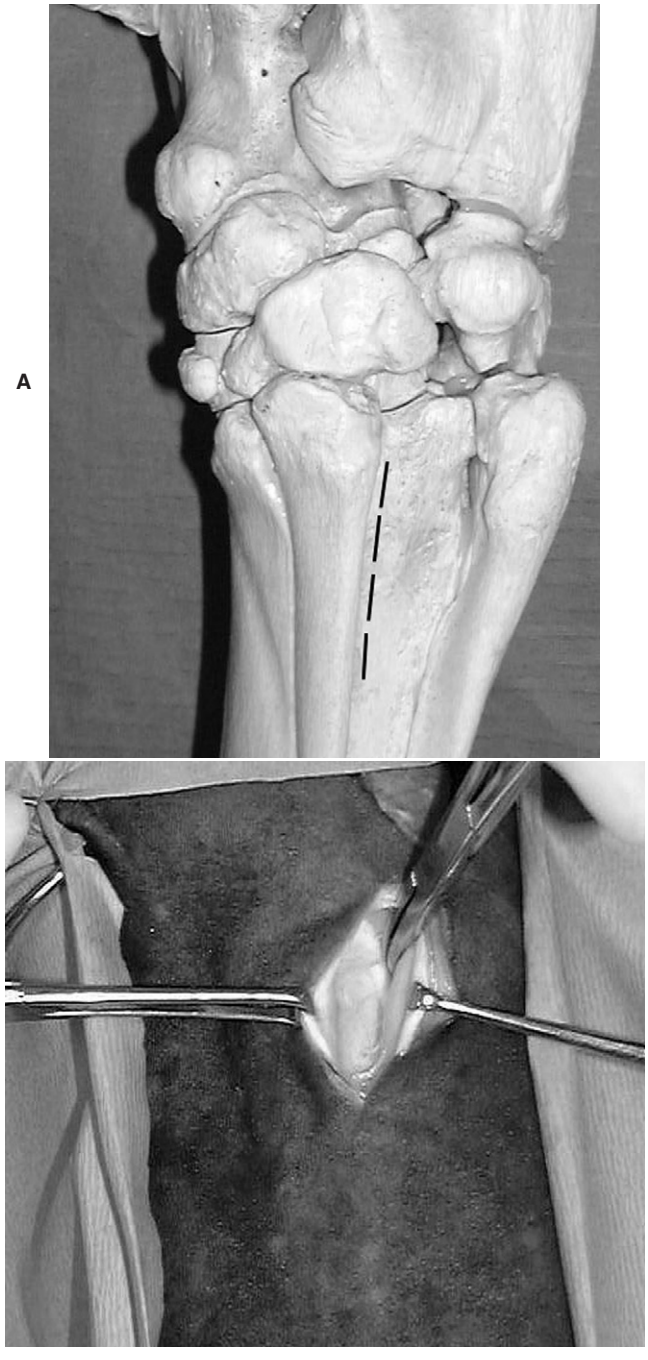

Fig. 44-5 **A,** Photograph of an anatomy specimen and **B,** a cadaver specimen showing the location and approach *(dotted line)* for bone marrow injection and fasciotomy used in management of horses with proximal suspensory and body desmitis. Dense metatarsal fascia is being retracted using Allis tissue forceps, exposing the origin of the suspensory ligament (curved Kelly forceps is positioned plantar to the suspensory ligament).

Tenosynovitis of the Digital Flexor Tendon Sheath

Tenosynovitis of the digital flexor tendon sheath is discussed in Chapter 75.

Desmitis of the Accessory Ligament of the DDFT

Desmitis of the ALDDFT is discussed in Chapter 72.

Deep Digital Flexor Tendonitis

Deep digital flexor tendonitis is discussed in Chapters 71, 77, and 79.

Superficial Digital Flexor Tendonitis

Tendonitis of the SDF is an unusual to rare cause of lameness, but it occurs in racehorses with chronic, progressive curb and in any type of sport horse as a result of direct trauma (see Chapter 79). SDF tendonitis may be obvious clinically or more subtle and requires careful palpation and ultrasonographic examination to confirm diagnosis. STB and TB racehorses with spontaneous SDF tendonitis invariably have sickle-hock conformation. The points of the hock should be compared, because in some horses loss of support accompanies SDF tendonitis and is a negative prognostic sign. Long-term rest has been successful in pacers, but trotters and TB racehorses have a poor prognosis for returning to racing. Most horses regardless of gait drop in race class.

Undiagnosed Metatarsal Region Lameness

In a small proportion of horses, lameness can be localized to the metatarsal region by high plantar analgesia, but comprehensive imaging fails to identify a source of pain. I believe most of these horses have proximal suspensory desmitis and pain from subtle swelling of the SL. Measurement of the cross-sectional area and comparison with the contralateral limb is useful. Horses with lameness inappropriate for the degree of injury confirmed using radiographic and ultrasonographic examinations should be referred for scintigraphic examination. Magnetic resonance imaging may be useful in the future to elucidate sources of plantar metatarsal pain.

Wounds of the Metatarsal Region

Wounds involving the metatarsal region are common and can be simple and involve the skin and subcutaneous tissues, but they are often complex and involve bone and deep soft tissue structures. Lacerations often involve the extensor and flexor tendons (see Chapter 82), and those involving the flexor tendons have serious implications for future performance and salvage. Laceration of the long digital extensor tendon often

delays healing of overlying wounds because movement of tendon ends exacerbates granulation tissue formation and delays wound contraction and epithelialization. Horses with long digital extensor tendon lacerations knuckle initially, but functional healing of the tendon most often results. A common wound is a distal-based flap wound that exposes the dorsal cortex of MtIII and often is associated with extensor tendon lacerations. Because the skin base is distal, much of the proximal, triangular-shaped section of skin becomes necrotic. The skin should not be removed prematurely. Osteitis of the MtIII from direct trauma, drying from exposure, superficial infection, and loss of blood supply may prolong healing. Occasionally, large areas of the dorsal cortex of MtIII have radiographic characteristics of sequestra, but in many horses surgical removal or curettage is not required. In my experience, many horses have received unnecessary surgery to remove damaged areas of MtIII cortex that if left in place would likely have healed uneventfully. However, horses that develop extensive proliferative changes of the MtIII usually have adhesions to the long digital extensor tendon and often have chronic lameness.

Small puncture wounds may result in localized osteitis of the MtII or MtIV. Removal and curettage should be reserved for those with purulent drainage refractory to management with antimicrobial therapy. Horses without drainage but with radiographic evidence of fragmentation within a radiolucent defect can be managed conservatively unless expedient resolution of the problem is mandatory. Horses with wounds and evidence of MtIII cortical crushing or obvious fractures should be managed with caution because catastrophic fracture could develop. Of substantial risk are those with acute, cortical fractures that resemble true saucer fractures. If horses are placed under general anesthesia to remove what is misinterpreted as devitalized pieces of cortex, there is a risk for catastrophic fracture during anesthetic recovery.

Acquired or secondary stringhalt is an unusual complication from metatarsal region wounds involving the long digital extensor tendon and sometimes the lateral digital extensor tendon. Horses at risk are those with extensive proliferation of the MtIII and adhesion formation with the overlying long digital extensor tendon. Mechanisms to account for the development of stringhalt include adhesion formation and interruption of the normal myotactic reflex.[12] Of 10 horses that developed stringhalt after dorsal metatarsal trauma, 6 horses developed stringhalt within 3 months, 3 developed stringhalt after 3 months, and in 1 horse time of injury to development of stringhalt was undetermined.[12] Of four horses managed with rest and progressive exercise, stringhalt resolved in one, improved in two, and remained the same in one horse.[12] Of five horses that received surgical management using lateral digital extensor myotenectomy, stringhalt resolved in two, improved in two, and remained the same in one horse.[12] In my limited experience with this condition, prognosis is guarded to poor for complete resolution of the gait deficit.

Diffuse Swelling in the Metatarsal Region

Diffuse swelling in the metacarpal region is discussed in Chapter 38, and this also applies to the metatarsal region.

Severe Cellulitis of the Metatarsal Region

Cellulitis of the hindlimb occurs frequently from kick wounds or other trauma. In many horses obvious signs of trauma are lacking, but severe lameness and signs of infection in the metatarsal region occur. A severe form of cellulitis occurs primarily in the TB racehorse in which severe lameness, swelling, and fever develop, apparently from a very small skin wound or excoriation, because an obvious wound is most difficult to find. This condition is similar to focal, peritarsal cellulitis (see Chapter 109). Initially it is difficult to differentiate cellulitis from infection of the tarsal sheath, SDFT, DDFT, and the DFTS, because swelling is diffuse and horses are severely lame. Within 48 to 72 hours, skin necrosis and sloughing can occur, which exposes underlying and sometimes infected SDFT. *Staphylococcus* and occasionally *Streptococcus* species are cultured. Laminitis in the contralateral limb is a risk initially. Skin sloughing can be pronounced and requires weeks to months of wound care and, in some horses, grafting. Involvement of underlying SDFT and other soft tissue structures is a poor prognostic sign.

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CHAPTER • 45

The Tarsus

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ANATOMY

The hock joint consists of numerous articulations, including the tarsocrural, talocalcaneal, talocalcaneal-centroquatral (proximal intertarsal), centrodistal (distal intertarsal), and tarsometatarsal joints. The bones consist of the talus and calcaneus and the central, first and second fused, third, and fourth tarsal bones.[1] The tarsocrural joint is a ginglymus joint based on the shape of deep grooves on the cochlear articular surface of the distal end of the tibia with the extensive surface of the trochlea of the talus. The articulation of these joints is at an angle of 12° to 15° dorsolateral to the sagittal plane of the limb. The proximal intertarsal, centrodistal, and tarsometatarsal joints are plane joints and are only capable of small amounts of gliding movement.

The fibrous part of the joint capsule attaches above the tibial articulation proximally and to the metatarsal bones distally. Distally the fibrous joint capsule attaches to the surfaces of the distal tarsal bones and blends in with the collateral ligaments.[2] The dorsomedial aspect of the joint capsule is thin and uncovered by any tendons or ligaments and forms a fluctuant swelling over the medial trochlea of the talus. The proximal aspect of the plantar fibrous joint capsule is also thin and extends proximally behind the distal end of the tibia for about 5 cm.[2] The plantar aspect of the distal joint capsule consists of the plantar and tarsometatarsal ligaments, which are thick and tightly adherent to the distal tarsal bones. The tarsus is composed of four synovial compartments. The tarsocrural compartment lubricates the tarsocrural joint and the cranial aspect of the proximal intertarsal joint. This is the largest compartment and is composed of four pouches: the dorsomedial, dorsolateral, plantaromedial, and plantarolateral. The proximal intertarsal synovial sac lines the talus and calcaneus proximally and the plantar aspect of the central and third tarsal bones distally and communicates cranially with the tarsocrural joint. The centrodistal joint lubricates the articulation between the central and third tarsal bones and the bones on each side, and the tarsometatarsal joint lubricates the third and fourth tarsal bones with the proximal metatarsal bones. There is communication between 8% and 30% of the centrodistal and tarsometatarsal joints, but not necessarily in both hocks of the same horse.[3-5] Communication between the distal tarsal joints and the tarsocrural joint occurs in about 3% of horses.[4]

Numerous ligaments surround the hock. The long collateral ligament is superficial and originates at the caudal aspect of the lateral malleolus of the distal tibia and attaches to the fourth tarsal and third (MtIII) and fourth metatarsal (MtIV) bones and forms a canal for the tendon of the lateral digital extensor muscle. The short lateral collateral ligament lies deep to the long lateral collateral ligament and originates from the cranial aspect of the lateral malleolus, passes caudally, and attaches to the lateral surface of the calcaneus. The long medial collateral ligament originates from the caudal aspect of the medial malleolus and attaches distally to MtII and MtIII and also attaches to the medial aspect of the distal tarsal bones. The short medial collateral ligament lies deep to the long medial collateral ligament and extends from the medial malleolus to the medial aspect of the calcaneus and sustentaculum tali. The plantar ligament is a strong, flat band that originates at the proximal plantar surface of the calcaneus and extends distally and attaches to the fourth tarsal bone and MtIV. The dorsal tarsal ligament spreads out distally from the distal tuberosity of the talus and attaches to the central and third tarsal bones and proximal aspect of the MtIII and MtIV bones. Numerous short ligaments connect adjacent bones of the tarsus and have connections between tarsal and metatarsal bones.

ARTICULAR DISEASES OF THE TARSUS

Osteoarthritis of the Distal Hock Joints

Distal hock joint pain is common in horses from all disciplines and is often associated with osteoarthritis. Distal hock joint pain is known colloquially as bone or jack spavin or occult or blind spavin in the absence of radiographic abnormalities. The term *juvenile spavin* has been used to describe early osteoarthritis that had a prevalence of 20% in a group of horses younger than 2 years of age.[6] Although it is usually seen in mature horses used for sport or pleasure, distal hock joint pain can occur in young Thoroughbred (TB) and Standardbred (STB) racehorses and Western performance horses (see Chapter 121). Distal hock joint pain may be a sequel to incomplete ossification of the central and third tarsal bones (see page 443). Certain conformational abnormalities (such as sickle-hock, in-at-the-hock, or cow-hock) or excessive straightness of the hindlimbs may predispose to distal hock joint pain, although this condition frequently occurs in normally conformed horses. Traditionally it has been proposed that osteoarthritis of the distal hock joints is caused by excessive compression and rotation of the distal tarsal joints as the horse jumps or stops, which results in abnormal tension on the intertarsal ligaments. However, this theory is not consistent with the common recognition of distal hock joint pain in pleasure horses or its high incidence in Icelandic horses, a breed in which osteoarthritis is thought to be a heritable condition.[7] Distal hock joint pain is classically thought to begin on the dorsomedial aspect of the joints and to progress dorsally. However, it is the Editors' experience that in early osteoarthritis, scintigraphic or radiographic abnormalities are frequently first identified only on the dorsolateral aspect of the joints. Nuclear scintigraphic studies of clinically normal mature athletes has shown mild increased uptake of radiopharmaceutical on the lateral aspect, which is consistent with increased modeling, presumably the result of a relative increased loading laterally compared with medially.[8]

The centrodistal and tarsometatarsal joints are most commonly affected, either individually or together, but osteoarthritis of the proximal intertarsal joint does occur, usually in association with osteoarthritis of the more distal joints. The condition may be unilateral but is often bilateral. Occasionally osteoarthritis of the talocalcaneal joint occurs in isolation[9] (see page 443).

History

Clinical signs of distal hock joint pain vary considerably among horses, ranging from a moderate to severe unilateral lameness to subtle changes in performance without overt lameness, (see Chapters 100 and 107 to 128). These signs include the horse becoming disunited in canter, an unwillingness to canter with a particular lead, and reluctance to turn or decelerate with proper engagement of the hindlimbs. The owner may comment that the farrier has experienced difficulties when shoeing the horse. Frequently a horse with bilateral distal hock joint pain has low-grade stiffness that wears off with work. Lameness frequently improves or resolves with rest but recurs when work is resumed. Treatment with non-steroidal anti-inflammatory drugs (NSAIDs) usually results in an improvement in lameness unless it is severe.

Clinical Signs

In many horses no abnormalities are detectable by visual inspection or palpation of the hock region. With more chronic distal hock joint pain there may be enlargement over the medial or dorsomedial aspects of the distal hock joints, which is the result of peri-articular soft tissue thickening. Distention of the tarsocrural joint capsule may occur either coincidentally or reflecting involvement of the proximal intertarsal joint. Frequently there is secondary soreness of the epaxial muscles in the lumbar region and sometimes caudal gluteal muscle soreness. The toe and branch of the shoe of the lame limb, or both, may wear abnormally. In the Editors' experience, shoe wear in this location, however, is not pathognomonic of distal hock joint pain. Some horses, if not properly trimmed, develop lateral flare of the hoof and mediolateral imbalance that is high medially. Flexion of the limb may be resisted slightly, but marked lifting of the limb during flexion is more likely to reflect stifle pain. The Churchill test (see Chapter 6) is helpful in identifying distal hock joint pain in some, but by no means all, horses. Soreness associated with specific acupuncture points (see Chapter 93) can also be suggestive of distal hock joint pain.

Lameness varies greatly in degree and is not necessarily correlated with the degree or type of osteoarthritic change detected radiographically. The horse should be assessed moving from the side, from behind, and from in front to appreciate all gait abnormalities. These abnormalities may include asymmetrical movement of the tubera coxae; reduced arc height of foot flight, with or without toe drag; shortening of the cranial phase of the stride; reduced extension of the fetlock; and a tendency for the limb to swing medially during protraction and slide laterally at ground contact. Lameness may be accentuated on a circle, in some horses with the lame or lamer limb on the outside, and in others with it on the inside. Gait abnormalities may be accentuated if the horse is ridden. However, none of these characteristics is pathognomonic for distal hock joint pain.

Kinematic gait measurements were recorded after endotoxin-induced lameness of the distal tarsal joints.[10] Both fetlock and tarsal joint extension during stance phase decreased, fetlock joint flexion and hoof height during swing phase increased, limb protraction decreased, and vertical excursion of the tuber coxae became more asymmetrical. These observations are not entirely consistent with the observations made in natural disease.

Proximal (upper) limb flexion tests (i.e., the so-called spavin test) are useful in accentuating lameness in some, but not all, horses with distal hock joint pain. The hindlimb should be held with the metatarsal region parallel to the ground for 60 to 90 seconds before the horse is trotted away. The limb may be held either by the toe of the foot or by cupping the hands behind the fetlock. Applying pressure to the flexor tendons should be avoided. The response to flexion should be interpreted in light of the discipline of the horse, the degree of accentuation of lameness, and its duration. Flexion of the non-lame or least lame limb should be performed first and the response compared with the contralateral limb. In some horses there is a paradoxical increase in lameness in the weight-bearing limb. Because of the reciprocal apparatus of the hindlimb, a positive response to proximal limb flexion is not specific for distal hock joint pain. The response to flexion may be recorded as none (0), mild (+1), moderate (+2), or severe (+3). A severe response is unusual in horses with distal hock joint pain.

Diagnostic Analgesia

Diagnostic analgesia is important to confirm the source(s) of pain. Although intra-articular analgesia is potentially more specific than perineural analgesia, the results can be misleading. A negative response to intra-articular analgesia does not preclude distal hock joint pain. Narrowing of the centrodistal joint space or peri-articular new bone can prohibit intra-articular injection. In the presence of extensive subchondral bone damage the response to intra-articular analgesia is very poor. Even in the absence of radiological change the response to intra-articular medication is sometimes substantially better than that to intra-articular analgesia, but the converse is also true in some horses.

However, intra-articular analgesia is very useful in many horses. The degree of communication between the centrodistal and tarsometatarsal joints is variable.[3,4] Therefore theoretically it is preferable to inject both the tarsometatarsal and centrodistal joints either separately or in combination. However, because it is considerably easier and safer to inject the tarsometatarsal joint, it is often preferable to block the tarsometatarsal joint first. Then if there is no response in lameness or only partial improvement, the centrodistal joint should be blocked. The techniques for intra-articular analgesia are described in Chapter 10. Improvement in lameness by 50% or more is considered a positive response. In the Editors' experience, lameness is often substantially improved within 10 minutes, although two authors (R.M.D. and G.K.C.) reassess lameness after 30 minutes. The Editors find reassessment of the response to flexion potentially misleading, although two authors (R.M.D. and G.K.C.) routinely repeat flexion tests. Perineural analgesia of the superficial and deep fibular and tibial nerves (see Chapter 10), although not specific for distal hock joint pain, can be very helpful in confirming hock pain, assuming that pain arising from the more distal aspects of the limb has already been excluded. With practice these blocks are reliable and safe and often result in a much greater degree of improvement in lameness than intra-articular analgesia. Improvement is generally seen within 20 minutes, but occasionally the response is delayed, and it is preferable not to proceed with further blocks until at least 1 hour has elapsed. Horses with specific performance problems associated with distal hock joint pain, rather than overt lameness, may respond better to intra-articular medication than either intra-articular or perineural analgesia.

Radiography and Radiology

Four radiographic views of the tarsus are required: lateromedial, dorsolateral-plantaromedial oblique, dorsomedial-plantarolateral oblique, and dorsoplantar.[11] Lesions may be detectable only in a single view, thus in the Editors' opinion, all four views should be obtained routinely. To cut through the centrodistal and tarsometatarsal joint spaces, it is important that the horse be standing with the metatarsal region vertically and bearing weight evenly on each hindlimb. Because the centrodistal and tarsometatarsal joints slope distally from laterally to medially, to reliably cut through the joint spaces in a lateromedial view, the x-ray beam should be angled 10° distally. Both hocks should be examined because the condition is often bilateral. If a horizontal x-ray beam is used, it can be difficult to cut through the entire centrodistal joint space in a

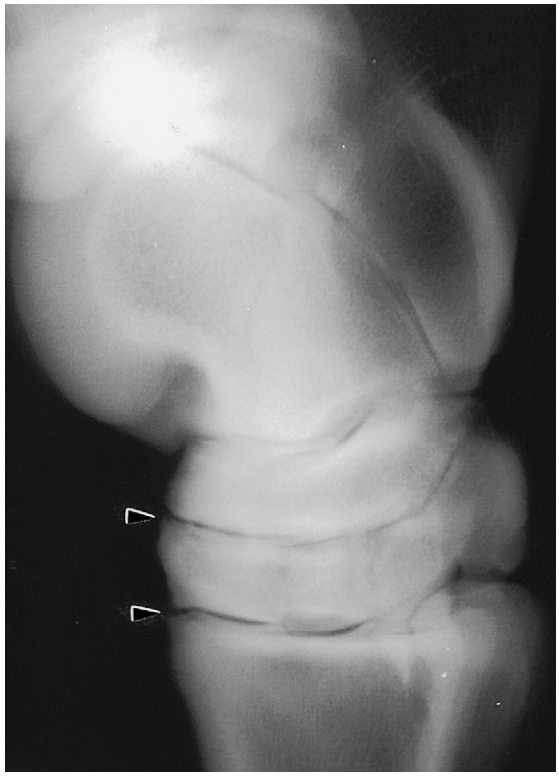

Fig. 45-1 Lateromedial radiographic view of the tarsus of a horse with mild peri-articular osteophyte formation *(arrows)* involving the centrodistal and tarsometatarsal joints.

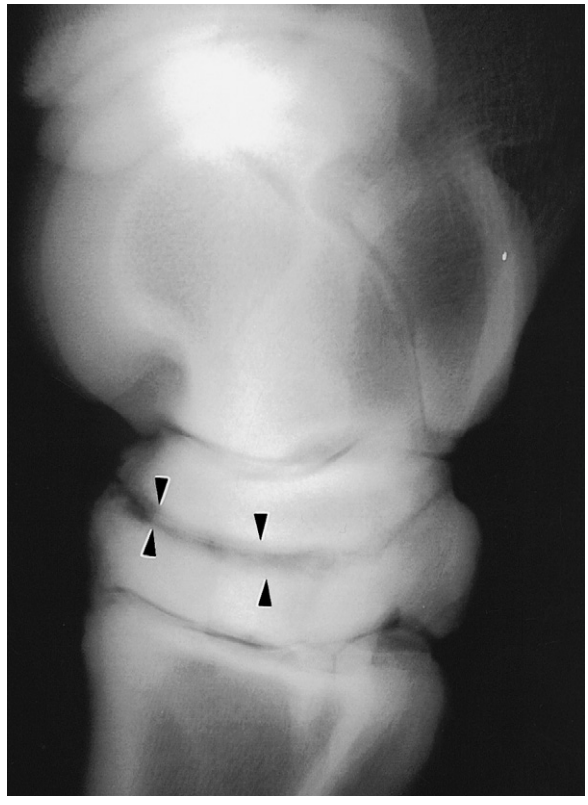

Fig. 45-2 Lateromedial radiographic view of the tarsus of a horse with osteoarthritis of the centrodistal and tarsometatarsal joints. There is modeling of central tarsal and third tarsal bones and irregularity of the centrodistal joint space *(arrows)* caused by subchondral lysis.

dorsoplantar projection, and it may appear that one side of the joint is narrowed. An additional dorsal 5° proximal-plantarodistal oblique view helps to determine whether there is genuine joint space narrowing. Involvement of the talocalcaneal-centroquatral joint merits a more guarded prognosis.

Radiographic abnormalities consistent with osteoarthritis include peri-articular osteophyte formation (Fig. 45-1), subchondral lucent areas (Fig. 45-2), subchondral sclerosis, periosteal new bone, and narrowing of joint spaces. The correlation between the degree of lameness and the extent of radiological abnormalities is poor.[12,13] Extensive changes may be present when lameness is first recognized. Some horses predominantly develop peri-articular changes, whereas others have abnormalities confined to the central and third tarsal bones and the proximal MtIII. The reason for these differences is currently unclear. Small osteophytes or entheseophytes on the dorsoproximal aspect of the MtIII can be incidental radiological abnormalities. Horses with severe subchondral lysis tend to be lamer and respond poorly to intra-articular analgesia and intra-articular medication.

Nuclear Scintigraphy

In many horses nuclear scintigraphy is unnecessary, but it can be very helpful in difficult horses and those with performance problems rather than overt lameness. A region of increased radiopharmaceutical uptake (IRU) may be focal or more diffuse. Very focal increased uptake may reflect intertarsal ligament enthesopathy rather than osteoarthritis. Intense IRU may be present in the absence of radiological abnormality, and these horses tend to respond poorly to intra-articular medication, despite a positive response to intra-articular analgesia. Because IRU may occur in association with primary lameness originating from pain elsewhere in the limb, results should be interpreted cautiously.

Treatment

The aim of treatment is to provide pain relief so that the horse can remain in work. Traditionally it has been suggested that by maintaining the horse in work, the affected joints will fuse. However, progressive radiological ankylosis is rarely observed, although radiography tends to underestimate the degree of joint fusion. Horses with involvement of the talocalcaneal-centroquatral joint and extensive subchondral bone lysis have a more guarded prognosis.

Treatment options include palliative therapy with NSAIDs; intra-articular medication with corticosteroids, hyaluronan, or polysulfated glycosaminoglycans (PSGAGs); with or without systemic treatment with PSGAGs, oral nutraceuticals, or both, combined with corrective trimming and shoeing[14]; and adaptation of the work program. Extracorporeal shock wave treatment has recently been described, but long-term results are lacking.[15] If medical therapy fails, surgical treatment, including cunean tenectomy, subchondral forage to reduce intra-osseous pressure,[16] drilling the affected joints to promote arthrodesis,[17] chemical induction of ankylosis using sodium monoiodoacetate,[18,19] or neurectomy[20] are options.

Selection of treatment depends on the degree of lameness, extent of radiological abnormalities, use of the horse, regulations for competition, response to previous treatment, time available, and financial constraints. Resting the horse usually is not beneficial. Palliative treatment with NSAIDs such as phenylbutazone is useful in pleasure horses; the lowest dose that alleviates lameness should be used. Long-term use of phenylbutazone, 1g twice daily for a 500-kg horse, is generally well tolerated. Any treatment should be combined with corrective trimming and shoeing to ensure correct mediolateral

balance and to facilitate breakover by shortening the toe, squaring and rolling the toe of the shoe, or setting the shoe back from the toe. A lateral trailer[14] or a lateral extension of the shoe provides symptomatic relief in some horses and tends to stop excessive twisting of the limb. However, these devices may be contraindicated in horses that have to stop and turn quickly, because the extension may dig into the footing and stop the distal limb abruptly, causing abnormal torque on the distal limb joints.

Intra-articular medication with corticosteroids, such as methylprednisolone acetate or triamcinolone, with or without hyaluronan, is extremely useful for management of horses with distal hock joint pain. However, horses with extensive radiological abnormalities often have a limited response. Horses with scintigraphic abnormalities in the absence of radiological change also respond poorly. Two authors (R.M.D. and G.K.C.) routinely treat both the centrodistal and tarsometatarsal joints of both hocks by using 60 to 80 mg of methylprednisolone acetate in each joint. If the competition schedule allows, the horse is turned out for 3 to 4 days, followed by 3 to 4 days of light riding before resuming normal activity. Phenylbutazone (2.2 mg/kg sid) is also recommended for 7 days to decrease the possibility of post-injection joint flare. Most horses are sound within 7 to 10 days and stay sound for 3 to 6 months, depending on severity of disease and level of horse use.

Similar results have been achieved using triamcinolone, 6 mg per joint (S.J.D.). There are no controlled clinical trials assessing the relative efficacy of different corticosteroids or the use of a combination of corticosteroids and hyaluronan, although some clinicians believe that combination therapy lasts longer. Hyaluronan alone is of dubious value. Corticosteroid injections can be repeated two or three times per year without promoting progressive radiological change. In some horses it is possible to predict when lameness will recur, and repeated treatment before this recurrence can maintain soundness. Although cunean bursitis is rarely recognized as a primary cause of lameness, some horses respond better if intra-articular treatment is combined with treatment of the bursa. A series of intra-articular injections of PSGAG (three injections given every 2 weeks) can be useful in competition and race horses with distal hock joint pain.

Systemic medication with PSGAG (500 mg IM once weekly for 6 weeks), hyaluronan (4 mg IV monthly), or oral nutraceuticals may be beneficial as adjunctive therapy. Treatment 3 to 4 days before an event may be useful. In some horses, increasing the dose of PSGAG to 1 g weekly gives better results.

Recently shock wave treatment that was performed with horses under general anesthesia, followed by 5 weeks of rest, resulted in an 80% improvement in lameness in horses 90 days later,[15] but long-term results are not available.

A variety of techniques for surgical arthrodesis of the distal hock joints[17,21,22] and subchondral forage[16] exist, and although subchondral forage can provide rapid pain relief in some horses, effective arthrodesis and resolution of lameness often takes up to 12 months. Careful surgical technique is essential to avoid the development of extensive peri-articular new bone, which can itself cause pain. Using the three-drill tract procedure,[17] a 66% success rate was reported.[21] However, the results in upper-level competition horses have been less favorable.[23] Cunean tenectomy alone may result in temporary improvement in clinical signs but is unlikely to restore soundness.[24] Chemical fusion with sodium monoiodoacetate has had excellent results in some horses,[18] but even if positive radiographic contrast studies do not show physical communication between the centrodistal and talocalcaneal-centroquatral joint, it is likely that in some horses, sodium monoiodoacetate can spread to these joints and result in progressive osteoarthritis. Arthrodesis of the centrodistal and tarsometatarsal joints can be followed by fracture of the central or third tarsal bone or the development of osteoarthritis in the talocalcaneal-centroquatral joint. Neurectomy of the deep fibular nerve and a partial neurectomy of the tibial nerves relieves the pain associated with osteoarthritis of the distal hock joints,[20] and approximately 60% of treated horses return to full athletic function.

Osteoarthritis of the Talocalcaneal Joint

Osteoarthritis of the talocalcaneal joint is unusual.[9] There are frequently no localizing signs. Lameness is resolved by perineural analgesia of the fibular and tibial nerves and may be improved by intra-articular analgesia of the tarsocrural joint. Radiographic abnormalities are confined to this joint and are best identified in lateromedial or dorsomedial-plantarolateral oblique views and include subchondral lucency or sclerosis and narrowing of the joint space. The prognosis is guarded to poor.

Osteoarthritis of the Tarsocrural Joint

Osteoarthritis of the tarsocrural joint may occur as a primary disease, develop secondary to osteochondrosis (see Chapter 58) or osteoarthritis of the distal hock joints (see page 440), or be a sequel to trauma. There may be distention of the tarsocrural joint capsule, but this is a variable feature. Lameness varies from mild to severe. Horses with longitudinal wear lines on the trochleas of the talus secondary to osteochondritis dissecans of the distal intermediate ridge of the tibia often have only mild lameness. However, horses with focal partial- or full-thickness cartilage erosions on weight-bearing parts of the trochleas of the talus may have severe lameness. Lameness usually is partially improved by intra-articular analgesia. In horses with primary osteoarthritis of the tarsocrural joint, usually there are no detectable radiological abnormalities and scintigraphic examination is generally negative. In racehorses, scintigraphic examination often reveals IRU involving the distal tibia and proximal talus and must be differentiated from subchondral trauma or fracture of the distal tibia and talus.[25] Definitive diagnosis is dependent on arthroscopic examination of the joint. The prognosis for resolution of lameness and return to full athletic function is generally guarded to poor. Response to intra-articular medication is often disappointing.

Incomplete Ossification of the Central and Third Tarsal Bones

Incomplete ossification of the central and third tarsal bones is most common in premature or twin foals, but it can occur in full-term singles and is characterized by a sickle hock appearance. It may occur unilaterally or bilaterally. Radiographically the bones are smaller and more rounded than usual. Early recognition and treatment are essential to avoid crushing of these bones with resultant progressive osteoarthritis. Cylinder tube casts can be used effectively with good results. In some horses the condition goes unrecognized until a young horse increases work intensity, although the horse may have a pre-existing curb-like appearance of the hock. Lameness may be sudden in onset despite the existence of advanced radiographic changes. Radiographic examination reveals a wedge shape of the central and third tarsal bones, which are narrower dorsally. One or more bones may be fractured. There is often advanced osteoarthritis. Although affected horses may be used for pleasure riding, the prognosis for competition use is guarded.

Distention of the Tarsocrural Joint Capsule

Among the many causes of acute or chronic synovitis of the tarsocrural joint (also known as *bog spavin*) are osteochondrosis, osteoarthritis, trauma, poor conformation, hemarthrosis, infection, and idiopathic causes. Signalment varies and

depends on origin. Clinical signs are excessive tarsocrural joint fluid, which is most easily recognized on the dorsomedial and plantarolateral aspects of the joint. Lameness may or may not be present and is dependent on etiology. Excessive distention of the joint capsule may result in mechanical lameness. Diagnosis is made on the basis of clinical appearance and the response to intra-articular analgesia if the horse is lame. Arthrocentesis may reveal hemorrhage. Radiographic examination should include four standard views, but if no radiological abnormality is detected, flexed lateromedial and flexed dorsoplantar (skyline) views may be helpful. Slightly oblique dorsoplantar views may be necessary to identify osteochondritic lesions of the medial malleolus of the tibia (see Chapter 58). In the absence of lameness or radiographic abnormalities, a diagnosis of idiopathic synovitis is made.

Treatment involves fluid drainage and intra-articular injection of either 80 mg of methylprednisolone acetate or 12 mg of triamcinolone. Combining hyaluronan with the corticosteroids may provide joint protection, but the strong anti-inflammatory effect of the corticosteroids seems most beneficial. Pressure bandages should be applied to help maintain joint decompression. Phenylbutazone (2.2 mg/kg) once daily for 7 days and confinement to a small area for 2 weeks is recommended. Approximately 50% of horses have resolution or decrease in effusion, although some horses may require retreatment. If joint effusion returns and the horse is not lame, the owner is advised that the horse has a cosmetic blemish that will probably not resolve completely. Horses that are lame and block to the tarsocrural joint but have no radiographic lesions are candidates for scintigraphic examination and diagnostic arthroscopy (see "Osteoarthritis of the Tarsocrural Joint," page 443). Hemarthrosis is discussed in Chapter 67.

Osteochondrosis of the Tarsocrural Joint

See Chapter 58 for a discussion of osteochondrosis of the tarsocrural joint (page 551).

Subchondral Trauma and Radiolucency of the Tibia and Talus

A relatively unusual lameness that is alleviated by intra-articular analgesia of the tarsocrural joint is associated scintigraphically with a focal region of IRU in the subchondral bone of either the distal tibia or the talus.[25] In some horses a progressive radiolucent defect may be identified radiographically, whereas in others no lesion could be identified radiographically but a lesion was recognized using computed tomography. These lesions are believed to be traumatic in origin. Conservative management has resulted in persistent lameness. Surgical curettage may offer a more favorable prognosis.

Fragments in the Talocalcaneal-Centroquatral Joint

The tarsocrural joint communicates directly with the proximal intertarsal joint in immature horses through a slit-like opening and in adult horses through a broad opening in the dorsal reflection of the synovium that is located at the distal dorsal aspect of the talus. The proximal intertarsal joint is approached through this synovial fenestration during arthroscopic exploration of the tarsocrural joint. A report describes 17 horses with either a free-floating fragment, which was suspected to have originated and dislodged from an associated osteochondritic lesion in the tarsocrural joint, or a fragment located at the distal end of the medial trochlear ridge with attachment to the synovial reflection that separates the tarsocrural and proximal intertarsal joints.[26] However, such fragments can be seen as incidental abnormalities and unassociated with lameness.[11] Fourteen of 17 horses returned to racing or intended performance activity after arthroscopic removal of the fragment.[26]

TARSAL BONE FRACTURES AND LUXATIONS

Fractures of the Distal Tarsal Bones

Fractures of the central and third tarsal bones occur most frequently in STB, cutting horses, or TB racehorses,[27-30] but they occasionally occur in other horses, sometimes secondary to previous fusion of the centrodistal and tarsometatarsal joints. These fractures cause an acute onset of a moderate to severe hindlimb lameness that is most noticeable when the affected limb is on the inside of a circle.[27-29] The lameness is exacerbated by hock flexion. The degree of lameness diminishes after 1 to 2 weeks of rest but returns if the horse returns to work. Some horses with incomplete fractures of the central tarsal and third tarsal bones can race a number of times before lameness becomes pronounced. On initial diagnosis, some fractures appear old in both the TB and STB racehorse. Horses with bilateral third tarsal bone fractures may be examined for poor performance rather than overt unilateral hindlimb lameness. Within STB racehorses, central and third tarsal bone fractures are more common than expected in pacers than in trotters, even if the normal 3:1 pacer/trotter ratio is considered. Heat, soft tissue swelling, and pain on digital palpation of the distal tarsal bones usually accompany the fracture. Synovial effusion of the tarsocrural joint may occur in horses with central tarsal bone fractures but is not usually seen in horses with third tarsal bone fractures.

Diagnosis is made by radiography, but a fracture may not be radiographically apparent until up to 10 days after injury, when demineralization of the fracture line occurs. Scintigraphy is useful in horses with mild or bilateral hindlimb lameness. Incomplete or bilateral fractures may be seen. Many fractures can be identified on a lateromedial radiographic view, but if no radiological abnormality is detectable in standard views, additional oblique views, including plantar 25° lateral-dorsomedial oblique, should be obtained (Fig. 45-3). However, the precise

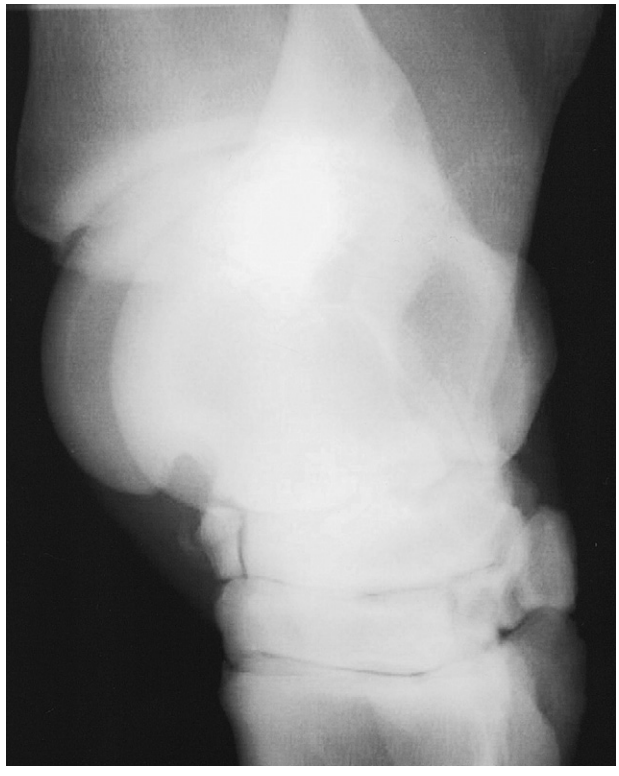

Fig. 45-3 Plantar 25° lateral-dorsomedial oblique radiographic view of a tarsus. There is a complete sagittal fracture of the central tarsal bone.

location and orientation of fractures seems to vary between disciplines and between the central and third tarsal bones. Thus if a fracture is suspected, additional oblique views may be required. In many fractures in STBs the fracture does not appear to extend to the distal articular surface. In Western performance horses the fracture line is generally located in a more plantar region of the third and central tarsal bones than is seen in racehorses. Third tarsal bone fractures are most commonly dorsal or dorsolateral. In TB racehorses a wedge-shaped conformation of the dorsolateral aspect of the third tarsal bone may be a risk factor for fracture.[31] Comminuted fractures are more common in the central tarsal bone and may be difficult to identify radiographically.[28] Nuclear scintigraphy is invaluable for identifying the likely presence of a fracture if it cannot be determined radiographically.

Horses may be treated conservatively[30,32,33] or surgically.[28,29,33] Seven horses with fractures of the third tarsal bone that were treated conservatively remained lame for 12 months after injury,[28,29] whereas three horses treated by lag-screw compression returned to racing within 6 months.[28] Six racehorses with central tarsal bone fractures remained lame, whereas six Quarter Horses treated surgically made a complete recovery.[29] However, conservative management resulted in return to training within 8 months for 73% of 45 TB racehorses with fractures of the central or third tarsal bone,[30] and this corresponds with the Editors' experience. Ten (71%) of 14 STBs and 2 (33%) of 6 TBs returned to racing and started at least 5 races after injury, and 4 (80%) of 5 Quarter Horses returned to previous athletic function with conservative management.[32] If surgical treatment is performed, case selection is important, because many central tarsal bone fractures have hidden fracture lines that could be inadvertently displaced during screw placement. The large head of a 4.5-mm cortical bone screw may cause soft tissue inflammation and a periosteal reaction. For third and central tarsal bone fractures, 3.5-mm cortical bone screws are preferred. One or two screws should be placed through stab incisions by using radiographic or fluoroscopic guidance. The use of a Herbert cannulated compression screw has been reported.[29] Horses with fragments that are too small to allow lag-screw fixation, or those with chronic fractures of the third tarsal bone causing osteoarthritis of the centrodistal or tarsometatarsal joints, can be treated by surgical drilling to facilitate arthrodesis of the joints.

Sagittal Fracture of the Talus

Sagittal fractures of the talus are rare, usually non-displaced, can be difficult to diagnose, and have been recorded most frequently in STB and TB racehorses,[34,35] but they can occur in any type of horse. Twelve racehorses had a history of chronic, mild hindlimb lameness that became acutely severe during a race,[34,35] but lameness in other types of horses may be associated with trauma. Lameness is moderate to severe and usually associated with distention of the tarsocrural joint capsule. Intra-articular analgesia may improve lameness but rarely removes it completely. Fractures in racehorses usually originate at the proximal aspect of the sagittal groove of the talus and are often incomplete. Non-displaced fractures can be difficult to identify radiographically; extra views, including a dorsal 10° to 20° lateral-plantaromedial oblique and a flexed dorsoplantar (skyline) view, can be helpful. Fractures following trauma are frequently comminuted, and the degree of damage may not be apparent radiographically. Nuclear scintigraphy can be helpful in confirming the presence of a fracture; there is usually intense focal IRU in the proximal aspect of the talus.[14]

Eleven horses with incomplete sagittal fractures of the talus were treated conservatively with a minimum of 1 month of stall rest followed by small paddock turnout. Seven returned to racing performance within 7 to 8 months after injury; approximately 50% had the same or improved performance.[33] Horses with complete sagittal fractures have been treated by lag-screw compression using two 4.5-mm cortical bone screws.[35] Prognosis may be favorable in horses with simple acute fractures, but the prognosis for horses with comminuted fractures is poor.

Fractures of the Fibular Tarsal Bone

Fractures of the fibular tarsal bone (or calcaneus) are uncommon and usually the result of trauma. Physeal fractures in foals and fractures through the body of the bone are easily diagnosed because of the obvious loss of gastrocnemius muscle function, which results in a dropped-hock appearance. Chip fractures involving the plantar aspect of the calcaneus can be difficult to diagnose unless soft tissue swelling is present or a draining tract secondary to sequestra formation exists. Surgical removal of small fragments may be necessary, depending on size and location of the fragment (intra-articular or extra-articular). Diagnosis is confirmed radiographically. Flexed lateromedial and skyline views of the calcaneus are recommended in addition to standard views (Fig. 45-4). Complete body and physeal fractures can be difficult to reduce and stabilize, but horses can be treated successfully with bone plates and wires by using the tension-band principle. It is not possible to place a bone plate directly caudally because of the presence of the superficial digital flexor tendon, but the plantarolateral or lateral surface of the calcaneus has been used successfully.[35] Conservative therapy using casting methods alone has been unrewarding and is not recommended. Horses with open, comminuted fractures have a grave prognosis and humane destruction should be advised. Prognosis for future performance activity in horses with physeal or full-body fractures is considered poor.

Fracture of the Lateral Malleolus of the Tibia

The lateral malleolus is considered to be the distal end of the fibula and develops as a separate center of ossification that fuses to the distal tibial epiphysis by 1 year of age. The long

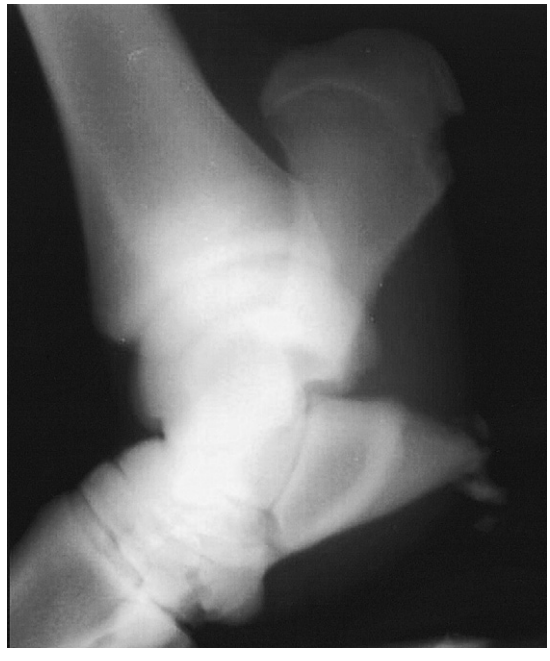

Fig. 45-4 Flexed lateromedial radiographic view of a tarsus. There is a complete, comminuted articular fracture through the calcaneus.

lateral and deeper short collateral ligaments originate on the lateral malleolus of the tibia, just plantar to the groove for the tendon of the lateral digital extensor muscle. Only a small portion of the lateral malleolus is intra-articular; therefore most fracture fragments are located within the joint capsule and collateral ligaments.

Lateral malleolar fractures are usually traumatic in origin. Small, well-rounded fragments, probably a manifestation of osteochondrosis, occur occasionally and should be differentiated because horses are often asymptomatic. Clinical and radiographic signs and results of surgical removal of the fragments were reported in 16 horses.[37] All were TBs with injuries incurred either during a fall in a race over fences or from being kicked. All horses had a moderate degree of lameness and tarsocrural effusion. Approximately 50% of the horses had periarticular swelling, thickening of the collateral ligament, and pain on digital palpation of the lateral malleolus. All fractures were visible on a dorsoplantar radiographic view (Fig. 45-5). Fourteen fractures were unilateral and two bilateral, with nine simple and nine comminuted. Thirteen of 16 fragments were displaced distally and rotated 90°.

Ultrasonographic evaluation is useful to determine the location of the fracture in a dorsoplantar plane and to identify a concurrent collateral desmitis. Small or minimally displaced fractures have been successfully managed conservatively. Surgical removal may result in a quicker recovery. The tarsocrural joint should be inspected arthroscopically to remove any debris, and surgical removal of some fragments is possible, but in some horses the fragment is removed via an incision through the lateral collateral ligament of the tarsocrural joint, depending on fragment orientation. If the fracture fragment is of adequate size, lag-screw fixation can be used. Thirteen of 16 horses treated by fragment removal returned to full athletic function.[37] With conservative or surgical management the prognosis is good.

Tarsal Joint Luxation

Complete luxation or subluxation of the tarsocrural, talocalcaneal-centroquatral, and tarsometatarsal joints may occur, with or without concurrent tarsal bone fractures.[38,39] Tarsal luxations are the result of severe trauma from kicks from other horses or limb entrapment in fixed objects, such as a fence or cattle guards. Proximal intertarsal and tarsometatarsal luxations are most common. Luxation causes severe, non–weight-bearing lameness, and there may be abnormal deviation of the limb at the tarsus. Crepitus may be present with a fracture, and palpation reveals tarsal joint instability. Diagnosis is confirmed by radiography. Dorsoplantar radiographic views obtained with medial or lateral limb traction are recommended to determine the extent of collateral ligament damage (Figs. 45-6, 45-7, and 45-8). Treatment consists of reduction of the luxation with the horse under general anesthesia and stabilization with the application of a full hindlimb cast extending from the foot to a point level with the tibial tuberosity. Internal fixation is rarely indicated but may be necessary with concurrent tarsal bone fracture. Tarsocrural or tarsal luxation with functionally intact collateral ligaments can be difficult to reduce, but manipulation of the tarsus with the limb in a flexed position may facilitate reduction. The limb should maintained in a cast for 4 to 6 weeks if the collateral ligaments are intact and 6 to 8 weeks if the collateral ligaments are ruptured.[38,39] After the cast is removed, the hindlimb is placed in a heavy cotton bandage with rigid splints for an additional 4 to 6 weeks before turnout in a small area is allowed. Of the seven reported horses with tarsal joint luxation (three proximal intertarsal, three tarsometatarsal, and one tarsocrural), all were treated with closed reduction and external coaptation, and all returned to either light riding, pasture soundness, or breeding activity. The presence of tarsal bone fractures did not affect outcome. All horses had a reduction in range of motion of the tarsus and osteoarthritis that

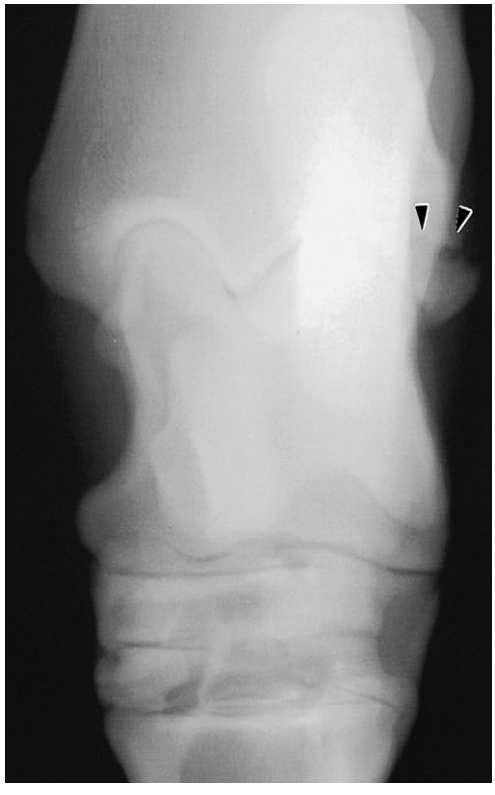

Fig. 45-5 Dorsoplantar radiographic view of a tarsus. There is a fracture of the lateral malleolus *(arrows)*.

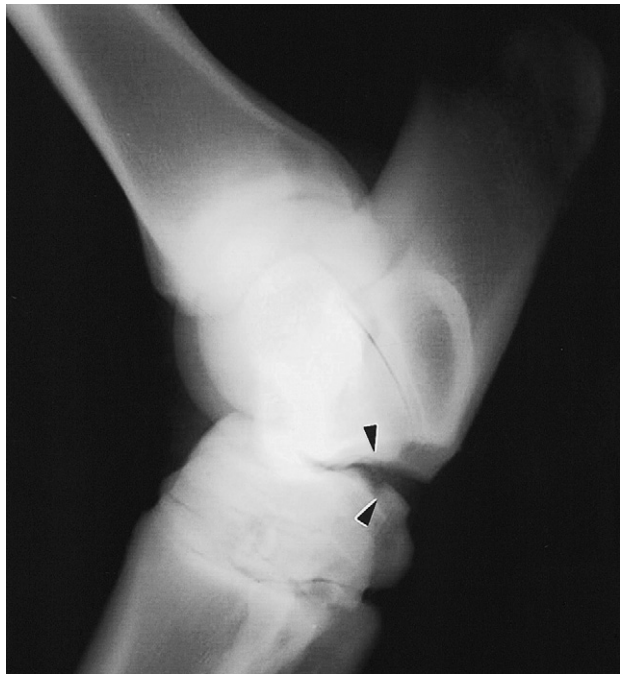

Fig. 45-6 Flexed lateromedial view of a hock. There is widening of the plantar aspect of the talocalcaneal-centroquatral joint *(arrows)*, which is indicative of luxation.

prevented the return to athletic soundness. Prognosis for horses with proximal intertarsal and tarsometatarsal joint luxations is reasonably good for pasture soundness.[38] A guarded prognosis is warranted for tarsocrural joint luxation because of the difficulty in reducing the luxation.

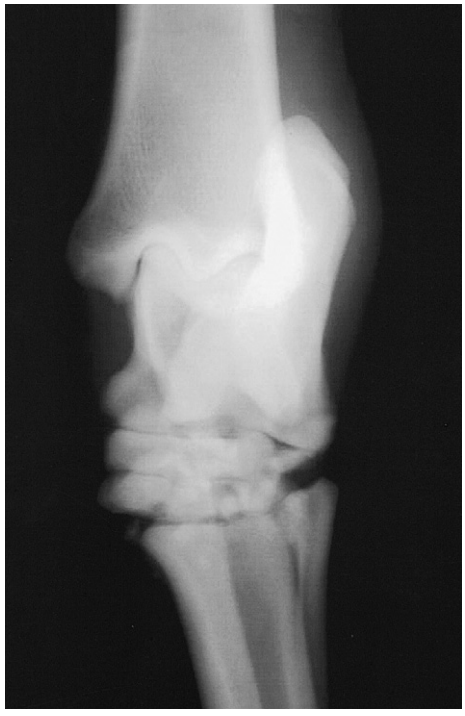

Fig. 45-7 Stressed dorsoplantar view of a hock. There is luxation of the tarsometatarsal joint, which is associated with fracture of the third and fourth tarsal bones.

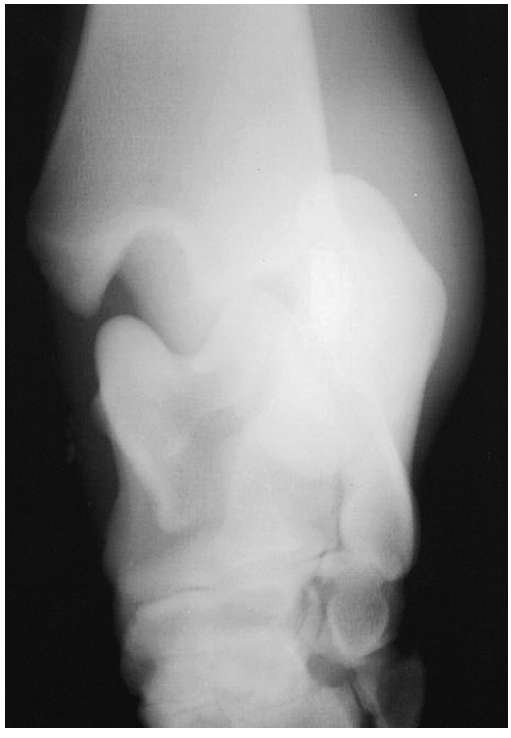

Fig. 45-8 Stressed dorsoplantar view of a hock. There is luxation of the tarsocrural joint.

SOFT TISSUE INJURY OF THE TARSUS

Various soft tissue injuries of the tarsus are discussed elsewhere: calcanean bursitis (see Chapter 80), cunean tendon bursitis-tarsitis (see Chapter 109), curb (see Chapter 79), subcutaneous calcaneal bursitis (capped hock; see Chapter 80), distention of the tarsal sheath (thoroughpin; see Chapter 77), luxation of the superficial flexor tendon from the tuber calcanei (see Chapter 81).

Collateral Ligament Damage

Complete extension of the tarsocrural joint is prevented by tension in the collateral ligaments. Collateral ligament injury is usually the result of trauma or a fall. Lameness varies from mild to severe, depending on the degree of damage. There is often distention of the tarsocrural joint capsule and peri-articular soft tissue swelling. Flexion is usually resented. Analgesic techniques are usually not required, but lameness may be improved in some horses by intra-articular analgesia of the tarsocrural joint. In an acute injury there often are no radiographic abnormalities; however, 4 to 6 weeks after injury, entheseophytes develop at the origin or insertions of the injured collateral ligaments. Nuclear scintigraphy is helpful in horses with mild injuries, because it demonstrates intense, focal IRU at the ligament attachment sites.[40] Ultrasonographic evaluation is used to identify the injured structure(s) and assess the severity of the injury (see Chapter 17). Initial treatment involves stall rest, cold water therapy, topical dimethylsulfoxide with or without corticosteroids, and NSAIDs. Long periods of rest (e.g., 6 months) followed by a controlled exercise program have been recommended.[40] Prognosis depends on the severity of injury, but horses with very extensive periosteal new bone have been able to return to full athletic function, although some residual enlargement of the hock has persisted.[23]

Entheseopathy of the Lateral Collateral Ligaments of the Tarsocrural Joint

Entheseopathy of the long, and less commonly, the short lateral collateral ligaments is a rare condition that is recognized most often in STB racehorses, especially pacers, and results in lameness that is improved by analgesia of the tarsocrural joint. There may be distention of the tarsocrural joint capsule, and subtle, localized soft tissue swelling may be present. Diagnosis usually depends on scintigraphic findings[41] that show focal, intense IRU on the lateral aspect of the calcaneus. Entheseous new bone is usually detectable radiographically. Local injections with methylprednisolone acetate and Sarapin, an extract of the pitcher plant, may result in improvement, but rest is the treatment of choice.

Rupture of the Fibularis Tertius

See Chapter 85 for a discussion of rupture of the fibularis (peroneus) tertius.

Stringhalt

See Chapter 49 for a discussion of stringhalt.

MISCELLANEOUS TARSAL INJURIES

Osteitis of the Calcaneus

Osteitis of the tarsal bones is uncommon. However, the enlarged end of the tuber calcanei has minimal soft tissue protection and is at risk of traumatic injury and subsequent infection. Management of these injuries can be complicated if the tarsocrural joint, calcaneal bursa, or tarsal sheath is involved. Most horses sustain a traumatic injury from kicking a fixed object or being kicked by another horse.[42] Osteitis usually

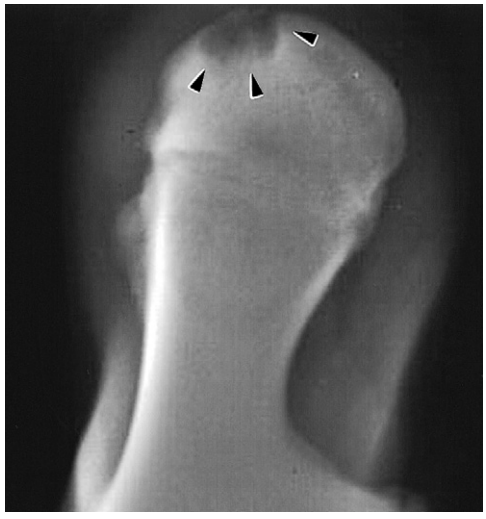

Fig. 45-9 Flexed plantaroproximal-plantarodistal radiographic view of the calcaneus. There is a osteolytic lesion in the tuber calcanei *(arrows)*. There is also new bone on the lateral aspect (to the left) of the calcaneus.

causes moderate to severe lameness, cellulitis, and tarsal tenosynovitis. The severity of lameness often decreases once drainage is established. Twenty-two of 28 horses had a wound or draining tract over the plantar aspect of the calcaneus.[42] Lateromedial, flexed lateromedial, and plantaroproximal-plantarodistal (skyline) radiographic views are the most helpful (Fig. 45-9). Radiographic signs of calcaneal osteitis or sequestra formation may take several days or weeks to appear, and thus sequential radiographic evaluation is recommended. Rarely is the medullary cavity of the calcaneus involved; hence the term *osteitis* rather than *osteomyelitis* is preferred. Ultrasonography is useful to assess calcanean bursa or tarsal sheath involvement. If tarsal sheath involvement is suspected, cytological evaluation of the tendon sheath fluid with aerobic and anaerobic microbial culture and susceptibility is recommended (see Chapter 77). Treatment is based on resolving the infection and debridement of any bony lesions. Broad-spectrum antimicrobials such as penicillin (22,000 IU/kg q12h IM) and gentamicin sulfate (6.6 mg/kg q24h IV) are recommended until culture results are known. Pure bacterial cultures were isolated in 14 of 28 horses in one report,[42] but mixed bacterial isolates do occur. The most common pure bacteria colonies isolated were *Escherichia coli*, *Streptococcus zooepidemicus, Staphylococcus aureus,* and *Enterobacter cloacae.* All isolates were susceptible to penicillin, gentamicin, or trimethoprim sulfamethoxazole. With acute infection, antimicrobial medication alone may be satisfactory, but surgical intervention to remove infected soft tissue and damaged bone or sequestra is usually required. Resection of the infected soft tissues and curettage of the bony lesion can resolve calcaneal lesions that do not involve the tarsal sheath. Infections involving the tarsal sheath require flushing the sheath and establishing drainage (see Chapters 24 and 77). Duration of antimicrobial therapy depends on clinical response, but should continue for at least 7 days after resolution of the lameness and wound drainage. Phenylbutazone (2.2 mg/kg PO) should be used as needed for pain relief.

The prognosis for horses with calcaneal osteitis depends on the structures involved and duration of infection before treatment. Quick and aggressive medical and surgical treatment improve the chances of horses returning to athletic function. In one study, 9 of 18 horses were used as broodmares and 9 returned to athletic function.[42]

Osteitis of the Sustentaculum Tali

The sustentaculum tali is on the plantaromedial aspect of the calcaneus. Injuries involving the medial aspect of the hock often involve the sustentaculum tali and adjacent tarsal sheath. Injury usually occurs from direct trauma, most commonly from a kick wound. Lameness is often severe. Diagnosis can be difficult because of the severe soft tissue swelling that accompanies this injury, especially when acute. Although unusual there can be communication of the site of infection or fragmentation of the sustentaculum tali with the tarsocrural joint. Radiographic examination should include dorsomedial-plantarolateral oblique, dorsoplantar, and plantaroproximal-plantarodistal views. Ultrasonography is useful to assess the deep digital flexor tendon (DDFT) and tarsal sheath. Infection of the tarsal sheath as a result of sustentaculum tali fragmentation (see Fig. 77-3) or lysis requires surgical curettage of the sustentaculum tali and lavage and flushing of the tarsal sheath. Horses with bony irregularities in the tarsal groove of the sustentaculum tali can be severely lame, even without infection, because of motion of the DDFT against the roughened bone, which results in severe tendonitis. The tarsal retinaculum can become constrictive if the tendon is inflamed and swollen, which causes further tendon damage and pain. Surgery with the horse under general anesthesia is recommended to transect the tarsal retinaculum, explore and curette necrotic bone involving the sustentaculum tali, and drain and flush the tarsal sheath.[43] The tarsal groove should be smoothed with a curette to avoid abrasion to the DDFT after surgery. Endoscopic examination of the tarsal sheath may be difficult in horses with severe swelling from concomitant infection. In horses with mild swelling, drainage localized to the region of the sustentaculum tali and without substantial tarsal tenosynovitis, a small incision should be made directly over the fragments. The fragments should be removed and the site curetted; the incision is closed primarily. In these horses, prognosis for future soundness is favorable. Horses should be treated with systemic antibiotics based on culture and susceptibility results. Antimicrobial therapy is continued for 7 days after resolution of clinical signs and may require 4 to 6 weeks of treatment. After surgery, horses should be confined to a stall or small run for 8 weeks with daily hand walking, followed by 6 more weeks of pasture turnout before returning to normal activity. Prognosis is good for returning to athletic activity. Seven of nine horses returned to previous use (three barrel racing, one cutting, and three pleasure riding) after surgery.[43]

Infectious Arthritis

See Chapter 66 for a discussion of infectious arthritis.

Peri-Articular Cellulitis

Peri-tarsal infection results in acute-onset, very severe non–weight-bearing lameness associated with localized swelling that is usually but not invariably on the dorsal aspects of the hock. The swelling is hot and exquisitely painful to palpation. These signs are associated with an elevated rectal temperature, neutrophilia, and hyperfibrinogenemia. In some horses, skin abrasions can be identified somewhere in the more distal part of the limb.

Prompt, aggressive treatment with oxytetracycline (5 mg/kg IV sid) or other antimicrobial agents and phenylbutazone (4.4 mg/kg) for at least 5 days, combined with forced walking exercise for as much as possible daily, usually results in rapid resolution of lameness. Horses can often start trotting within 3 days of the onset of clinical signs and should be trotted for 10- to 15-minute periods repeatedly through the day. The prognosis is usually favorable; 1 of 10 horses developed osteomyelitis of the third tarsal and fourth tarsal bones, although this horse ultimately made a complete recovery.[44]

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CHAPTER • 46

The Crus

Mike W. Ross

ANATOMY

In the horse the crus is located between the stifle and hock joints. Anatomy of the hock and stifle are discussed in Chapters 45 and 47. The medial tibia lacks muscle covering and is easily palpated, but muscles and tendons cover the cranial, lateral, and caudal aspects. The tibia has proximal and distal physes and a separate center of ossification for the tibial tuberosity.

The fibula is lateral, does not share axial load, and fuses with the tibia to form the lateral malleolus. The proximal fibula may develop from two or more separate centers of ossification, and fibrous union may persist throughout life. The result of this union is transverse radiolucent lines that are evident radiographically and should not be confused as fractures.[1,2]

Muscles and tendons of the crus are important in locomotion and support of the hindlimb. The long digital extensor muscle originates from a common tendon with the fibularis tertius from the extensor fossa of the femur and is located cranially and laterally in the crus. The fibularis tertius courses distally to divide into dorsal and dorsolateral tendons of attachment. Avulsion injury of the tendon of origin of the fibularis tertius and long digital extensor causes a classic disruption of the hindlimb reciprocal apparatus. The cranialis tibialis muscle is deep to the long digital extensor muscle and distally splits into two parts, the medial of which is called the *cunean tendon* (jack tendon) and may play a minor role in horses with distal hock joint pain. The lateral digital extensor muscle originates proximally and its tendon courses laterally over the tarsus and joins the long digital extensor muscle in the proximal metatarsal region. Myotenectomy of the lateral digital extensor tendon and muscle is performed for management of stringhalt.

The paired gastrocnemius muscles originate from the distal, caudal femur and share a single, strong tendon that courses distally and inserts on the calcaneus. The superficial digital flexor and deep digital flexor muscles and tendons arise caudally. The superficial digital flexor tendon (SDFT) begins medial to the gastrocnemius tendon and courses from medial to caudal to attach to the calcaneus before continuing distally. The deep digital flexor tendon (DDFT) is deep to both the SDF and gastrocnemius tendons and courses distally over the plantaromedial aspect of the calcaneus, the sustentaculum tali. The combined SDFT and gastrocnemius tendons form the common calcaneal tendon, the major extensor of the hock, and injuries can cause partial or complete loss of hock support. The tarsal sheath begins in the distal, caudal aspect of the crus and surrounds the DDFT.

The fibular (peroneal) nerve originates from the sciatic nerve and branches in the proximal crus to become the superficial and deep fibular nerves that run between the long digital extensor and lateral digital extensor muscles and tendons. The tibial nerve is palpable in the distal, caudal aspect of the crus, cranial to the common calcaneal tendon.

CLINICAL CHARACTERISTICS AND DIAGNOSIS OF LAMENESS OF THE CRUS

Degree of lameness can vary from the subtle, high-speed lameness seen in horses with early stress-related bone injury to acute, non–weight-bearing lameness, swelling, and deformity seen in horses with complete tibial fractures. Horses with tearing of the gastrocnemius tendon, musculotendonous junction, origin, and insertion have varying degrees of hyperflexion of the tarsocrural joint and partial loss of the reciprocal apparatus. Because the hock drops during weight bearing, the degree of pelvic hike is less than expected and the degree of lameness may be underestimated. Foals with fibularis tertius and long digital extensor avulsion injury lose integrity of the reciprocal apparatus and have swelling on the lateral, proximal aspect of the crus. Fibularis tertius injury in adult horses may cause swelling of the distal, cranial aspect of the crus near the tarsocrural joint, but lesions in the mid-crus are not associated with swelling. Calcinosis circumscripta, which are round to oblong, non-painful mineralized masses, are attached to the distal aspect of the lateral patellar ligament and lateral femorotibial joint capsule or collateral ligament.

No gait characteristics are pathognomonic of pain associated with the crus. Horses with tibial stress fractures have a shortened cranial phase of the stride and, most often, a stabbing type of hindlimb gait when viewed from behind. Injuries of the crus are suspected when lameness is pronounced, but sites in the rest of the limb are eliminated by using diagnostic analgesia. In Thoroughbreds (TBs), in which the risk of tibial stress fractures is high, a tentative diagnosis of stress fracture is made when lameness is pronounced and recurrent after work.

Palpation of the crus should be done with the limb in both the standing and flexed positions, but this often yields no information. The medial side of the tibia is most easily felt, and occasionally in horses with tibial stress fractures there is mild swelling and pain is elicited by deep compression. Unfortunately, most tibial stress fractures involve the caudolateral cortex, and it is difficult to compress this area during palpation. Digital tibial percussion sometimes elicits a painful response in horses with tibial stress fractures, but there are many false-positive and false-negative responses.

Diagnostic Analgesia

There is no practical method to use diagnostic analgesia in the entire crus. The distal portion is blocked when fibular and tibial nerve block is performed, and pain from injuries involving the distal tibia and caudal soft tissues may be abolished, but this block is unreliable. Lameness of the crus becomes more likely in horses in which perineural and intra-articular techniques for the rest of the limb have been exhausted. In at least three TB racehorses, pain associated with tibial stress fractures has been abolished or diminished by intra-articular analgesia of the femorotibial joint. An explanation is not readily apparent.

Imaging Considerations

Large cassettes and film (35 × 43 cm) should be used to obtain radiographs of the entire length of the tibia. There are normal areas of modeling involving the cranial cortex of the tibia that appear as layers or a mound of bone, but stress fractures do not occur here. Occasionally, an obvious bony proliferation is seen involving the caudal or caudolateral tibial cortex, under the fibula in normal horses. Periosteal and endosteal proliferation of the caudal and lateral (rarely medial cortex) and oblique linear radiolucency are changes that may be seen in horses with tibial stress fractures. Enostosis-like lesions appear as single or numerous medullary radiopacities. In some horses the fibula has one or more transverse radiolucent lines through the proximal one third of the bone that should not be mistaken for fractures. Stress-related bone injury of the tibia is most easily imaged and diagnosed using scintigraphic examination. Without scintigraphy the diagnosis can be easily missed radiographically. Enostosis-like lesions may be associated with single to numerous areas of increased radiopharmaceutical uptake (IRU) in the medullary cavity and should be differentiated from the cortical uptake associated with tibial stress fractures.

Ultrasonographic examination is useful in evaluating the gastrocnemius muscle and tendon. Patellar desmitis at the attachments can be diagnosed by using ultrasonographic and scintigraphic examinations. Ultrasonographic examination is useful in horses with fibularis tertius injury, thoroughpin or "false" thoroughpin, and to evaluate the tarsal sheath (see Chapter 6).

SPECIFIC CONDITIONS OF THE CRUS

Tibial Stress Fractures

Tibial stress fractures are the most common lameness condition of the crus and occur most commonly in TB racehorses. In my experience, tibial stress fractures are rare in other sport horses, including the Standardbred (STB) racehorse. In an 8-year period, of 1020 STBs in which scintigraphic examination was performed, only 3 horses (2 of which were trotters) had tibial stress fractures.[3] Thirteen STB racehorses, 11 pacers and 2 trotters, with tibial stress fractures were reported in one study,[4] but based on my experience, this is a highly unusual clustering of horses. Pacers were over-represented and factors such as breeding, track size, training methods, and referral bias may have played a role. The caudal tibial cortex appears prone to stress-related bone injury because it is under compressive forces when loaded. The highest compressive forces were recorded in the mid-diaphysis at the walk, but loading at the gallop, pace, and trot was not determined.[4-6]

Tibial stress fractures usually occur in 2- and 3-year-old racehorses. Tibial stress fractures occur later in training than stress fractures of the humerus, can occur when horses are racing, and also occur in older horses. Usually lameness is unilateral. Stress-related bone injury is more advanced in the lame limb, but scintigraphic evidence of stress-related bone injury can be bilateral. Typically TB racehorses with tibial stress fractures usually become acutely lame after training or racing, only to become reasonably sound within 3 to 5 days. Lameness recurs after another work session or race, but overt clinical signs other than lameness are subtle or lacking. Horses with pelvic stress fractures and those with stress-related bone injury of the distal third metatarsal bone manifest similar signs. Lameness may be severe initially, but within a few days horses can be trotted and show grade 2 to 4 lameness (of 5). Occasionally, severe non–weight-bearing lameness is present, which usually indicates the presence of a spiral fracture.

Tibial stress fractures are seen as focal areas of IRU of the caudal or caudal lateral tibial cortex and are usually singular, but can be multiple and bilateral (see Chapter 19). Caudomedial tibial stress fractures occur rarely. In horses in North America, tibial stress fractures are usually located from mid-diaphysis to the distal tibia, but occasionally a fracture is seen in the proximal, caudal metaphyseal region. In TBs racing in Europe the proximal caudal site is affected more commonly.[7] Occasionally a spiral fracture of the distal tibial cortex is seen. In general, intensity of IRU is inversely proportional to the amount of radiographic change. In horses with stress-related bone injury a continuum of bone changes occur that precede and eventually lead to fracture. If radiographs show proliferative changes and an oblique fracture line, IRU is usually mild to moderate. In horses that develop sudden, severe lameness, focal or spiral intense IRU is seen, but radiographic changes are equivocal or mild. Authentic tibial stress fractures do not occur in the cranial cortex, but IRU from a previous fibular nerve block can produce an artifact resembling fracture (see Fig. 19-15).

Most horses are given 4 weeks of stall rest, followed by 4 weeks of stall rest with hand walking, then 8 weeks of turn out in a small paddock before returning to race training. Earlier return to race training predisposes to recurrence of stress-related bone injury and fracture. Fracture of the contralateral limb the next racing year is possible, but recurrence of ipsilateral fracture is unusual, unlike recurrence seen early in training in horses with humeral stress fractures. Horses with severe lameness may have difficulty rising in the stall and should be bedded on good footing or kept in the standing position for several weeks by the use of cross ties or other suitable restraints.

Tibial Diaphyseal Fractures in Adult Horses

Tibial fractures in adult horses occur from trauma from falling, a spill sustained while performing, being kicked, or while attempting to rise after general anesthesia. Horses with tibial stress fractures, usually those with severe initial lameness or spiral fracture, can develop comminuted fractures if turn-out exercise is given too soon, or if a horse struggles to rise in a stall. One of the only reported successful repairs of an adult horse with a displaced tibial fracture was performed after the horse was anesthetized for radiographs of the pelvis and coxofemoral joint only to develop a displaced, closed tibial fracture as a result of displacement from a tibial stress fracture. The injury was repaired using two dynamic compression plates and bone screws.[8] In general, however, prognosis is grave for adult horses with displaced, comminuted tibial fractures, because comminution is severe, additional fracture lines often propagate proximal and distal from the fracture site, fractures are often open, and implant failures are common (Fig. 46-1). Prognosis for horses with comminuted fractures involving the distal tibia and tarsocrural joint is hopeless. A horse with a closed, simple, or mildly comminuted mid-shaft oblique or transverse fracture may be a candidate for an attempt at internal fixation. However, prognosis is poor to grave, because even with double-plating or triple-plating techniques, implant failure is common and fixation usually fails during anesthetic recovery even with a pool recovery system. Given the extremely poor prognosis, transportation of a horse with a flail leg should be avoided. Most horses should be immediately destroyed.

Tibial Fractures in Foals

Suckling and weanling foals with displaced, comminuted tibial fractures have a reasonable prognosis for salvage and some become performance horses and racehorses. The size of the foal is critical, and complications in those heavier than 225 to 325 kg may be similar to adult horses. Most foals with tibial fractures sustain kick trauma from mares or have other accidents.

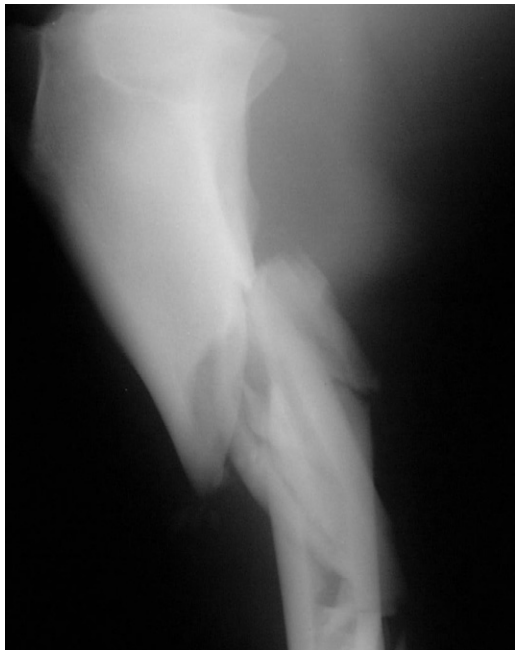

Fig. 46-1 Lateromedial radiographic view of an adult tibia. There is a typical comminuted tibial fracture. The prognosis is grave and the horse should be humanely destroyed.

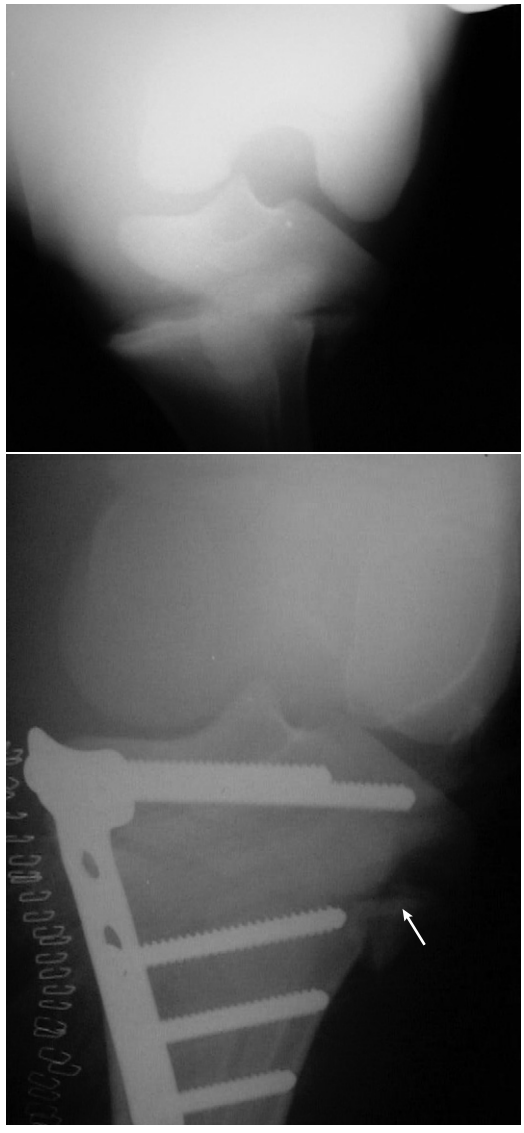

Fig. 46-2 Caudocranial radiographic projection of a foal with a Salter-Harris type II fracture of the proximal tibial physis before **(A)** and after **(B)** surgery. Note the lateral metaphyseal component *(arrow)*.

Proximal Physeal Tibial Fractures

Salter-Harris type II fractures occur in sucklings, weanlings, and rarely in yearlings. These fractures result in acute-onset lameness and swelling of the proximal, medial aspect of the crus and stifle. Within 1 to 3 days, foals are often weight-bearing and may be surprisingly comfortable. Radiographs reveal a simple Salter-Harris type II fracture with a lateral metaphyseal component (Fig. 46-2). Occasionally mild comminution exists laterally. Foals with minimal displacement can be managed conservatively, but progressive displacement, as evidenced by valgus deformity, usually occurs. Foals should be treated surgically. Many methods have been used, but the most stable repair is a medial approach, in which either a T plate or two short dynamic compression plates should be applied by using 5.5-mm bone screws in the epiphyseal component. A long metaphyseal component can be engaged using screws placed in lag fashion. The tension side of the proximal tibia is medial and the T plate should be applied in this location. Because the soft tissue covering is minimal, chronic drainage from the wound is common until the plate(s) is removed. Prognosis for life is good to excellent and for soundness is fair to good. Prognosis for racing is not established and sequelae from differences in limb length are unknown, but racing is not out of the question.

Mid-diaphyseal Tibial Fractures

Similar considerations for degree of comminution, location of fracture, and whether the site is open or closed apply to foals and adult horses, but in foals prognosis associated with repair is considerably better than in adults. Foals with closed, mid-shaft, transverse, or oblique fractures with minimal comminution are the best surgical candidates, but successful repair of those with comminution has been achieved (Fig. 46-3). Foals with open fractures that are associated with a small wound and minimal contamination are also surgical candidates. A cranial approach is used to place two dynamic compression plates on the craniolateral and craniomedial aspects and additional screws to repair loose fragments. In nine foals whose injuries were repaired in this manner, results were considered excellent in six, and good or fair in two.[9] If fractures heal, implants should be removed, and if contralateral angular deformity does not develop, prognosis for racing should be fair to good.

Distal Physeal Fractures of the Tibia

Distal physeal fractures of the tibia do occur but are rare. There is acute severe lameness, swelling, and angular and rotational deformity of the limb. Salter-Harris type II fractures are most common, but the small size of the epiphysis complicates surgical repair. Substantial tibial shortening can be expected and prognosis is poor.

Tibial Malleolar Fractures

Medial malleolar fractures are rare but occasionally occur from trauma and may be associated with substantial collateral ligament injury. When collateral ligament injury is minimal, fragments can be removed arthroscopically, but visibility can be difficult without proper instrumentation. After fragments are removed, the arthroscope can usually be advanced to the plantar pouch. Prognosis is good in horses without substantial collateral ligament damage and tarsocrural instability. However,

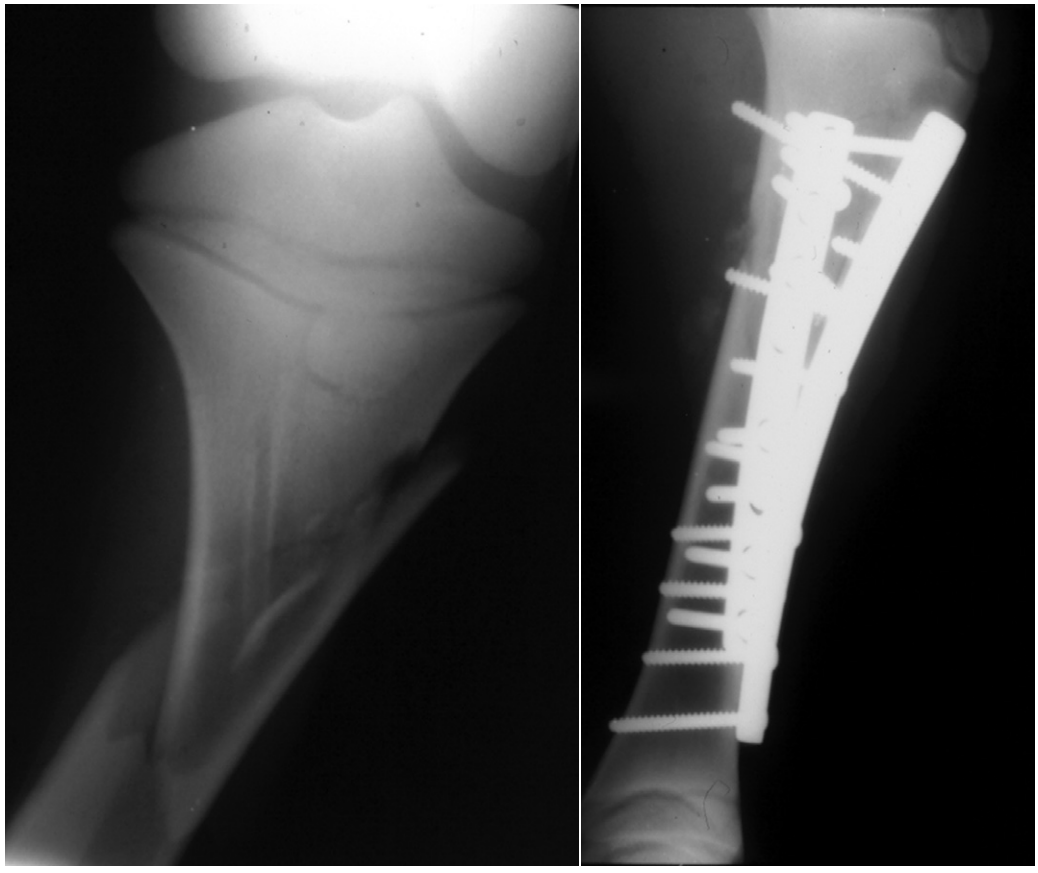

Fig. 46-3 A, Preoperative and B, postoperative craniocaudal radiographic views of a 2-month-old Standardbred foal with a diaphyseal tibial fracture.

if there are additional loose fragments within the joint, this may indicate further damage of the tarsocrural joint.

See Chapter 45 for a discussion of lateral malleolar fractures.

Tibial Tuberosity Fractures

Direct trauma causes tibial tuberosity fractures, which occur most often in field hunters and jumpers. Fractures are usually non-articular and can vary in length, width, and depth. In most horses with injuries that are managed conservatively, fractures heal functionally but without radiographic evidence of union. Surgical repair of non-articular fractures in six horses resulted in a successful outcome.[10] Large fractures involving the femorotibial joint surfaces can be repaired, but surgical repair is not necessary in most horses and can be associated with catastrophic fracture of the tibia if occult fracture lines are not recognized and implant failure occurs[11] (see Fig. 47-12). In one study, 10 of 14 horses with non-articular tibial tuberosity fractures that were managed conservatively were sound and horses performed at the expected level[12] (Fig. 46-4).

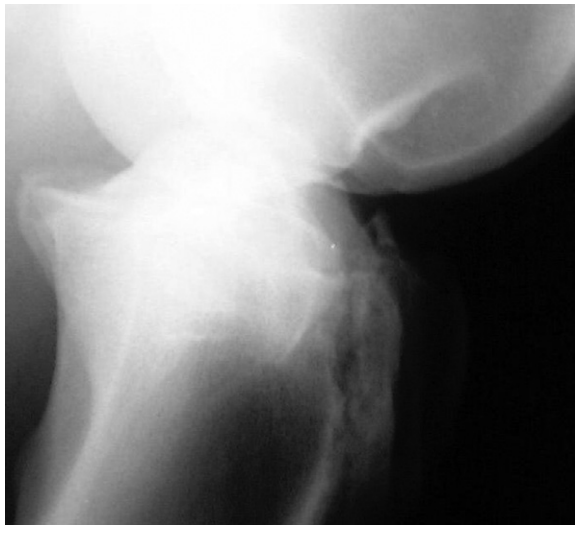

Fig. 46-4 Lateromedial radiographic view of the proximal tibia in a horse with chronic lameness and fracture of the tibial tuberosity. Conservative management of this horse resulted in a successful outcome, although radiographic evidence of fracture union was never achieved.

Enostosis-like Lesions of the Tibia

Enostosis-like lesions of the tibia are diagnosed by scintigraphic and radiographic examinations and can be a cause of chronic, mild hindlimb lameness.

Osteochondroma of the Tibia

Osteochondroma of the tibia is a rare radiographic finding in the distal, caudal tibia and may be incidental or cause tarsal tenosynovitis. I have seen one adult horse with osteochondroma that as a foal had repair of a tibial fracture. The condition has rarely been reported.[13]

Osseous Cyst-like Lesions of the Proximal Tibia

Osseous cyst-like lesions of the proximal tibia are unusual, may be the result of osteochondrosis, and frequently communicate with the lateral femorotibial joint. Some adult horses

develop radiolucent changes caused by osteoarthritis, but in these horses osseous cyst-like lesions are acquired, not developmental. Some adult horses develop sudden-onset lameness associated with a well-established osseous cyst-like lesion that was previously asymptomatic with no evidence of primary osteoarthritis. Clinical signs are similar to those in horses with other subchondral bone cysts or osseous cyst-like lesions of the femorotibial joint, but lameness is often pronounced in adult horses with osteoarthritis. In most horses with osseous cyst-like lesions involving the lateral femorotibial joint, curettage is difficult or impossible without elevating or severing the lateral meniscus. Some of these horses respond to conservative management and intra-articular injections of hyaluronan and corticosteroids. Intralesional injection with corticosteroids has also been reported. In horses with acquired osseous cyst-like lesions related to osteoarthritis, the lesion is usually medial and prognosis is poor because cartilage damage of the proximal tibial condyle and distal medial femoral condyle is usually severe. Of 12 horses with osseous cyst-like lesions, 6 were believed to be the result of osteochondrosis and 3 of the 6 horses that received surgical debridement became athletes. Only 2 of 6 in which osseous cyst-like lesions were caused by osteoarthritis returned to work after surgery.[14]

Osseous Cyst-like Lesions of the Distal Tibia

Osseous cyst-like lesions of the distal tibia are rare injuries and appear to be acquired as the result of trauma or stress-related bone injury. I have seen osseous cyst-like lesions in this location in a jumper and a STB racehorse. In both horses, there was acute-onset lameness and tarsocrural effusion. Scintigraphic examination revealed focal, moderate to intense IRU of the distal tibia. Horses were rested and later performed at a level similar to pre-injury level. Computed tomography was useful in elucidating the lesion in other horses (see Chapter 20).

Physitis of the Distal Tibia

A discussion of physitis of the distal tibia is in Chapters 59 and 129.

Fibular Fractures

Authentic fibular fractures occur rarely and are often fortuitously diagnosed scintigraphically or radiographically when images are obtained in horses with undiagnosed hindlimb lameness or in those with suspected tibial stress fractures. Fractures occur either proximally or in the mid-diaphyseal region and can appear as chronic non-unions with bulbous proliferation on each side of the fracture. Rest is recommended. The entire limb should be evaluated carefully to exclude another site of pain causing lameness. Six horses with proximal fibular fractures made complete functional recoveries; horses were sound within 3 months, but a fracture line persisted radiographically until 4 to 6 months after the injury.[7]

Soft Tissue Injuries of the Crus

Fibularis tertius injury in foals and adult horses is discussed in Chapter 81, and gastrocnemius tendonitis is discussed in Chapter 81. Stringhalt is discussed in Chapter 49.

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CHAPTER • 47

The Stifle

John P. Walmsley

ANATOMY

Developmental Anatomy

The seven centers of ossification in the stifle of the foal are the metaphyses of the femur and tibia, distal femoral epiphysis, proximal tibial epiphysis, patella, tibial tuberosity (the apophysis), and fibula. The proximal tibial physis closes at about 2½ years of age. The tibial apophyseal-epiphyseal physis closes by 1 year of age. The apophysis does not fuse with the metaphysis until 3 years of age. This apophysis is an important radiographic feature in a young horse and can be mistaken for a fracture. The distal femoral physis closes by about 2½ years of age. In young foals the margins of the femoral trochleas and the patella are irregular for the first 3 months of life because of incomplete ossification. The fibula is not evident radiographically until about 2 months of age, and a high percentage of adult horses have one and occasionally up to three horizontal radiolucent lines in the fibula distal to its head. These should not be confused with fracture lines.[1]

Reciprocal Apparatus

The reciprocal apparatus has a significant influence on the action of the stifle. Extension of the joint exerts a pull on the superficial digital flexor tendon, which originates in the supracondyloid fossa of the femur and is mostly tendonous. Because this flexor tendon has an insertion on the calcaneus, the hock must extend simultaneously with the stifle. On the cranial aspect of the stifle the fibularis (peroneus) tertius, also a tendonous structure, originates in common with the long digital extensor tendon between the lateral trochlea and the lateral condyle of the distal femur.[2] The fibularis tertius passes through the extensor sulcus in the lateral part of the tibial head, associates closely with the deeper tibialis cranialis, and inserts on the third tarsal and third metatarsal bones and the fourth tarsal bone and calcaneus. Flexion of the stifle necessitates hock flexion because of the action of the fibularis tertius.[3] Weight bearing is achieved without muscular effort by the parapatellar fibrocartilage of the medial patellar ligament hooking over the medial femoral trochlea by contraction of the quadriceps femoris. The patella is released by contraction of the quadriceps femoris, combined with the lateral pull of the tensor fascia latae and biceps femoris.

Femoropatellar Joint

The articulation between the patella and the trochleas of the femur forms the femoropatellar articulation. The patella has three straight ligaments. The medial patellar ligament attaches to the medial border and the base of the patella through the parapatellar fibrocartilage. Arthroscopically the ligament can be viewed beneath the joint capsule. Distally the medial patellar ligament attaches medial to the groove on the cranial aspect of the tibial tuberosity. The middle patellar ligament originates on the cranial part of the patella just proximal to the apex and inserts in the distal part of the groove of the tibial tuberosity. The lateral patellar ligament extends from the lateral aspect of the patella to the lateral part of the tibial crest. The biceps femoris has a tendon of insertion on this ligament.[3] Femoropatellar ligaments also reinforce the joint capsule medially and laterally, the lateral ligament being the more distinct.

The medial trochlear ridge of the femur is larger and more rounded than the lateral trochlear ridge and articulates with the medial part of the patella and the fibrocartilage of the medial patellar ligament. The joint capsule has a large suprapatellar pouch and inserts abaxially on the trochlear ridges forming lateral and medial recesses, the lateral being the smaller. A large fat pad is cranial to the joint capsule, proximal and distal to the patella. In my experience, most horses have a slit-like opening into the femorotibial joint at the distal end of the medial trochlear ridge and frequently the lateral trochlear ridge as well. Latex passes from the femoropatellar to the medial femorotibial joint in 60% to 65% of horses and in the reverse direction in 80% of horses.[3] Diffusion of mepivacaine between all the compartments may occur in about 75% of horses.[4]

Femorotibial Joints

The medial and lateral femorotibial joints are separate compartments, which are divided by an intact median septum in a healthy joint but which may communicate after trauma.[5] The crescent shaped, fibrocartilaginous medial and lateral menisci lie between the respective femoral and tibial condyles to form a congruent articulation.[2] Both are attached to the tibia, cranial to the intercondylar eminences, by cranial ligaments. The medial ligament wraps around the cranial aspect of the medial intercondylar eminence before inserting on the tibia. The medial meniscus is also attached caudally to the medial intercondylar eminence by the caudal ligament, which can be seen arthroscopically in the caudal part of the femorotibial joint. The lateral meniscus attaches caudally to the popliteal notch of the tibia and through the strong meniscofemoral ligament to the caudal part of the intercondylar notch of the femur. Only the cranial and caudal poles of the menisci can be viewed arthroscopically because of the close apposition of the tibia and femur, but the respective cranial ligaments are clearly visible. During flexion the menisci slide caudally, and during extension they slide cranially.

The cranial cruciate ligament has its tibial attachment cranial to the medial intercondylar eminence, and its femoral attachment in the lateral part of the intercondylar notch. The cranial cruciate ligament lies beneath the median septum and usually cannot be directly viewed arthroscopically without removing the septum. A better view is often gained from the lateral compartment. The caudal cruciate ligament originates in the popliteal notch of the caudal tibia and runs proximally, medial to the cranial cruciate ligament, to insert cranially in the intercondylar notch of the femur.[7] The ligament can be viewed beneath the septum in the cranial and caudal medial compartment of the femorotibial joint. In the dog the cranial cruciate ligament is under tension during extension of the femorotibial joint,[8] and this may be so in the horse.

The collateral ligaments both originate proximally on the respective epicondyles of the femur. The medial collateral ligament inserts distally on the tibia distal to the medial condyle and has attachments to the medial meniscus. The lateral collateral ligament lies over the popliteal tendon and inserts distally on the head of the fibula. The popliteal tendon originates close to the lateral collateral ligament on the femur and courses distally and caudally, in close apposition to the femoral condyle, to the triangular insertion of the muscle on the caudal aspect of the proximal tibia. The tendon is viewed arthroscopically in the cranial aspect of the lateral femorotibial joint and also in the caudal part, which it effectively divides, limiting the arthroscopic accessibility. The tendon of origin of the long digital extensor muscle can be followed arthroscopically from its origin to the extensor notch of the femur and is usually invested within the joint capsule, although in some traumatized joints, the tendon appears separate.[5]

DIAGNOSIS

General Considerations

The history may give an important lead to diagnosing the cause of lameness. For example, a young horse is a candidate for osteochondrosis or subchondral cystic lesions. Acute-onset stifle lameness in a horse at pasture or during work is more likely to be a traumatic injury involving ligaments or bone. Sudden reduction of work or poor condition may predispose to upward fixation of the patella. Palpation of the patellar ligaments and the outline of the patella, collateral ligaments, long digital extensor tendon, tibial crest, and medial and lateral tibial condyles should be possible. Many horses with stifle injuries manifest no abnormalities on physical examination of the joint. If severe trauma has occurred, the whole region may be swollen, making appreciation of the individual structures difficult. The horse may guard the limb so strongly that instability may not be obvious. Because of the ligamentous structures around the joint, distention is only readily palpable over the cranial aspect of the femoropatellar joint and over the medial femorotibial joint cranial to the medial collateral ligament.

Gait and Manipulative Tests

Differentiating stifle lameness in the horse by studying the gait is difficult, because the reciprocal apparatus coordinates the movement of the whole limb. In my view attributing the cause of certain gait changes to stifle pain is not possible. Some horses with stifle pain may carry the stifle slightly abducted, but this is not specific. A careful analysis of the whole limb is required to establish the site of pain. Other gait changes that may be seen with stifle lameness, but which are also not specific, are a reduced cranial phase of the stride and a reduced flexion of the limb in flight. Many horses with stifle pain dislike going downhill. Horses with delayed release or upward fixation of the patella tend to avoid fully extending the limb and appear to have a crouching gait. Flexion of the upper limb exacerbates lameness in horses with stifle lameness and abduction of the limb may be painful. When performing proximal limb flexion tests, holding the limb at mid-metatarsal level, rather than by the foot, helps to differentiate between upper and lower limb pain.

Three specific manipulative tests have been described for the stifle. These are the cruciate test, collateral ligament test,[9] and patellar displacement test. Most horses with significant stifle pain resent these tests, which makes the tests difficult to perform and interpret. All manipulation or flexion tests should be done on the contralateral limb first. For the cruciate test the affected limb should be weight bearing. The head of the tibia is pushed caudally and then released 5 to 10 times before trotting the horse. Laxity is supposed to be appreciated and lameness exacerbated if severe cruciate injury exists. I have never found this test effective. Pain in the affected joint provokes strong guarding by the horse so that the procedure is impossible to perform. The medial collateral ligament test involves abducting the distal limb against shoulder pressure exerted on the femorotibial joint 5 to 10 times before trotting the horse. Horses with ruptured medial collateral ligaments are so painful and instability is so great that this test is inappropriate, but it can be useful for a sprain of the ligament. The lateral collateral ligament is less often affected, but it can be tested by pulling the distal limb medially. Lameness associated with problems with patella release may be worsened by pushing the patella proximally several times with the horse weight bearing, before trotting, but again this is an unrewarding test.

DIAGNOSTIC ANALGESIA

In many horses with low-grade stifle lameness, positive diagnostic analgesia is the only way to localize the site of pain, so it is an important test. Because diffusion of local anesthetic solution between the three joint compartments is so variable,[4] all three must be blocked to ensure a valid test. Alleviation of lameness after analgesia of one compartment does not necessarily infer that that compartment is definitely the source of pain.

I use a 5-cm, 19-gauge needle for each joint compartment and up to 30 ml of local anesthetic solution, because experience has shown that 20 ml in each compartment might be incompletely effective in a 600-kg horse. Strict aseptic procedure should be followed. Arthrocentesis of the femoropatellar joint is well tolerated and performed first. An intradermal bleb is usually unnecessary. However, an inexperienced veterinarian may find one helpful for the medial femorotibial compartment, because some horses are sensitive about injection at this site. My preferred approach for the femoropatellar joint is between the middle and medial patellar ligaments. Synovial fluid is infrequently retrieved from this site unless the joint capsule is distended. If a synovial fluid sample is required, it may be retrieved more easily through a lateral approach.[10] The lateral cul-de-sac is entered caudal to the caudal edge of the lateral patellar ligament and 5 cm proximal to the tibial condyle. The medial femorotibial compartment is entered over the medial tibial condyle between the medial patellar ligament and the medial collateral ligament. A small outpouching of the joint capsule may be palpated. The lateral femorotibial compartment is best approached just cranial or caudal to the long digital extensor tendon, and close to the tibial plateau. Less space is available between the meniscus and the joint capsule in the latter approach, so the former is preferred. An improvement in lameness can be expected in 30 minutes, but the clinician is wise to allow at least 1 hour for the final assessment.

A number of conditions causing lameness in the stifle respond incompletely or not at all to intra-articular analgesia. Horses with medial collateral ligament injuries may be unaffected. Horses with subchondral bone cysts in the medial femoral condyle show a variable response, ranging from resolution of lameness to little change, and analgesia can take much longer to take effect. Horses with conditions that cause severe lameness are often only partially improved by analgesia; these conditions include infections; fractures, particularly patellar and tibial crest fractures; advanced osteoarthritis; and severe cruciate and meniscal tears.

IMAGING CONSIDERATIONS

Radiography

Although many stifle injuries are not associated with detectable radiographic changes, radiography is usually the first imaging mode to be employed once the site of lameness has been established. An x-ray machine capable of producing

at least 90 kV and 20 mAs is required. In larger horses adequate definition will only be achieved with even higher-powered x-ray generators. Fast-screen film combinations can be used particularly for caudocranial views. Using as slow a combination as possible is always worthwhile, commensurate with safe practice, to achieve the best definition on the radiograph. Large cassettes are necessary and should preferably be held in a cassette holder with a long handle. Because of the difficulty of aligning the cassette perfectly in the standing horse, using a grid is impractical, although one can be used if the horse is anesthetized. Many horses dislike having cassettes placed close to the stifles, so great care must be taken with this procedure. If any doubt exists about the horse's temperament, the horse should be sedated.

Five standard views are most commonly used: lateromedial, flexed lateromedial, caudocranial, caudolateral-craniomedial oblique, and cranioproximal-craniodistal oblique (skyline).[1]

Lateromedial View

The horse should be standing naturally for this view. The x-ray beam is directed perpendicular to the stifle. The stifle is naturally rotated slightly laterally in most horses, which predisposes to the beam being directed from too far cranially. The x-ray beam should pass just proximal to and parallel to the tibial plateau. The landmark on which to target the x-ray beam is the lateral condyle of the tibia. The cassette has to be pushed as proximal as possible, which can be difficult in a well-muscled horse. In a well-positioned x-ray the femoral condyles are superimposed on each other.

Flexed Lateromedial View

The limb is held in the farrier's position with the tibia parallel to the ground. If the stifle is held with its axial plane vertical, directing the x-ray beam perpendicular to the joint is easier. The same landmarks are used as for the standing lateromedial view. When the x-ray beam is correctly positioned, the femoral condyles are superimposed. The flexed view reveals a greater area of the medial intercondylar eminence of the tibia and the cranial part of the femoral condyles and also allows more complete imaging of the patella.

Caudocranial View

The caudocranial view requires relatively high exposure factors. A key feature for correct positioning is the angle of the tibia, because the x-ray beam should be perpendicular to the tibia. Placing the horse in its natural stance or with the limb slightly caudal to the contralateral limb facilitates correct alignment. The x-ray beam should divide the limb in the caudocranial plane and pass just proximal to the level of the lateral tibial condyle. The natural lateral rotation of the stifle should also be taken into account. Thus the x-ray beam is usually aimed craniodistally and craniolaterally and meets the caudal musculature of the thigh surprisingly proximally. The correct view defines the femorotibial joint spaces and clearly images the intercondylar eminence of the tibia within the supracondylar fossa of the femur.

Caudal 30° Lateral-Craniomedial Oblique View

For the caudal 30° lateral-craniomedial oblique view the x-ray beam is directed 30° from the caudocranial plane and slightly from proximal to distal, so that it crosses parallel to the tibial plateau, which it should bisect. The main value of the view is imaging the lateral femoral trochlea, with the advantage of highlighting the medial femoral condyle, and it can be used to screen for osteochondrosis lesions and subchondral cystic lesions.

Cranioproximal-Craniodistal Oblique View

The cranioproximal-craniodistal oblique view is a skyline view of the patella and femoral trochlear ridges and may be the only view on which a patellar fracture may be seen. In a standing horse the limb is held in the farrier's position with the tibia horizontal. The x-ray beam is aimed along the articular surface of the patella, but it may be impeded by the horse's flank. Twisting the metatarsal region medially, which rotates the stifle laterally, sometimes allows better access to the patella. The cassette is held along the cranial proximal aspect of the tibia, and the x-ray beam is directed almost vertically. Checking the position of the patella on a previous flexed lateromedial view helps to decide on the correct beam angle. In an anesthetized horse the leg is flexed with the horse in dorsal recumbency and the x-ray beam is directed from distal.

The contours of the femoral trochlear ridges and the patella are irregular in young foals. In most foals this irregularity is present up to 11 weeks of age, and in 45% of foals, up to 25 weeks.[11] In foals older than 5 months of age, irregularity of the femoral trochleas is abnormal. Irregularity of the femoral or tibial condyles is abnormal at any age.

Ultrasonography

A significant proportion of stifle lameness is caused by soft tissue damage, so ultrasonography has a potential diagnostic role in defining the injury and has several advantages over other imaging diagnostic techniques. At present ultrasonography is the only method of assessing soft tissue injury in the stifle in a standing horse. The disadvantages are that ultrasonography requires experience to be used effectively and a good-quality ultrasound scanner with a sector and a linear array transducer is necessary. Transducer frequencies of 7.5 and 5 MHz are needed to image the cranial aspect of the stifle, but a 3-MHz transducer is required to image the caudal part of the stifle.[12-14] When it becomes available, magnetic resonance imaging is likely to be a superior imaging technique for soft tissues of the equine stifle.[15]

Ultrasonography can be valuable for differentiating joint capsule distention from extra-articular swelling. Soft tissue structures that can be imaged include the patellar ligaments, the menisci and the respective cranial ligaments, the collateral ligaments, the cranial and caudal cruciate ligaments, the meniscofemoral ligaments, the long digital extensor tendon of origin, and the popliteal tendon. The articular cartilage and bony outline of the femoral trochlear ridges and the cranial and caudal one third of the femoral condyles may also be imaged.[12-14]

The patellar ligaments and the collateral ligaments can be imaged longitudinally and transversely with a 7.5- to 10-MHz linear array transducer with the horse weight bearing. The middle patellar ligament is the most obvious of the three and is a useful landmark. The menisci can also be imaged with this transducer from caudal to the medial and the lateral patellar ligaments. They appear as wedge-shaped structures of moderate echogenicity with the base of the wedge closer to the transducer. The cranial ligaments of the menisci are more easily imaged with a small convex array or sector transducer, which can be aimed more perpendicular to the meniscal ligaments and which can be more easily positioned between the patellar ligaments. The cruciate ligaments are difficult to image, because aligning the transducer perpendicular to the fibers of the ligament is difficult. Only a small length of ligament can be imaged at a time, which can make interpretation equivocal. Cruciate ligaments can only be viewed with the stifle flexed, using a convex array or sector scanner. From caudal a sector transducer is preferable for imaging the caudal cruciate ligament and the meniscofemoral ligaments and in large horses a 3-MHz transducer is necessary.[13]

Scintigraphy

Scintigraphy has been used on horses for more than 20 years, but little published evidence assesses its specificity and sensitivity for conditions of the stifle. For most stifle conditions scintigraphic findings are variable, and although positive findings are obviously helpful, negative findings can also add useful information. The significance of any scintigraphic result should be confirmed as exhaustively as possible by other tests, particularly diagnostic analgesia, because false-positive results

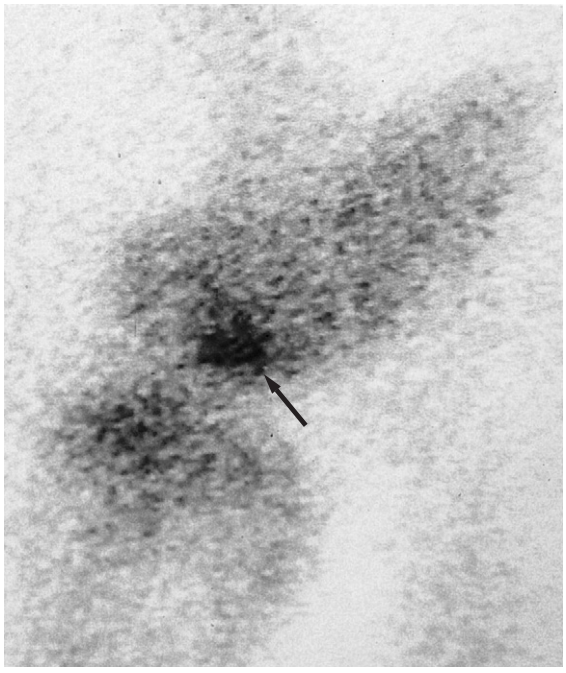

Fig. 47-1 Lateral delayed (bone) phase scintigraphic image of the left stifle of 9-year-old Thoroughbred cross gelding. There is focal increased radiopharmaceutical uptake in the distal femoral condyle *(arrow)*. A subchondral bone cyst in the medial femoral condyle thought to be causing lameness was seen radiographically.

also occur. In the normal adult stifle the caudal tibial epiphysis often has highest uptake of radiopharmaceutical.

Nuclear scintigraphy is most consistently valuable in diagnosing incomplete fractures or avulsions associated with the stifle.[16] However, although positive results may be obtained within 24 hours of injury, in some horses at least 3 days must elapse before significant increased radiopharmaceutical uptake occurs. Subchondral cystic lesions that are causing lameness may be scintigraphically positive or negative (Fig. 47-1). The lesions are more likely to be detected in older horses, using a caudal view, than in an immature horse with high background activity. Absence of radiopharmaceutical uptake was thought to be due to osteoclasts being the dominant cells in certain stages of the condition.[17,18] Although scintigraphy has been used to assess osteochondrosis in people,[19] bone scan findings are not well documented in horses. I have had positive and negative scintigraphic results associated with clinically significant osteochondrosis of the lateral femoral trochlea. Soft tissue injuries of the stifle are often scintigraphically negative, but in my experience increased radiopharmaceutical uptake can occasionally be encountered in these horses, especially in association with enthesopathy.

Scintigraphy is certainly a useful tool as a diagnostic aid for lameness in the stifle and can be useful for evaluating the bone activity of lesions. Occasionally horses may respond positively to intra-articular analgesia, but no significant abnormalities are found on radiography, ultrasonography, and arthroscopy. Increased radiopharmaceutical uptake in the stifle may be the only finding. Making a definitive diagnosis in these horses is difficult, although conceivably subchondral bone pain may be a possible cause of the lameness. One should bear in mind that many stifle conditions are negative scintigraphically. Conversely the joint may be scintigraphically positive when the cause of lameness is elsewhere in the limb.

ARTICULAR DISEASES

Femoropatellar Joint

Osteochondrosis

Osteochondrosis has been recognized in horses for 50 years and is an important cause of stifle lameness in young horses. The exact cause of the disease is still not well defined, but several factors are known to influence its development. Adequate dietary copper is important. Mare's milk is relatively low in copper, and because the foal relies on copper stored in its liver during late pregnancy, it will be deficient if the mare's diet contains insufficient copper. The zinc/copper ratio is also important, because zinc inhibits the absorption of copper.[20] Foals on high-energy diets are more prone to the disease.[21] Insufficient or excessive exercise and trauma may be factors that influence the development of the disease. Genetic factors may predispose to osteochondrosis in the hock of Swedish Standardbreds.[22] Large, fast-growing males are more susceptible. Osteochondrosis is most frequently seen in Thoroughbreds and Warmbloods. The lesions probably develop in the first 7 months of life,[23] but sometimes clinical signs may not be manifest until the horse is brought into work. In the sports horse this may be as late as 5 years of age or even older, when a mild to moderate lameness may develop as the horse begins more serious work. In my experience, surprisingly severe lesions can remain undetected until this time. Lesions are most commonly seen on the lateral trochlear ridge of the femur[24] but do occur on the medial trochlea, the intertrochlear groove, and the patella and are often bilateral.

Signs and diagnosis Osteochondrosis may be present asymptomatically. Lameness is often acute in onset and varies from a subtle gait deficit to marked lameness. Lameness is often more acute in onset and more severe in foals and young yearlings than in older horses. Distention of the femoropatellar joint capsule is often present and can be severe, especially in foals and yearlings. Gluteal muscle atrophy is seen in horses with severe lesions. Flexion tests are mostly positive. Synovial fluid may be hemorrhagic, but it is often normal. Intra-articular analgesia of the femoropatellar joint should improve lameness.

Radiographic changes include the following: no detectable signs; slight loss of contour or loss of outline of the lateral trochlear ridge; irregular defects in the trochlear ridge; radiopaque fragments within the defect (Fig. 47-2); round radiopaque bodies loose in the joint; and more rarely, irregularities on the patellar apex or the medial trochlear ridge.[1,25,26] Lateromedial and caudolateral-craniomedial oblique views are the most useful projections. Horses with radiographic lesions do not always show lameness.[25,27] Some horses have no detectable radiographic abnormalities, but osteochondrosis lesions are diagnosed on arthroscopic examination. Lesions without fragmentation in young foals may resolve with time[25] but many progress.[26]

Treatment Conservative management, with confinement and correction of dietary imbalances, or arthroscopic debridement of the lesions are the main treatment options. Conservative management is appropriate for horses with mild lesions. Because of the insensitivity of radiographic evaluation of osteochondrosis lesions, an argument can be made for arthroscopic examination of most horses with radiographic lesions or persistent femoropatellar effusion and lameness that fails to respond to conservative treatment.[27] Arthroscopic examination would ensure that radiographically silent cartilage lesions are treated effectively. Osteoarthritis can also be evaluated. For horses with fragmentation and significant subchondral bone lysis, arthroscopic surgery is probably the treatment of choice.[24] In my opinion more caution is required when treating foals arthroscopically, because some foals with large radiographic lesions are found to have intact cartilage at arthroscopy. Debridement of such lesion can leave a large

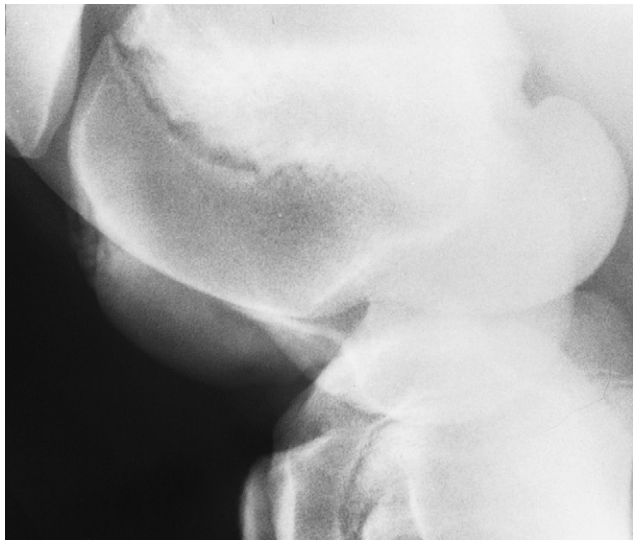

Fig. 47-2 Lateromedial radiographic view of the femoropatellar joint of a yearling Thoroughbred colt. There is a large irregular defect in the subchondral bone of the lateral trochlear ridge containing several radiopaque fragments.

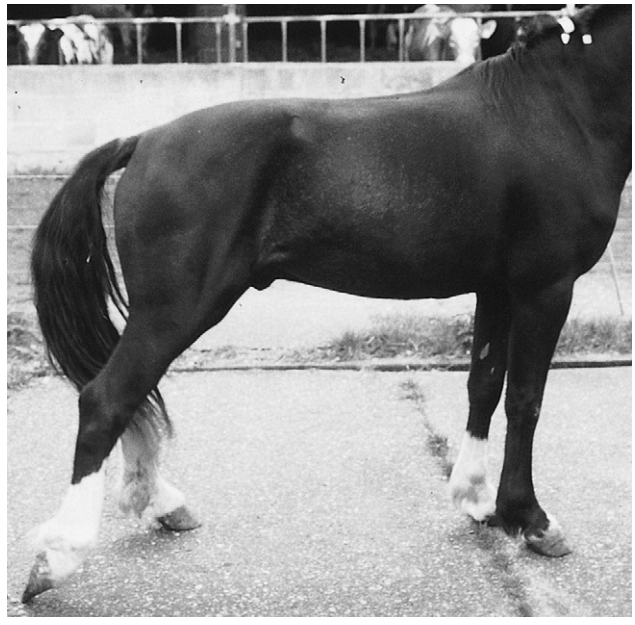

Fig. 47-3 Three-year-old Welsh Cob gelding with the typical stance of upward fixation of the patella in the right hindlimb.

deficit in the lateral trochlear ridge. Leaving the lesions intact may be preferable, because some healing may take place. If the clinical signs are persistent, these horses should be re-examined arthroscopically at a later date.

A variety of lesions are seen arthroscopically. The articular cartilage may appear intact, but if probed, large flaps may lift from the subchondral bone. Large defects on the lateral trochlear ridge may contain nodules of mineralized cartilage, which are readily removed, and often large tufts of fibrillated cartilage are associated with the lesion. The exposed subchondral bone is frequently soft and crumbly. Extensive areas of fibrillated cartilage over the adjacent lateral trochlear ridge and the opposing patella are often present in older horses.[5] Loose cartilage should be removed and the lesion should be curetted to healthy subchondral bone, which is a source of pluripotential cells. The cartilage perimeter should be vertical to allow better attachment of tissue regrowth.[28] A thorough lavage to remove all debris at the completion of surgery is essential. Postoperatively 6 weeks of stable rest with daily hand walking after 2 weeks is recommended. The horse should then be left for another 4 to 5 months at pasture before returning to work.

Prognosis Mild lesions in foals have been shown to heal with conservative management.[25] For more severe lesions better results have been reported with arthroscopic debridement.[24] Sixty-four percent of 161 horses were able to perform athletically. In this series, treatment of horses with mild to moderate lesions and treatment of older horses was most successful, which has also been the experience in my practice. The presence of extensive secondary articular cartilage fibrillation did not have a significant effect on the outcome in horses more than 2 years of age.[5]

Upward Fixation of the Patella and Delayed Patellar Release

Upward fixation of the patella occurs when the stifle subtends an angle of approximately 145° and the medial patellar ligament hooks over the medial trochlea of the femur, thus locking the reciprocal apparatus with the limb in extension. The condition is more common in horses with a straight hindlimb conformation with a stifle angle nearer 140° (in the normal horse the angle is about 135°), so that only a small degree of extension is required for upward fixation to occur.[29] Upward fixation is not a luxation of the patella, despite being commonly described as such. Predisposing straight hindlimb conformation or the condition itself may be hereditary.[30] Upward fixation of the patella is more commonly seen in young horses and ponies, especially if they are in poor condition, when weak thigh musculature fails to release the patella. Upward fixation can occur in older animals after trauma to the stifle region, is most frequently manifest when the affected horse is stabled, and sometimes occurs in fit horses that are suddenly given box rest. Lameness may develop in horses with more severe or long-standing lesions. I recognize delayed patellar release as a condition in which delayed release of the patella occurs, without complete upward fixation, and which presumably is a less severe form of the disease.

Signs and diagnosis A horse with upward fixation of the patella stands with the hindlimb locked in extension with the fetlock flexed (Fig. 47-3). The leg releases with a snap, usually unaided, but occasionally the horse needs assistance. Some horses merely show intermittent delayed release of the patella, especially when turned toward the affected limb. This can be mistaken for stringhalt. Delayed patellar release is manifested by a catching of the patella as the limb is protracted, usually as the horse moves off. Delayed release of the patella may be evident as a rather jerky movement of the patella when the horse moves over in the stable or as it decelerates from canter to trot or trot to walk. Horses in which the condition is chronic develop stifle soreness and may be resistant to work, especially in deep going. If the condition is more serious, horses try to avoid extending the hindlimbs while walking uphill or downhill. Femoropatellar joint distention may be present. Diagnosis may depend on the history and the owner's description if the horse does not lock the patella during the examination. Locking the patella manually may be possible by pushing it proximally, although this can be difficult and is resented by many horses. A careful search should be made for concurrent stifle disease such as osteochondrosis or soft tissue injury, and the site of any lameness should be confirmed by diagnostic analgesia. X-ray examination of both stifles is prudent, because any pathological condition caused by upward fixation or concurrent with it affects treatment and prognosis.

Treatment For a horse that has the patella locked, pushing the patella medially and distally and backing the animal is recommended but difficult to do. Pulling the limb forward with a side line may provide relief. If the upward fixation of the patella is intermittent and not causing lameness, a conditioning program should be undertaken. This includes an exercise regimen, the administration of anthelmintics, an increased plane of nutrition, and dentistry as appropriate for each horse. The exercise regimen depends on the specific circumstances for each horse. Daily lunging should be instituted to a level that is appropriate for the age and type of horse. Stable rest is usually contraindicated, and turning the horse out to pasture as much as possible is preferable. Immature horses should be allowed time to outgrow the problem. Injection of counter-irritants containing iodine into the medial and middle patellar ligaments has been used at this stage of the disease.[31] Delayed patellar release requires the same management.

Because of potential complications, which include fragmentation of the patella and lameness, surgery is only indicated when the following criteria have been fulfilled:

1. Upward fixation of the patella persists despite an appropriate conditioning program.
2. Lameness has developed because of the disease.
3. The condition in an immature horse does not resolve as the animal matures.

The surgery can be performed under local anesthesia in the standing horse.[32] A small incision is made over the distal part of the medial patellar ligament. I find that minimizing the incision length seems to reduce the incidence of postoperative swelling. A curved Kelly forceps is then advanced caudally under the ligament, developing a path for a blunt-ended bistoury. The bistoury is passed until its end can be palpated caudal to the medial patellar ligament, before the ligament is severed close to its tibial insertion. Stringent asepsis should be observed. Once the ligament is severed, the border of the tendon of the sartorius muscle is palpable caudally. Some surgeons prefer to perform this surgery with the horse under general anesthesia, which allows for complete asepsis: the ligament can be exteriorized before severing, and the fascia overlying the ligament can be sutured before skin closure.

Postoperatively I prefer to confine the horse for 2 months to allow the patella to settle in its new position in the intertrochlear groove. Hand walking can be introduced during the second month.

Prognosis A substantial number of horses respond to conservative treatment. Occasional recurrences happen if the horse has enforced stable rest, but many grow out of the condition. After surgery and in the absence of degenerative changes in the joint, the horse should return to normal use, although some appear to have a slightly restricted gait. Recurrence of the condition is unusual. Some fibrous thickening is palpable at the surgical site for many years in most horses and indefinitely in some horses. Significant complications including lameness, local swelling, fragmentation of the patella, and even fracture of the patella have been reported[33-36] and have also been produced experimentally. If fragmentation causes lameness, it can be treated arthroscopically with good results.[33] In my experience up to 30% of horses have minor fragmentation of the patella, without clinical signs, after medial patellar desmotomy.

Fragmentation of the Patella

This condition is generally considered to be a sequel to medial patellar desmotomy or to be associated with upward fixation of the patella, and it is manifested by fragmentation of cartilage and bone off the apex of the patella.[33,36] This condition is not chondromalacia of the patella, which could be a form of osteochondrosis, and has not been specifically reported in horses. If the condition follows desmotomy, clinical signs can appear from 3 weeks to more than 12 months postoperatively. Horses can develop fragmentation within a few weeks of surgery without clinical signs.[5] A medial patellar desmotomy has been performed experimentally in 12 horses, and eight of these developed fragmentation.[36] The lesions may be caused by instability of the patella after medial patellar desmotomy.

Signs and diagnosis Lameness varies from a stiff hindlimb action to an obvious lameness. Flexion is resented and worsens the lameness. Synovial effusion is common and excessive fibrous tissue reaction may be present at the surgical site. Radiographically, small bone fragments are present close to the apex of the patella (Fig. 47-4), often combined with subchondral bone lysis and roughening or spurring of the cranial surface of the patellar apex.

Treatment Arthroscopic debridement is indicated. The lesions are most commonly on the lateral surface of the apex and may be partly obscured by synovial villi, so a careful arthroscopic examination should be performed before deciding on the site of the instrument portal. Debridement of the lesion to healthy subchondral bone is required.

Prognosis The prognosis for athletic function is reasonable, although some horses anyway appear to have a slightly stiff hindlimb action after medial patellar desmotomy. Ten of 15 horses returned to athletic use.[33] In my experience the incidence of the condition has decreased in the last 8 years, which may be because fewer patellar desmotomies are being performed or because more care is taken during convalescence.[5]

Luxation of the Patella

Lateral luxation of the patella in the foal is considered to be an inherited condition caused by a recessive gene.[37] Luxation in the adult is likely to be traumatic in origin.[38] Because the medial trochlear ridge is larger, only severe trauma induces medial displacement. Hypoplasia of the lateral trochlea is often present,[39] but the condition also occurs in foals with apparently normal conformation.[37] Luxation is most common in miniature breeds but has been reported in Standardbreds, Thoroughbreds, and an Arabian foal,[38-40] and I have seen it in a Welsh Cob.[5]

Signs and diagnosis The condition may be unilateral or bilateral and has been graded as follows:

- Grade 1: The patella can be manually luxated but readily reduces itself.
- Grade 2: The patella is usually in place but luxates intermittently.

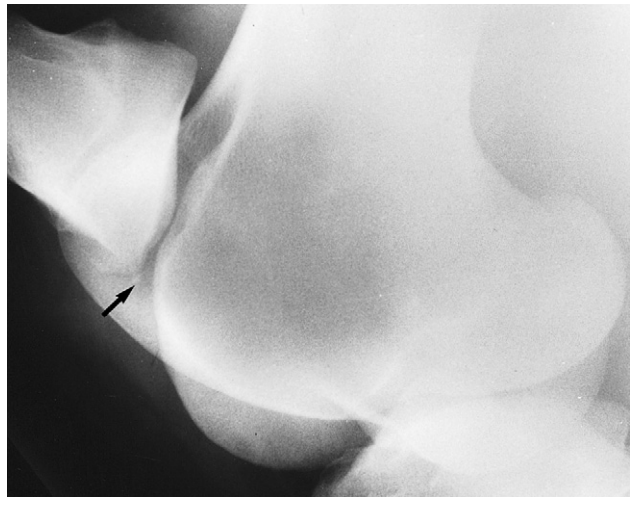

Fig. 47-4 Lateromedial radiographic view of the stifle of a 7-year-old Thoroughbred cross gelding that had previously had a medial patellar desmotomy to treat intermittent upward fixation of the patella. The apex of the patella is fragmented (*arrow*).

- Grade 3: The patella is usually luxated but can be manually relocated.
- Grade 4: The patella is continuously luxated and cannot be relocated.[39]

Severely affected foals are unable to extend the stifle and stand in a characteristic crouching position. If less severe, the condition may not be obvious clinically, but horses are usually reluctant to flex the stifle and have a stiff gait in the affected limb. Some horses show little evidence of the disease until degenerative changes provoke lameness. I have seen a Thoroughbred yearling with severe osteochondrosis of the lateral trochlear ridge, which led to a diminution of the ridge and allowed the patella to luxate laterally. Lateromedial, cranioproximal-craniodistal oblique, and caudocranial radiographic views are the most useful to ascertain the position of the patella and to evaluate the trochlear ridges.

Treatment Lateral release and medial imbrication of the femoropatellar compartment are necessary to maintain the patella in the intertrochlear groove.[38-40] Both lateral release and medial imbrication appear to be important. Recession sulcoplasty is valuable in the presence of hypoplasia of the lateral trochlear ridge of the femur.[40]

Prognosis Poor success can be expected with surgery in the presence of osteoarthritis and in larger horses. In foals there is a moderate prospect of achieving athletic function. Aggregating the results from the 3 case series mentioned, the patella maintained its position postoperatively in 8 out of 11 foals.[3,4] Breeding from affected animals should be discouraged.

Osteoarthritis

Any injury to the femoropatellar joint can potentially cause osteoarthritis. The injury could be simple trauma or result from any of the previously mentioned conditions if they have caused irreversible changes.

Signs and diagnosis The clinical signs depend on the cause and severity of the condition. Lameness is usually persistent, and chronic joint distention may be present. If osteochondrosis is responsible for osteoarthritis, radiographic changes typical of chronic osteochondrosis are likely to be present (see page 458). Cartilage fibrillation, ranging from focal tufting to widespread involvement of the trochlear or patellar articular cartilage, may be seen arthroscopically and full-thickness defects of the articular cartilage may be present in more severely affected horses.

Treatment Initially treatment depends on the inciting condition and is described in the appropriate sections. However, once chronic osteoarthritic changes have developed, realistically therapy is likely only to be palliative. Intra-articular medication with small doses of corticosteroids, such as 10 mg of triamcinolone acetonide combined with hyaluronan, may give temporary relief. A controlled exercise regimen may help, and in some horses an arthroscopic debridement prolongs working life.

Prognosis The femoropatellar joint is more forgiving than the femorotibial joints, and although osteoarthritis will not resolve, if mild it may be tolerated enough for a horse to perform useful work. More severely affected horses are unlikely to return to athletic use.

Distal Luxation of the Patella

One report of distal luxation of the patella has been made.[41] The horse had the affected limb flexed and non–weight-bearing. The patella was relocated with the horse under general anesthesia, and when the horse was re-examined 3 months later, no lameness was seen.

Patellar Ligament Injuries

Patellar ligament injuries are rare. Sprain of the middle patellar ligament is the most common, although lateral patellar ligament injuries also occur.[42] There may be an association between middle patellar desmitis and previous medial patellar desmotomy. Jumping horses are most commonly affected.

Signs and diagnosis Lameness can be severe in the acute phase but varies. Sometimes no localizing signs are apparent, but in some horses femoropatellar effusion, periarticular thickening, or edema are seen. Intra-articular analgesia of the femoropatellar joint may not affect the lameness. The diagnosis is confirmed by ultrasonography. Proximal, mid-body, and distal lesions have been identified. Radiographic changes are unusual and one should bear in mind that entheseous change on the cranial aspect of the patella may be seen as an incidental finding during radiography of the stifle. Scintigraphy may show increased radiopharmaceutical uptake in the patella or proximal tibia in association with proximal or distal lesions.

Treatment Horses with severe injuries may require rest for up to 6 months.

Prognosis Lameness is often slow to resolve and is recurrent in some horses.

Femorotibial Joint

Subchondral Cystic Lesions

The cause of subchondral bone cysts and other osseous cyst-like lesions is still unknown. The distal weight-bearing aspect of the medial femoral condyle is the most frequent site for subchondral bone cysts.[43] Osseous cyst-like lesions have been identified in the caudal femoral condyles in foals.[44] Osseous cyst-like lesions also occur in the proximal tibia. Lesions thought to result from osteochondrosis were in the lateral aspect of the tibia, and those associated with osteoarthritis were more common medially in a report of 12 horses.[45] Subchondral bone cysts and osseous cyst-like lesions may be a form of osteochondrosis,[46] but trauma is also probably an important factor. Subchondral bone cysts in the medial femoral condyle have been produced experimentally after development of full-thickness defects in cartilage and subchondral bone.[17] Lesions did not form if the defects were made only in cartilage. The lining of these lesions has been shown to contain active inflammatory enzymes that cause bone resorption.[47] In the literature the syndrome in the young horse is emphasized, but in my experience subchondral bone cysts and osseous cyst-like lesions occur in any age group, mostly apparent as a primary lesion but sometimes as a sequel to osteoarthritis.

Signs and diagnosis The condition is reported to be more common in horses under 4 years old,[43] but in my practice is encountered in horses of all ages.[5] Lameness is mild to moderate but can be severe at first and acute in onset. Lameness may be intermittent, especially in older horses. Affected horses seem to be lamer when turning. Occasionally mild medial femorotibial effusion occurs, but often no signs are obvious on physical examination. Intra-articular analgesia of the medial femorotibial joint may be required to diagnose the site of pain causing lameness. Because the subchondral bone is probably the site of pain, local anesthetic solution must diffuse into the lesion and may not be completely effective. A 50% improvement in lameness justifies radiography.

The caudocranial, caudolateral-craniomedial oblique, and flexed lateromedial radiographic views are the most useful for demonstrating the lesion. Subchondral bone cysts in the medial femoral condyle are relatively round or oval, with a variably sized base and communication with the joint (Fig. 47-5). A sclerotic rim is usually seen only in horses with older lesions. Radiographic signs of osteoarthritis may also be apparent in some horses. The osseous cyst-like lesions in the tibia have a similar morphology. Subchondral bone cysts can also occur as incidental radiographic findings in sound horses. Lesions in the caudal femoral condyles in foals may appear as localized or extensive osseous defects on radiography. Scintigraphic findings in horses with subchondral bone cysts are inconsistent,[17] but in some horses the lesions are clearly depicted. Flattening or indentation of the distal medial femoral condyle is sometimes seen radiographically and may be a precursor of a

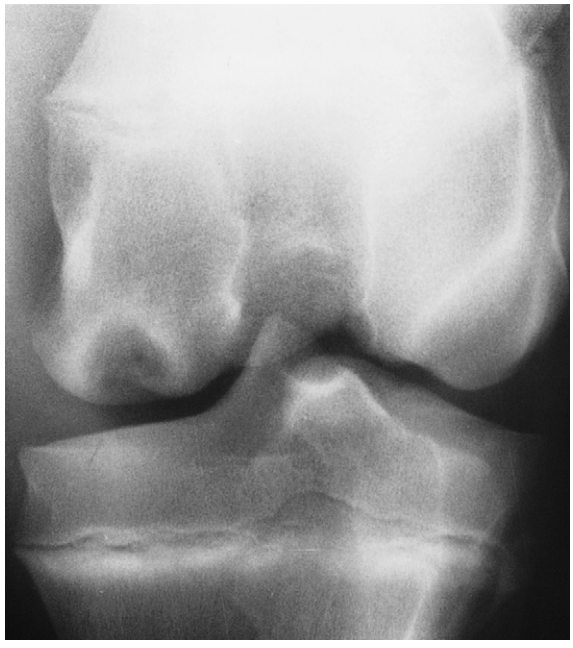

Fig. 47-5 A caudocranial radiographic view of the right stifle of a yearling Thoroughbred colt. Medial is to the left. There is a subchondral bone cyst in the medial femoral condyle with a sclerotic rim and a wide base that communicates with the medial femorotibial joint.

subchondral bone cyst. These lesions are relatively common and frequently do not cause lameness. Occasionally increased radiopharmaceutical uptake is seen in these defects.[5] In my experience positive scintigraphic findings, together with a positive response to diagnostic analgesia, are good indications that the defect is a cause of lameness, but some scintigraphically positive lesions do not result in clinical signs, although the Editors consider this unusual.

Treatment Conservative treatment involves stable or pasture rest, with or without non-steroidal anti-inflammatory medication or intra-articular corticosteroids.[48,49] Horses with non-articular osseous cyst-like lesions or small articular defects are probably best treated conservatively. If the horse does not respond to conservative treatment, and for articular lesions, arthroscopic debridement is indicated.[43,46,50] Debridement of the cyst lining and removal of all debris from the joint is the goal. Experimentally the use of cancellous bone grafts did not improve the outcome.[51] Postoperative intralesional corticosteroid has been advocated to suppress the inflammatory mediators.[47] Subchondral bone forage is contraindicated, because reports indicate that it may worsen the lesion.[43] Debridement of small medial condylar defects should be approached with caution, because this may provoke a worsening of the lesion. Tibial osseous cyst-like lesions can be debrided arthroscopically, but many of them lie beneath the cranial meniscal ligament, which must be divided to gain access.

Prognosis A 50% success rate has been reported with conservative treatment of horses with medial femoral condylar subchondral bone cysts, but remodeling of bone may be more prolonged than with surgical treatment.[48] Arthroscopic treatment carries a 70% to 75% success rate in horses under 3 years old.[43,50] An assessment of the results in older horses is required. Improvement of clinical signs after surgery frequently occurs without radiological resolution. Limited reports of surgical treatment of tibial osseous cyst-like lesions[45] suggests that the lateral osteochondrosis associated lesions may respond best, and this is also my experience.

Meniscal and Meniscal Ligament Injuries

The cause of meniscal injuries in horses is not well documented, but in dogs meniscal injuries are considered to be caused by crushing forces combined with tibial rotation and flexion or extension of the stifle.[8] A few reports of the condition in horses have been published.[52-55] At my hospital 55 meniscal injuries in 52 horses have been diagnosed arthroscopically as the primary cause of lameness. The medial meniscus was affected in 75% of horses. The cranial ligaments of the menisci were involved in nearly all horses.[56] Concurrent injury to other structures in the stifle was diagnosed in 44 of 55 horses and included articular cartilage damage on the femoral condyles in 26 horses, cruciate injury in 9 horses, cartilage lesions on the medial intercondylar eminence of the tibia in 7 horses, and disruption of the long digital extensor tendon of origin in 4 horses. Secondary medial collateral ligament injury was not diagnosed. Primary medial collateral ligament rupture with secondary meniscal injury was not included in the series. Twenty-seven of 79 other horses that were examined arthroscopically and that had other stifle injuries, but no evidence of meniscal damage, did have fibrillation of the axial borders of the cranial ligaments of the menisci.[52] The significance of this is uncertain.

Signs and diagnosis Lameness is often acute and severe in onset after trauma but mostly becomes low grade and persistent. Distention of the femoropatellar or medial femorotibial joints can be expected in 66% of horses. Lameness is exacerbated by flexion of the limb in about 90% of horses.[55] Diagnostic analgesia is usually necessary to confirm the site of pain causing lameness but may not render the horse completely sound. Radiographic signs occur in about 55% of horses. New bone formation on the cranial aspect of the medial intercondylar eminence of the tibia is the most frequent finding (33%)[56] and appears to be more common with meniscal than cruciate ligament disease[55] (Fig. 47-6). Dystrophic mineralization and osteoarthritic changes may be seen in severe lesions. Ultrasonography demonstrates some of the more severe lesions.[57]

Arthroscopy offers the best chance of a definitive diagnosis and an assessment of concurrent damage. Because only the cranial and caudal poles of the menisci can be seen, assessment of meniscal injury is limited. Owing to these limitations, I graded changes visible from cranially as follows:

1. Tears extending longitudinally from the cranial ligament into the meniscus, with minimal separation
2. Tears involving the cranial pole in which the extent of the injury is visible (Fig. 47-7)
3. Severe tears that extend beneath the femoral condyle and that cannot be completely assessed or debrided.[55]

An injury of a caudal meniscal pole would require further classification but has not been reported.

Treatment Horses with acute stifle injuries that have no definitive diagnosis should be managed with rest and anti-inflammatory medication, followed by controlled exercise. Arthroscopy is indicated if conservative therapy is unsuccessful or for severely lame horses. Grade 1 meniscal tears can only be superficially debrided. Intra-articular suturing for some horses may be a way forward. Grade 2 and 3 tears are debrided as effectively as possible by removing all loose tissue. A careful assessment of visible concurrent damage should be made. Postoperatively controlled exercise begins at 2 to 3 weeks and can gradually be increased over 3 months, depending on progress and the extent of the injury. Free exercise at pasture should be avoided for 6 months.

Prognosis Return to full athletic use can be expected in about 50% of horses overall, though improvement may be seen in another 10%.[55,56] Full function was regained in 54% of 28 horses with grade 1 tears, 50% of 14 horses with grade 2 tears, 25% of 8 horses with grade 3 tears, and neither of

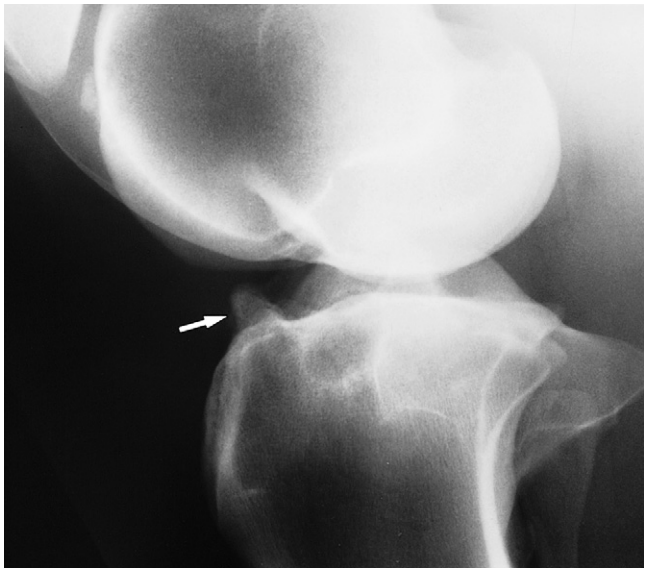

Fig. 47-6 Lateromedial radiographic view of the left stifle of a 7-year-old Thoroughbred cross gelding. There is new bone on the cranial aspect of the intercondylar eminences of the tibia (*arrow*). Note also the large lucent zone in the proximal tibia. There is an old osteochondrosis lesion involving the lateral trochlear ridge of the femur. Arthroscopic examination revealed a tear of the medial meniscus.

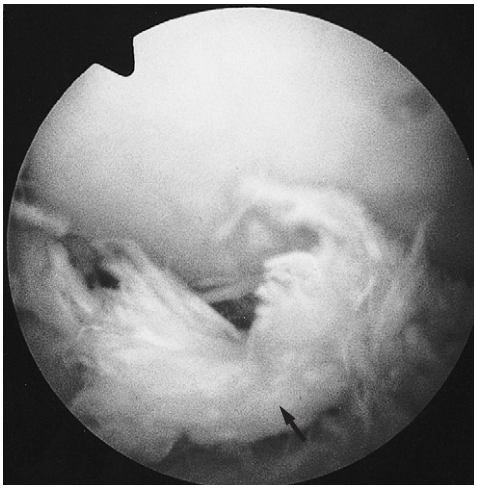

Fig. 47-7 Arthroscopic view of the left lateral femorotibial joint of 6-year-old eventer gelding. The lateral meniscus has a grade 2 tear *(arrow)*.

2 horses with two menisci affected. Concurrent articular cartilage damage appeared to worsen the prognosis, and only 3 of 8 horses with concurrent cruciate ligament injury became sound. Horses treated arthroscopically within 2 months of injury fared 25% better than those treated later than 2 months.[56] If radiographic signs of osteoarthritis were present, the prognosis was poor and only 1 of 4 horses with dystrophic mineralization improved after arthroscopy.

Cranial and Caudal Cruciate Ligament Injuries

In the dog the cranial cruciate ligament can be injured when the stifle is in hyperextension, or after sudden rotation with the stifle in flexion,[8] and this is likely to pertain to the horse. Direct trauma to the joint or degenerative change in the ligament can also lead to cruciate injury in the horse.[5,58] Experimentally[59] and in my clinical experience[5] the cranial cruciate ligament fails most frequently at mid-body level, but avulsions of its tibial and less often of its femoral attachments have been reported.[58,60] Caudal cruciate ligament injuries occur less frequently.[61] Concurrent injuries, including damage to the medial collateral ligament, the menisci, and articular cartilage, are often associated with cruciate tears.[58,62]

Signs and diagnosis Lameness is often associated with a history of trauma and is usually acute in onset and severe at first. Distention of the femoropatellar or the femorotibial joints is sometimes present. In severe injuries crepitus may be detectable. The cruciate draw test appears to be valueless in the conscious horse, but the flexion test is usually positive, especially with severe injury. With mild injuries diagnostic analgesia is required to confirm the site of pain causing lameness. In my series of 48 horses with cranial and caudal cruciate ligament injuries diagnosed at arthroscopy, radiographic signs included the following[5]:

- No changes in at least 50% of horses.
- Fracture of the intercondylar eminence of the tibia; however, this can occur without significant cruciate injury.
- New bone formation cranial to the intercondylar eminence of the tibia. This finding was three times more common in horses with meniscal injuries than those with cruciate ligament injuries in my series.
- Bony resorption or fragmentation at the femoral attachment of the cruciate ligaments.
- Cranial displacement of the tibia with cranial cruciate ligament rupture.
- Osteoarthritis, or mineralization of the ligament, associated with chronic lesions.

Ultrasonographic evaluation of the cruciate ligaments is difficult. Arthroscopy offers the best evaluation of the cruciate ligaments. The cranial cruciate ligament is covered by the median septum, which may make the ligament difficult to assess, but the septum is often disrupted in cranial cruciate ligament injury. Motorized debridement of the septum may be required to see the ligament. The caudal cruciate ligament is viewed from the cranial and caudal aspects of the medial femorotibial joint. Mild cranial cruciate ligament changes are difficult to quantify because of inflammatory changes in the median septum, but fiber disruption is seen in moderate injuries (Fig. 47-8). With acute, severe injuries of the cranial cruciate ligament, inflammatory debris must be removed before the torn ends of the ligament can be seen. Concurrent articular cartilage or meniscal damage within the joint is common.

Treatment With acute injuries the same treatment criteria apply as for meniscal injuries. The indications for arthroscopy and for postoperative management are also similar. Arthroscopic debridement is best performed with a motorized synovial resector. Repair of complete cruciate ligament rupture has not been reported in horses, and the best treatment that can be offered is good debridement of loose tissue to allow less severe injuries the best chance to heal.

Prognosis Prognosis is poor in horses with moderate to severe injuries. In one report, 2 of 10 horses were pasture sound and 1 raced,[58] and in another report 2 of 6 were pasture sound.[62] In my series lameness persisted in 4 horses with severe injuries; 6 of 17 horses with moderate injuries became sound and 21 of 27 with minor superficial changes became sound.[5]

Collateral Ligament Injuries

Injury of the medial collateral ligament is more frequently diagnosed than injury of the lateral collateral ligament. Such injury is associated with acute trauma, which, as in the dog, probably involves a medial or lateral force on the joint or distal limb.[62] Concurrent injury of the menisci or cruciate lig-

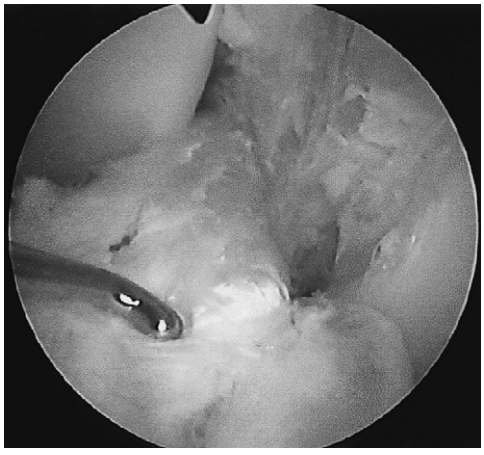

Fig. 47-8 An arthroscopic view of the right femorotibial joint of an 8-year-old Warmblood gelding. The median septum has been removed with the motorized synovial resector to reveal a chronic, low-grade fiber disruption of the cranial cruciate ligament.

aments commonly occurs in horses with severe injuries,[62,63] but not necessarily in those with more moderate injuries.

Signs and diagnosis The most easily diagnosed collateral ligament injuries are complete ruptures of the medial collateral ligament, which manifest as an acute, severe lameness after trauma. Instability of the stifle occurs with swelling and pain over the ligament. Sprains of the collateral ligaments are less obvious. Heat and pain may be palpable and chronic thickening develops in some horses. The collateral ligament test can be useful but is not specific. Flexion of the limb is usually painful, but intra-articular analgesia of the medial femorotibial joint may be negative. If complete rupture of the ligament has occurred, stressed caudocranial radiographs are diagnostic. Widening of the joint space occurs on the affected side (Fig. 47-9). Entheseophyte formation at the origin and insertion may be seen in chronic injuries. Ultrasonography can be helpful in diagnosing and assessing the injury.

Treatment Horses with mild sprains, in which no instability occurs, are treated with stall rest for 6 weeks and anti-inflammatory medication until the inflammation subsides, followed by controlled exercise for another 6 weeks. One report describes the repair of a complete rupture of the medial collateral ligament using braided polyester material attached to two 6.5-mm cancellous screws in the femur and tibia to stabilize the joint.[64] Orthopedic wire was originally used, but it dislodged postoperatively. The surgery is a salvage procedure and is not well documented, and concurrent injury and patient compliance should be carefully assessed before undertaking surgery.

Prognosis Horses with mild sprains have a moderate chance of returning to athletic use, depending on the extent of any concurrent injury. Results are not well documented, and in my view the prognosis is probably poor if entheseous change develops at the medial collateral ligament attachments. The prognosis for complete ruptures is grave. The horse described above that was treated surgically became lame after falling 9 months postoperatively, but it had been comfortable at pasture until then. Because associated meniscal or cruciate injury is common, a high chance exists of osteoarthritis in the long term, even if the medial collateral ligament heals.

Articular Cartilage Trauma

Articular cartilage defects on the medial or lateral femoral condyles or on the axial part of the tibial plateau are frequently encountered during arthroscopic examination. Usually they accompany other femorotibial injuries, but occasionally they are the only finding, in which case they may be considered the primary cause of lameness.[65] Cartilage lesions were diagnosed as the primary cause of lameness in approximately 25% of horses undergoing diagnostic arthroscopy of the stifle in my series.[5] Primary lesions may be seen on both the medial and lateral femoral condyles.

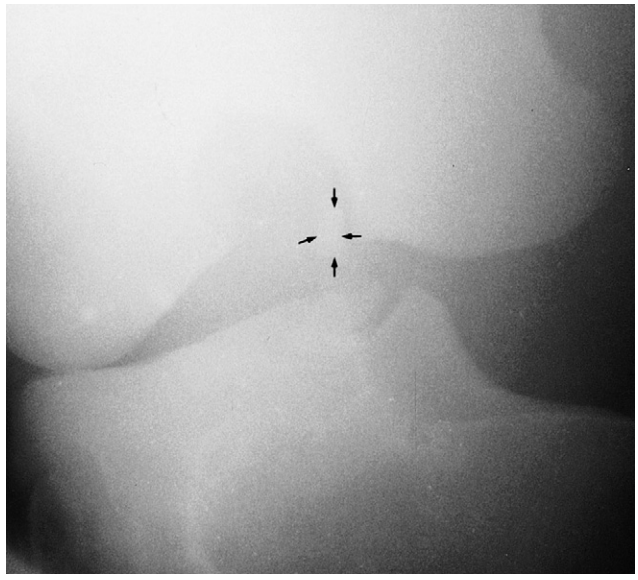

Fig. 47-9 Stressed caudocranial radiographic view of the left stifle of 10-year-old riding horse with rupture of the medial collateral ligament of the femorotibial joint. Medial is to the right. There is widening of the medial joint space. Note also the osseous opacity proximal to the intercondylar eminences (*arrows*).

Signs and diagnosis Horses in which articular cartilage lesions are a primary cause of lameness usually have mild to moderate lameness. Diagnostic analgesia is required to define the site of pain. Abnormalities are rarely seen radiographically. At arthroscopy a variety of lesions may be present. These include thickening, softening, and creasing of the cartilage, widespread fibrillation, focal tufts of fibrillated cartilage, circumscribed nodules of cartilage, apparent tears of the articular surface, and areas of exposed subchondral bone.[56,65,66]

Treatment Doubt about the long-term effect of debridement of cartilage lesions still exists.[67] Small, full-thickness lesions are probably best debrided to subchondral bone; micropicking the bone appears to improve healing to a degree.[68] The effect of smoothing partial thickness defects is still controversial and may provoke the development of an osseous cyst-like lesion.[43,67] Debris should be thoroughly lavaged from the joint.

Prognosis The prognosis for return to soundness for horses with cartilage defects is generally guarded. Six of 7 horses with focal defects returned to racing, but 5 horses with larger lesions remained lame.[65] In my practice a full-thickness articular cartilage defect was diagnosed arthroscopically in 17 horses as being the primary cause of lameness. Five of 8 horses for which follow-up information was available became sound.[5] Of 16 horses in which generalized articular cartilage fibrillation was the cause of lameness, 7 of 8 horses for which follow-up information was available became sound.

Osteoarthritis

Osteoarthritis of the femorotibial joint can be a sequel to any injury described in this chapter that has the potential to damage the articular cartilage. Osteoarthritis is likely to develop when the initial tissue damage is severe, is not treated, or is treated ineffectively.

Signs and diagnosis Many horses have a history of lameness that has already been treated or chronic lameness of varying intensity. Diagnostic analgesia may be required to identify the site of pain, but in many horses joint effusion and thickening are palpable on the medial aspect of the joint. Flexion of the limb is painful and some horses resent holding up the limb for shoeing. Radiography reveals typical changes associated with osteoarthritis, including osteophytosis, flattening of the articular surfaces of the femoral condyles, sclerosis and lucent zones in the subchondral bone, narrowing of the joint space, and dystrophic mineralization of soft tissues.

Treatment Management of osteoarthritis is at best palliative. If possible, the inciting lesion should be treated appropriately in an attempt to prevent worsening of the changes. Intra-articular corticosteroids or glycosaminoglycans, and in some horses arthroscopy, may give temporary relief.

Prognosis Once radiographic changes of osteoarthritis are manifest, the prognosis for athletic soundness is poor. With careful management less severely affected horses may be able to perform light work or be kept at pasture.

Fractures Involving the Stifle

Fractures of the Patella

External trauma is the commonest cause of patellar fracture in the horse.[69] Contraction of the quadriceps muscles may be the cause of some avulsion fractures. The majority of horses have a history of direct trauma, such as a kick, or an impact on the stifle by hitting a fixed fence while jumping. If the trauma occurs while the stifle is in flexion, the patella is fixed against the trochlear ridge of the femur, which is thought to render it more vulnerable to fracture.[70]

A variety of fracture morphology is seen.[70,71] Sagittal fractures of the medial aspect of the patella are probably the most frequent. These are usually articular and the fragment often involves a significant area of the attachment of the parapatellar fibrocartilage of the medial patellar ligament. Fragmentation of the base of the patella sometimes accompanies these fractures[69] or may be seen separately. Non-articular fractures are mostly seen as mildly displaced fragments on the cranial aspect. Complete horizontal fractures are severe injuries, and the massive forces exerted by the extensor muscles have a significant bearing on their management. Complete sagittal fractures are less catastrophic because the distracting forces are not as strong. Comminuted fractures of the whole body of the patella occasionally occur after severe trauma.

Signs and diagnosis The history and clinical signs often strongly suggest the possibility of patellar fracture. Initially lameness may be severe and remains so in horses with midsagittal and horizontal fractures. In horses with many of the smaller avulsion fractures, or fractures of the medial aspect, lameness becomes moderate to mild after a few days. Often evidence of trauma and local swelling are apparent in the stifle region. Crepitus is occasionally present, and pain may be elicited on palpation of the patella. The femoropatellar joint is usually distended. Horses with chronic, less severe fractures often have surprisingly little direct evidence of the injury. Flexion of the limb is usually painful.

Radiography is essential to confirm the diagnosis and to evaluate the fracture. Lateromedial views may give no indication of the fracture, so a cranioproximal-craniodistal view is essential (Fig. 47-10). Severely injured horses greatly resent positioning for this radiographic view and may require analgesia. Occasionally general anesthesia is required, but it should not be attempted without treatment options being in place.

Treatment Horses with open wounds must be treated with debridement and antibiotics, and it is vital to establish if joint infection exists and treat it appropriately. Horses with small non-displaced, non-articular fractures may be treated conservatively with stable rest. Most heal by fibrous union only. Horses may return to work in 8 weeks. Fragments off the base of the patella need arthroscopic debridement if debris is loose in the joint. Small fragments at the base of the patella, which are embedded in the soft tissue attachments, may not require removal.[72] Articular fractures of the medial aspect are best removed.[69] This can be done by arthrotomy or arthroscopy (Fig. 47-11) and requires careful dissection of the fragment from the medial patellar ligament. The distal end of the fracture is clearly seen arthroscopically. Concurrent joint damage should be evaluated and can have an important bearing on the outcome. Complete horizontal fractures must be treated by internal fixation.[70,73,74] Lag screw fixation with 5.5-mm cortical screws or partially threaded 6-mm cancellous screws, possibly with reinforcement with tension band wiring, is necessary to combat the huge distraction forces of the extensor muscles. Anesthetic recovery has to be carefully controlled by delaying attempts of the horse to stand by use of sedation, to avoid breakdown of the repair. Sagittal fractures can also be fixed using the lag screw principle.[75]

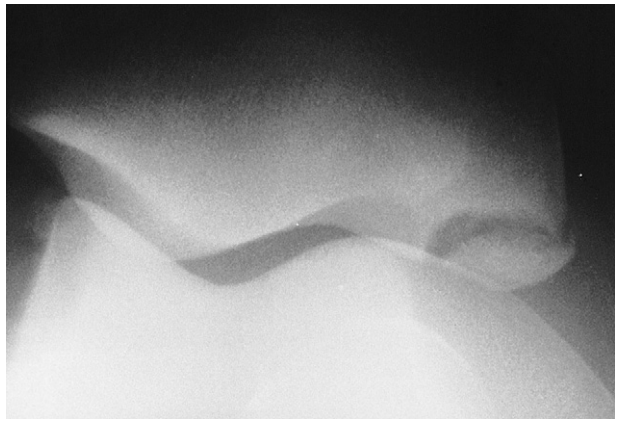

Fig. 47-10 Craniopoximal-craniodistal radiographic view of the right patella of 10-year-old eventer gelding that hit a cross-country fence with the right stifle. Medial is to the right and cranial is to the top. There is a displaced fracture of the medial pole of the patella.

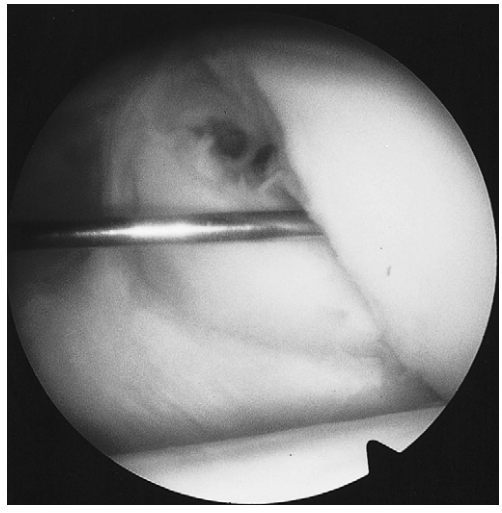

Fig. 47-11 Arthroscopic view of the left patella of the horse in Fig. 47-10 showing the fracture line (*with probe inserted*) in the medial aspect of the patella.

Prognosis Horses with small, non-articular fractures usually have a good prognosis with conservative management.[72] Ten out of 12 horses with medial fractures without concurrent joint disease treated by partial patellectomy returned to athletic use.[69] My experience has been similar. Internal fixation of horizontal and mid-body vertical fractures can be rewarding, but the risk of breakdown during anesthetic recovery is significant, especially in the horizontal fractures. Severely comminuted fractures have a poor prognosis.

Fracture of the Intercondylar Eminence of the Tibia

These fractures have been described as avulsion fractures of the insertion of the cranial cruciate ligament.[58,76,77] However, because the insertion of the cranial cruciate ligament is cranial to the eminence, they are probably not avulsions but may be caused by trauma to the joint involving a lateral force from the medial femoral condyle on the intercondylar eminence.[78]

Signs and diagnosis The history and presenting signs are similar to other acute, severe stifle injuries, and confirmation of the fracture is made radiographically. The caudocranial and flexed lateromedial views are the most useful. Once the acute inflammation has subsided, lameness can be mild to moderate in horses that do not have significant injuries to vital soft tissue structures.

Treatment An arthroscopic assessment is essential for evaluating the fracture and concurrent soft tissue damage in the joint, which is extensive in some horses. If accompanying soft tissue injuries have a reasonable prognosis, treatment of the fracture can be rewarding. Small fragments should be removed. Horses with large fragments can be treated by internal fixation.[78] Alternatively, large fragments can be removed, but this depends on fragment configuration and the involvement of soft tissue structures, such as the cranial cruciate ligament or the cranial ligament of the medial meniscus that wraps around the cranial surface of the eminence.[76,77]

Prognosis If soft tissue damage is reparable, the prognosis for fracture treatment can be good. The possibility always exists of articular cartilage or soft tissue damage that is not detected at arthroscopic examination[78] but that may ultimately cause chronic lameness.

Fracture Fragments off the Femoral Trochlear Ridges or Femoral Condyles in Adult Horses

These injuries usually result from direct external trauma, such as hitting a fence while jumping, a penetrating wound, or a kick.[72,79]

Signs and diagnosis Characteristically there is a sudden-onset, moderate to severe lameness, with a history of acute trauma. Effusion of the femoropatellar joint is usually present. With condylar fractures there is also effusion of the medial femorotibial joint. Crepitus may be present and flexion of the joint causes pain. In the femoropatellar joint fragments arise most commonly from the distal lateral trochlea and are best seen on flexed and standing lateromedial radiographic views. Caudocranial views are also important for identifying the position of fragments in the cranial or caudal femorotibial joints, and cranioproximal-craniodistal oblique views are necessary to rule out concurrent patellar fractures. The fracture site may not be obvious. Because these fractures are often caused by direct trauma, often the stifle region has a wound, and in these circumstances a strong chance of infection in the joint exists. Infection should be ruled out by examining the synovial fluid. Infection can also occur by extension into the joint, hours or even days after the injury. If a sudden increase in lameness occurs, synoviocentesis must be repeated.

Treatment Fragments should be removed to prevent the development of osteoarthritis. Most can be removed arthroscopically,[72,79] but arthrotomy may be necessary for excision of large slabs.[80] In many horses I prefer to wait a few days before examining the joints arthroscopically, because the initial intra-articular hemorrhage hinders surgery. Many horses have a severely inflamed synovium in which fragments may be embedded and difficult to find. Fragments in the femoropatellar joints are often loose, whereas in the femorotibial joints they more often have soft tissue attachments, but conditions vary greatly, and intra-operative radiography to ensure removal of all fragments can be useful. Evaluation of the remainder of the stifle joint for concurrent injuries is important.

Prognosis Horses with trochlear ridge fractures have a good prognosis after fragment removal.[72,79] If no significant soft tissue damage occurred, the prognosis is also favorable after removal of fracture fragments in the femorotibial joint. One should bear in mind on the initial assessment that some of these injuries are not as catastrophic as the radiographic changes suggest.

Salter-Harris Fractures of the Femur, Types III and IV

Type III and IV Salter-Harris fractures of the femur tend to occur in older foals and usually result from a fall with the limb in adduction or from an external trauma. From limited reports, type II fractures, which are not discussed in this section, are the most common physeal fracture; but of the articular fractures, type IV fractures appear to be more common.[81-83] Type IV fractures often have a characteristic configuration in which the fracture begins in the trochlear ridges in the horizontal plane and exits through the caudal lateral diaphyseal cortex.[81]

Signs and diagnosis Foals with minimally displaced fractures may be weight bearing, but the others usually have acute, severe lameness, swelling, pain, and often crepitus in the affected joint. Radiography confirms the fracture and should include enough views to establish its configuration.

Treatment Conservative treatment may be appropriate for horses with stable, minimally displaced fractures.[81] Internal fixation is indicated for horses with displaced or unstable fractures. Repairs using lag screw fixation, cobra head bone plates, and angle blade plates have been reported.[81-84] Implanting screws through the articular cartilage of the femoral trochlea is sometimes necessary, and crossing the physis with an implant does not seem to have affected growth in the distal femoral physis in older foals.[81] The dynamic condylar screw plate may be of value.

Prognosis From the few published reports[81] comes evidence that foals with minimally displaced fractures have a good chance of achieving athletic use with conservative treatment. If surgery is required, the outcome is more equivocal, but good success has been achieved in some horses.

Fractures of the Tibial Tuberosity

The tibial tuberosity is a relatively exposed structure and is susceptible to fracture after direct trauma, such as a kick, or a collision with a fence. These fractures do not seem to be associated with the growth plate of the tibial tuberosity or with avulsions of the insertions of the patellar ligaments, though the lateral patellar ligament is often involved. They may not be true avulsion fractures, but because of the patellar ligaments, some do displace proximally.[85] Traction apophysitis similar to Osgood-Schlatter disease in people has been reported in horses[86] but is rare. In the horse reported, lameness was mild and small fragments detached from the cranial aspect of the tibial tuberosity were visible radiographically. A variety of fracture configurations occur, from small fragments on the cranial proximal aspect, to large fractures extending from distal to the tibial crest to proximally into the femorotibial joint.

Signs and diagnosis Most horses are acutely, severely lame immediately after sustaining the fracture, but in some this may settle into a milder lameness if left untreated. Swelling and crepitus are common. A wound on the lateral tibia may be present and intra-articular fractures may be associated with joint effusion. Care should be taken to rule out other injuries

in the stifle, such as fracture of the patella, or joint infection if an open wound exists. Infection can even develop by extension into the joint through a skin graze. Because of the lateral curvature of the tibial tuberosity, several radiographic views are often required to identify the contour of the fracture, the caudolateral-craniomedial oblique view being the most useful. The tibial tuberosity does not fuse until at least 3 years of age,[87] and its physis should not be confused with a fracture.

Treatment Non-displaced fractures may heal with stable rest, but one should bear in mind that displacement can occur some time after the injury because of the pull of the quadriceps muscles on the patellar ligaments. Because of this, preventing the horse from lying down for 2 weeks is wise. Small fragments can be removed if they do not involve disruption of the middle patellar ligament. Horses with large fractures heal satisfactorily if treated conservatively in the Editors' experience, provided they are not displaced. Large fractures can also be treated by internal fixation. Lag screw fixation does not provide a secure enough repair, so tension band wiring,[88] a lateral narrow dynamic compression plate,[89] and a cranial narrow dynamic compression plate[85] have been used. I believe that the most secure repair is achieved with the latter (Fig. 47-12). The plate should extend well distal to the distal extremity of the fracture to withstand the distraction forces of the quadriceps muscles. Because of the shape of the cranial proximal tibia, contouring of the plate demands considerable twisting and bending, but this does not seem to affect its efficacy. A technique for preoperative plate bending has been reported as a way of saving surgery time,[90] but in my experience this is not especially time consuming. Most horses are substantially more comfortable postoperatively, but they should be prevented from lying down for 2 weeks.

Prognosis Conservative treatment of horses with non-displaced, small fragments results in a good prognosis. Horses with larger fractures may respond to conservative treatment with complete return to soundness, but convalescence is often prolonged. Good results for return to full athletic function have been reported after repair with a cranially applied plate,[85] and in my view this is the treatment of choice. A potential risk exists for failure of the repair during anesthetic recovery, or the early postoperative period,[89] or for tibial fracture. This emphasizes the importance of adequate fixation and restriction of the horse during early convalescence.

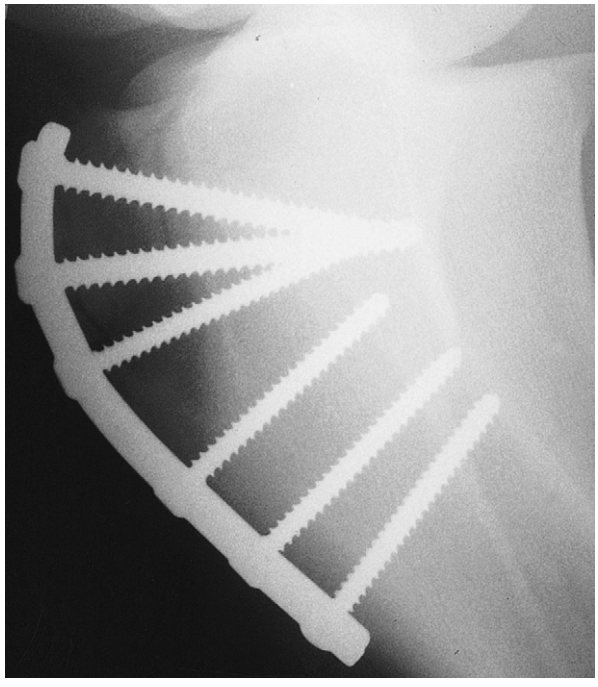

Fig. 47-12 Postoperative lateromedial radiographic view of the right tibia of 13-year-old pony gelding that had sustained a fracture of the tibial tuberosity. The fracture had been treated by internal fixation with a narrow dynamic compression plate.

Fractures of the Fibula

Fracture of the fibula is usually caused by direct trauma and is a rare injury. Lameness is often moderate to severe at first. Palpation of the region may not elicit obvious pain, but radiography should be diagnostic. The caudocranial view is the most useful, but great care must be taken to differentiate a fracture from persistent radiolucent lines that result from separate ossification centers in the fibula. As many as three of these lines can be present, and they may be irregular. Scintigraphy is a useful aid to diagnosis in the absence of localizing clinical signs or if one has difficulty interpreting the x-ray films. From limited experience, confinement of the horse for 2 to 4 months is usually long enough for reasonable healing to take place, and the prognosis is good.

Other Diseases

Avulsion of the Origin of the Fibularis (Peroneus) Tertius and Long Digital Extensor Tendon

Avulsion of the origin of the fibularis tertius and long digital extensor tendon is a relatively rare condition in horses and is usually caused by sudden, forceful hyperextension of the hindlimb. Mid-substance tears are probably more common than avulsions of the common tendon of origin of the fibularis tertius and long digital extensor muscles, but the latter have been reported in foals.[91-93] Avulsions of the origin of the fibularis tertius and long digital extensor muscle occasionally occur in adult horses,[5] although more distal injuries are commoner. Foals may be more likely to suffer avulsions because of weaker bone.[92] Minor disruption of the common tendon of origin may be seen at arthroscopy.[5]

Signs and diagnosis The classical sign of rupture of fibularis tertius and avulsion of its origin is the ability to extend the hock while the stifle is flexed, resulting in a dimpling of the Achilles tendon. Synchronous flexion of the hock and stifle has been reported with avulsions of the origin, presumably because the avulsed bone is held by the joint capsule.[92] Horses may bear weight normally, but during the protraction phase of a stride, the distal limb appears flaccid. Lameness can be severe at first and distention of the femorotibial and femoropatellar joints may be present. Radiography is necessary for diagnosing avulsion fractures. Minor disruptions of the tendon of origin manifest as a non-specific lameness associated with pain in the stifle and can be diagnosed by arthroscopic examination; they may accompany other stifle injuries.[5]

Treatment Horses with injuries to the tendon of origin are usually best evaluated arthroscopically, which also offers the opportunity to check for concurrent injury. Small bone fragments can be removed, but large fragments embedded in the joint capsule may be better left.[92] Six weeks of stable rest followed by controlled exercise for 3 months is recommended after surgery.

Prognosis Horses with avulsion injuries have a guarded prognosis for full soundness, but successful outcome has been reported.[92] Minor disruptions of the origin may heal, but much depends on concurrent injuries.

Calcinosis Circumscripta

Calcinosis circumscripta lesions are described as calcified, granular, amorphous deposits that induce fibrous reaction.[94] They usually lie in the subcutaneous tissue close to joints or tendon sheaths. The lateral aspect of the stifle adjacent to the fibula is the predilection site for these lesions,[94-96] but they have been reported on the dorsolateral surface of the hock,[94]

the neck, and the shoulder.[94,95] Calcinosis circumscripta is an uncommon condition, more often seen in young horses. The cause is unknown.

Signs and diagnosis The usual reason for presentation is an unsightly lesion that has been slowly increasing in size. Occasionally the lesion appears to affect the horse's gait. Lesions are usually hard, well circumscribed, and firmly attached to underlying tissue. The overlying skin is not involved. Caudocranial radiographs show a circumscribed, roughly oval lesion of mineralized tissue (Fig. 47-13) lying close to the lateral aspect of the femorotibial joint, with the bulk of the lesion distal to this. These lesions may be seen incidentally on scintigraphic examination as focal areas of intense increased radiopharmaceutical uptake on the lateral aspect of the proximal tibia. The lesion is clearly visible in a caudal view in a subcutaneous location, superficial to the tibial cortex, but in a lateral view could be confused with a proximal tibial stress fracture.

Treatment Treatment is only indicated if the lesion is causing clinical complications or for cosmetic reasons. Surgical excision is necessary to remove the mass. Care should be taken when embarking on excision because some lesions are attached to the femorotibial joint capsule, which may be entered inadvertently. Steps should be taken to prevent postoperative seroma formation by closing dead space, suturing a stent bandage over the wound, and preventing the horse from lying down for several days postoperatively. The mass usually has a thick fibrous capsule and contains deposits of gritty material.

Prognosis In horses that are not treated, the mass remains but may not cause complications. A good result can be expected after surgical excision. Regrowth has not been reported, but postoperative problems with wound healing could be serious if the femorotibial joint is involved.

Hematoma in the Stifle Region

The stifle region is vulnerable to external trauma, particularly when horses are jumping. Profound injuries to vital structures may result, but frequently external trauma causes only superficial bruising or a subcutaneous hematoma. The clinician's task is to differentiate between a swollen stifle that has a potentially career-threatening injury and one that has a superficial lesion such as a hematoma.

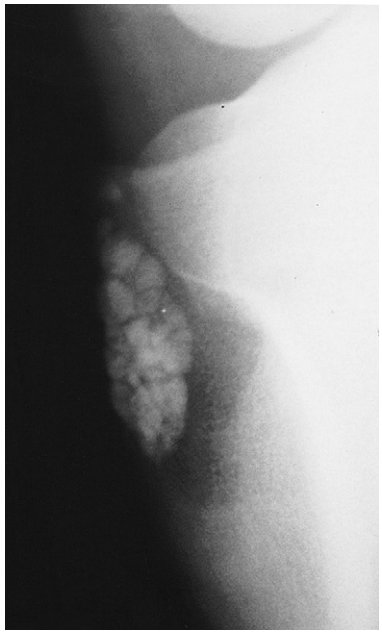

Fig. 47-13 Caudocranial radiographic view of the stifle of an 8-year-old riding horse with an oval mass of mineralized tissue lateral to the head of the left tibia. The lesion is typical of calcinosis circumscripta.

Signs and diagnosis A hematoma is manifested as a variably sized swelling, usually on the cranial aspect of the stifle, and may take several hours to reach maximum size. Smaller swellings may be difficult to differentiate from femoropatellar joint effusion. A hematoma is only painful on palpation until the initial bruising and swelling has settled. An uncomplicated hematoma does not usually cause lameness once the discomfort associated with the initial bleeding has passed. Other concurrent injury should be suspected if the horse is significantly lame, especially if lameness persists for several days. A hematoma may occasionally involve only the stifle initially but appear to spread to the inguinal and ventral abdominal areas. Huge swellings can cause oozing from the skin and skin loss. A careful search should be made for open wounds, which could lead to infection in the hemotoma or cellulitis. Cellulitis usually takes at least 24 hours to form, the swelling is firmer than a hematoma, hot, and painful. Lameness worsens as infection progresses.

Ultrasonography is a helpful tool for diagnosing hemotoma, which can be confirmed by aseptic centesis of the swelling. In the early stages, frank blood is retrieved, but as the blood clots, the fluid collected will be serum. Radiographic examination is indicated if lameness is severe or persists.

Treatment In the initial phase, when hemorrhage is likely, the horse should be confined and cold applied to help to slow hemorrhage. Phenylbutazone can be given for its anti-inflammatory effect. Antibiotics should be administered if the horse has an open wound. Although some clinicians feel that draining these hematomas is contraindicated, in my experience hematomas resolve better after effective drainage. Drainage should not be attempted until the hemorrhage has stopped and the free blood has clotted, usually 3 to 4 days after the injury. A 2- to 4-cm scalpel incision at the most dependent point of the hematoma ensures that drainage continues until the cavity has filled in.

Prognosis In the absence of concurrent injury or infection and once the hematoma has resolved and is free of fluid, the prognosis for athletic use is good.

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CHAPTER • 48

The Thigh

Dan L. Hawkins

THE GLUTEAL SYNDROME

Pain that can be demonstrated with digital pressure along a line between the wing of the ilium and the greater trochanter of the femur was first recognized as a cause of lameness 45 years ago.[1-3] Various aspects of the origin, diagnosis, and management of the syndrome have been appreciated since its initial recognition by Dr. Churchill and myself.[1-5] The condition has also been referred to as *sacrosciatic lameness* or *pelvic myositis*. We have preferred to use the term *gluteal syndrome* because we believe that there may be more than one abnormality that will result in this type of pain.

Gluteal syndrome has been diagnosed in most breeds and uses of horses. Standardbred (STB) racehorses seem to have the highest incidence, but it is not uncommonly seen in Thoroughbred (TB) racehorses. One risk factor for STBs is related to the more recent harness and race bike designs that tend to decrease the weight borne by the forelimbs while transferring weight to the hindlimbs. Gluteal syndrome has been diagnosed in horses that are used for hunting, jumping, eventing, and endurance racing.

Based on dissections of cadavers, the accessory head of the middle gluteal muscle was identified as the deep structure that best corresponds anatomically to the characteristic pattern of pain. This muscle originates on the concave surface of the wing of the ilium near the tuber coxae. At its caudal extent, the accessory head of the middle gluteal muscle forms a flat tendon that passes over the cranial aspect of the greater trochanter of the femur and inserts on the crest below it. The accessory head is directly related to other aspects of the middle gluteal and superficial gluteal muscles superficially and is important in propulsion of the hindlimbs[6] (Fig. 48-1).

The pathological process associated with the accessory head of the middle gluteal muscle is uncertain. It is known that in horses with some acute injuries a hematoma may form in the area between the ilium and greater trochanter. Small, hypoechogenic areas may be demonstrated ultrasonographically in muscle tissue in the same area. In other horses the muscle may become partially detached from the ilium. Horses with the latter condition develop a depression in the musculature caudal to the tuber coxae after the acute stage. In horses with chronic pain the affected muscle mass may become very firm, presumably as a result of fibrosis. Post-mortem evaluation of two horses with chronic refractory gluteal syndrome did not provide any conclusive information. Other potential changes such as fasciitis, gluteal tendonitis, or injury to one or more adjacent muscles have been considered. Whatever the cause, the source of pain is consistently confined to the area described.

To date, the most reliable, practical method of diagnosis is by careful, systematic physical examination of the area between the wing of the ilium and the greater trochanter of the femur with the horse standing on the limb. More specifically, the musculature should be palpated and examined for changes in resilience, swelling, or fibrosis. The position and outline of the tuber coxae should be evaluated. Next, gentle, deep pressure should be applied along the entire area

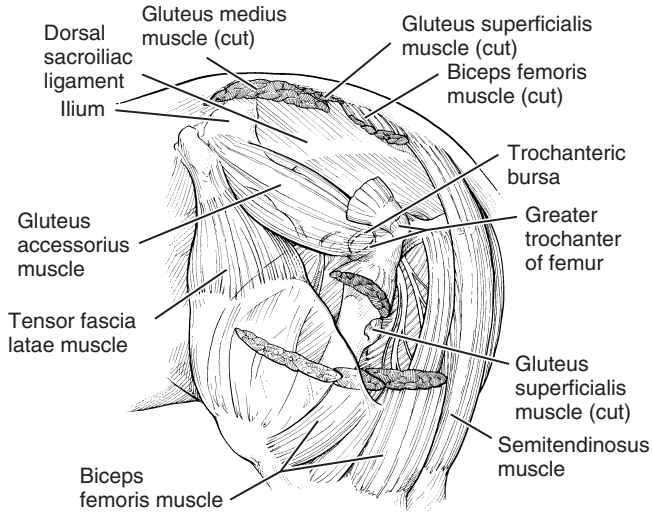

Fig. 48-1 Drawing of the anatomy of the lateral pelvic and proximal thigh regions showing the accessory head of the middle gluteal muscle, the most likely source of pain associated with gluteal syndrome. The tendon of the accessory head of the middle gluteal passes over the cranial portion of the greater trochanter where there is a bursa. Inflammation in this region causes trochanteric bursitis.

Fig. 48-2 Careful palpation for pain is the only way to establish the clinical diagnosis of gluteal syndrome.

described with the tips of eight digits (Fig. 48-2). Initially, light digital pressure should be applied, but then the pressure is gradually increased to firm, deep pressure. The pressure is maintained briefly to evaluate the horse's response. Finally the area caudal to the ilium, the central portion, and the caudal part of the accessory head of the middle gluteal muscle should be examined similarly. Each one third of the area should be considered separately to determine whether the area is affected generally or if the injury is confined to one or more

sections. A sound horse will not respond to the examination of any of the areas. An affected horse leans away from the pressure while showing a reluctance to voluntarily bear full weight on the limb as long as the pressure is maintained. A positive response for this syndrome as described is specific and should not be confused with that for trochanteric bursitis (see section entitled "Trochanteric Bursitis").

Most horses with acute gluteal syndrome tend to drag the toe of the affected limb on the ground at a walk. Horses with chronic gluteal syndrome do not drag the toe at a walk, but at a slow trot they break over on the inside of the toe and swing the leg medially and then laterally, finally landing on the outside branch or toe of the shoe. At this gait the lameness produced appears identical to that caused by trochanteric bursitis or tarsitis. When the horse trots, paces, or runs at high speed, the abnormality starts off as described, then the limb appears to tire and is carried more in abduction the further in the race or competition the horse proceeds. It appears that early in the race the horse is able to tolerate the gluteal pain and continue to use the limb for propulsion. However, with sustained speed or intensity of effort over distance, compensatory mechanisms within the limb fatigue as pain from the gluteal injury becomes overwhelming. At this point a trotter or pacer may make a break or freeze on a line, a TB may bolt or stop trying in the race, and a show horse may refuse to jump or crash when it tries. Many of these horses are believed to have a stifle problem because the quadriceps muscles fasciculate when they pull up. Others are believed to have exercise-induced pulmonary hemorrhage because they stopped like a bleeder, but no blood is seen endoscopically. Regardless, speed, intensity of work, and distance are key factors in how this problem affects an athletic horse. Some horses that accommodate to the injury, or are treated and do not recover to total soundness, can perform reasonably well at slower speeds or less intense work.

There is not a specific cause for gluteal syndrome. The onset is frequently directly associated with being cast in a stall, slipping or falling while playing in a paddock, or going down in a trailer or van and struggling to get up while the head is tied. Gluteal syndrome also develops when there is a chronic, pre-existing problem in the distal aspect of the same limb, such as hock lameness, suspensory desmitis, or other problem that causes the horse to carry its weight on the toe at high speed. Slipping or losing footing on a soft racetrack or other muddy surface is also associated with development of the clinical signs.

Horses with this injury are best treated while in moderate, controlled exercise, regardless of the treatment approach. Rest alone, even for extended periods of time, has not been successful as a treatment. Several weeks of careful exercise management with repeated evaluations provide the best chance for a favorable response to treatment. Horses with acute injury, hemorrhage, and severe lameness should be rested until they are walking well and the seroma has resorbed before resuming exercise and treatment. Intramuscular injections of a counterirritant, acupuncture, or therapeutic ultrasound are the best treatment options. At present, I still believe that injections of a counterirritant into the area of sensitivity yield the best results. The injection site is prepared with a surgical scrub followed by rinsing with 70% isopropyl alcohol. An injection grid is marked on the horse by drawing three lines in the wet hair over the accessory head of the middle gluteal muscle (Fig. 48-3). One hundred milliliters of a 2% iodine diluted in sesame oil solution is injected intramuscularly into 20 sites (5 ml per site) on and between the grid lines with a 4-cm, 19-gauge needle. If the pain is concentrated in the cranial or caudal one third of the area between the tuber coxae and the greater trochanter of the femur, the injection sites can be limited to the cranial or caudal half of the area, respectfully. After injection moderate, controlled exercise is recommended on a daily basis beginning the day after injection for all horses. After injection horses with acute or recent gluteal syndrome are walked and jogged for 3 weeks and then re-examined. If the lameness is improved, but the area is still sensitive or if there has been no improvement, the injection is repeated in the same manner. Horses with chronic gluteal syndrome are walked and jogged for only 1 week before light training is resumed. Some horses respond dramatically to the treatment and appear to be pain-free within less than a week. The injury cannot heal within that time frame; therefore the temptation to resume full training too quickly must be resisted. When the treatment is successful and horses return to full work and racing, 10% to 20% may experience a recurrence of the problem. These horses generally respond well to treatment as they did previously.

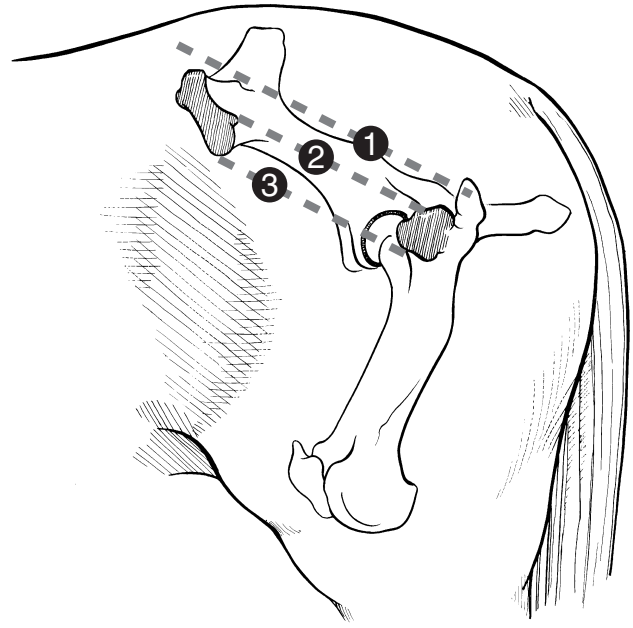

Fig. 48-3 Diagrammatic representation of the injection pattern for intramuscular injections of counterirritant solution to manage gluteal syndrome.

Lameness that fails to improve after two or three injections at 3-week intervals is considered refractory to this mode of treatment. For these horses, I suggest that the trainer or owner try another form of treatment, such as therapeutic ultrasound or acupuncture. Approximately 10% to 15% of horses do not respond to any mode of treatment and never recover from the injury. The prognosis is guarded initially for unilateral injury and poor if the problem is bilateral or if there is a chronic condition more distally in the same leg. In these more complicated cases, failure is related to the aggravating effect of each injury on the other.

Shoeing can be a factor in the management of the gluteal syndrome. Slipping on the racetrack, cross-country course, or show jumping arena is a major factor in development and recurrence of the injury. I recommend that TB racehorses that have or have had this condition be shod with block heels on both branches of each hind shoe. Calks applied near the end of both branches of the hind shoes during training and competition can be used in other types of horses.

TROCHANTERIC BURSITIS

Pain elicited by firm digital pressure over the greater trochanter of the femur and the musculature immediately caudal to it is attributed to inflammation of the trochanteric bursa and is a cause of lameness in horses.[1,2] Historically this condition has

been referred to as trochanteric bursitis, trochanteric lameness, and whorl bone lameness.[7] Anatomically the bursa is interposed between the flat tendon of the accessory head of the middle gluteal muscle and the cranial portion of the greater trochanter, which is covered with cartilage at this point. The tendon inserts on the crest below the greater trochanter (see Fig. 48-1).[7]

Although confident, positive diagnosis is achieved by physical examination in almost all horses, a technique of injection of local anesthetic solution into the bursa has been described.[8] The condition is relatively common for all breeds and uses of athletic horses and is generally considered to be a secondary problem. Trochanteric bursitis often is associated with tarsitis or other problems in the affected limb in which the horse lands on the toe. An additional cause that has been overlooked is that associated with over-reaching with the hindlimbs to compensate for a chronic problem in the forelimbs. A horse that is chronically sore in front (e.g., navicular disease) will try to carry more of its weight on the hindlimbs by over-extending the ipsilateral limb, which results in excessive strain on the tendon of the middle gluteal muscle. Wet, cold weather conditions appear to have a positive association with the incidence and response to treatment.

Trochanteric bursitis causes a gait abnormality that is similar to that produced by tarsitis. A horse with purely trochanteric lameness breaks over on the medial aspect of the toe; carries the leg medially, even crossing the midline; and then the leg moves laterally to land on the lateral aspect of the toe and branch of the shoe. The length of stride is shortened and the hindquarters are carried toward the opposite side, which is sometimes described as "dog trotting" at a slow gait. Trochanteric lameness especially affects horses when they are racing around turns, jumping, circling, or performing precision dressage movements.

Trochanteric bursitis is generally a chronic condition that is diagnosed at the same time as a primary condition in the lower limb or ipsilateral forelimb. The more distal condition (e.g., tarsitis) should be addressed before the bursitis is treated. Occasionally trochanteric bursitis is identified without an associated primary condition, or it may spontaneously resolve after correction of the associated primary lameness.

Rest is of no value in treating trochanteric bursitis and may intensify the lameness when the horse returns to training. The most effective, practical treatment of trochanteric bursitis is injection of the soft tissues over and around the bursa with a counterirritant solution. The area for injection is prepared with a surgical scrub followed by rinsing with 70% isopropyl alcohol. Approximately 6 ml of a 2% iodine diluted in sesame oil solution should be injected into 8 sites with a 4-cm, 19-gauge needle (Fig. 48-4). The horse should be maintained in mild, controlled exercise for 1 week before resuming normal work. Normally within 5 to 7 days after treatment the horse should become sound behind and no longer resent palpation over the bursa. Horses that require reinjection are more commonly encountered during cold, wet climatic conditions. The prognosis is good for obtaining a cure for trochanteric bursitis provided the associated primary lameness is corrected. Alternatively, solutions of a corticosteroid can be injected in the same manner. In my experience injection of corticosteroids directly into the bursa is not necessary to alleviate the lameness.

Trochanteric bursitis has been addressed here as a performance-limiting condition in the athletic horse. Trochanteric bursitis that results from external trauma, infection associated with a wound, or a fracture of the greater trochanter of the femur is not discussed.

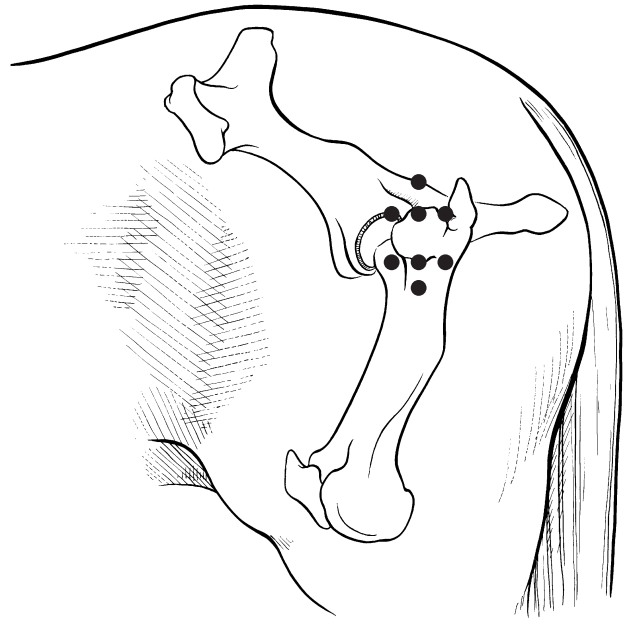

Fig. 48-4 Diagrammatic representation of the injection pattern to manage horses with trochanteric bursitis.

FRACTURES OF THE FEMUR

Fractures of the femur occur most commonly in foals and weanlings, although they may be seen in older horses. Most femoral fractures result from external trauma, such as a kick or fall with the limb in adduction or abduction. In young foals fractures may be associated with breaking to lead. In most horses the traumatic incident is not witnessed, but the horse is found with an unstable, non–weight-bearing lameness in the affected hindlimb. Because of the energy required to fracture the femur of an adult horse, the fracture generally has a severely comminuted configuration. Although foals and weanlings most commonly acquire diaphyseal femur fractures, fractures involving the proximal or distal femoral physis are not uncommon. Salter-Harris type II distal femur fractures are most commonly seen in older weanlings and yearlings.

Diagnosis

In most horses the diagnosis can be made after a brief physical examination. Femur fractures result in an acute onset of severe lameness, with swelling in the area of the femur. Rotational instability is elicited when internal or external rotation is applied by grasping the proximal tibia and calcaneal tuber. Crepitation may be appreciated with manipulation of the limb. However, swelling and hemorrhage in the large muscle mass of the thigh may keep the fracture ends separated and make it difficult to elicit crepitus. The large muscle mass also makes open fractures uncommon. Swelling is most consistent with diaphyseal fractures, but it is often less obvious with proximal physeal fractures and minimally displaced distal physeal fractures. In most horses the distance between reference points, such as the patella and the greater trochanter of the femur, is shortened because of the overriding of the fracture fragments that is caused by contraction of the quadriceps muscles. Foals with proximal physeal fractures may be able to bear some weight on the affected limb.

Radiographic evaluation is required to obtain a definitive diagnosis and determine the severity of a femoral fracture. Radiography of horses larger than young foals requires use of general anesthesia unless the fracture is located in the distal femur. Many small foals can undergo radiography safely when recumbent under heavy tranquilization or anesthesia. Because induction and recovery of large yearlings and adults cannot be assisted sufficiently to prevent complications and discomfort, anesthesia is contraindicated for assessment of unstable femur fractures. Ultrasonography can be used to confirm a displaced femoral fracture in a standing horse.

Once the horse is in lateral recumbency with the affected limb down against the cassette and the opposite limb abducted, the x-ray beam should be directed from medial to lateral. Radiography of the diaphysis and proximal femur requires a grid and high-capacity radiographic generator except for examination of small foals and the distal femur of larger horses. Two exposures are generally required for the mediolateral images because of the variation in soft tissue mass around the distal and proximal femur. Craniocaudal views are also required to assess the severity of the fracture and the degree of overriding. Evaluation of the proximal physis requires that the horse be positioned in dorsal recumbency with the cassette and grid under the coxofemoral area.

Case Selection for Treatment and Prognosis

The decision to treat horses with femoral fractures by surgical intervention depends on several variables. The size of the patient is the single most important consideration, regardless of fracture type. Successful surgical treatment of diaphyseal or proximal physeal fractures has been accomplished only in foals and small ponies.[9] Distal physeal fractures have been successfully repaired in yearlings.[10,11] However, adult horses or young horses weighing more than 200 kg (440 lb) with displaced diaphyseal fractures warrant humane destruction.[12] Fracture location and configuration, soft tissue injury, temperament of the horse, client expectations, and economic considerations are other important determinants for case selection. Compromise of the vascular supply to the distal limb that is caused by the fracture, damaged major vessels, and swelling can result in failure due to necrosis of the distal limb.[13]

Surgical techniques, complications, and the prognosis for horses with diaphyseal and proximal and distal physeal femur fractures have been reviewed.[9,12,14] Of foals with diaphyseal fractures that were repaired with double plating, 50% of the fractures healed and 75% of the horses were able to perform the intended use.[15] The most common complications were seroma formation and infection. Foals with a successful outcome were younger than 3 months of age. Additionally, an intact caudal cortex was considered necessary for a successful outcome. Conservative management of horses with diaphyseal fractures can result in fracture healing. However, limb shortening, rotational deformity, and serious varus deformity in the contralateral limb are common complications of this approach. The prognosis of horses with distal fractures was more guarded than that for diaphyseal fractures because of the limited amount of bone for implant purchase. Conservative management of horses with distal fractures can result in a satisfactory outcome if the fracture is minimally displaced and stable.[15]

Although proximal femoral physeal fractures account for 16% of physeal fractures in the horse,[16] sufficient reported results of surgical treatment to establish good prognostic information are lacking. Currently the prognosis for successful repair is guarded at best. Conservative management is unlikely to result in a comfortable horse. Unstable fixation, necrosis of the femoral head, and osteoarthritis of the coxofemoral joint are the more common complications associated with failure of surgical or conservative approaches.

Other less common types of proximal femoral fractures that cause less severe lameness in older horses include the third trochanter (see Chapter 52) and greater trochanter. Ultrasonography is helpful in confirming these fractures. Unless these fractures are associated with a wound or become sequestrated, they should be managed with stall rest.[9] Stress fractures of the femur are rare but occasionally occur in the proximal cranial femoral cortex (Editors).

OSSIFYING MYOSITIS

See Chapter 81 for a discussion of ossifying myositis.

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CHAPTER • 49

Mechanical Lameness in the Hindlimb

Beth A. Valentine

GENERAL CONSIDERATIONS

Mechanical lameness reflects altered biomechanical forces affecting limb function. The biomechanics of limb function depend on normal functioning of peripheral nerves, muscles, tendons, and ligaments. Appropriate muscle contraction and relaxation are important factors in limb biomechanics. A muscle that cannot contract with sufficient force, or a muscle that cannot relax and stretch, can result in a significantly altered gait. Mechanical lamenesses are typically most obvious at the walk and often become less evident or disappear at the trot. Gait alterations are usually not evident at the canter, although a horse with a mechanical lameness may not be capable of generating a smooth canter and may appear to be hopping or "off" behind at the canter. Mechanical lamenesses include altered gaits caused by decreased and increased joint flexion.

The importance of proper functioning of the neuromuscular system for a normal hindlimb gait is exemplified by horses with chronic motor neuron disease and myopathy from equine polysaccharide storage myopathy (EPSSM). Both of these relatively recently recognized neuromuscular disorders can cause a fibrotic myopathy type or a stringhalt type of gait. Horses with EPSSM may develop prolonged or intermittent upward fixation of the stifle. Underlying myopathy was found in horses with shivers (shiverers).[1,2] Neuropathy is known to be a cause of stringhalt[3,4] and was found in horses with fibrotic myopathy.[5] Trauma to peripheral nerves may also cause a mechanical lameness. The possible role of altered proprioceptive input to the affected limb is an intriguing one and is discussed under appropriate headings.

Equine protozoal myelitis or myeloencephalitis (EPM) with involvement of motor neurons causing selective denervation may also cause a mechanical lameness. Horses with lameness from EPM should, however, also exhibit proprioceptive deficits and ataxia. EPM is unlikely to cause an obvious mechanical lameness without accompanying ataxia. I have difficulty accepting that EPM could cause selective damage to motor neurons sufficient to cause a mechanical lameness without also causing concurrent spinal cord white matter damage and ataxia. However, neurological deficits may be subtle early in the disease. Careful neurological evaluation by a clinician with expertise in equine neurological evaluation is an important part of examining a horse with a mechanical lameness, because distinguishing between gait abnormality caused by mechanical lameness and gait abnormality caused by neurological disease may be difficult.

Electromyography and muscle biopsy may aid in determining the cause of a mechanical lameness. Concentric needle electromyography of denervated muscle often reveals abnormal spontaneous activity such as positive sharp waves, fibrillations, and myotonic bursts. Abnormal spontaneous activity may also be present in muscles of horses with myopathy, but these findings are generally mild and may be absent. Biopsy of the semimembranosus or semitendinosus muscle is useful for evaluating evidence of denervation atrophy and of EPSSM. However, if denervation of a single muscle or part of a muscle causes the gait alteration, sampling error may result in a false-negative result.

Mechanical lameness does not usually cause pain and horses usually do not respond to anti-inflammatory therapy. Methocarbamol therapy is indicated only when muscle cramping from central nervous system disease is suspected. Phenytoin therapy has proved to be useful in some horses with mechanical lameness, particularly those with stringhalt.

Mechanical lameness in the horse is from abnormal structure or function of the musculoskeletal system, or more commonly, of the neuromuscular system. Possibly a variety of underlying problems can result in the same type of mechanical lameness.

ANATOMICAL CONSIDERATIONS

A number of unique anatomical features in the equine hindlimb contribute to mechanical lameness. The configuration of the patellar ligaments in the stifle joint allows locking of the stifle into an extended position. The stay apparatus minimizes the muscular effort required for a horse to stand for long periods of time. The reciprocal apparatus links the actions of the stifle and hock, primarily through the fibularis (peroneus) tertius and superficial digital flexor tendons, such that an abnormal action in either joint affects the action of both.

Innervation patterns to the muscles of the hindlimbs may also contribute to mechanical lameness. The semitendinosus muscle, for example, receives innervation from two different nerves, the caudal gluteal and sciatic nerves. Often more than one muscular branch innervates long muscles such as the semitendinosus and possibly the semimembranosus. Damage to one nerve or to a muscular branch can result in partial denervation of a large muscle, with resultant altered biomechanical forces leading to abnormal limb action.

UPWARD FIXATION OF THE PATELLA (LOCKING STIFLE OR FIXATION OF THE PATELLA)

Clinical Characteristics

Horses with upward fixation of the patella are periodically unable to flex the stifle or the hock and drag the extended limb behind them on the toe (Fig. 49-1). The condition occurs most commonly after a period of standing still and tends to decrease with continued exercise. An intermittent form of patellar fixation, called *delayed release of the patella*, also occurs, in which the patella appears occasionally to catch briefly, followed by exaggerated flexion of the stifle and hock. This gait resembles the abnormal gait of stringhalt and shivers and may occasionally occur at the trot and canter, in addition to at the walk. Affected horses may appear to be in some discomfort, particularly if the joint is locked for an extended time, and can become somewhat irritable.

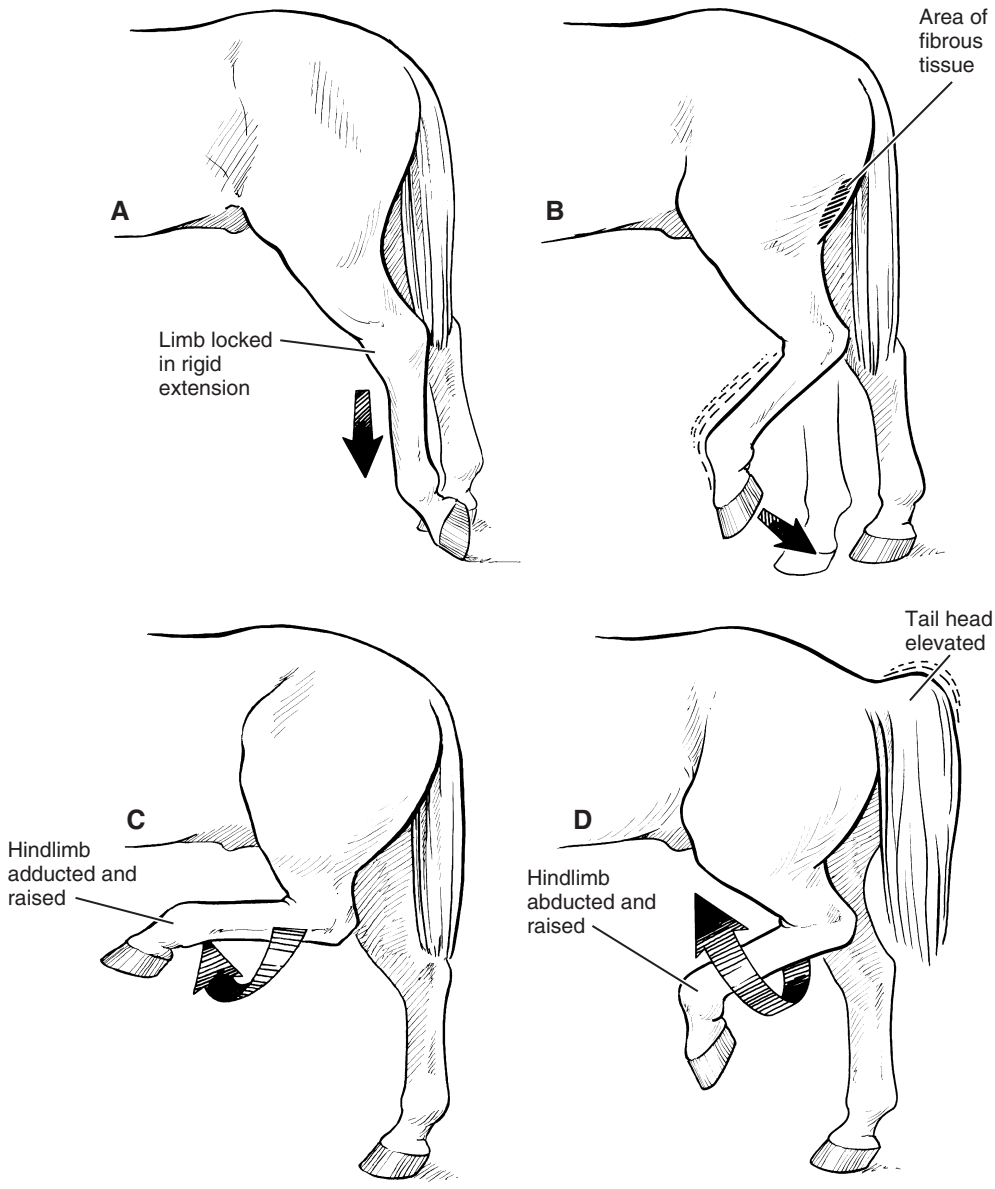

Fig. 49-1 Diagrammatic representation of the common mechanical hindlimb gait abnormalities. **A,** In horses with upward fixation of the patella the hindlimb is periodically held in rigid extension. The fetlock joint is flexed, and the horse walks on the toe. Horses with intermittent upward fixation can have a gait similar to shivers (see **D**). **B,** Horses with fibrotic myopathy show slapping of the affected hindlimb downward just before the end of the cranial phase of every walk stride. Hock flexion is near normal, but fibrous tissue in the caudal thigh region limits the cranial phase of the stride. **C,** Horses with stringhalt exaggerate flexion of the hock with the hindlimb in a normal (adducted) position. This gait occurs at every walk stride. Excessive hock flexion causes some horses to hit the ventral abdomen with the dorsum of the hindlimb. **D,** Horses with shivers often have abnormal tail head elevation and hold the affected hindlimb in a flexed and raised position away from the midline (abducted). This abnormal gait occurs sporadically, not at every walk step, which helps to distinguish it from stringhalt.

Low-grade intermittent upward fixation or delayed release of the patella is relatively common, but it is often difficult to detect. Affected horses have a slightly jerky movement of the hindlimb that may be most apparent during deceleration from trot to walk and when the horse is working in deep footing, especially on turns. Warmbloods, Thoroughbreds, and Standardbreds may be predisposed to upward fixation of the patella. The episodic nature of intermittent upward fixation of the patella adds to the difficulty of detecting a horse with low-grade signs. Careful observation while moving the horse over from side to side, or walking down an incline, can be particularly useful.

Cause

Upward fixation of the patella has been attributed to abnormal laxity and to increased tenseness of patellar ligaments. Decreased force of muscle contraction of the thigh muscles (quadriceps and biceps femoris) that move the patella out of

the locked position may also cause upward fixation of the patella. This may be the explanation for upward fixation of the patella occurring in horses with underlying myopathy, causing weak or stiff muscles. Horses and ponies with overly straight hindlimbs may be prone to upward fixation of the patella, because such conformation can result in overextension of the stifle joint, leading to patellar locking.[6] Permanent upward fixation of the patella rarely occurs secondary to luxation or subluxation of the ipsilateral coxofemoral joint.

Biomechanical Basis

Upward fixation of the patella is caused by failure of the medial patellar ligament to unhook from the medial ridge of the femoral trochlea, causing the stifle to lock in extension. Abnormalities of the fibrocartilage at the medial aspect of the patella also possibly make the joint prone to locking. Once the stifle is locked in extension, the hock is also fixed in extension through the action of the reciprocal apparatus. The biomechanical basis for stifle hyperflexion after the brief period of patellar catching in intermittent upward fixation of the patella (or delayed release of the patella) is not entirely clear and may be from increased force of contraction of the biceps and quadriceps muscles during the transient locking phase. Clearly, therapy for this disorder should be directed at correcting the underlying cause. Unfortunately, for many horses an exact cause is never determined.

Medical Therapy

Exercise is an important part of therapy for patellar fixation. Conditioning exercise, including work on hills, may result in complete resolution of the problem in some horses. Uphill work may be more beneficial than downhill exercise. Horses that respond to conditioning exercise indicate that muscle dysfunction can play an important role in the development of upward fixation of the patella. If underlying myopathy such as EPSSM is the cause, diet change to one that is high in fat and low in starches and sugars[7] and continued exercise conditioning may be beneficial. The most effective diets are those that provide at least 20% to 25% of total daily energy from fat and less than 20% of total daily energy from starches and sugars.

Another medical therapy is administration of estrogenic compounds. The rationale is presumably that estrogens can cause tendon and ligament relaxation. Whether horses with upward fixation of the patella have overly tense patellar ligaments and whether estrogen has any effect on patellar ligaments and tendons is unclear. Estrogen effects on muscle cell metabolism and muscle tone are possible. Anecdotal evidence suggests that some horses may benefit from this type of therapy. Intramuscular injection of 1 mg estradiol cypionate per 45 kg of body weight (i.e., 11 mg/500 kg) once weekly for 3 to 5 weeks has been recommended.

Injection of iodine-containing counterirritants into and around the medial and middle patellar ligaments has also been advocated. The rationale is also unclear; presumably the sclerosing action of these compounds acts to tighten loose ligaments. Injection of a 2% solution of iodine in oil or of ethanolamine oleate has been advocated. Injection of 1 to 1.25 ml of these compounds into multiple sites in and around the distal aspect of the medial and middle patellar ligaments is recommended.

Surgical Therapy

Medial patellar desmotomy may be necessary, although this should be considered only in horses with severe upward fixation of the patella that are unresponsive to conditioning exercise or medical therapy or for horses that are persistently locked for several days. Because the transected ligament often eventually heals with a fibrous union, the main benefit of surgery may be temporarily to alter the action of the stifle joint such that the horse is capable of conditioning exercise. Development of fragmentation of the apex of the patella (sometimes referred to as patellar chondromalacia) is possible after medial patellar desmotomy (see Fig. 47-4). Middle patellar desmitis is also a potential untoward sequela.

FIBROTIC MYOPATHY

Clinical Characteristics

The abnormal gait of fibrotic myopathy is characterized by a shortened cranial swing phase of the affected limb while walking, with an abrupt catching of the forward swing, a slight caudal swing, and slapping of the hoof onto the ground (see Fig. 49-1). This abnormal gait is present at every walk stride, but it is often much less apparent at the trot and canter. Affected horses may have a palpable thickening behind the stifle in the area of the insertion of the semitendinosus and semimembranosus muscles or farther distally, but this is not a consistent finding. Osseous metaplasia may accompany fibrosis in some horses. Affected muscles may exhibit some degree of atrophy. Some horses have an obvious dimple or depression in the affected muscle. Ultrasonographic evaluation of the caudal thigh musculature can be useful[8] and may detect focal hyperechoic areas of fibrosis when none are palpable or hyperechoic areas with underlying acoustic shadowing associated with mineralization.

Fibrotic myopathy is most often a unilateral problem but can occur bilaterally. Fibrotic myopathy may occur first in one hindlimb, followed by development of a similar abnormal gait in the other hindlimb months to years later. This gait abnormality does not appear to be painful or distressing to the horse, and signs do not increase from anxiety or decreased ambient temperature. Fibrotic myopathy occurs most often in adult horses but has also been described in neonates. Fibrotic myopathy in foals does not appear to be associated with palpable fibrosis of the distal semitendinosus.

Cause

Fibrotic myopathy has been attributed to tearing at the insertion of the semitendinosus muscle during activities that result in extreme tension, such as sliding stops in reining horses and struggling from catching a leg in a halter or tether. Traumatic injuries to the semitendinosus, or inflammatory processes such as abscesses at injection sites, can cause fibrotic myopathy. Less commonly, damage to the semimembranosus or gracilis is a cause.[9] Fibrotic myopathy present at birth has been speculated to be caused by trauma at or soon after birth.[10] Adhesions between the semitendinosus and the semimembranosus or biceps femoris muscles causing restriction of muscle action have been proposed.[11] Careful study of a small series of horses with fibrotic myopathy found that underlying traumatic or degenerative neuropathy causing denervation of the distal semitendinosus can result in fibrotic myopathy.[5] Two horses with minimal fibrosis had bilateral fibrotic myopathy from degenerative neuropathy of unknown cause. One horse had unilateral fibrotic myopathy caused by nerve damage after fracture and scarring of the caudal aspect of the greater trochanter. This horse had characteristic fibrosis of the distal aspect of semitendinosus. Underlying neuropathy is the most likely cause in horses in which fibrotic myopathy develops in one hindlimb and progresses to involve the other hindlimb.

Biomechanical Basis

Functional shortening caused by denervation, muscle scarring, or adhesions to adjacent muscles results in increased muscle tension that does not allow full extension of the stifle, and secondarily the hock, during the cranial swing phase.

Medical Therapy
No medical therapy has been shown to be useful in treating fibrotic myopathy. Exercise does not appear to alleviate or exacerbate the abnormal gait.

Surgical Therapy
Surgical resection of the affected distal semitendinosus muscle (semitendinosus myotomy),[12] affected muscle and tendon of insertion (semitendinosus myotenectomy),[9,11] or semitendinosus tenotomy at the tibial insertion[10] have all resulted in some degree of immediate gait improvement. Myotomy and myotenectomy have been associated with a relatively high degree of postoperative complications compared with simple tenectomy. Semitendinosus tenectomy may result in satisfactory results in many horses, although others may require myotenectomy or myotomy. If portions of muscle are excised, submission of these samples to a histopathologist with expertise in the pathology of neuromuscular disease may aid in determining the cause. At least partial recurrence of fibrotic myopathy after surgery occurs in about one third of horses, although the resultant gait deficit may not interfere with full return to function.[12] If progressive neuropathy is the cause, recurrence of signs after any type of surgery is likely.

STRINGHALT

Clinical Characteristics
The abnormal gait of a horse with stringhalt is an exaggerated upward flexion that occurs at every walk stride (see Fig. 49-1). The affected leg is brought up and in (adducted) underneath the horse, such that the fetlock may contact the ventral abdominal wall. This abnormal gait often lessens considerably during the trot and usually, but not always, disappears at the canter. Affected horses may have difficulty backing and in horses with mild disease the gait abnormality may only be apparent while backing. In such horses a diagnosis of mild shivers should also be considered. Stringhalt may affect one or both hindlimbs. The Australian form of stringhalt is bilateral and occurs in groups of horses on pasture containing dandelions known as flatweed (*Hypochoeris radica*).[3] The toxic principle is as yet unknown. Forelimbs may also be affected in horses with Australian stringhalt. A similar syndrome has been seen in the Pacific Northwest and in Europe. Horses with stringhalt may or may not appear distressed by the gait abnormality. Similar to shivers (see the following discussion), anxiety and cold weather are reported to increase the severity of clinical signs in horses with stringhalt.[4,11]

Cause
True stringhalt, or sporadic, or pasture-associated, stringhalt are most likely caused by underlying neuropathy. The Australian form of stringhalt has been clearly shown to be from underlying neuropathy.[3] All horses with sporadic stringhalt in which I examined muscle or peripheral nerve have had evidence of neuropathy and denervation atrophy, although the cause has not been apparent. Histopathological evaluation of the lateral digital extensor muscle after lateral digital extensor myotenectomy has been particularly useful in detecting evidence of denervation atrophy. Horses with equine motor neuron disease have generalized denervation atrophy and can develop stringhalt that may be bilateral. Denervation atrophy of multiple muscles of the affected hindlimb is seen in horses with stringhalt from EPM. Careful examination of multiple nerves from horses with sporadic[13] or pasture-associated stringhalt[3] has revealed widespread neuropathy. Laryngeal hemiplegia associated with stringhalt is common, indicating a predisposition for abnormalities of long nerves. The condition is similar to peripheral neuropathies in dogs, in which long nerves such as the recurrent laryngeal and sciatic nerves can be preferentially involved, and the cause is often not known. Lesions occur in the left recurrent laryngeal nerve in about 60% of horses of Australian stringhalt.[3] Clinical signs of laryngeal dysfunction may or may not be apparent. Development of stringhalt has been reported in horses after trauma to the dorsal hock and metatarsal regions in the area of the extensor tendons.[14] If proprioceptive deficits and ataxia are detected in a horse with stringhalt, peripheral neuropathy caused by EPM should be considered likely.

Biomechanical Basis
Although underlying neuropathy is well established as a cause of stringhalt, the biomechanical basis for the exaggerated flexion is still somewhat perplexing. Altered proprioception from neuropathy was suggested[6] and this hypothesis is appealing. Neuropathy may lead to paresthesia or to altered input to or activity of muscle spindles. Given the exaggerated flexion of the hindlimbs of most horses after placement of hind limb shipping (traveling) boots, altered sensation leading to limb hyperflexion is plausible. Other proposed causes include tendon adhesions around the tarsocrural joint or altered tendon reflexes after trauma[14] and hyperexcitability of motor neurons.[4]

Medical Therapy
For horses with stringhalt associated with plant toxicity, removal from the pasture may be curative, although recovery may take months to years and may not be complete.[4] Trauma-induced stringhalt may resolve with exercise therapy.[14] Oral phenytoin at 15 to 25 mg/kg once or twice daily may also be effective in some horses. The beneficial actions of phenytoin may include stabilization of neuronal, peripheral nerve, or skeletal muscle membrane electrical activity or decreased anxiety from sedation.[4]

Surgical Therapy
Horses with residual gait abnormality after plant toxicity or traumatic neuropathy and horses with stringhalt caused by degenerative peripheral neuropathy may benefit from lateral digital extensor myotenectomy. Although some affected horses may return to full and high-level athletic performance after surgery, for many this should be considered a salvage type of operation to allow a more normal gait for breeding horses, pasture pets, and horses in which the owner's expectations of performance are limited to light hacking.

SHIVERS (SHIVERERS)

Clinical Characteristics
First described in Draft breeds, this disorder also occurs with some frequency in Warmbloods. Anecdotal evidence suggests a heritable predisposition. Shivers also occurs sporadically in other breeds, including Thoroughbreds, Quarter Horses, Arabians, and Morgans. In my experience with 25 shiverers approximately 80% were stallions or geldings.

The clinical features of shivers, particularly in horses with early or mild disease, may resemble stringhalt or intermittent upward fixation of the patella. Affected horses may be described as stringy, especially by Draft horse owners. Affected horses exhibit an episodic hyperflexion of the hindlimb and often abduct the limb before placing the hoof on the ground (see Fig. 49-1). The affected limb may be held up in a flexed and abducted position for several seconds. Frequently the tail is simultaneously raised (Fig. 49-2). The horse may stretch its head up or forward during these episodes and flickering of the eyelids and ears may occur. The muscles of the affected limb may tremble, but more commonly the tail quivers. As opposed to stringhalt, this abnormal gait is sporadic rather than occur-

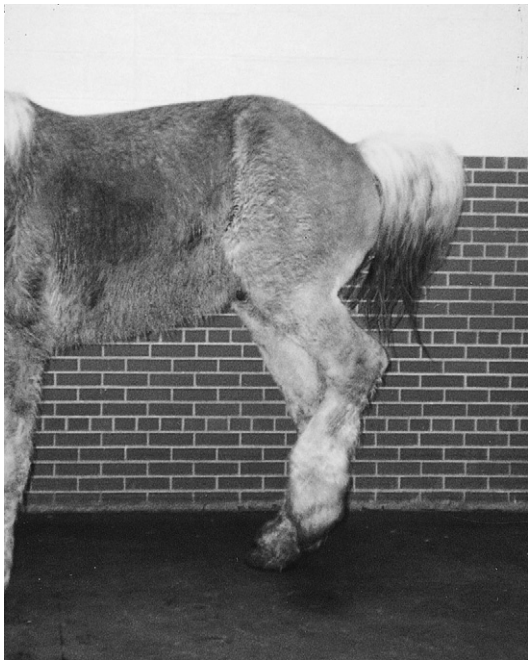

Fig. 49-2 Belgian Draft horse exhibiting the raised tail and hindlimb overflexion characteristic of severe shivers.

ring at every stride and is not apparent at the trot or canter. The abnormal gait is most often seen when backing, turning tight circles, in the first walk stride after standing, and in the last walk stride before halting. The clinical signs may also be seen periodically while the horse is standing still. The abnormal gait is exacerbated by lack of exercise, cold ambient temperature, and increased anxiety in the horse. Neurological examination does not reveal evidence of postural reflex abnormalities or of ataxia.

The farrier may first diagnose shivers, because trimming and shoeing the hindlimbs is often difficult in these horses. They may be reluctant or appear unable to hold the affected limb up, or to stand on three limbs, for any length of time. When asked to lift a hind foot, the horse often hesitates, followed by an exaggerated flexion.

Shivers most often starts as a hitch in one hind limb. Shivers is a progressive disorder that eventually involves both hindlimbs and may progress to involve forelimbs. Severely affected horses develop generalized muscle atrophy and weakness and may exhibit periodic cramping of each leg in succession, with occasional leaning of the body such that the horse appears ready to fall. Onset is often between 2 and 4 years of age, but this varies extremely, as does the rate of progression. The most severe form of progressive shivers occurs in Draft horses. A somewhat milder form occurs in Warmbloods, in which sporadic abnormal hindlimb action is seen, but Warmbloods with shivers rarely exhibit a raised and quivering tail. Clinical signs in such horses are often mistaken for stringhalt or intermittent upward fixation of the patella. Affected horses may appear unaware of the problem and continue to compete in harness or at high levels in dressage or jumping for many years. Prepurchase evaluation of these high-performing shiverers can be particularly vexing, because the expected progression of signs is completely unpredictable.

A recently recognized and poorly understood syndrome known as "stiff-horse syndrome" may resemble shivers. Affected horses have a stiff gait and muscle spasms in the epaxial muscles of the lower back and in the muscles of the hindlimbs. Spasms may be precipitated by voluntary movements or by someone picking up a limb. Increased levels of antibodies to glutamic acid decarboxylase have been reported.[15]

Cause

Although an underlying neuropathy was proposed, careful study of two Draft horses with shivers failed to reveal evidence of peripheral or central nervous system disease. Examination of muscle from these and a small but growing number of shivers horses of various breeds has revealed underlying EPSSM. Contiguous groups of glycogen-depleted skeletal muscle fibers in muscles of the proximal thigh and back have been seen in a small number of horses studied and indicate that this disorder may involve episodic muscle contracture (cramping).[1]

Biomechanical Basis

As with the exaggerated gait of stringhalt and of intermittent upward fixation of the patella, explaining the abnormal gait of shivers is difficult. The possible role of episodic muscle cramp or abnormal sensations within the affected leg is worthy of further study. Horses with an abscess in a hind hoof may show an abnormal flexing of the limb while standing that resembles shivers, and hind hoof abscesses often increase the abnormal action of the affected hindlimb in horses with shivers.

Medical Therapy

Regular exercise may help alleviate some of the signs of shivers and may even slow the progression. To date, the only medical therapy that appears to reverse the signs of shivers has been a change to a high-fat and low starch and sugar diet.[2,7] Whether this therapy acts by decreasing muscle cramping, decreasing anxiety in the horse, a combination of these effects, or some other mechanism is still unknown. In my experience approximately 80% of horses diagnosed with shivers (and many misdiagnosed as stringhalt) have responded to some degree after 4 to 6 months of diet change. These horses include those in which muscle biopsy revealed characteristic changes of EPSSM and horses with no apparent abnormalities in the muscle samples examined. Improvement is often only partial, but most owners have been pleased with results, and dietary therapy may help to slow the progression of the disease.

Surgical Therapy

No surgical therapy is available for shivers.

OTHER MECHANICAL LAMENESSES

The biomechanical basis of mechanical lameness resulting in decreased flexion of the stifle and hock, such as fibrotic myopathy and upward fixation of the patella, are readily understood. These gaits are also characteristic and readily distinguished from other mechanical lamenesses. The mechanical lamenesses resulting in hyperflexion—that is, intermittent upward fixation of the patella, stringhalt, and shivers—however, have clinical similarities that can make them difficult to distinguish. One may also safely say that altered gaits from abnormal muscle or nerve function may not fall into one of the previously defined categories. A horse may appear stringy, locked up, have a goose waddle or stiff, stabbing hindlimb gait, or just appear off in a hindlimb because of mechanical lameness. A slight reduction in hock flexion at the walk may be considered normal for the horse, but this gait may also reflect a mechanical lameness.

In all horses with perplexing hindlimb lameness, the possibility of a form of mechanical lameness should be considered. This is especially true in breeds such as Draft, Quarter Horse, and Warmblood-related breeds that are recognized to have a predilection for underlying myopathy. Because underlying myopathy or neuropathy is likely to be the most common cause of mechanical lameness, electromyography and muscle biopsy should be considered in all horses with mechanical lameness of the pelvic limbs.

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CHAPTER • 50

Estrous Cycle and Performance in Athletic Mares

Joan S. Jorgensen and Richard A. Mansmann

Some fillies and mares periodically suffer from a variety of problems that may be linked to the estrous cycle and can have serious consequences on performance,[1,2] including alterations in behavior that affect training or riding, lameness, back pain, abdominal pain, or an increased propensity for muscle spasms and exertional rhabdomyolysis (ER) (tying up).[3] These conditions have been associated with the estrus (heat) or diestrus phases of the cycle, but in some horses the problems cannot be linked to any stage of the estrous cycle.[2]

The estrous cycle has two phases: estrus and diestrus. Typical behavioral signs of estrous include the adoption of a urination stance and passage of intermittent spurts of urine, tail raising, and allowance of stallion contact.[4-6] Concentrations of estrogen and luteinizing hormone increase, and serum progesterone levels decrease.[6,7] In contrast, mares in diestrus kick at stallions and do not allow contact.[4,6] Serum estrogen and luteinizing hormone concentrations are low, and progesterone dominates the hormonal profile. Progesterone suppresses the manifestation of estrus behavior and is considered to dominate the behavioral effect of estrogen.[4,8-10] Thus most treatments for abnormalities associated with the reproductive cycle include some form of progestin.[3]

PROBLEMS ASSOCIATED WITH THE REPRODUCTIVE CYCLE

A recent survey of the American Association of Equine Practitioners revealed that the most frequently reported clinical sign of a change in performance caused by the estrous cycle was a change in attitude.[11] Other signs included tail swishing, difficulty in training, squealing, horsing, urinating excessively, kicking, and a general decrease in performance. Anecdotal reports from Thoroughbred racehorse practitioners suggested that some young nervous fillies were predisposed to ER. Furthermore, anxious mares may be at an increased risk for orthopedic problems because they are often worked for longer to reduce the anxiety and improve performance.

Difficulty in training or riding, lameness, back pain, or ER are most commonly associated with musculoskeletal or neurological disorders. Similarly, abdominal pain is usually linked to gastrointestinal disease. Thus these problems should be ruled out before incriminating the reproductive system. Mares with colic-like discomfort, hindlimb lameness, or back pain when the rider is in the saddle may have painful ovaries, especially in the peri-ovulatory period.[2,12] Indeed, pressure applied

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Difficulty in training or riding, lameness, back pain, or ER are most commonly associated with musculoskeletal or neurological disorders. Similarly, abdominal pain is usually linked to gastrointestinal disease. Thus these problems should be ruled out before incriminating the reproductive system. Mares with colic-like discomfort, hindlimb lameness, or back pain when the rider is in the saddle may have painful ovaries, especially in the peri-ovulatory period.[2,12] Indeed, pressure applied

to the ovaries per rectum can elicit a pain response.[13] Palpation of a large ovulatory follicle or a follicle that has recently ovulated, other genital tract characteristics of estrus, and concomitant behavioral signs of estrus may support this diagnosis. The discomfort should subside shortly after ovulation, but it may recur with future cycles.[12]

Peri-ovulatory discomfort is thought to be caused by a combination of extremely enlarged follicles that affect neighboring tissues and the release of follicular fluid during ovulation, some of which may leak into the peritoneal cavity and cause localized inflammation. These factors may result in localized pain that lasts approximately 24 to 48 hours.

The estrous cycle is also characterized by changes in the hormonal profile. Specific balances between gonadotropins and sex steroids are necessary for smooth transitions between phases of the reproductive cycle. We propose that any alteration in the ratios between luteinizing hormone and follicle-stimulating hormone; androgens, estrogens, and progestins; or both results in erratic behavior that leads to poor performance. Ongoing studies of behavior and performance in athletic mares are intended to test this hypothesis.

TREATMENT

Treatment of performance problems related to the estrous cycle is directed toward stabilizing the hormonal profile by medical, physiological, or surgical means.

Medical

The most common form of medical therapy is some form of progestin: oral, injectable, or pellet implant (Table 50-1). Progestin therapy has long been the standard for preventing ovulation. However, treatment for performance problems in mares does not necessarily require suppression of estrus. Altrenogest (Regu-Mate) is given orally, once daily (0.044 mg/kg), and is the only drug that is approved for use in mares. Administering all other forms of progestins is considered extra-label usage. Care must be taken in handling this drug because it can be absorbed through the skin and may potentially affect the menstrual cycle of women. Pelleted implants for use in cattle containing either 100 mg progesterone and 10 mg estradiol benzoate (Synovex C), or double those dosages (Synovex S), have been used successfully for performance problems for up to 3 months. The implants are placed subcutaneously in the side of the neck beneath the mane. A potential complication is development of implant site reactions or abscesses.[3]

Other steroidal medications have been used successfully but much less frequently. Glucocorticoids (dexamethasone) interfere with ovulation in many species, including mares.[14] Dexamethasone can be administered orally or by injection. However, dexamethasone must be given at least once daily and has been associated with serious complications, such as an increased susceptibility to infection and laminitis.[15] Dexamethasone (15 mg IM bid) has suppressed estrus signs in ovariectomized pony mares.[16,17] Mares that have a predisposition to ER have been treated successfully with anabolic steroids such as testosterone and boldenon undecylenate (Equipoise).

Physiological

Progesterone levels obtained during pregnancy are generally sufficient to suppress sexual behavior in mares,[18] thus breeding a problem mare may temporarily resolve performance problems. Most mares can train and continue to perform until the eighth month of pregnancy. However, the undesirable behavior may be passed on to female offspring, and several risks are associated with breeding, pregnancy, parturition, and foal management.

Table • **50-1**

Drugs Used to Treat Hormone-Related Performance Problems in Mares

Form of Drug	Progestins	Glucocorticoids
Oral	Altrenogest (Regu-Mate) 0.044 mg/kg PO q24h Hoechst-Roussel Agri-Vet Co.	Dexamethasone (Azium) 10 mg/500 kg PO bid Schering-Plough Animal Health Corp.
	Megestrol acetate (Ovaban) 65-85 mg/500 kg PO q24h Schering-Plough Animal Health Corp.	
Injectable	Medroxyprogesterone acetate (Depo-Provera) 200-250 mg/500 kg IM q8-14d Pharmacia	Dexamethasone solution 15 mg/500 kg IM bid Vedco, Inc.
	Progesterone impregnated microspheres (Lutamate Plus) 1.25 g progesterone + 100 mg estradiol-17β/500 kg IM q12-14d Unavailable in United States	
Pellet implant	Synovex C 6-8 pellets (100 mg progesterone, 10 mg estradiol benzoate) Synovex S 3-4 pellets (200 mg progesterone, 20 mg estradiol benzoate) Implant subcutaneously q3mo Fort Dodge Laboratories	

Surgical

Ovariectomy is irreversible and considered a therapy of last resort. The surgery may be successful in treating hormone-related performance problems, but many reports indicate that mares continue to exhibit undesirable traits after surgery.[3,16,17,19,20] These findings suggest that other systems may play an important and as yet uncharacterized role in erratic performance problems. For example, the adrenal gland is an important source of sex steroid production. Further investigation into performance problems related to the reproductive cycle and the associated hormone producing organs is necessary to fully understand this problem.

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CHAPTER • 51

Diagnosis and Management of Pelvic Fractures in the Thoroughbred Racehorse

Robert C. Pilsworth

Injuries to the pelvis of the horse have in the past been considered uncommon.[1-3] They have also previously been described as invariably resulting from external trauma.[4] Only recently have pelvic fractures been recognized as a common cause of lameness in the racing Thoroughbred (TB). Stover et al.[5] confirmed a high incidence of stress fractures in the pelvis of American racing TBs examined at post mortem. Similarly in a clinical study performed in Newmarket, England, pelvic fractures were found to be common in the young racing TB.[6,7] We are gradually realizing that pelvic fractures in the TB are most often the end stage of a cycle of bone fatigue and injury, commonly called stress-related bone injury. This has focused attention on early detection, before the development of a full-blown displaced fracture. This section outlines steps to aid in early detection and management strategies subsequent to diagnosis.

PELVIC ANATOMY

The pelvis comprises the symmetrical halves and the sacrum in the midline. The left and right halves of the pelvis are joined in the ventral midline at the pubic symphysis. Although technically a joint, this becomes mineralized with age and is in most horses a firm bony union. A second joint occurs on each side between the pelvis and the head of the femur forming the coxofemoral joint. The third joint is that between the ventral surface of the pelvis and the sacrum, the sacroiliac joint. Only the coxofemoral joint has any significant degree of movement. The sacroiliac joint does have a synovial membrane, but in many horses it is largely immobile. In most horses the sacroiliac joint is spanned by dense bands of fibrous connective tissue and, despite having a cartilage surface, little or no movement takes place in the normal sacroiliac joint. Each half of the pelvis comprises three bones, which meet at the acetabulum, the ilium cranially, the ischium caudally, and the pubis medially. The ilium has a large wing of bone extending from the tuber sacrale in the midline to the tuber coxae at the lateral extremity. The blade of the ilium narrows to form the shaft, which extends back to form the cranial segment of the coxofemoral joint. Caudal to the coxofemoral joint, and forming its caudal margin, is the ischium. Joining the two acetabula, forming the floor of the pelvis with its contralateral counterpart, is the pubis. The bones of the pelvis form a large fulcrum on which most of the gluteal muscle mass originates, to exert considerable propulsive forces on the hindlimbs. Although the pelvis can fracture anywhere as a result of external trauma or a fall, as is common with many other stress injuries, the forces involved in locomotion create predilection sites for stress fractures. These sites are associated with the concentration of forces involved in load bearing at speed and the biomechanics, innate structure, and form of the bone.

DIAGNOSTIC TECHNIQUES

Clinical Examination

A thorough working knowledge of the anatomy of the equine pelvis is essential for clinical examination to be useful. Because of the large muscle mass over the horse's hindquarters only the bony extremities of the pelvis can be palpated. However, it is often possible to gain information about horses with pelvic injuries by studying the position of these bony landmarks. For instance, the normal position and angle of the tuber coxae in the racehorse is often disturbed in horses with fracture or sacroiliac joint instability. The position of the tubera coxae can be assessed by viewing the horse from behind, with an assistant placing fingertips on the craniodorsal extremity of the tubera coxae (Fig. 51-1). It is important that the horse stands completely level, with both hind feet together, for this test to be meaningful. Some horses, however, show asymmetry viewed in this way that is not linked to lameness. Similarly, careful palpation of the tubera sacrale can give information about the possible involvement of the sacral wing of the ilium or disruption of the sacroiliac joint.

Ventral displacement of one tuber sacrale is commonly encountered in ilial wing fractures, where the overlap of the fracture fragments seems to allow the tuber sacrale to move ventrally. Often a pain response is associated with palpation of a horse with tuber sacrale displacement, and sometimes movement of the bone itself may be felt if the fracture is complete. Fracture of a tuber coxae is often produced by external trauma, usually after a fall, but can also occur as a stress-related athletic injury. This usually results in a cranioventral displacement of the fracture fragment, because of the distrac-

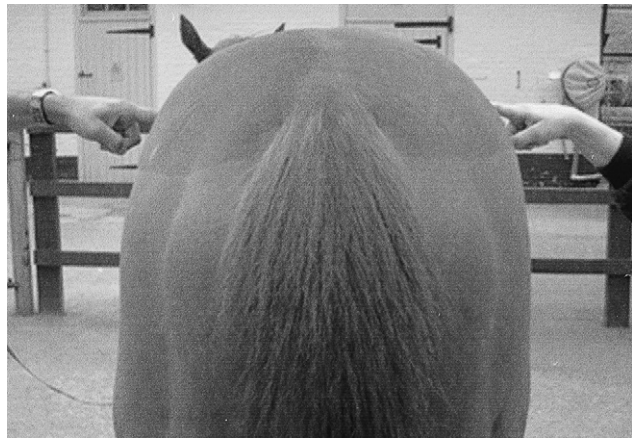

Fig. 51-1 Assessment of the position of the bony pelvis by fingertip levels placed on the dorsal extremity of the tubera coxae on either side.

tive forces of associated musculature. The tuber coxae in these horses can often be felt situated in the sub-lumbar fossa, and the remnant fracture bed can be palpated at the original site. Fractures of the ischium can sometimes be felt by manual palpation, although the extensive muscle spasm and protective boarding which is associated with these fractures often precludes this examination. Finally, muscle tone in the tail and anus should be evaluated, because fractures involving the sacrum can involve neural elements that supply these structures and cause flaccid paralysis of the rectum and anal ring and of the vulva in the filly, the cauda equinum syndrome (Fig. 51-2). Bilateral ilial wing fractures can produce the same neurological appearance associated with severe nerve root damage consequent on movement of the pelvis in relation to the sacrum.

Rectal examination allows direct manual assessment of the integrity of the pubis, internal surface of the wing of the ilium, and ventral border of the sacroiliac joint. Sometimes an obvious, sharp discontinuity in the bone surface can be felt, particularly in horses with fracture of the pubis. Gentle rocking of the horse by an assistant, while the clinician maintains digital contact with the bone surface of the pelvis per rectum, sometimes allows an appreciation of relative movement of adjacent bones, or more commonly, a sensation of crepitus. In horses with fracture of the ilial wing, a soft asymmetrical swelling can often be felt at the fracture site, representing a sub-fascial hematoma. The more serious hemorrhage that occurs when the iliac artery is severed by the sharp dorsal edge of the fractured ilium and that can be fatal often cannot be detected per rectum. If not immediately fatal, this free blood often percolates ventrally to cause massive swelling and edema of the thigh musculature.

Some horses have several stress fractures identified scintigraphically, only one of which may have initially collapsed, leading to overt lameness. Progressive collapse of the pelvis may then occur during the convalescent period, as incomplete fractures become complete and displaced because of bone resorption and weakening of the fracture site. The degree of lameness seen in these horses varies enormously, depending on the type and extent of the fracture, and is considered separately in the after discussions of each class of fracture.

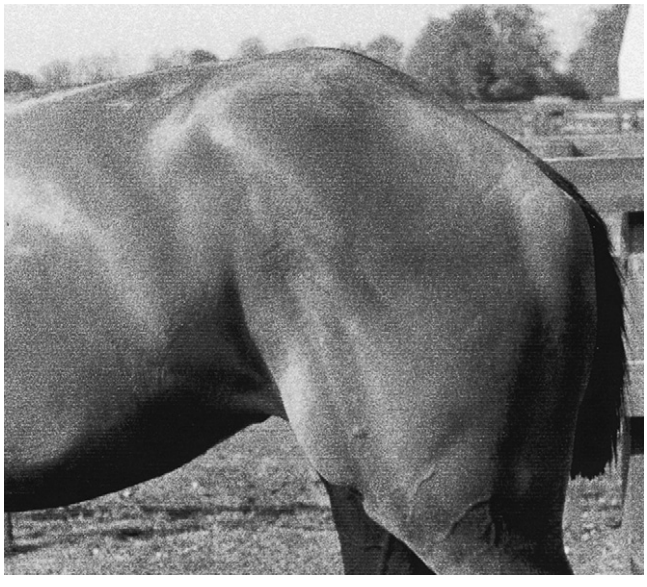

Fig. 51-2 A horse with a sacral fracture. Note the abrupt angle change just behind the tubera sacrale, where the sacrum and coccyx have moved ventrally. The tail has complete flaccid paralysis. This filly had urine and fecal retention.

Diagnostic Ultrasonography

Ultrasonography is useful for diagnosing pelvic fractures and has proved especially useful in demonstrating fractures to the ilial wing, ilial shaft, and tuber coxae. Ultrasonography is quick, easy, and within the capability of clinicians with a suitable ultrasound machine. Ultrasonography may eliminate the requirement for a horse to travel to a referral center for diagnosis, and the risk associated with radiography under general anesthesia can be avoided. Ultrasonography has obvious limitations. For example, adequate imaging of the sacral wing, sacroiliac joint, and the femoral head is not possible. Fractures with minimal displacement or poorly developed callus are also difficult to image, as are incomplete fractures involving the ventral surface of the ilium. For this reason ultrasonography should not be regarded as a stand-alone imaging modality for identifying a fracture of the pelvis but should be used with a thorough clinical examination and, if available, scintigraphy. In many horses the exact site and extent of the fracture can be determined, which allows improved prognostic and management advice to be given. The healing process can be monitored by serial examinations, allowing the management program to be tailored to the individual horse. A longitudinal- or sector-array ultrasound transducer can be used, provided it has a deep enough penetration to see the bone surface (i.e., a 3.5- or 5-MHz transducer). The muscle mass lying above the bone structures acts as a natural "standoff," bringing the bone surfaces into the focal zone of the ultrasound beam. A separate standoff may be required to evaluate the position of the tubera sacrale and to detect any displacement. In thin-coated horses no clipping is required, provided adequate saturation of the coat is achieved by degreasing with a detergent solution (chlorhexidine), or by soaking in surgical spirit, followed by application of a coupling gel before scanning. Horses with thicker hair coats must be clipped to obtain images of adequate quality. Images can be difficult to produce in horses with large amounts of subcutaneous fat because of the attenuating properties of this tissue. Numerous blood vessels running through the musculature can create acoustic shadows, which may be confused with a discontinuity of the bone surface. Identifying the bony landmarks such as the tubera sacrale, tubera coxae, cranial and caudal margins of the ilial wing, and greater trochanters of the femur allow anatomical orientation. A dry pelvic specimen is also useful in orientation. Both sides of the pelvis should be evaluated, because the normal side can be used for comparison. However, keep in mind that bilateral ilial wing stress fractures occur and both sides may be abnormal. For recording and reference purposes the area of the ilial wing imaged is referred to as line A, B, or C and the distance from the tuber sacrale is measured. Scans aligned longitudinally along the ilial shaft are referred to as line D. This simple system is useful, especially in follow-up examinations. A systematic method for recording ultrasonographic findings has been published elsewhere.[8]

Radiography

Radiography of the pelvis is easily carried out in a foal when it can be performed under heavy sedation, for instance, with a combination of detomidine and butorphanol, with the foal allowed to stand again shortly after the procedure. Standard projections include a ventrodorsal view with the foal lying on its back and a lateral view with the foal lying on its side on top of the cassette. A grid is mandatory to reduce scattered radiation produced by the large mass of soft tissue covering the pelvis. Radiography in the adult horses is considerably more difficult. General anesthesia is required for thorough radiographic examination of the pelvis for numerous reasons, including the safety of the horse, personnel, and radiographic equipment. Administering general anesthesia to a horse suspected of having a pelvic fracture is contraindicated because

of the possibility of the horse displacing the fracture on recovery. Fracture displacement can lead to fatal hemorrhage or worsening of clinical signs, which is particularly true in horses with an incomplete fracture of the wing and shaft of the ilium. It is also difficult to get high-quality radiographs of the pelvic bones without extensive prior starvation of the horse to allow emptying of the gastrointestinal tract. Small, incomplete, pelvic stress fractures are extremely difficult to see radiographically. Standing radiography in the horse has been described.[9] However, in our hands this has proved difficult, presents real risks of damage to the equipment, and is a significant radiation hazard to attending personnel. The standing examination is also limited to examination of the acetabulum and ilial shaft, and images of the ilial wing are obscured by the sub-lumbar and overlying dorsal lumbar musculature. Image quality obtained in this way is also often poor. Although radiography used to be the imaging technique of choice for assessing horses with pelvic fractures, in our hospital it has been replaced by ultrasonographic and scintigraphic evaluation.

Scintigraphy

Scintigraphy is considered the most sensitive method of assessing acute bone damage in the horse.[3,10-12] A full review of veterinary nuclear medical techniques applicable to the horse has recently been published (see Chapter 19).[13] Special techniques for examining the pelvis have also been described, including an oblique projection of the ilium, which is useful in diagnosing incomplete stress fracture in this site.[14] Sacroiliac joint luxation has also been described as a scintigraphic diagnosis, the appearance of which is extremely similar to the ilial wing fractures observed by others.[3] To examine the equine pelvis fully using a gamma camera, dorsal projections of the entire pelvis are mandatory. The lateral projection of the pelvis tends to be difficult to interpret because of the apparent focal increased radiopharmaceutical uptake (IRU) observed at the tuber coxae and the coxofemoral joint in a normal horse. The massive muscle mass interposed between the slender bones of the ilial shaft and the gamma camera result in extensive shielding and poor images. Radioactive urine in the underlying bladder contributes to extensive background radiation, further decreasing image quality. Movement is a key factor in limiting image quality, but motion correction software can be used to minimize this complication. However, the lateral oblique projection described by Hornof et al.[14] usually gives better images of the ilial wing and shaft. A caudal oblique projection is also employed by some to give better images of the coxofemoral joint and acetabulum. A method for scintigraphic examination of the pelvis using a handheld probe has also been described.[15] If a pelvic fracture is strongly suspected and initial scintigraphic examination is performed within 3 to 5 days of injury and is negative, a follow-up examination should be performed at least 10 days from the time of injury. Early fractures, even those with associated displacement, may not be evident scintigraphically for several days after injury.

FRACTURES OF THE TUBER COXAE

Fractures of the tuber coxae are often described as a knocked-down hip. They are relatively common and fairly straightforward to diagnose by clinical examination of the bony landmarks of the pelvis. They may occur after direct trauma or a fall but also as an athletic injury in training or racing.

Clinical Signs

On the first day of injury, the horse is moderately to severely lame, even at the walk. Often the horse shows intense muscle spasm and guarding of the affected hindquarter. The horse may sweat and scrape the ground, which can mimic the signs of colic. The affected tuber coxae is often displaced cranioventrally and is palpable in the region of the sub-lumbar fossa. The parent bed from which the fragment originated is often palpable in the original site of the tuber coxae. Lameness rapidly resolves, and after only 24 to 48 hours many horses are walking sound, but they will still trot lame. Hemorrhaging often occurs, which may present as a subcutaneous hematoma. Occasionally the sharp spiculated end of the ilium wears through the overlying skin, leading to development of a compound fracture, which can be extremely difficult to treat, because the skin overlying the sharp fragment will not heal and infection becomes a problem.

Scintigraphic Examination

Scintigraphic examination is probably not indicated in most horses, because the clinical diagnosis is straightforward. If performed, scintigraphic examination reveals significant focal IRU associated with the displaced tuber coxae and also a fairly obvious distortion of the normal anatomy of the pelvis on the dorsal image. In the Editors' experience, photopenic regions associated with ventral displacement of the fracture fragments may be present.

Ultrasonographic Examination

Directly after injury, horses with tuber coxae fracture have clinical signs similar to those with other fractures of the pelvis, and an ultrasonographic examination helps to confirm the diagnosis and to define better the limits of the fracture. The fracture is easily seen as a disruption to the normally smooth and continuous contour of the ilial wing as it approaches the tuber coxae. A standoff may be required for better imaging of the bone immediately beneath the skin. It is important to perform a thorough examination of all the bony structures to eliminate fractures in other regions of the pelvis.

FRACTURES OF THE ILIAL WING

Fractures of the ilial wing appear to be among the most commonly encountered type of athletic stress fractures in the skeletally immature TB racehorse.[16]

Clinical Signs

A horse with a complete ilial wing fracture often shows lameness, but the initial lameness varies from lame at the walk to grade 1 to 4 out of 5 at the trot. Lameness often resolves rapidly within 24 to 48 hours, and the horse then has a slight gait abnormality, walking with the back hunched up, but with no overt lameness. A horse with an ilial wing fracture often plaits with the hindlimbs at the trot and has a shortened stride. Although a complete fracture may be present on only one side, evidence of a sub-clinical stress fracture in the same site on the contralateral limb is common, and this may contribute to the peculiar gait shown by the horse. Obviously, pain from both sacroiliac joints considerably affects the freedom of movement of the hindlimbs. A horse with bilateral lesions may appear to have signs of exertional rhabdomyolysis, but plasma creatine kinase levels are often normal or only slightly raised (500 to 1500 IU/L).

Incomplete fractures may occur on one or both sides, and this can often show as poor propulsion and a poor hindlimb action in the absence of clinical lameness. In a horse with a complete fracture, ventral displacement of the ipsilateral tuber sacrale is often apparent and can be detected by careful digital examination of the midline of the spine and both tubera sacrale. Firm pressure in the previous site of the tuber sacrale often evinces a marked pain response, with associated

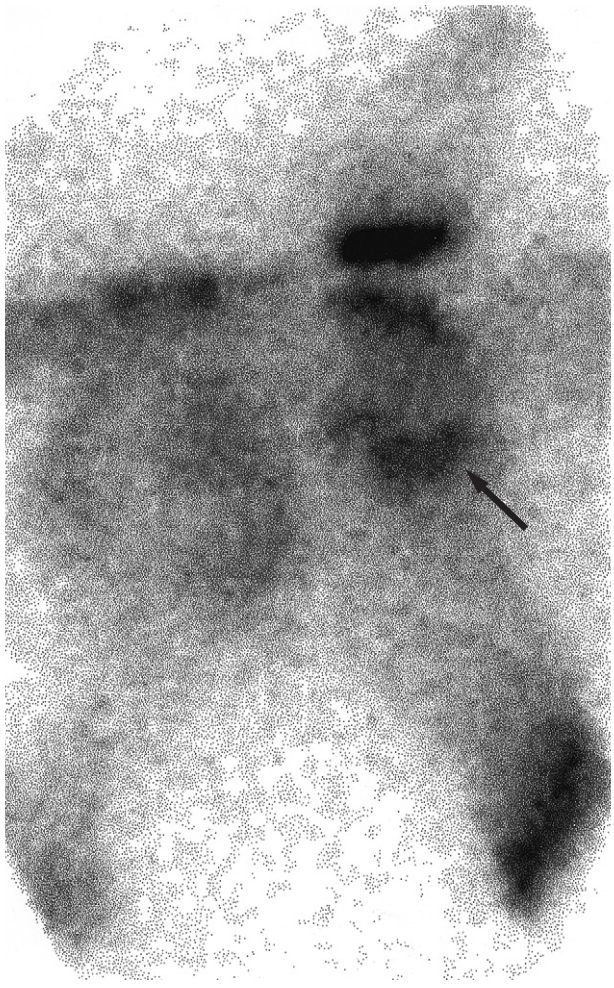

Fig. 51-3 Dorsal lateral oblique delayed (bone) phase scintigraphic image over the left side of the pelvis of a horse with a stress fracture of the ilial wing. Cranial is to the right. Focal increased radiopharmaceutical uptake *(arrow)* appears about a fourth of the way between the tuber sacrale and tuber coxae, the most common predilection site for stress fractures of the ilial wing.

muscle spasm over the sacroiliac joint region. Palpating the tip of the tuber sacrale in its ventrally displaced position is sometimes possible. Often fairly profound muscle wastage is associated with this injury in the first 2 weeks, and this contributes significantly to the apparent asymmetry of the horse when viewed from behind. Bilateral complete fractures lead to profound stiffness, unwillingness to walk, and boarding of the muscles of the pelvis in response to digital manipulation. They can lead to collapse and inability to rise, with significant neurological deficits in severe injuries.

Scintigraphic Examination

Scintigraphy usually reveals marked focal IRU associated with the ilial wing, usually 10 to 15 cm from the midline (Fig. 51-3). Increased radiopharmaceutical uptake may be bilateral or unilateral. A clear fracture line and the displacement of the ilial wing are sometimes clearly visible. The lateral oblique projection[14] is particularly useful in determining whether the fracture extends to the cranial and caudal cortices of the ilial wing.

Ultrasonographic Examination

The normal ultrasonographic appearance of the ilial wing is a smooth and regular concave hyperechoic line, extending between the tuber sacrale and the ipsilateral tuber coxae. Acoustic shadowing and refraction artifacts can be created by the many blood vessels within the musculature and must not be confused with disruption to the bone contour. Ilial wing fractures appear as a disruption to the ilial wing contour, with changes ranging from a clear fracture gap to small irregular echogenic areas on the dorsal surface, representing prodromal periosteal new bone formation. The fractures normally run sagitally and involve the bone dorsal to or in close proximity to the sacroiliac joint. The bone abnormalities often appear more severe toward the caudal margin of the fracture. Sometimes hemorrhage at the site of fracture appears as an anechogenic defect in the normal granular texture of skeletal muscle (Fig. 51-4).

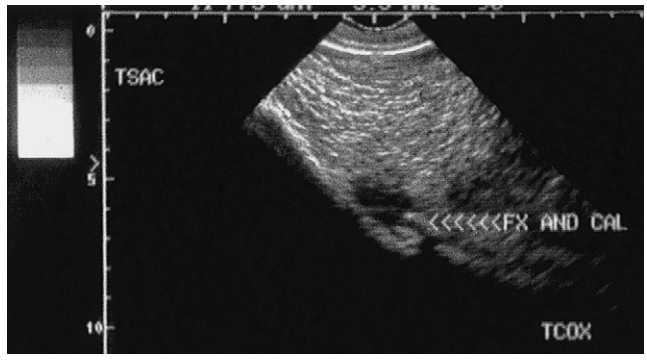

Fig. 51-4 Ultrasonographic image of the left ilial wing obtained using a 3.5-MHz sector transducer. There is a stress fracture of the ilial wing *(FX AND CAL)*. The fracture appears as a discontinuity in the pelvis surface. Adjacent to the fracture is irregular echogenic material (callus), and just dorsal to the fracture is a large anechogenic defect in the muscle (hemorrhage).

FRACTURES OF THE ILIAL SHAFT

The ilial shaft is a common site for fracture after a fall or as a spontaneous athletically induced injury.

Clinical Signs

Fracture of the ilial shaft is extremely painful, producing non–weight-bearing lameness, and is often associated with shock from extreme pain and rapid blood loss. The two tubera coxae often show marked asymmetry when viewed from behind, although encouraging the horse to bear weight equally to allow this examination to be carried out can be difficult. Sometimes with incomplete or minimally displaced fractures, no displacement of the tuber coxae is apparent initially, but in these horses the pelvis often collapses early in convalescence, at which time asymmetry becomes obvious. Collapse of the pelvis can often make the fracture less painful for the horse for reasons that are unclear. Rectal examination is often useful, and gentle rocking of the horse while the arm is inserted in the rectum often allows clear crepitus to be felt with the fingertips on the ilial shaft of the affected side. A hematoma next to the fractured ilium can also be easily palpated in many horses. Not uncommonly horses damage the iliac arteries with the sharp edges of the comminuted fracture fragments, and this can result in severe and sometimes fatal hemorrhage.

Horses usually do not resent palpation and flexion of the distal limb, although forced abduction of the limb including the hock and stifle may elicit a painful response. Palpation of the hindquarters usually results in intense muscle spasm and guarding on the affected side. Bizarre consequences of the

severe lameness and change in functional anatomy after some of these fractures include permanent upward fixation of the patella, contracture of the hamstring group of muscles and spastic hyperextension of the tarsus, and are not uncommon. These complications invariably result in a grave prognosis.

Scintigraphic Examination

Many horses are too lame to allow transport to an equine clinic for scintigraphic examination, and a combination of clinical signs and ultrasonographic findings can establish the diagnosis. Probe point-counting techniques usually show a fairly massive and widespread IRU on the affected side of the pelvis, and seeing clearly from the plot exactly where the fracture has taken place is difficult. Gamma camera studies give much more detail than do probe point-counting techniques, and a caudal oblique projection is often extremely useful in illustrating the extent and nature of the fracture. Because pelvic anatomy is directly visible on a gamma camera scan, the displacement of the fracture fragments and of the ilial wing is often easily appreciated.

Ultrasonographic Examination

The ilial shaft is examined by aligning the ultrasound beam in a craniocaudal direction and following the ilial shaft from the flat surface of the ilial wing toward the greater trochanter of the femur. With ultrasonography a displaced ilial shaft fracture is easily detected as a discontinuity to the bone surface. Passive movement of the limb may show independent movement of the fracture margins in real time scanning. Hypoechogenic areas may be present around the fracture line, which represent hemorrhage into the musculature. As with other fractures, if displacement is minimal, an ultrasonographic examination may fail to detect any pathological condition.

FRACTURES OF THE PUBIS AND ISCHIUM

Discrete fractures of the ischium are relatively uncommon. The horse usually has unilateral lameness with obvious swelling over the caudal thigh. A fall, falling backward, or rearing over are often noted in the history. Often palpable crepitus and a marked pain response on firm digital pressure to the site are apparent. The tail may be held to one side, because of muscle spasm associated with the painful lesion. Often focal sweating occurs on a patch of skin on the back of the thigh, presumably resulting from nerve damage. Fractures of the pubis are only encountered with other multiple pelvic fractures, or after a fall, and are uncommon.

Scintigraphic Examination

Scintigraphy shows focal IRU associated with the ischium and sometimes clear displacement of a segment of bone. Isolated fractures of the pubis can be difficult to see because of the mass of bone and muscle interposed between the fracture site and the gamma camera. For this reason a handheld probe, used per rectum, can give useful information. A bladder artifact produced by delayed excretion of urine may give the appearance of an IRU in this site.

Ultrasonographic Examination

The pubis is the least rewarding of the pelvic bones to examine by ultrasonography. A rectal probe is required, and only marked changes are detectable. The ultrasonographic examination is limited by the small degree of probe movement that is possible within the rectum, coupled with the irregular normal contour of the bone at this site.

Part of the ischium is also examined internally using a rectal transducer. This approach may be the only way to detect any ultrasonographic changes associated with an acetabular fracture. The caudal-most portion of the ischium and the ischiatic tuberosity can be examined percutaneously. Normally a smooth concave contour extends caudally from the greater trochanter of the femur. Ultrasonographic changes similar to those described in other sites where bone damage has taken place can be seen in this area. Comparing the contralateral ischium to the fractured one is useful.

FRACTURES INVOLVING THE ACETABULUM

Horses with fractures of the ilial shaft and ischium that involve the acetabulum have the poorest prognosis of all for return to athletic function as a racehorse and normally develop osteoarthritis of the coxofemoral joint, which results in permanent and progressive lameness. Acetabular fractures commonly result from trauma, often subsequent to slipping over or being cast. This is one of the more frequent causes of lameness in a horse found not bearing weight on a hindlimb in the stable or paddock, with no obvious inciting cause.

Clinical Examination

The horse is extremely lame and is often unwilling to move. The horse has extremely short protraction of the hindlimbs at the walk and often hops rather than attempting to bear weight. Pain occurs on abduction of the limb, and often firm palpation of the muscle mass around the coxofemoral joint causes muscle spasm and pain. Crepitus may be felt with displaced fractures if the hand is applied to the greater trochanter of the femur while the horse is walked, although commonly these fractures are incomplete and crepitus is not apparent. In horses younger than 2 years of age separation of the femoral head can occur from trauma and produces similar clinical signs to a comminuted acetabular fracture.

Scintigraphic Examination

Scintigraphic examination reveals focal IRU associated with the coxofemoral joint, greater trochanter of the femur, and ilial shaft on the affected side. The IRU is particularly visible on a caudal oblique view in which both coxofemoral joints can usually be seen using a large field of view gamma camera. Osteoarthritis of the coxofemoral joint contributes significantly to IRU in horses with a chronic fracture. These fractures are not associated with intense focal IRU initially and early examination may give false-negative results (Fig. 51-5, *A*). At least a week is required before scintigraphic abnormalities are detectable and often this time period is necessary anyway to allow humane transport of the horse. If the clinician is doubtful, the scan should be repeated 14 days later, when bone uptake will be maximal[17] (Fig. 51-5, *B*). Separation of the femoral head, which occurs infrequently in yearlings usually after a fall, produces similar scintigraphic changes, with significant IRU associated with the coxofemoral joint.

Ultrasonographic Examination

Because of the great depth of the acetabulum and the many changing bone contours, the acetabulum is probably the most difficult area of the pelvis to image by ultrasonography. Subtle pathological conditions may well be missed. The greater trochanter of the femur provides a good landmark by which to orient the image. The normal acetabular region is represented as a smooth and regular hyperechoic line of the acetabular rim. The greater trochanter of the femur is close to the skin surface as a hyperechoic line extending down and toward the acetabular rim. In some horses a hypoechogenic region represents the joint space. In foals and immature animals a hyperechoic convex line extends from the greater trochanter and represents the femoral head.

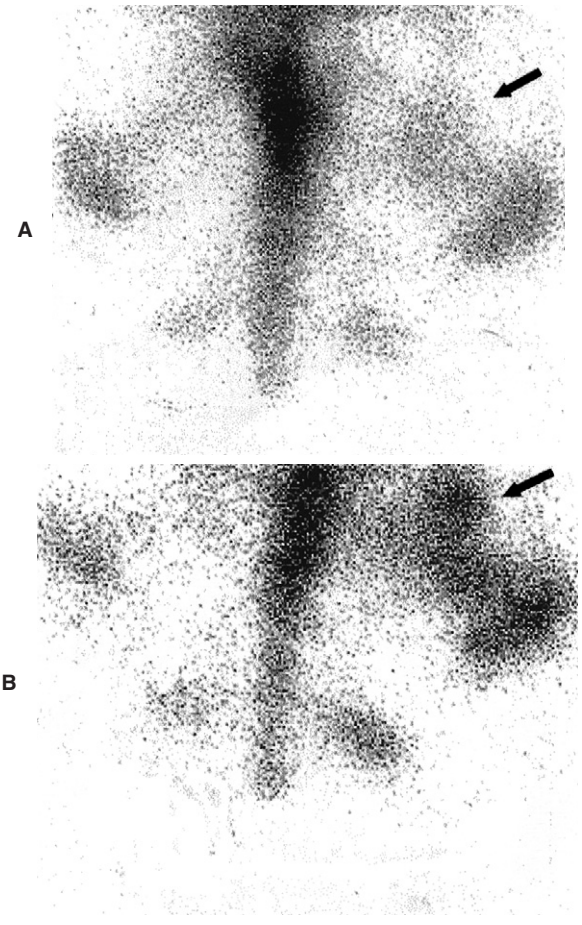

Fig. 51-5 Caudal-dorsal oblique delayed (bone) phase scintigraphic image of the pelvis of a horse with a fracture of the right acetabulum. **A,** An image acquired about 5 days after injury. **B,** An image acquired 3 weeks later. There is poor radiopharmaceutical uptake of this fracture *(arrow)* in the acute stage compared with that at the second examination.

A fracture can appear as a discontinuity or irregular roughening of the acetabular rim and will be particularly obvious if greater degrees of callus are present.

PRINCIPLES OF TREATMENT OF HORSES WITH PELVIC FRACTURES

Surgical repair of pelvic fractures is not currently a realistic option in the adult horse. The period of box rest required for bony union ranges from 2 to 3 months, judged by monitoring fracture healing with ultrasonography. The end result in terms of the functional anatomy of the pelvis depends on the degree of displacement in the initial insult and the extent of subsequent distraction of the fracture fragments by subsequent muscle contracture. For instance, tuber coxae fracture fragments heal by becoming adherent to the cranial wing of the pelvis through fibrous union. Despite the fact that the position of the tuber coxae often changes by several centimeters and the tuber coxae comes to rest in the sub-lumbar fossa, many horses make a full return to athletic function and race with success. Fractures of the sacral wing of the ilium are often non-displaced and can make an extremely good, smooth bone union after healing. Often an abnormal angle is apparent on the ilial surface, because of distraction of the fragments by muscle contraction before healing. Although the abnormal angulation of the pelvic blade cannot help the stability of the sacroiliac joint immediately beneath the fracture, many affected horses do go on to train and race successfully. In a review of 20 horses with ilial fractures, 15 horses made a full recovery to advanced race training and 11 of these raced successfully.[6] Involvement of the ilial shaft considerably worsens the prognosis for racing, though many heal adequately to allow retirement to stud. Some general principles of the care and management of horses apply equally to the different types of pelvic fractures and can be outlined as follows:

- Pain should be controlled during the initial phase by administration of non-steroidal anti-inflammatory drugs (NSAIDs), such as phenylbutazone, until the horse appears comfortable walking around the stable.
- The risk that normal recumbency in the box will result in displacement of an already fractured pelvis is always present. Unfortunately the proximity of the sharp edge of the fractured ilium to the iliac arteries makes this a potentially life-threatening complication. Therefore a horse suspected of major pelvic bone injury should be tied up by the head during the initial convalescent period. When doing so, it is important that the horse has enough rope to move around comfortably, without a sufficient length to encourage it to attempt to lie down. A "break string" should always be between the rope and the head collar, so that if a horse tries to lie down, it can do so without risk. Tying the horse in a position where it can see outside events is best, so that the horse is not alarmed by things happening behind it, to avoid the horse being startled and the risk of displacement of a lesion.

  Horses should remain tied up for no more than 1 month. Many horses cope with being tied up without problems, but while they are tied up, horses should be fed from the floor several times a day and be held by an attendant while they eat. This encourages drainage of bronchial secretions down the trachea and may help to prevent pleuritis and pleuropneumonia, which can develop with prolonged periods of being tied up.[18] During the period that NSAIDs are being administered by mouth, any rise in rectal temperature will be masked. For this reason it is vital to take regular (every 48 hours) blood samples to assess changes in white blood cell count, plasma viscosity, and fibrinogen level, which may signal an early onset of pneumonia or pleuritis. Subsequent to NSAID therapy, rectal temperature should be monitored twice daily. When a horse is untied after a month, the fracture fragments possibly still can displace, leading to fatal hemorrhage. However, displacement is rare, and the clinician must balance the potential risk to the horse against the humanitarian aspects of keeping a horse tied up continuously for more than a month. Although I feel strongly about tying up horses with pelvic fractures, others feel differently, and many horses with pelvic fractures have been managed successfully without being tied up.
- Once untied, a horse requires a minimum of another 2 months of stable rest, combined with daily walking exercise to encourage normal blood flow to the feet, before turnout is safe. Pasture rest for another 2 months is then advisable for horses with a severe, displaced fracture, even before contemplating covering. Pregnancy and parturition can severely test fracture healing, therefore a prolonged convalescent period is required. Often questions arise about the suitability of mares undergoing pregnancy and parturition after pelvic fractures. In my experience the main concerns are lameness, the ability to get up and down, and

maintaining weight, rather than callus formation and the physical inability to deliver a foal.

- If a pelvic fracture has led to an extreme unilateral lameness, the contralateral foot is at real risk of developing laminitis. Many horses will not allow the contralateral limb to be raised long enough to fit a frog support. It is therefore vital that the horse is bedded on deep litter (bedding), so that the bedding can pack up under the foot to give some support. A frog support should be placed as soon as possible and the shoes should be removed. Once the horse is able to bear weight on the affected limb, short periods of walking exercise help prevent vascular stasis and lymphangitis that often accompany weight bearing on one limb for prolonged periods of time. Walking may also help prevent supporting limb laminitis, but only if the horse is not profoundly lame in the affected limb. Support bandaging of the contralateral limb in horses with severe unilateral lameness is probably worthwhile and certainly helps to prevent the occurrence of lymphedema.
- Administration of acetylpromazine in small doses (25 to 50 mg PO bid or 0.02 to 0.04 mg/kg injected IM bid, judged on degree of tranquilization by the first dose) to a lame horse helps for three reasons: preventing laminitis; relieving some anxiety and allowing the horse to rest, which is important for recovery; and preventing upward fixation of the patella, to which the horses are prone, presumably by causing mild muscle relaxation.
- Most horses with incomplete or minimally displaced complete ilial wing stress fractures become sound rapidly. I do not normally keep these horses tied up, but nonetheless a small but real risk of fatal hemorrhage exists in these horses, even when they are apparently sound. Displacement is rare, but from the outset the risk should be explained to and understood by all involved in the management decisions.
- Adult horses with fractures involving the acetabulum have the poorest prognosis for return to athletic function, although horses often become sound enough to be retired to pasture. Compromise of the diameter of the pelvic canal diameter can be a complication, especially if the ilial shaft is involved and the fracture has healed with abundant callus. Even for experienced clinicians, being categorical about the ability of a mare with a narrowed pelvic canal to breed can be difficult. Often a trial mating to an inexpensive stallion is the most pragmatic management choice, if the clinician has doubts.

  Careful thought should be given to each individual horse to make sure that the horse's future is worthwhile, in terms of quality of life for the horse and the economic realities of the situation, before subjecting a horse to a prolonged and by necessity, painful convalescent period.

  Prognosis in foals with acetabulur fractures is considerably better than in adult horses. Treating a foal with an acetabular fracture is definitely worthwhile, because at this age the pelvis appears capable of healing with little chance of developing osteoarthritis in the coxofemoral joint. Foals are treated with 12 weeks of box rest initially and progressive turnout in a nursery paddock.

  Separation of the femoral head in a young horse carries a hopeless prognosis and, if confirmed, should lead to immediate euthanasia.
- As with any horse that has had a prolonged period of box rest, the clinician must remember that although the pelvic fracture may have healed, the rest of the horse's skeleton will have decalcified significantly because of disuse. It is therefore vital that horses are treated as "naive skeletal specimens" when training is resumed. The exercise program should increase gradually, beginning with walking only for the first 2 weeks and building up gradually through walking and trotting to slow speed cantering, eventually re-attaining race speed training. Clinicians should remember that the skeleton takes approximately 1 month at each gait to adapt to the loads placed on it. Bringing a horse back from injury often takes as long as the period of box rest itself, if further orthopedic problems are to be avoided.

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CHAPTER • 52

Pelvic Injuries in the Non-Racehorse

Sue J. Dyson

ANATOMICAL CONSIDERATIONS

Detailed anatomy of the ilium, ischium, pubis, sacrum, coxofemoral joint, sacroiliac joint, nerves, and major vessels is described elsewhere (see Chapter 53, page 501).

CLINICAL SIGNS

History

Pelvic injury in a mature athletic horse is a comparatively unusual cause of lameness, except as the result of trauma from a fall, rearing and falling over backward, becoming cast in the stable, or sustaining an injury during transport. When the horse has no history of trauma, diagnosis can be difficult, and excluding all possible sources of pain in the distal limb is frequently necessary before focusing on the pelvic region.

Horses with significant pathological conditions of the sacroiliac joints frequently have a history of progressive reduction in performance, difficulty in engaging the hindlimbs, and poor hindlimb impulsion, especially when ridden.

Clinical Examination

Clinical assessment of individual structures of the pelvic region by visual examination and palpation is not easy, especially in Warmblood and draft breeds, because of the large muscle mass of the hindquarters. Frequently, only the tubera coxae and tubera sacrale can be palpated. Large muscle mass may prohibit palpation of the greater trochanter of the femur. Atrophy of the hindquarter musculature is non-specific and can reflect disuse because of pain arising anywhere in the limb, although atrophy of the muscles around the tail head often reflects injury to the tuber ischium, or local nerve damage. Asymmetry of the height of the tubera sacrale is a common finding in horses in full work, free from lameness, although it may be seen along with poor performance or alterations in hindlimb gait. Apparent asymmetry may actually reflect differences in size of the dorsal sacroiliac ligaments. Alteration in muscle mass in the proximity of the tubera sacrale superficially can give a false impression of asymmetry of the tubera sacrale. Asymmetry of the tubera coxae may reflect a previous injury unassociated with ongoing pain. Poor muscle development in the lumbar region and over the hindquarters may make the tubera sacrale and the summits of the dorsal spinous processes of the lumbar vertebrae appear abnormally prominent. This should alert the clinician to the possibility of thoracolumbar or pelvic pain; however, this finding is non-specific and may reflect the horse's work history.

The pelvic region should be appraised visually and palpated systematically, and although preliminary assessment is usually best performed in the stable, for accurate evaluation of symmetry of the musculature and bony elements of the pelvic region the horse should be standing completely squarely behind on a firm, level surface with the horse looking straight ahead. In a horse with severe lameness this may not be possible, because the horse may be unwilling to load the lame limb fully. Careful differentiation should be made between muscular and bony asymmetry. Significant muscular atrophy can make accurate assessment of symmetry of the pelvic bones difficult. To evaluate accurately the levelness of the tubera coxae, two assistants each must place an index finger on the craniodorsal aspect of each tuber coxae and extend the finger horizontally, or the tubera coxae should be marked using tape. Elevation of the tail may be necessary to identify muscle atrophy around the tail head, which may be seen along with nerve damage, or injuries of the ipsilateral tuber ischium.

Assessment of symmetry when the horse is unwilling to bear weight evenly on both hindlimbs is not easy, but particular attention should be paid to the way in which the limb is positioned. An abnormally straight limb may reflect luxation of the coxofemoral joint and secondary upward fixation of the patella. The greater trochanter of the femur of the lame limb may appear higher than that of the contralateral limb.

The muscles of the lumbar and pelvic regions should be assessed carefully to identify any area of abnormal muscle tension, pain on palpation, or unusual firmness. Firm stroking of the muscles first with a finger and then with a blunt-ended object (e.g., artery forceps) is useful to determine whether muscle spasm or muscle fasciculation are induced. Palpation of the caudal muscles of the crus is also important, because abnormal pain or tension can reflect primary muscle injury or an injury of the ipsilateral tuber ischium.

Firm pressure should be applied to the bony prominences to see if pain or an abnormal reaction, such as sinking on the hindlimbs when pressure is applied to the tubera sacrale, can be induced. Both tubera coxae should be grasped simultaneously, and the horse rocked from side to side to determine whether crepitus can be detected by palpation or auscultation, bearing in mind that the absence of crepitus does not preclude a fracture.

Careful, systematic examination of the pelvic canal region per rectum is also indicated to assess the aorta and iliac arteries, psoas musculature, the caudal aspect of the ilial shaft, and the pubis and ischium.

Pelvic injuries should also be considered when a clinician examines a recumbent horse that has fallen over a fence. Palpation of the pelvic region is even more difficult in these circumstances. Even with a severe fracture, palpating any abnormality may be impossible. The clinician should bear in mind that in the acute phase, local reflexes such as the patella reflex and the withdrawal reflex may be suppressed, which does not necessarily reflect a spinal cord injury. Major fractures may be associated with rupture of one or more large vessels, resulting in potentially fatal internal hemorrhage. Thus it is important to assess the recumbent horse as a whole, monitoring pulse rate, color of mucous membranes, and capillary refill time. Increased pulse rate and progressive pallor of the mucous membranes is a good indicator of major vessel rupture, such as laceration of the iliac artery after fracture of the ilial shaft.

Manipulation of the limb may be resented if pain is associated with the coxofemoral joint, but generally the responses

to flexion of the limb, protraction, retraction, or abduction are rather non-specific. A horse with pain associated with the sacroiliac joints or a coxofemoral joint may be reluctant to stand on one limb, with the other limb raised, and may behave awkwardly in anticipation of discomfort. However, the reaction is non-specific, and one must bear in mind that some horses present difficulties in picking up the hindlimbs in the absence of any sign of lameness or poor performance. Difficulties in picking up hindlimbs may be caused by reluctance to accentuate weight bearing on the lamest limb, reluctance to flex the lame limb, or may be psychological. If the horse is a shiverer, unilaterally or bilaterally, the response to hindlimb flexion can be difficult to assess.

The degree and character of lameness depends on the underlying cause. Fractures or luxation of the coxofemoral joint results in acute onset, severe lameness. Lameness associated with other lesions in the pelvic region may vary in degree, not only among horses with similar lesions, but also within and between examination periods. Pain from the coxofemoral joint frequently results in the horse moving on three tracks, with the non-lame limb being placed between the two forelimbs. On the lunge the horse may be inclined to break to canter, rather than move with adequate hindlimb impulsion, but this is not specific for pelvic pain and is typical of many horses with hindlimb lameness. Pain associated with the coxofemoral joint or the greater trochanter of the femur sometimes results in the horse carrying the lame limb in canter. Lesions associated with the sacroiliac joints frequently result in the horse crossing over each hindlimb at the trot (i.e., plaiting), but this is not pathognomonic and some horses move with a base wide hindlimb gait.[1] The horse may move with reduced hindlimb impulsion rather than overt lameness. Although acute fractures of the tuber ischium invariably cause lameness, chronic injuries may result in loss of performance (e.g., jumping to the right) rather than overt lameness.

The response to flexion tests is non-specific. The clinician should bear in mind that increased weight bearing on one limb, caused by flexing the contralateral limb, may accentuate lameness in the weight-bearing limb. Turning the horse in small circles, inducing rotational forces on the coxofemoral joint, may accentuate lameness associated with the coxofemoral joint.

Ridden exercise is invaluable in horses with a history of poor performance, reduced hindlimb impulsion, or low-grade lameness, because frequently the lameness is accentuated. Some horses with sacroiliac pain or pain associated with new bone on the caudal aspects of the wings of the sacrum (see pages 495 and 497) show extreme reluctance to go forward freely. However, care must be taken to differentiate these horses from those with bilateral hindlimb lameness, thoracolumbar pain, or recurrent low-grade exertional rhabdomyolysis and those performing poorly because of the rider (see Chapter 100), previous poor schooling, or a combination of boredom and an unwilling temperament.

Analgesic Techniques

In horses with chronic lameness, reduced hindlimb impulsion, or poor performance, excluding the distal limb as a source of pain by performing perineural analgesia of the fibular and tibial nerves and intra-articular analgesia of the three compartments of the stifle joint may first be necessary. If the response is negative, intra-articular analgesia of the coxofemoral joint may be indicated. This is relatively straightforward to perform if the horse is not well muscled and the greater trochanter of the femur is readily palpable. However, in the majority of heavily muscled, mature competition horses, needle placement must be guided by ultrasonography. Even if the needle is accurately positioned, retrieval of synovial fluid may be difficult. Extra-articular deposition of local anesthetic solution may result in transient paralysis of the obturator nerve and instability of the limb. The technique is described in Chapter 10 (page 120).

Clinicians have anecdotally described positive responses to infiltration of local anesthetic solution in the region of the sacroiliac joints, but I do not believe that intra-articular injection can be achieved reliably. However, generalized infiltration of local anesthetic solution around the sacroiliac joint region in part may alleviate pain associated with the joint and periarticular structures and has been useful in some horses. The techniques are described on page 506. Infiltration of local anesthetic solution around the sacroiliac joint regions resulted in significant improvement in all 34 horses in which it was performed. These horses had clinical signs suggestive of sacroiliac joint pain, and the majority also had abnormal radiopharmaceutical uptake in the sacroiliac joint region.[1]

Serum Muscle Enzyme Concentration

Measuring serum concentration of creatine kinase (CK) and aspartate aminotransferase (AST) is invaluable for diagnosing horses with acute and chronic rhabdomyolysis (tying up). In horses with chronic tying up, comparing resting levels with concentrations reached after maximum exercise may be necessary. Peak levels of CK are likely to occur 3 hours after exercise. If a horse has had a tying-up episode within the last 4 to 5 days, AST levels almost invariably will be raised. Some horses with chronic recurrent problems have constantly elevated levels of CK and AST. The degree of elevation of muscle enzyme concentrations may show a poor correlation with the severity of clinical signs.

DIAGNOSTIC IMAGING

Radiography

Radiographic examination of the pelvic region of a horse, anesthetized and positioned in dorsal recumbency or in the standing position, is described in depth elsewhere.[2] Since the advent of nuclear scintigraphy and diagnostic ultrasonography the indications for radiographic examination have decreased. If the source of pain has been localized to the coxofemoral joint, radiographic examination is indicated to determine the nature of the pathological condition and hence prognosis. High-quality radiographs can only be obtained with the horse positioned in dorsal recumbency under general anesthesia. Evaluation of the sacroiliac joints can be difficult because of the superimposition of abdominal viscera. Identification of new bone formation on the caudal aspect of the joint and irregular joint space width are poor prognostic indicators. Nuclear scintigraphic examination gives accurate information about bone turnover, but anatomical detail is less well defined. Therefore radiography of a horse with a suspected acetabular fracture may be indicated at least 6 weeks after the onset of lameness to determine whether a suspected fracture involves the coxofemoral joint, which merits an extremely guarded prognosis for return to full athletic function in a mature horse.

Radiographs of the coxofemoral joint obtained in the standing position may be satisfactory for confirmation of luxation or major fractures of the joint.

Ultrasonography

Diagnostic ultrasonography of the pelvic region can be performed transcutaneously or per rectum. The choice of transducer frequency depends on the structures to be imaged. Transcutaneous evaluation of the bony elements of the pelvis and the deep musculature requires a 5- or 3.5-MHz transducer, depending on the size of the horse, whereas evaluation of the dorsal sacroiliac ligaments, sub-lumbar musculature, ventral aspect of the lumbar and sacral vertebrae, and aorta and iliac arteries is better performed using a 5- or 7.5-MHz

transducer. Evaluation of nerve roots per rectum may be best achieved using a 10-MHz transducer.

Diagnostic ultrasonography is useful in evaluating fractures (see Chapter 51, page 485), assessing muscles (see Chapter 84) and the sacroiliac ligaments (see page 497), determining blood vessel patency (see page 497), assessing the lumbar vertebrae and articulations (see Chapter 54), and evaluating nerve roots.

Nuclear Scintigraphy

Nuclear scintigraphic evaluation of the pelvic region is useful for identifying fractures, stress reactions in bone, increased bone modeling associated with osteoarthritis and other bony lesions, and evidence of rhabdomyolysis; for evaluating blood flow in the aorta, iliac, and femoral arteries; and for assessing the sacroiliac joints. Sensitivity of the technique in part depends on the angle of the gamma camera to the area of interest and the degree of overlying muscle mass. It is important to recognize that some lesions may be bilateral; therefore recognition of the normal scintigraphic appearance of the region in horses of different ages and different disciplines is important. The clinician should bear in mind that superficial bony structures such as the tubera coxae and tubera sacrale always appear "hotter" than deeper structures. Unilateral muscle atrophy may also confound interpretation.

Radioactive urine in the bladder may complicate interpretation; therefore the judicious use of furosemide to induce urination before examination is essential.

Complete evaluation of the pelvic region requires dorsal views of the sacroiliac joints, oblique views of the ilial wings, caudodorsal and caudal views of the tubera ischii, and lateral views of the coxofemoral joints. Care must be taken in interpreting the appearance of the sacroiliac joints, because age-related changes occur in normal horses. Swaying movement of the horse during image acquisition can result in images that mimic abnormalities and using motion correction software is invaluable.

DIFFERENTIAL DIAGNOSIS

Fractures

Clinical features, diagnosis, and treatment of fractures of the pelvis in the young Thoroughbred (TB) racehorse have been dealt with in depth (see Chapter 51), and this section focuses on differences in mature athletic horses. The incidence of stress or fatigue fractures of the pelvic region in the mature horse is low, except in horses that race over fences, which have a substantial incidence of ilial stress fractures. The majority of fractures result from external trauma. The clinical features are similar to those in the young racehorse (see Chapter 51).

Tuber Ischium

Fractures of the tuber ischium sometimes occur in event horses that fall when jumping up onto a bank, may also occur in any horses as a result of a fall on the flat, and also have been recognized in horses from other disciplines with no known history of trauma. Lameness is usually acute in onset and severe. Mild localized swelling is easily overlooked unless the tuber ischium is suspected as a site of injury. The ipsilateral semimembranosus and semitendinosus muscles are usually sore to palpation. Atrophy of the muscles around the tail head often develops within 7 to 10 days. Usually crepitus is not palpable. In horses with chronic lameness, pain on palpation may not be evident, although the tubera ischii may appear asymmetrical, and the lameness may be only mild or moderate.

Diagnosis of a fracture of the tuber ischium can be confirmed using nuclear scintigraphy. Dorsal oblique and caudal views are useful. Usually increased radiopharmaceutical uptake and an abnormal pattern of uptake are apparent.[3,4] In some horses determining whether the fracture is complete and whether it has become significantly displaced may be possible. Discontinuity of the bone outline may also be confirmed using diagnostic ultrasonography. Radiographic examination can be performed, but it is most easily and safely done with the horse under general anesthesia.

Treatment by restriction to box rest usually results in a satisfactory outcome, although occasionally sequestration of the fracture fragment occurs, necessitating surgical removal.

Occasionally a horse shows no obvious lameness, but it has reduced performance and a tendency to jump drifting consistently to one side because of pushing off unevenly with each hindlimb. This symptom is associated with reduced muscle development over one of the tubera and increased radiopharmaceutical uptake in the tuber ischium (see Fig. 12-4). The outline of the tuber ischium appears irregular in an ultrasonographic image.

Greater Trochanter of the Femur

Fracture of the greater trochanter of the femur is an unusual injury causing severe lameness. A tendency exists for the fracture fragment to be displaced cranially because of the pull of the attachments of the deep and middle gluteal muscles.

Usually no localizing clinical signs are apparent, unless the horse is poorly muscled and the greater trochanter is readily palpable. The diagnosis is based on nuclear scintigraphic examination with or without diagnostic ultrasonography. The prognosis for return to athletic function with conservative treatment is guarded.

Sacrum

Fractures of the sacrum may be complete or incomplete and result in bilateral hindlimb lameness. If the fracture is complete, the contour of the hindquarters when viewed from the side changes, so that the rump has abnormal angulation (Fig. 51-2). Associated neurological signs include flaccid paralysis of the tail, reduced sensation around the tail head, urine dribbling in a mare, and loss of anal tone. The onset of neurological signs may be delayed for several weeks after the primary fracture. The fracture may be palpable per rectum. The fracture may be confirmed radiographically and using diagnostic ultrasonography per rectum. The prognosis for return to athletic function is guarded.

Equine Rhabdomyolysis (Tying Up)

Equine rhabdomyolysis is considered in depth elsewhere (see Chapter 84). This section focuses on aspects of diagnosis and differential diagnosis of lameness associated with the hindquarters and pelvic injuries in sports horses used for different disciplines and is restricted to recurrent exertional rhabdomyolysis (RER).

The spectrum of clinical signs associated with RER is enormous. A horse may have mild bilateral hindlimb stiffness or loss of freedom of action that may deteriorate slightly with work, without the horse becoming unduly distressed, but sometimes requires a prolonged recovery period after work. Such horses may have no palpable firmness of the hindquarter musculature or pain induced by firm palpation and a normal pattern of sweating. The horse may be able to compete but performs below expectations. These clinical signs are not unique to exertional rhabdomyolysis and may be seen in horses with severe thoracolumbar discomfort associated with impinging dorsal spinous processes, pain associated with the sacroiliac joints, or bilateral hindlimb lameness. Some horses show progressive agitation during ridden exercise and awkwardness to ride, without recognition of a gait abnormality. Careful clinical evaluation usually reveals a progressive shortening of stride.

Alternatively a horse may start to jump poorly during, for example, the cross country phase of a Three Day Event and pull up with a unilateral hindlimb lameness, with no

localizing clinical signs, that resolves within 24 hours. In contrast a different horse may have recurrent acute onset, severe episodes—often provoked by competition, for example, during the speed and endurance phase of a Three Day Event—that may be so bad as to result in recumbency.

The incidence of RER depends to some extent on the discipline in which the horse is involved, occurring most commonly in racehorses (see Chapters 84, 107, and 108), endurance horses (see Chapter 119), and event horses (see Chapter 118). RER is unusual in show jumpers and dressage horses, but occasionally occurs in competitive TB-type ponies. In all these horses the results of muscle biopsies usually indicate no evidence of abnormal glycogen metabolism as in polysaccharide storage myopathy,[3] which occurs more commonly in Draft and Draft crossbreeds and Quarter Horses. In the TB evidence indicates that RER may be a heritable condition, as an autosomal recessive trait with variable expression.[5] A familial trait has also been observed in some part-TB event horses.[3] However, RER in many event and endurance horses does not become apparent until the horse is middle aged, or even older. The condition is manifest more frequently in mares than in stallions or geldings.

Diagnosis is based on measuring raised serum muscle enzymes (CK and AST) after exercise (see page 723). The degree of muscle enzyme elevation frequently does not correlate with the severity of clinical signs. Interpretation is not always straightforward because asymptomatic endurance horses frequently have extremely high levels of CK and AST during and after a ride. Horses with low-grade RER, which occurs daily, often have constantly raised levels of CK and AST, even if rested.

Nuclear scintigraphy can be useful in horses with low-grade hindlimb stiffness and poor performance to validate a diagnosis of suspected RER,[6] to evaluate blood flow to the hindlimb musculature,[7] and to exclude any other concurrent musculoskeletal lesions. In association with RER in some, but not all horses, abnormal uptake of ^{99m}Tc–methylene diphosphonate occurs in the affected muscles in the bone phase of the scan, appearing usually as diagonal streaking (Fig. 52-1), but in horses with more severe, long-standing RER, affected muscles may show large areas of intense radiopharmaceutical uptake. Lesions are not restricted to the gluteal muscle mass, but they may also affect quadriceps, biceps femoris, semitendinosus, semimembranosus, and longissimus dorsi muscles. Lesions may be symmetrical or asymmetrical. However, normal radiopharmaceutical uptake does not exclude RER.

Muscle biopsy is used to determine whether any evidence exists of polysaccharide storage myopathy. Feeding horses a high-fat and low-carbohydrate diet may reduce the frequency and severity of attacks after a period of 3 to 6 months.[8]

Measuring fractional excretion of electrolytes (sodium, potassium, calcium phosphorus, and chloride) can be useful to identify those horses that seem unable to absorb or to utilize normally specific dietary electrolytes and that might benefit from dietary supplementation.[9,10] For accurate interpretation of results it is important that the horse is consuming its normal diet and has recovered from any recent acute attack of exertional rhabdomyolysis when blood and urine samples are collected. A mid-stream urine sample should be collected, preferably freely voided rather than by catheterization. Results are only valid if serum creatinine is within the normal range and the fractional excretion of creatinine is normal. Results should be compared with the normal ranges for a horse on a similar diet, because results for horses eating an oat- and hay-based diet are different for those on a cube-based diet or a diet high in alfalfa. Fractional excretion ratios for potassium tend to be high if the horse is allowed access to pasture. Low values for fractional excretion ratios for sodium or calcium indicate that the diet should be supplemented with sodium chloride or either calcium carbonate or calcium gluconate, respectively. Appropriate dietary supplementation may help to prevent further attacks.

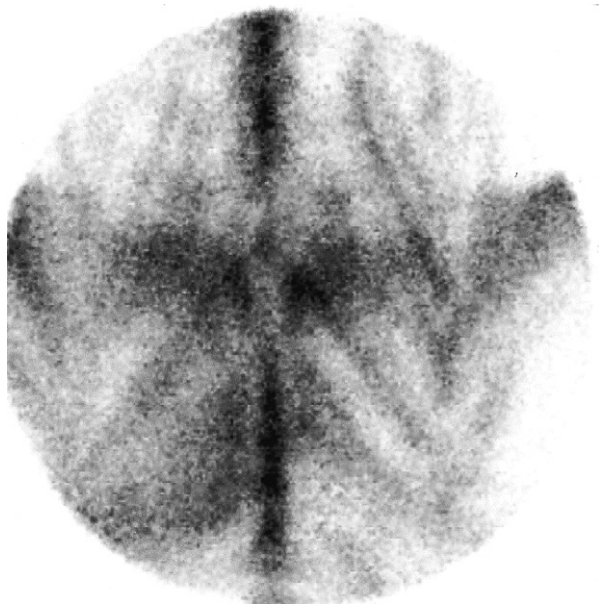

Fig. 52-1 Dorsal bone-phase scintigraphic view of the pelvic region of 12-year-old Thoroughbred mare, a former winner of the European Three Day Event Championships. The mare had shown slight but significant loss of performance but no overt lameness. She had never shown any signs typical of classical tying up. Abnormal radiopharmaceutical uptake appears in the gluteal muscles. This is compatible with recurrent exertional rhabdomyolysis and was asociated with persistently elevated levels of creatine kinase and aspartate aminotransferase.

Management practices may also help to prevent attacks in some horses. These include daily work, with a long, slow warm-up period, turnout as much as possible, and avoiding undue stress, especially in highly strung, nervous individuals. However, some horses, especially those with daily low-grade clinical signs that are not stress provoked, prove intractable to successful management.

Muscle Injury

Muscle soreness is frequently unassociated with any recognizable histopathological changes within the muscle and is often secondary to some other cause of lameness because of the altered way in which the horse is moving. Muscle soreness can often be induced by overuse of an undertrained muscle and can result in localized soreness and stiffness for several days. Focal intense muscle spasm and pain can cause sudden onset reduction in performance and, if primary, are usually alleviated by manipulation to relieve the muscle spasm, producing rapid amelioration of clinical signs.

Focal muscle soreness associated with localized swelling may be due to intramuscular hemorrhage, muscle fiber tearing, or exertional rhabdomyolysis. Diagnosis of the cause may be determined by measuring serum muscle enzyme concentrations and by ultrasonographic evaluation (Fig. 52-2). Hemorrhage results in an area of diffuse increase in echogenicity within the muscle. This should be differentiated from hyperechoic regions that result from chronic muscle fibrosis. Serum muscle enzyme concentrations are usually not elevated in those horses with hemorrhage, muscle fiber tearing, or fibrosis.

Damage to deep muscles of the hindquarters is difficult to identify because localizing clinical signs are frequently not

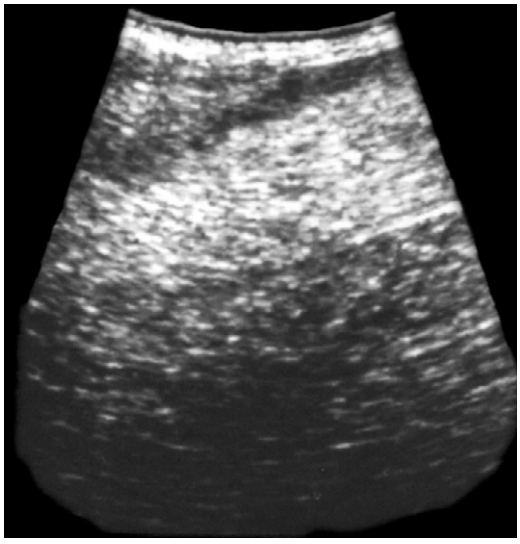

Fig. 52-2 Ultrasonographic image of the semimembranosus muscle of 6-year-old Thoroughbred with acute-onset, severe left hindlimb lameness. The horse had palpable enlargement of the muscle and localized soreness. The increased echogenicity reflects hemorrhage.

apparent to alert the clinician to the possible site of damage. Thermographic evaluation can be useful to help identify superficial muscle injury.

Some horses with reduced performance have pain on palpation of the psoas muscles per rectum. Pain may be primary or secondary to pathological conditions of the lumbosacral or sacroiliac regions and is an indication for nuclear scintigraphic evaluation of the pelvic region and ultrasonographic examination of the lumbosacral vertebrae.

Sacroiliac Joint Injury

See Chapter 53 for further discussion of sacroiliac joint injury. Detailed anatomy of the sacroiliac joints is discussed elsewhere (see page 501).

The diagnosis of sacroiliac joint disease has tended historically to be a dustbin diagnosis, frequently made by exclusion when the clinical signs could not be explained by any other condition. The high incidence of pathological degenerative lesions found in the sacroiliac joints of TB racehorses[11] and in mixed-breed horses[12] indicates that degenerative disease of the sacroiliac joint is likely to be a significant clinical problem, but definitive diagnosis remains difficult. In a recent study of 74 horses with sacroiliac joint pain, affected horses were older than the normal clinic population, and there was a high proportion of Warmblood horses.[1] Horses used for dressage or show jumping predominated. Affected horses were also of great body weight and height.

Pathological changes of the sacroiliac joint include lipping, cortical buttressing, and osteophyte formation, together with enlargement of the joint surfaces. These signs are thought to be a response to chronic instability of the joints, although no evidence of ligamentous laxity has been identified.[11,13] Erosion of the articular cartilage may also occur, but joint ankylosis has not been documented. A positive association has been recognized between the severity of the impingement of thoracolumbar dorsal spinous processes and lumbar transverse processes and the severity of sacroiliac lesions and also between the severity of articular process degeneration and the degree of pathological conditions of the sacroiliac region.

A separate entity of enthesophyte formation at the site of attachment of the sacroiliac ligaments has been described unassociated with pathological conditions of the sacroiliac joints.[14] The clinical significance of this finding has not been well defined.

Subluxation of the sacroiliac joint is comparatively rare[14,15] and results from acute, traumatic disruption of the dorsal sacroiliac ligament and the sacroiliac joint capsule. Subluxation should be differentiated from an acute ilial wing fracture resulting in depression of the ipsilateral tuber sacrale (see page 486).

The prominence of the tubera sacrale varies between horses and in part reflects the conformation of the back and hindlimbs. However, poor development of the epaxial muscles in the lumbar region caused by the horse not using its back and hindlimbs properly may make the tubera sacrale appear more prominent. Poor muscle development may be a reflection of pain or of the horse's previous training: if the horse has never been asked to engage the hindlimbs properly and work through its back, these muscles will not be well developed.

Many apparently clinically normal horses have some degree of asymmetry of the height and or shape of each tuber sacrale and the overlying soft tissues. Careful clinical appraisal of a small proportion of these horses may reveal subtle hindlimb gait abnormalities and mild discomfort induced by pressure applied over the tubera sacrale or by picking up one hindlimb and swaying the horse on the other. These findings are more likely to be detected in horses that are not fully fit and are often ameliorated when the horse is fitter and has greater muscle support. They may reflect mild instability of the sacroiliac joints but are not necessarily synonymous with degenerative change.

Osteoarthritis of the sacroiliac joints may be present despite symmetry of the tubera sacrale, is usually a bilateral condition, and is rarely associated with unilateral hindlimb lameness.[1] Osteoarthritis often manifests as reduced performance, failure properly to engage the hindlimbs, and back stiffness. Signs are often greatly accentuated when the horse is ridden. In horses with mild clinical signs the loss of hindlimb power and lack of suppleness through the horse's back may be much easier for the rider to feel than for an observer to appreciate. Gait irregularities may be most apparent as the horse changes direction through a tight circle. Specific movements such as half pass or sequence flying changes may be difficult. The horse may become progressively more unwilling to work under saddle.

A comprehensive clinical evaluation is essential to preclude other conditions that may present similarly, such as bilateral distal hock joint pain. Precluding the hindlimbs as a potential source of pain by performing perineural analgesia of the tibial and fibular nerves and intra-articular anesthesia of the femorotibial and femoropatellar joints of one hindlimb may be necessary to see if lameness becomes apparent in the contralateral limb.

Using a 9- to 15-cm needle inserted on the midline at the lumbosacral space and directed approximately 20° caudally, local anesthetic solution (20 ml per side) can be infiltrated in the direction of the sacroiliac joints (see page 506). This may improve clinical signs in some horses and resulted in significant change in all 34 horses with suspected sacroiliac pain in which local analgesia was performed.[1] However, in my experience a negative response does not preclude significant sacroiliac joint disease.

Nuclear scintigraphic examination may facilitate diagnosis by helping to preclude the presence of other significant causes of poor hindlimb action and by giving a positive indication of abnormalities of the sacroiliac joints.[16-18] Profile analysis and quantitative evaluation using regions of interest are required for accurate diagnosis. The results ideally should be compared with horses of similar age and work history because of apparent significant age-related variability between clinically normal

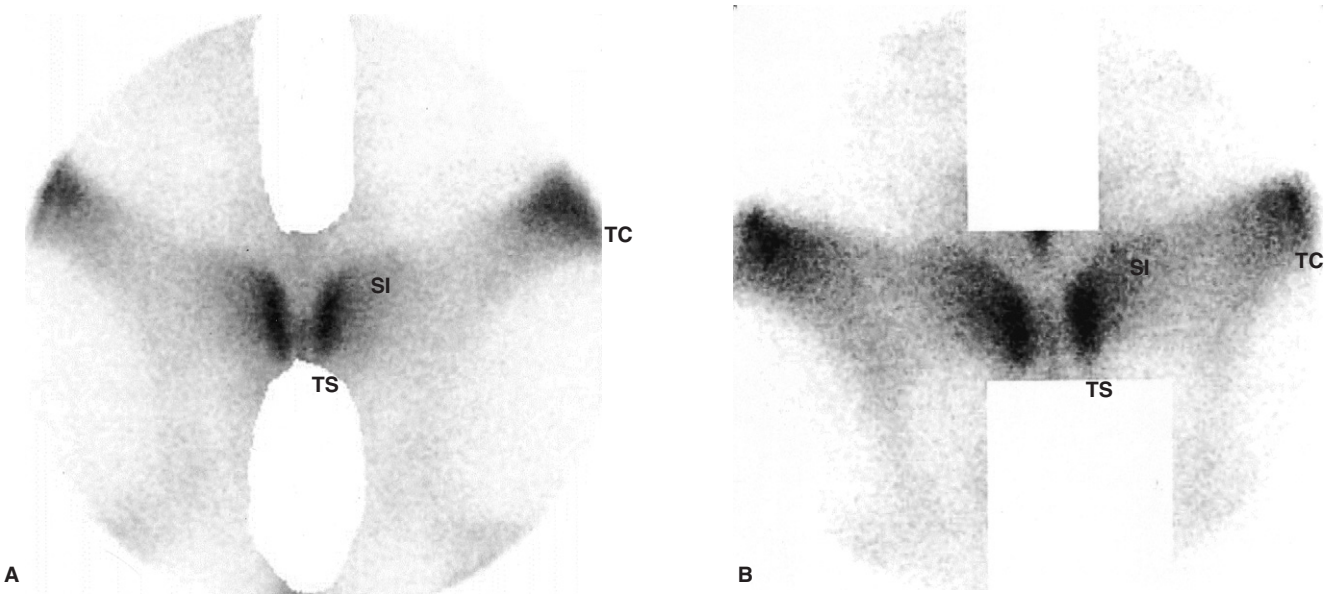

Fig. 52-3 Dorsal delayed (bone) phase scintigraphic images of the sacroiliac regions of (**A**) a normal 2-year-old Thoroughbred and (**B**) a normal 8-year-old Warmblood. The images were acquired as 35, 2-second frames, motion corrected and summed. *SI*, Sacroiliac joint region; *TS*, tuber sacrale; *TC*, tuber coxae.

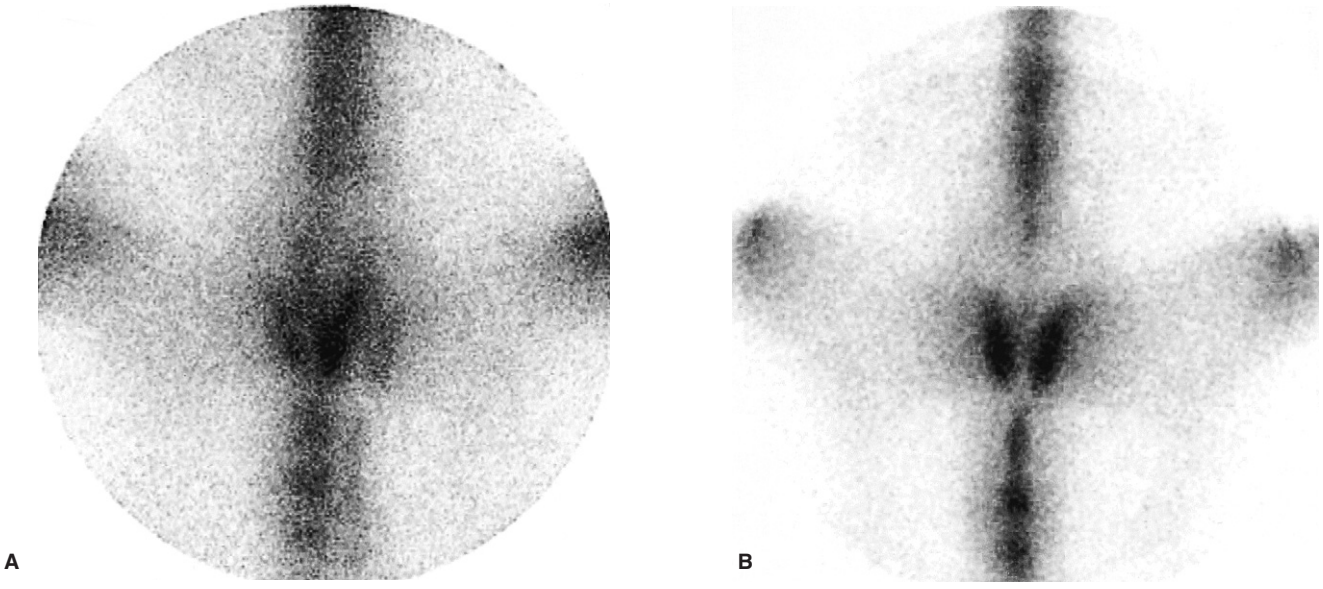

Fig. 52-4 Dorsal delayed (bone) phase scintigraphic image of the sacroiliac joint region of a normal 6-year-old Thoroughbred acquired dynamically as 35, 2-second frames. **A,** The images have been added, but without motion correction. The image is not of diagnostic quality. **B,** The image after motion correction. The oval-shaped tuber sacrale have the most intense radiopharmaceutical uptake, being closest to the gamma camera. The surrounding regions of the sacroiliac joint have less intense activity.

horses[17] (Fig. 52-3). Considerable overlap in ratios of uptake between each sacroiliac joint region and a standard reference site also occurs in clinically normal horses, horses with lameness unrelated to the sacroiliac joint, and horses with clinical signs compatible with sacroiliac joint disease.[18] However, in a normal horse radiopharmaceutical uptake is bilaterally symmetrical. Excessive motion during image acquisition can result in images that are not of diagnostic quality and may mimic abnormalities (Fig. 52-4); therefore, ideally, images should be acquired dynamically, and motion-correction software should be used.[17] Residual radiopharmaceutical in the bladder may also confound interpretation. Abnormalities are recognized as abnormal patterns of radiopharmaceutical uptake (Fig. 52-5) and asymmetrical ratios of uptake between the left and right

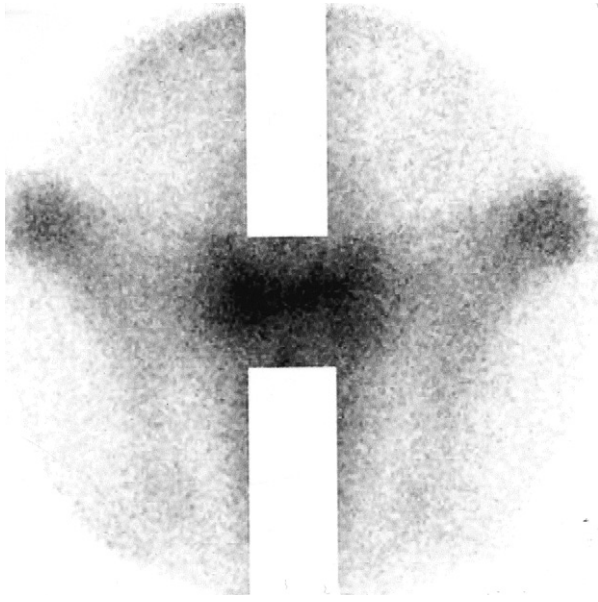

Fig. 52-5 Dorsal delayed (bone) phase scintigraphic image of a 9-year-old Warmblood with clinical signs compatible with sacroiliac joint disease. Compare with Fig. 52-3, *B*; the image is no longer symmetrical in appearance and there is no clear definition between the region of the tuber sacrale and sacroiliac joint regions. This is a motion corrected image. The diagnosis was confirmed post mortem.

sacroiliac joint regions.[18] Care must be taken to differentiate between uptake in the tubera sacrale and uptake associated with the region of the sacroiliac joints.

Radiographic examination requires general anesthesia and can be frustrating, because superimposition of abdominal viscera can preclude evaluation of the sacroiliac joints. However, detection of joint space irregularities or spur formation on the caudal aspect of the joints are poor prognostic indicators.

Ultrasonographic examination should be performed transcutaneously to evaluate the dorsal sacroiliac ligaments[19,20] and per rectum[20] to eliminate pathological conditions of the lumbosacral vertebrae.

Management is essentially palliative, and prognosis is generally inversely correlated with the severity of clinical signs. Local infiltration of a phenol-based sclerosing agent, P2G or a combination of corticosteroids (methylprednisolone acetate, 200 mg) and Sarapin appears to provide relief in some horses. Some horses benefit from treatment with non-steroidal anti-inflammatory drugs, which provide partial pain relief, enabling the horse to work better and develop increased muscle strength. Daily work on the lunge using side reins or a chambon, together with exercise in a cage horse walker in which the horse is free and not tied, and no ridden exercise for several weeks may be of significant benefit. Acupuncture therapy helps some horses. Affected horses should be maintained in work at all times to maintain muscular fitness.

Sacroiliac joint disease may occur with other conditions; therefore, careful appraisal of the whole horse is essential if a successful management strategy is to be achieved.[1] Nuclear scintigraphic evaluation should include the thoracolumbar region. Concurrent impingement of dorsal spinous processes in the mid- or caudal thoracic or cranial lumbar regions may occur with or without associated pain (see page 514). Infiltration of local anesthetic solution between or around the impinging dorsal spinous processes may be necessary to determine their contribution to the clinical problem. Careful evaluation of the synovial articulations, especially close to the thoracolumbar junction, is also important. Occasionally sacroiliac disease has been seen together with osteoarthritis of the coxofemoral joints. This warrants a guarded prognosis.

Desmitis of the Dorsal Sacroiliac Ligament

Desmitis of the dorsal sacroiliac ligament has been recognized in horses that have back pain, with or without focal pain on palpation.[19,20] Usually lesions have been restricted to one side and have been characterized by ultrasonography as enlargements of the ligament and disruption of normal architecture, with hypoechoic regions in transverse images and loss of parallel alignment of echoes in longitudinal images. Enthesophyte formation may occur on the tuber sacrale. Such lesions have been seen alone or with other causes of back pain.

New Bone on the Caudal Aspect of the Wing of the Sacrum

A small number of mature competition horses used for dressage or show jumping have insidious onset loss of hindlimb action, progressing to unwillingness to maintain a proper trot. These horses have appeared considerably worse when ridden compared with evaluation in hand or on the lunge. The horses have shown abnormal sensitivity to palpation in the general areas of the sacroiliac joints. However, careful analysis of nuclear scintigraphic images has shown relatively normal radiopharmaceutical uptake in the region of the joints themselves but increased uptake abaxial to the joints. The horses have responded poorly to treatment with non-steroidal anti-inflammatory or other analgesic drugs. Post-mortem examination has revealed spurs of new bone on the wings of the sacrum just caudal to the auricular surface but not involving the sacroiliac joint itself. This has been seen in association with fusion of the transverse processes of caudal lumbar vertebrae and osseous proliferation involving intervertebral foramina or synovial articulations.[3,14]

Aorto-Iliaco-Femoral Thrombosis

Aorto-iliaco-femoral thrombosis is a relatively uncommon cause of exercise-induced hindlimb lameness of variable severity.[21] Clinical signs may be sudden and severe in onset or subtle initially and slowly progressive. Horses of all ages may be affected. The incidence is higher in male horses than mares. The pathogenesis is unknown.

Clinical signs vary depending on the site(s) of thrombus formation, its size, and the degree of vessel occlusion. Lesions occur most commonly at the terminal aorta, but they may also involve the internal and external iliac arteries and the femoral arteries, unilaterally or bilaterally. Lesions restricted to the femoral artery have not been documented. With mild lesions the horse may show poor performance, early fatigue, or slight loss of hindlimb action during a work period. If the lesion(s) is predominantly unilateral, the horse may show episodic hindlimb lameness induced by work. With more advanced lesions progressive shortening of hindlimb stride may occur with exercise, followed by development of distress and sweating, except over the hindlimbs (Fig. 52-6, *A*). If the horse is allowed to stop, it may repeatedly flex its hindlimbs and stamp the feet to the ground. The affected limbs feel cool, and delayed filling of the saphenous veins may be seen (Fig. 52-6, *B*), with reduced pulse amplitudes in the dorsal metatarsal artery. Clinical signs usually resolve if the horse is allowed to stand still for a few minutes.

In horses with advanced thromboembolism, palpating a thrombus per rectum in the terminal aorta, which feels abnormally firm, is usually possible. Pulses in the iliac arteries may be reduced or absent. With less advanced lesions diagnostic ultrasonography is required for identification of the lesion (Fig. 52-7). Ultrasonography is also useful to determine the

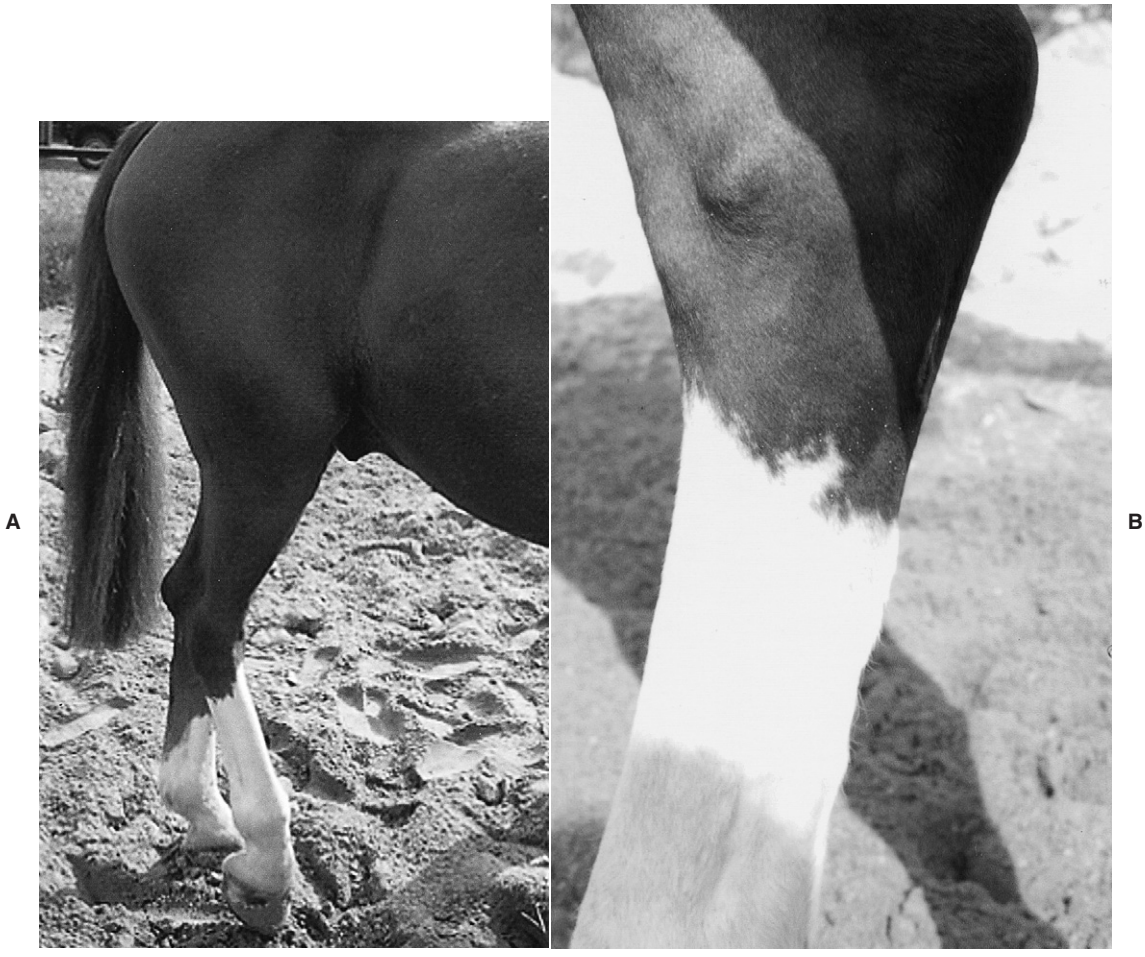

Fig. 52-6 A 9-year-old gelding with aortoiliac thrombosis. Clinical signs were provoked by just 5 minutes of lunging exercise. **A,** The horse repeatedly flexed each hindlimb, stamping it to the ground. No evidence of sweating over the hindquarters was apparent, but the horse was sweating profusely over the neck and flanks. **B,** The saphenous vein cannot be seen because of delayed filling.

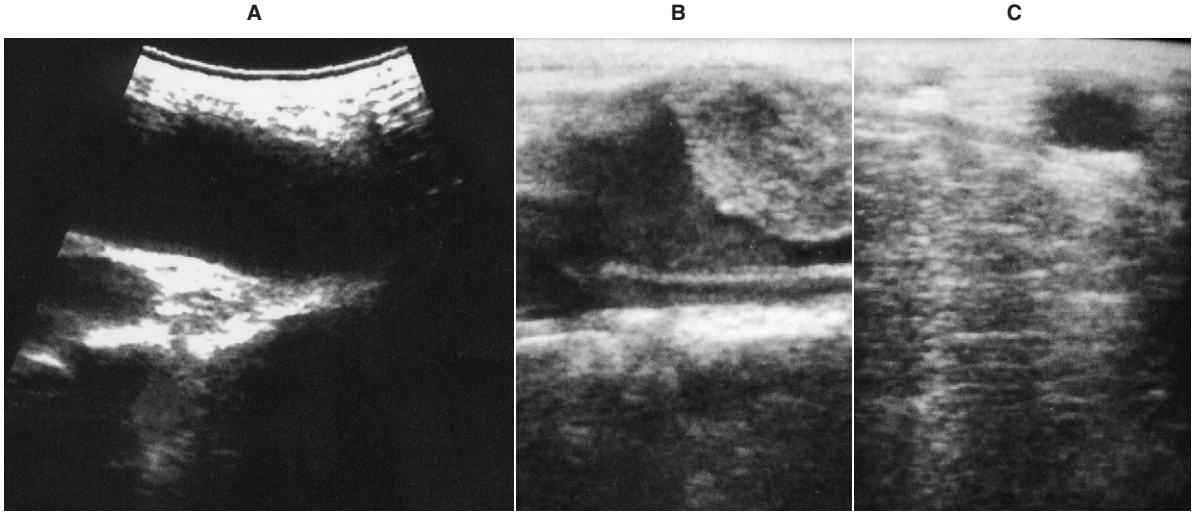

Fig. 52-7 Longitudinal ultrasonographic images of the terminal aorta of (**A**) a normal horse and (**B**) a horse with aortoiliac thrombosis. Cranial is to the left. A large echogenic mass appears in the aorta of the clinically affected horse. **C,** Transverse ultrasonographic image of the right external iliac artery. This appears normal but at post-mortem examination, an extensive thrombus was identified farther distally.

extent of the lesions.[22] Examination of part of the femoral artery can be performed transcutaneously on the medial aspect of the crus,[23] and the use of Doppler ultrasonography to measure blood flow characteristics should help to determine if a more proximal site of obstruction exists.[24]

First-pass radionuclide angiography can be used to determine blood flow in the aorta and iliac arteries[7,25] and also the femoral arteries.[3] However, the sensitivity of the technique for detecting subtle lesions has yet to be determined.

The aims of treatment are to try to prevent further thrombus formation and to promote the development of a collateral blood supply for adequate perfusion. No drugs are available that alter a pre-formed thrombus. Treatment rationale has been based on pain relief and the use of anti-inflammatory drugs, platelet inhibitors, anthelmintics, fibrinolytic agents, and anticoagulant drugs. Those most commonly used include phenylbutazone (2.2. mg/kg BID for 2 months), aspirin (5 mg/kg SID for several months), and isoxsuprine (1 mg/kg BID for 3 months). Successful treatment presumably is based on development of an effective collateral blood supply.

I have successfully managed some mildly affected horses with long-term aspirin therapy, but horses with more severe lesions are usually refractory to treatment. Only 2 of 29 horses returned to former athletic function, with resolution of clinical signs, following medical treatment.[21] Surgical removal is feasible,[26] although the rate of recurrence is high.

Coxofemoral Joint

The coxofemoral joint is rarely a source of pain causing lameness in the horse.

Dysplasia

Dysplasia of the coxofemoral joint is rare, usually occurs bilaterally, and predisposes relatively young horses to develop osteoarthritis. The prognosis for athletic function is poor. Dysplasia may be a heritable condition in the Norwegian Dole.

Osseous Cyst-Like Lesions

An osseous cyst-like lesion occasionally has been identified in young horses and associated with lameness.[3,27] The lesions have resulted in chronic lameness.

Osteoarthritis

Osteoarthritis of the coxofemoral joint is an unusual cause of hindlimb lameness in the horse, usually occurring unilaterally, but occasionally bilaterally. Osteoarthritis may occur secondary to dysplasia, rupture of the teres ligament (see page 500), or trauma.

Lameness varies from moderate to severe. The horse resents flexion of the limb and is unwilling to stand on the limb for long periods with the contralateral limb picked up. The degree of gluteal muscle disuse atrophy may be more than that from pain in the more distal part of the limb. Lameness may be characterized by a tendency for the horse to move on three tracks.

Intra-articular analgesia usually improves lameness, but it is rarely alleviated fully.

Nuclear scintigraphic evaluation may help in highlighting the coxofemoral joint as abnormal, especially if the results of intra-articular analgesia are equivocal. However, it is important to be aware that loss of muscle over the hindquarter of the lame limb may confound image interpretation. I have found it useful to evaluate ratios of radiopharmaceutical uptake in the region of the coxofemoral joint and another standardized location and compare this with values for normal horses. The clinician should be aware that radiopharmaceutical uptake associated with the coxofemoral joint is much less than that of the cranial and caudal parts of the greater trochanter of the femur.

Definitive diagnosis of osteoarthritis requires radiographic examination, and high-quality radiographs can only be achieved with the horse in dorsal recumbency under general anesthesia. Abnormalities include periarticular osteophyte formation, new bone formation along the femoral neck, lucent zones in the subchondral bone of the acetabulum or femoral head, and loss of congruity between the acetabulum and femoral head (Fig. 52-8). Care should be taken not to confuse the depression in the femoral head and underlying radiolucent zone at the site of insertion of the teres ligament as a lesion.

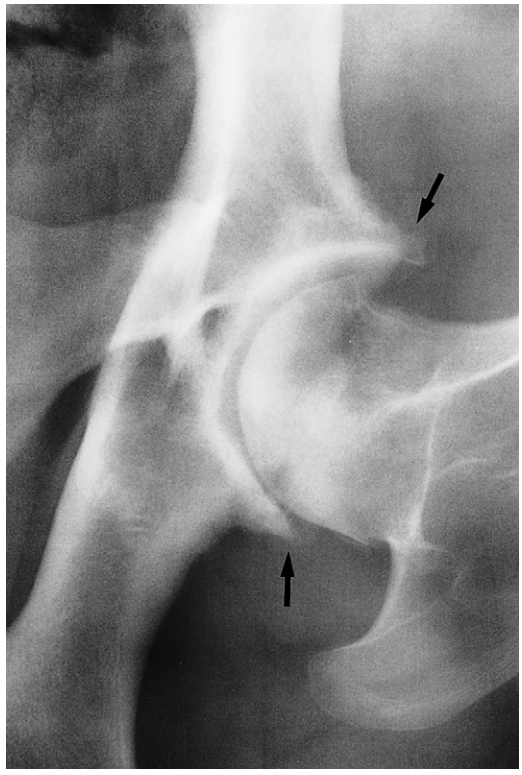

Fig. 52-8 Ventrodorsal radiographic view of the left coxofemoral joint of 9-year-old steeplechaser with osteoarthritis. The horse had raced successfully the previous season, but it now showed moderate left hindlimb lameness at trot and carried the left hindlimb at canter. Note the marked periarticular osteophyte formation on the pelvic acetabulum (*arrows*) and the irregular subchondral bone opacity.

Intra-articular medication of a coxofemoral joint with radiographic abnormalities has yielded disappointing results. The prognosis for return to athletic function is guarded.

Trochanteric Bursitis

Trochanteric bursitis has been described in the older literature, but the Editors have not recognized this condition. The condition is discussed elsewhere (page 472).

Luxation with or without Secondary Upward Fixation of the Patella

Luxation or subluxation of the coxofemoral joint is an unusual injury and can occur as a primary injury or secondary to an unstable fracture of the ilial shaft. The trauma causing luxation also can result in articular fractures of the acetabulum. Permanent upward fixation of the patella may develop as a sequel because of displacement of the femur. The femur is displaced proximally; therefore the greater trochanter of the femur appears higher on the lame limb. However, this can be difficult to assess, because the horse is usually reluctant to bear weight on the limb. Upward fixation of the patella results in an abnormally straight hindlimb stance and an inability to flex the limb. The diagnosis can be confirmed by radiography, and with high-output x-ray machines, diagnostic radiographs

can be obtained with the horse standing; however, determining if concurrent fractures exist may be difficult. The prognosis for athletic function is hopeless.

Rupture of the Teres Ligament

Rupture of the teres ligament results from trauma. It has been seen in a horse that tried to get up prematurely from general anesthesia while the limbs were still restrained in hobbles.[3] Lameness is severe, with the horse being unwilling to bear weight on the limb. Secondary osteoarthritis rapidly ensues. Definitive diagnosis is only possible post mortem. The prognosis for athletic function is hopeless.

Displacement of the Femoral Head

Displacement of the femoral head has been seen occasionally in young horses with severe lameness and reluctance to bear weight on the limb. Diagnosis is confirmed radiographically. The prognosis is hopeless.

Fracture (or Enthesopathy) of the Third Trochanter of the Femur

Fracture of the third trochanter of the femur is a relatively unusual injury, resulting in acute onset, severe lameness that often improves rapidly with box rest. In a lean, poorly muscled horse, eliciting pain by palpation may be possible, but in a well-muscled Warmblood type, eliciting pain is usually not possible, even in acute injuries. No particular gait characteristics are apparent. Nuclear scintigraphy is particularly valuable for tentative diagnosis of a fracture.[3,4] Increased radiopharmaceutical uptake and sometimes a change in the pattern of uptake occur. Diagnosis may be confirmed radiographically, but good-quality radiographs can only be achieved under general anesthesia. Fractures are often longitudinal, occurring at the base of the trochanter.[2-4] Displacement is usually minimal. Diagnostic ultrasonography has not been useful in my experience in helping to confirm a fracture. However, ultrasonography is useful to exclude abnormalities of the superficial gluteal muscle, which inserts on the third trochanter. Insertional tears of this muscle may be associated with a similar scintigraphic appearance. Treatment is box rest for 2 months, followed by walking exercise for another month. Healing may occur by osseous or fibrous union. Prognosis is good.[3]

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CHAPTER • 53

Diagnosis and Management of Sacroiliac Joint Injuries

Kevin K. Haussler

ANATOMICAL AND FUNCTIONAL FEATURES

The sacroiliac joint is a synovial articulation located at the junction between the ventral wing of the ilium and the dorsal wing of the sacrum (Fig. 53-1). The sacroiliac joint functions in pelvic attachment to the axial skeleton, providing support during weight bearing and helping to transfer propulsive forces of the hindlimb to the vertebral column. The sacroiliac joint is an atypical synovial articulation because of hyaline cartilage on the sacral articular surface and a thin layer of fibrocartilage on the ilial articular surface.[1] The articular surfaces of the sacroiliac joint are nearly flat and closely apposed to support gliding movements. The sacroiliac articular surfaces diverge cranially at about 40° from a transverse plane and are angled craniodorsally to caudoventrally at about 60° from the horizontal plane. The joint capsule is thin and closely follows the margins of the sacroiliac articular cartilage. The sacroiliac joint capsule is reinforced ventrally by the ventral sacroiliac ligament.[2] A small amount (<1 ml) of synovial fluid is normally present in the joint.[1] Because of articular surface remodeling, the size and shape of the sacroiliac joint margins vary considerably according to age and body weight.[3] Typically the sacroiliac joint outline is L-shaped, with the convex border directed caudoventrally.

The pelvis is firmly attached to the axial skeleton by sacroiliac and sacrosciatic ligaments, which form a strong ligamentous sling (see Fig. 53-1). The weight of the caudal vertebral column is suspended from the sacroiliac ligaments, which function similarly to the fibromuscular sling found between the proximal forelimb and the lateral thoracic body wall. Subsequently the sacroiliac articular cartilage may never be fully weight bearing, unlike most articular cartilage. The sacroiliac joint is supported by three pairs of strong ligaments: the dorsal, interosseous, and ventral sacroiliac ligaments. The dorsal sacroiliac ligament consists of dorsal and lateral portions (Fig. 53-2). The dorsal portions form two round cords that span from the dorsal aspects of the tubera sacrale to the dorsal apices of the sacral spinous processes. The lateral portion forms a sheet of connective tissue that spans from the caudal margin of each tuber sacrale and iliac wing to the lateral border of the sacrum. The lateral portion of the sacroiliac ligament is continuous ventrally with the sacrosciatic ligament. The interosseous ligament of the sacroiliac joint is robust and consists of a series of vertical fibers that connect the ventral wing of ilium to the dorsal wing of the sacrum (see Fig. 53-1). The interosseous sacroiliac ligament provides the major resistance to vertically oriented weight-bearing forces acting on the sacrum. The ventral sacroiliac ligament interconnects the ventral wings of the ilium and sacrum. The ventral sacroiliac ligament is thin and closely applied to the ventral margins of the sacroiliac joint capsule.

Neurovascular structures adjacent to the sacroiliac joint include the sciatic nerve, cranial gluteal nerve, and cranial gluteal artery and vein. These structures collectively travel

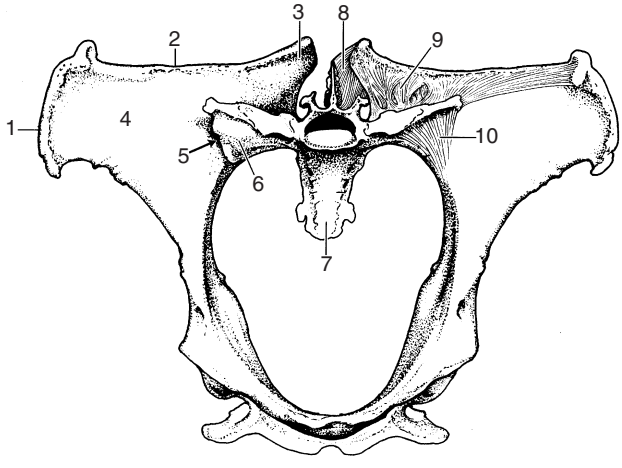

Fig. 53-1 Diagram of the pelvis, sacrum, and sacroiliac ligaments (cranial view). (Modified from Dyce KM, Sack WO, Wensing CJG: *Textbook of veterinary anatomy*, ed 2, Philadelphia, 1996, WB Saunders.) *1*, Tuber coxae; *2*, ilial crest; *3*, tuber sacrale; *4*, ventral surface of the iliac wing; *5*, sacroiliac articulation; *6*, sacral wing; *7*, sacrum; *8*, dorsal sacroiliac ligament; *9*, interosseous sacroiliac ligament; *10*, ventral sacroiliac ligament.

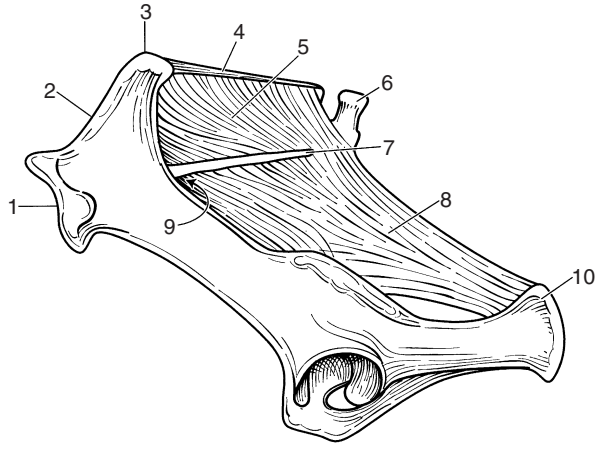

Fig. 53-2 Diagram of the pelvis, sacrum, and sacroiliac ligaments (lateral view). *1*, Tuber coxae; *2*, ilial crest; *3*, tuber sacrale; *4*, dorsal sacroiliac ligament (dorsal portion); *5*, dorsal sacroiliac ligament (lateral portion); *6*, first coccygeal vertebrae; *7*, lateral portion of sacrum; *8*, sacrosciatic ligament; *9*, greater sciatic foramen; *10*, ischial tuberosity.

through the greater sciatic foramen, ventromedial to the sacroiliac articulation (see Fig. 53-2). The cranial gluteal artery and vein arise from the internal iliac vessels and travel over the ventral sacroiliac ligament and the caudomedial aspect of the sacroiliac joint before emerging at the greater sciatic foramen. The cranial gluteal artery and vein continue dorsally into the gluteal musculature. The middle and accessory gluteal muscles originate from the dorsal iliac wing and have attachments near the caudomedial aspect of the sacroiliac joint. Within the pelvic canal a portion of the internal obturator and iliacus muscles cover the ventral sacroiliac joint margins.

The robust sacroiliac and sacrosciatic ligaments limit motion at the sacroiliac articulations.[4] Shear forces would be expected at the sacroiliac articular surfaces more than compressive forces, which are common in most limb articulations.[5] Sacroiliac joint movements are restricted to small amounts of flexion (nutation) and extension (counternutation), with an apparent axis of rotation oriented transversely near the caudomedial aspect of the joint. Lateral movements at the sacroiliac articulations are severely restricted, primarily because of lateral attachments of the ventral sacroiliac ligaments, and to a lesser degree the lateral portion of the dorsal sacroiliac ligaments and the sacrosciatic ligaments. The wings of the ilium overlay the sacral articular surfaces dorsally and laterally, which precludes any axial rotation from occurring at the sacroiliac joint. Dorsal displacement of the ilial wings is limited by the robust interosseous ligament, the ventral sacroiliac ligament, and lateral portion of the dorsal sacroiliac ligaments.[6] Propulsive forces of the hindlimb are transmitted dorsally and cranially to the vertebral column by the articular configuration of the overlapping and divergent ilial wings over the sacrum and by reinforcement from the dorsal and interosseous sacroiliac ligaments. The dorsal sacroiliac ligament also provides resistance against contraction of the powerful longissimus muscle and robust thoracolumbar fascia, which attach along the cranial border of the ilial wing.

PATHOLOGICAL CONDITIONS

The antemortem diagnosis of sacroiliac joint injury in horses is difficult and often based on a diagnosis of exclusion.[7] Diagnosis is complicated by anatomical inaccessibility, mild chronic clinical signs so that opportunities for correlation with necropsy findings are uncommon, and ongoing controversies over the clinical significance and prevalence of pathological conditions of the articular surface and ligaments.[4] Terms used to describe pathological conditions of the sacroiliac joint include sacroiliac sprain or instability,[8,9] sacroiliac joint subluxation,[10] and sacroiliac arthrosis.[11,12] The prevalence of pathological conditions of the sacroiliac joint in performance horses is probably high and many may go undiagnosed.[4,11,13] In a necropsy survey of 36 Thoroughbred (TB) racehorses with no known back or sacroiliac joint problems, we observed various degrees of degenerative sacroiliac joint changes in all specimens.[14] The clinical significance of osseous sacroiliac joint pathological conditions is difficult to determine, because many presumed normal horses have degenerative joint changes similar to horses with known back or sacroiliac problems.[1,4] Possibly the majority of sacroiliac joint pathological conditions are subclinical; however, if similar findings were noted in any other musculoskeletal location, the articular changes would be considered clinically significant and a likely contributing cause of lameness. A more likely scenario is that deep sclerotogenous pain (e.g., vertebral or sacroiliac joint osteoarthritis) is often poorly localized and perceived as deep aching pain, based on reports of similar pathological conditions of the sacroiliac joint in affected people. Clinically the most common reported signs of sacroiliac joint disorders in horses are poor performance, lack of impulsion, and a mild, chronic hindlimb lameness, which can easily be overlooked or dismissed as not clinically significant.[4,7] Obvious signs of lameness and localized pain or inflammation are not typical clinical characteristics of sacroiliac joint or pelvic injuries, unless pelvic fractures or substantial joint disruption are present.

Osseous changes of the sacroiliac joint include (in apparent order of increasing severity) articular surface lipping, cortical buttressing, articular recession, osteophytes, and intra-articular erosions (Fig. 53-3). In our survey of 36 TB racehorses, sacroiliac degenerative changes were classified as mild in 8% of specimens, moderate in 61%, and severe in 31%. Age has not been associated with the overall prevalence or severity of sacroiliac joint degenerative changes.[14] Osseous changes are usually bilaterally symmetrical and most commonly located at the caudomedial aspect of the articulation.[1,14] The pathogenesis of proliferative sacroiliac joint changes is uncertain, but it is thought to be related to chronic instability resulting in gradual remodeling and subsequent enlargement of the caudomedial joint surfaces. Histologically the caudomedial extensions consist of apparently normal cancellous bone.[4] Articular cartilage erosion is a lytic process of articular surfaces that presumably leads to eventual sacroiliac joint ankylosis.[14] However, ankylosis of the sacroiliac joint has not been reported, which is surprising based on the limited joint motion and the potential severity of osseous pathological conditions present.[1,14,15] Fibrous interconnections between the articular surfaces have also been reported in presumed normal sacroiliac joints.[1] Articular cartilage discoloration, a presumed indicator of sacroiliac joint degeneration, is common in racehorses, but it has a reported higher prevalence in Standardbreds (STBs) compared with TBs.[11] One theory is that these changes are caused by differences in pelvic and sacroiliac joint biomechanics associated with pacing and trotting (i.e., lateral bending or shear forces) in STBs compared with galloping (i.e., flexion and extension movements) in TBs.[16] Biomechanical studies are warranted to support or refute these claims.

Sacroiliac desmitis, the most common soft tissue injury, has been documented by ultrasonography in the dorsal portion of the dorsal sacroiliac ligament.[17] A diagnosis of sacroiliac desmitis is based on loss of normal echogenicity on a transverse view and a decrease in parallel fiber pattern on a

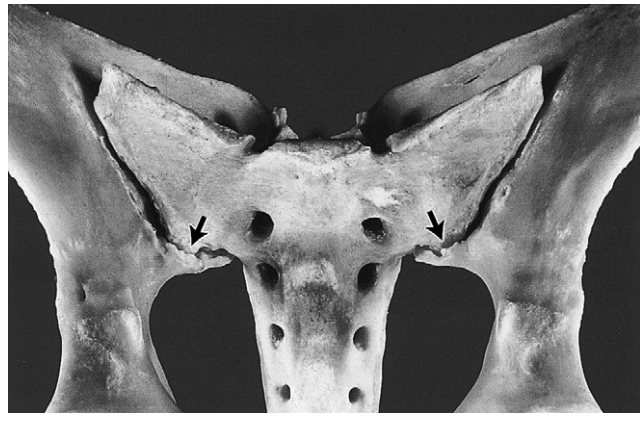

Fig. 53-3 Bilateral osteoarthritis of the sacroiliac articulations (ventral view). Severe proliferative changes of the caudomedial one third of the sacroiliac joints (*arrows*) in 8-year-old racing Thoroughbred gelding that was euthanized because of an acute colic episode. Normal sacroiliac joint margins are typically smooth and linear as represented by the cranial half of the sacroiliac articulations.

longitudinal view. Sacroiliac ligament injuries usually occur because of acute trauma, but few horses with documented injury have been reported.[14,18]

Complete sacroiliac ligament disruption is most likely caused by substantial trauma, such as flipping over backward, or catastrophic musculoskeletal injuries associated with race training.[14] Post-mortem findings associated with traumatic sacroiliac ligament injuries include either unilateral or bilateral joint capsule disruption, avulsion fractures of the sacroiliac ligament attachment sites, and noticeable sacroiliac joint laxity. Dorsal or ventral sacroiliac ligaments can be affected, depending on the inciting mechanism of injury.[6,14,18] Complete sacroiliac ligament disruption may produce unilateral or bilateral dorsal displacement of the tubera sacrale, depending on the extent of injury (Fig. 53-4).

Acute sacroiliac ligament injuries have been reported to contribute to development of chronic sacroiliac joint instability.[8,18] However, the presence and significance of chronic sacroiliac ligament injury and sacroiliac joint laxity are controversial. Rooney et al. reported on chronic sacroiliac joint injuries of the cranial portion of the ventral sacroiliac ligaments, which were found to be elongated or torn on the affected side.[18,19] Desmitis of the insertion site of the dorsal portion of the dorsal sacroiliac ligament at the insertion on the tuber sacrale has been reported.[6] In other studies osseous changes were found at the caudomedial sacroiliac joint margins in horses suspected of having chronic sacroiliac injury; however, no obvious sacroiliac ligament laxity was observed.[4,8,9] Radiographically some of these horses had an apparent increase in the sacroiliac joint space; however, no visible sacroiliac ligament injury, joint laxity, or subluxation was observed at necropsy.[4,9] In our necropsy survey of 36 TB racehorses no evidence of chronic ligament injury or sacroiliac joint subluxation was observed.[14] However, this could be related to the prior removal of horses from race training with poor performance or hindlimb lameness associated with chronic sacroiliac joint injury.

The pathogenesis of apparent spontaneous or insidious differences in tuber sacrale height needs to be further researched.[4] Unilateral or bilateral dorsal displacement of the tubera sacrale is often a presumed indication of sacroiliac subluxation.[20] In my opinion, variable degrees of tubera sacrale height asymmetry occur frequently and may be caused by chronic asymmetric muscular or ligamentous forces acting on the malleable osseous pelvis and not by direct sacroiliac ligament injury.[21] Tubera sacrale height asymmetries are common in horses without documented sacroiliac joint injuries (see Fig. 53-4). In only a few horses have tubera sacrale height asymmetries been associated with chronic sacroiliac ligament injuries or joint laxity.[19] In a study of 4-year-old STB trotters with tubera sacrale height asymmetries of more than 1 cm, associations were found with poor performance, but otherwise the asymmetry was of questionable clinical significance.[13] If substantial tubera sacrale height differences are identified, which side is affected is unclear and difficult to determine: the seemingly dorsally displaced tuber sacrale or the less prominent tuber sacrale on the opposite side.[4,13] Presumed sacroiliac joint subluxation produces an elevated tuber sacrale on the affected side, whereas complete ilial wing fractures (the most common type of pelvic fracture) typically produce a palpably depressed tuber sacrale on the affected side.[22]

CLINICAL PRESENTATION

Horses with sacroiliac joint injuries vary in clinical presentation, usually based on the duration and extent of injury present. The history of acute sacroiliac joint injury usually includes slipping, falling, or trauma that causes pelvic rotation or induces high stresses.[20] Horses with acute sacroiliac joint injuries often have noticeable lameness and localized sensitivity to palpation of the surrounding soft tissues or tubera sacrale (Fig. 53-5), which must be differentiated from other sources of back, pelvic, or hindlimb pain. Diagnosis of acute sacroiliac ligament rupture and subsequent sacroiliac joint subluxation or luxation is based on history, physical examination, and diagnostic imaging. Because the majority of horses have some degree of tuber sacrale height asymmetry, a diagnosis of acute sacroiliac joint subluxation can only be confirmed if symmetry of the tuber sacrale was documented immediately before the injury, and subsequently gross asymmetry of the tubera sacrale is noted with localized signs of pain or inflammation.

Significant historical findings in horses with chronic sacroiliac joint injuries usually involve repetitive overuse

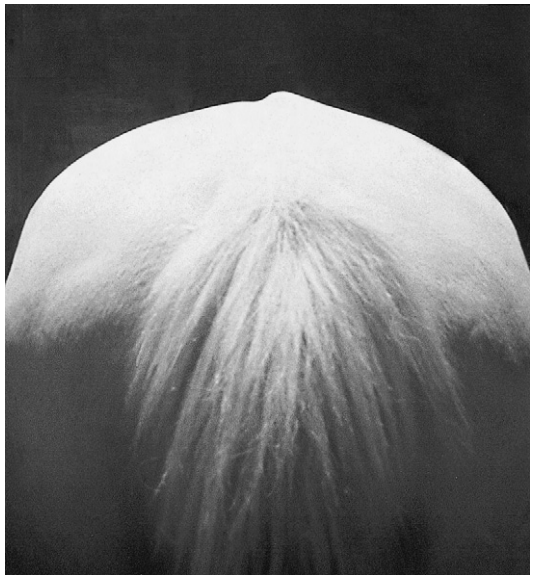

Fig. 53-4 Photograph of tubera sacrale height asymmetry (caudal view). The tuber sacrale on the right side is dorsally displaced, with apparent gluteal muscle symmetry. (Courtesy Al Kane, Ft. Collins, Colo.)

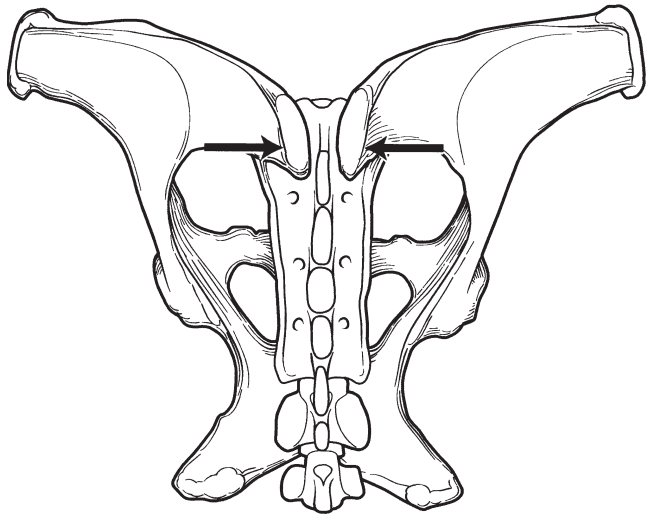

Fig. 53-5 Diagram of sacroiliac joint provocation test (dorsal view). Firm pressure is applied bilaterally with both hands, compressing the dorsal aspects of the tubera sacrale.

versus a single traumatic event.[11] The most consistent clinical feature of chronic sacroiliac joint injury is a prolonged, nonprogressive history of poor performance.[4] Changes in performance include back stiffness, resisting jumps, and lack of impulsion from one or both hindlimbs. Subtle gait asymmetries may be noticed at slow speeds during ground work or dressage movements and in harness horses at racing speeds.[7] Chronic sacroiliac joint injuries usually produce a low-grade or intermittent hindlimb lameness or stiffness that cannot be localized by routine lameness examination techniques and is improved only temporarily with anti-inflammatory medications.[4] The affected sacroiliac joint usually corresponds to the side of lameness or reduced cranial swing phase of the affected hindlimb, but bilateral gait abnormalities can occur. Gait asymmetries involve subtle differences in pelvic movement or reduced hindlimb flexion or stride lengths. Affected horses commonly resent standing on the affected hindlimb while the contralateral hindlimb is lifted off the ground during hock flexion or shoeing.[7] Affected horses may also drag a hind foot and have mild hindlimb abduction before hoof contact (i.e., plaiting or rope walking).[4] Disuse atrophy of the gluteal musculature is an inconsistent finding; however, some horses have pelvic asymmetry with a lower tuber coxae and noticeable gluteal muscle atrophy on the affected side.[4,23] Observation of the tubera sacrale may reveal unilateral or bilateral (i.e., hunters' or jumpers' bumps) differences in height or dorsal prominence (see Fig. 53-4).

PHYSICAL EXAMINATION

Clinical signs of sacroiliac joint injury vary. The sacroiliac joint is relatively inaccessible to direct evaluation or palpation, and the normally small amount of sacroiliac joint motion is difficult to detect clinically.[24] Horses with acute sacroiliac joint injuries may have localized sensitivity to palpation of the surrounding soft tissues in the dorsal croup region.[4] A localized region of edema may occasionally be palpated over the lumbosacral junction[25]; however, this is not a specific finding related to sacroiliac joint injury. Protective muscle spasms may be palpated in the adjacent middle gluteal musculature and the vertebral portions of the biceps femoris, semitendinosus, and semimembranosus muscles. In acute sacroiliac injuries asymmetry in gluteal muscle development is uncommon, unless pronounced osseous pelvic asymmetry is also present. Pain may be elicited by applying firm digital pressure over the dorsal aspects of the tubera sacrale or caudal lumbar and sacral dorsal spinous processes. Unilateral or bilateral prominence of the tubera sacrale may be noted, but it is not usually clinically significant unless associated with clinical signs of localized pain or inflammation, or positive findings on diagnostic imaging (e.g., scintigraphy). Normally the tubera sacrale move in unison during pelvic movement in locomotion. A palpable or visible independent movement of the tubera sacrale at a walk or during treadmill locomotion indicates sacroiliac joint luxation or a complete pelvic fracture. Crepitus associated with sacroiliac joint instability, or complete pelvic fracture, may be palpable or auscultated with a stethoscope placed over the gluteal musculature as the pelvis is repeatedly rocked laterally. Horses with acute sacroiliac joint injuries may also resent flexion of the hindlimb on the affected side or rectal palpation in the region of the sacroiliac joint.

Horses with chronic sacroiliac joint injuries often have compensatory stiffness and pain in the proximal hindlimb.[20] Induced upper limb flexion (i.e., spavin test) is often negative.[4] Rectal examination for chronic sacroiliac joint subluxation is usually unrewarding and will not be diagnostic unless bone proliferation, excess joint motion, or joint crepitus during externally applied movements are identified.[20] A pain response or palpable muscle hypertonicity in the iliopsoas muscles may be noted during rectal examination. Serum chemistry indicators of skeletal muscle injury or inflammation (i.e., creatine kinase and aspartate aminotransferase) are often negative.

The apex of the second sacral spinous process is a reliable landmark used to evaluate relative unilateral or bilateral tubera sacrale displacement. The robust dorsal portion of the dorsal sacroiliac ligament spans between the tubera sacrale and the sacral spinous processes. Normally the dorsal apices of the tubera sacrale and the second sacral spinous process lie in close apposition and follow the contour of the croup. Using palpation, ultrasound, or radiographs, a physical discrepancy in height often can be identified between the dorsal profile of the sacral spinous processes (which should remain constant, unless fractured) and the potentially dorsally or ventrally displaced tuber sacrale. In this manner, unilateral (i.e., tubera sacrale height asymmetry) or bilateral tubera sacrale displacement (i.e., hunters' or jumpers' bumps) can be diagnosed, depending on if one or both tubera sacrale are elevated relative to the apices of the sacral spinous processes. Bilateral tubera sacrale displacement has an unknown clinical significance and may be occur in many high-level competition horses.[25] Theoretically the hunters' or jumpers' bumps may provide a longer lever arm for the strong longissimus and thoracolumbar fascia to produce extension at the lumbosacral joint, resulting in increased impulsion and range of hindlimb motion, with subsequent improved performance.

Firm digital pressure applied to the dorsal aspects of each tuber sacrale has been reported to produce a variable and inconsistent pain response.[7] In my experience, dramatic pain responses have been produced in affected horses with specific provocation tests, which are useful to establish a presumptive diagnosis of pelvic stress fracture or sacroiliac joint injury. The first procedure involves simultaneous manual compression of the dorsal aspects of both tubera sacrale, which induces a bending moment on the iliac wing and presumably compresses the sacroiliac articulations (see Fig. 53-5). Acutely affected horses may have a dramatic reaction to this manipulation and demonstrate sudden hindlimb flexion and an apparent inability to bear weight in the hindlimbs when pressure is applied. Clinicians should gradually apply increasing pressure, because affected horses may actually collapse in the hindlimbs and fall to the ground if excess force is applied to the painful tubera sacrale. A negative response is characterized by minimal pain response and slight extension of the lumbosacral joint during manual compression of the tubera sacrale. This test is not specific for pathological conditions of the sacroiliac joint, because horses with incomplete or stress fractures of the ilial wing may respond even more dramatically to the applied pressure.

A second procedure used to identify sacroiliac ligament injury involves rhythmically applying a ventrally directed force over the lumbosacral dorsal spinous processes in an effort to stress the supporting sacroiliac ligaments. This procedure requires the clinician to get up on an elevated surface (e.g., mounting block) so that the applied forces can be directed vertically over the sixth lumbar and second sacral dorsal spinous processes. Horses with sacroiliac ligament injuries would be expected to resent the induced movement because it specifically stresses the interosseous sacroiliac ligament (i.e., ligamentous sling of the sacropelvic junction). Horses with lumbosacral vertebral joint dysfunction (i.e., localized pain, reduced joint motion, and muscle hypertonicity without structural pathological conditions) may also resent this procedure. Rhythmically applied ventrally directed forces over the dorsal spinous processes at the sacrocaudal junction would be expected specifically to stress the dorsal portion of

the dorsal sacroiliac ligament. A positive response to this test combined with positive ultrasound findings of desmitis of the dorsal sacroiliac ligament would be highly suggestive of clinically significant sacroiliac ligament injury.

A similar procedure involves rhythmically applying a ventrally directed force over the tuber coxae to induce general sacroiliac and lumbosacral joint motion. A normal response to the induced movement is fluid vertical motion of the lumbosacral region, with an amplitude of 1 to 2 cm of dorsoventral movement measured over the lumbar dorsal spinous processes. Affected horses have a noticeable pain response, resent the induced movement, or have protective gluteal or sublumbar muscle spasms. The vertically directed force also induces movement at the lumbosacral junction, which must be differentiated from sacroiliac joint injury.

Additional procedures used to localize sacroiliac joint or ligament injuries involve indirectly evaluating pain and ligamentous laxity in the sacroiliac joint using laterally applied forces (Figs. 53-6 and 53-7). These procedures are similar to valgus-varus stress tests used to evaluate collateral ligaments of the distal limb articulations. Caution should be taken not to apply excessive force because of the long lever arm action of

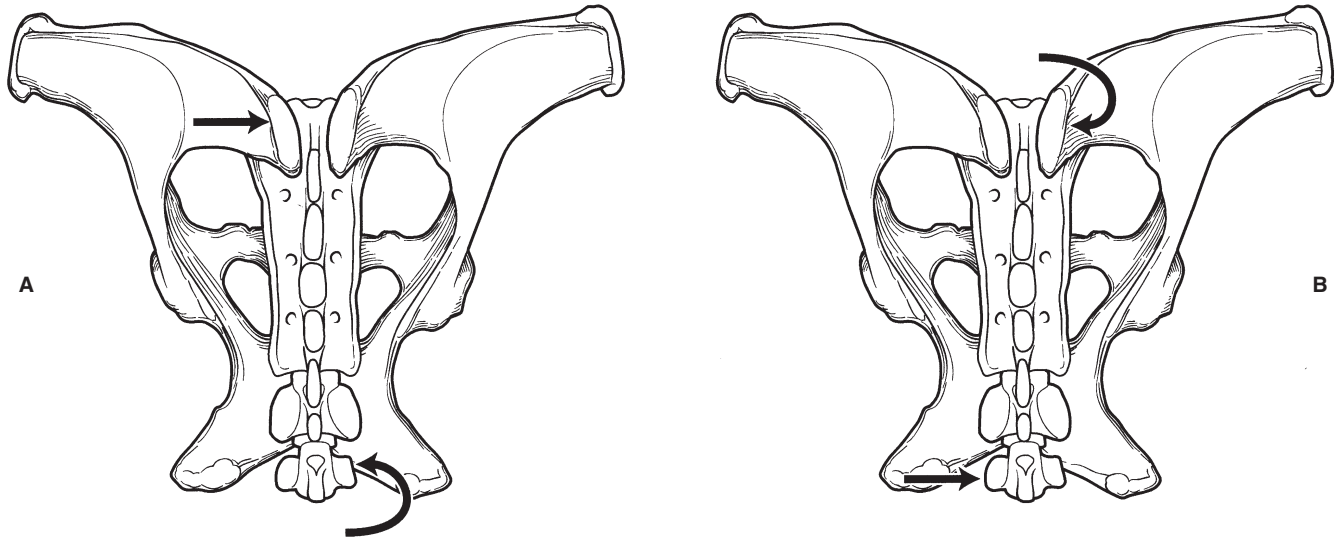

Fig. 53-6 Diagram of second sacroiliac joint provocation test (dorsal view). **A,** Firm pressure is applied by both hands, pushing with the hand at the ipsilateral tuber sacrale away from the clinician, while simultaneously pulling with the hand at the tail head toward him or her. **B,** Firm pressure is applied by both hands, pulling with the hand at the contralateral tuber sacrale toward the clinician, while simultaneously pushing with the hand at the tail head away from him or her.

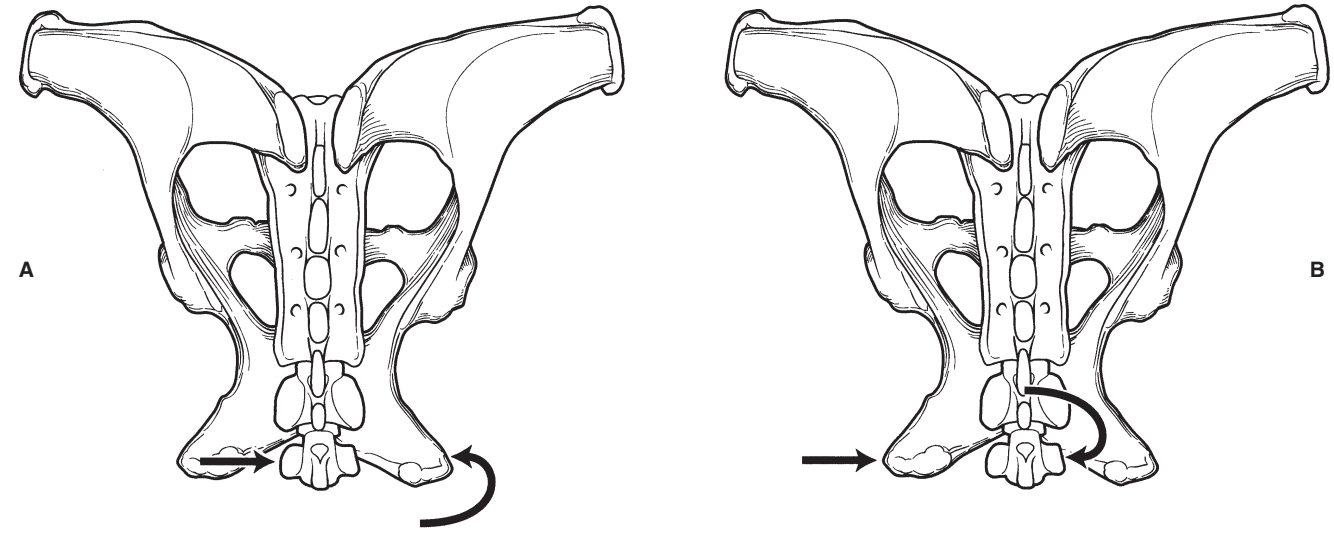

Fig. 53-7 Diagram of third sacroiliac joint provocation test (dorsal view). **A,** Firm pressure is applied by both hands, pushing with the hand at the tail head away from the clinician, while simultaneously pulling with the hand at the contralateral ischial tuberosity toward him or her. **B,** Firm pressure is applied by both hands, pulling with the hand at the tail head toward the clinician, while simultaneously pushing with the hand at the ipsilateral ischial tuberosity away from him or her.

the sacral apex on the sacroiliac ligaments, which can unduly stress unstable or partially torn ligaments or aggravate an acutely inflamed sacroiliac joint. The proposed mechanism of action of these tests is to use the base of the tail and sacrum as a handle to apply a lateral (horizontal plane) stress to the sacroiliac joint as the wing of the ilium is stabilized. The technique involves two parts. First, the base of the hand closest to the horse's head is placed over the lateral aspect of the tuber sacrale. The hand closest to the tail grasps the base of the tail head (the second and third coccygeal bones). The sacroiliac joints are then evaluated as firm pressure is simultaneously applied by both hands, pushing with the hand at the tuber sacrale away from the clinician and pulling with the hand at the tail head toward him or her (see Fig. 53-6, *A*). Theoretically, this maneuver produces compression of contralateral sacroiliac articular surfaces and distraction of the ipsilateral sacroiliac articular surfaces.

The second portion of the technique involves repeating the procedure and reversing the direction of the applied forces (see Fig. 53-6, *B*). The fingers of the hand closest to the horse's head are placed over the contralateral tuber sacrale, and the base of the hand closest to the tail is placed against the ipsilateral base of the tail head (second and third coccygeal bones). The sacroiliac joints are again evaluated as firm pressure is applied by both hands, pulling with the hand at the tuber sacrale toward the clinician and pushing with the hand at the tail head away from him or her. Theoretically the contralateral sacroiliac articular surfaces are distracted and the ipsilateral sacroiliac articular surfaces are compressed. A pain response to the induced movements may be identified unilaterally or bilaterally, depending on the extent of inflammation or injury present. In general, sacroiliac joint compression would be expected to aggravate osteoarthritic changes, whereas joint distraction would be expected to stress any injured or inflamed sacroiliac ligaments.

A variation of this basic technique uses repeatedly applied laterally directed forces over the ischial tuberosity instead of the tuber sacrale (see Fig. 53-7). This procedure incorporates two long lever arms to stress the supporting sacroiliac ligaments. Again caution should be taken not to apply excessive force because of the long lever arm action of the sacral apex and ischial tuberosities on the sacroiliac ligaments. Lastly, proximal hindlimb manipulation is done to assess coxofemoral and stifle joint range of motion and the willingness to actively extend the hindlimb. Concurrent joint stiffness or muscle hypertonicity can be identified and localized as the proximal hindlimb is passively protracted, abducted, flexed, and retracted.

REGIONAL ANALGESIA

Intra-articular analgesia of the sacroiliac joint is nearly impossible because of its deep anatomical location and inaccessible joint capsule. Experimentally, intra-articular sacroiliac injections have been accomplished by drilling a hole through the wing of the ilium, dorsal to the sacroiliac joint.[7] Regional perfusion with local anesthetic solutions or anti-inflammatory drugs for diagnostic or therapeutic purposes has been described. Approaches from the cranial wing of the ilium and the lumbosacral junction are typically used[25]; however, because of the wide overlying wing of the ilium, the proximity and diffusion of the medication near the sacroiliac joint is questionable.

I recommend a caudomedial approach to the sacroiliac joint region because of anatomical and pathological considerations. The caudomedial portion of the sacroiliac joint is the most frequent site of pathological conditions of the sacroiliac joint.[1,4,14] Therefore diagnostic analgesia or therapeutic analgesia or anti-inflammatory drugs should be directed toward the caudal

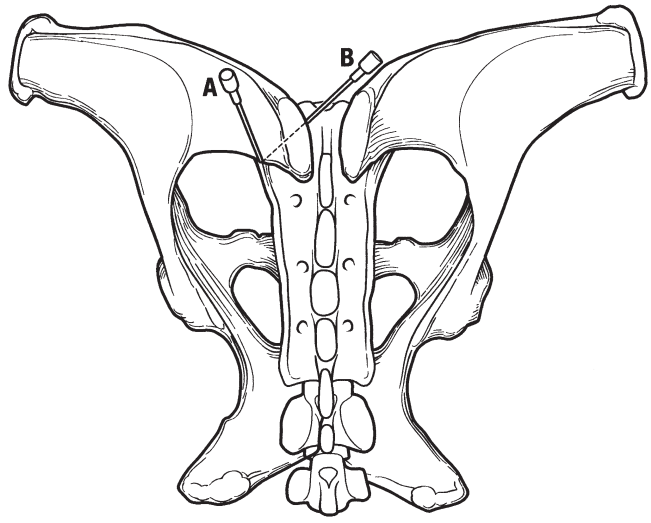

Fig. 53-8 Diagram of two approaches to the sacroiliac joint for diagnostic analgesia and therapeutic analgesia or administration of anti-inflammatory drugs (dorsal view). **A,** Dorsal approach over the ipsilateral iliac wing near the caudomedial aspect of the affected sacroiliac joint. **B,** Medial approach from the cranial aspect of the contralateral tuber sacrale with the needle directed toward the caudomedial aspect of the affected sacroiliac joint.

aspect of the sacroiliac joint in the region of documented pathological condition. Two approaches are described, using 20 to 25 cm long needles, depending on the depth and conformation of the gluteal musculature (Fig. 53-8). Surgical preparation of the injection site is recommended, similar to other joint injection techniques. Ultrasound guidance is also recommended to allow the caudomedial aspect of the sacroiliac joint to be seen and identification of neurovascular structures that must be avoided at the greater sciatic foramen (see Fig. 53-2). The cranial gluteal artery and nerve emerge laterally around the caudal margin of the ilial wing and travel dorsocaudally, adjacent to the sacroiliac joint. Other vital structures, including the sciatic nerve, lie ventral to the sacroiliac joint and travel on or within the substance of the sacrosciatic ligament.

The first approach involves needle placement dorsally over the iliac wing, ipsilateral to the affected sacroiliac joint (Fig. 53-8, *A*). This approach is technically easier, but it potentially has a higher risk of injury to the adjacent neurovascular structures. The needle is directed ventrally, toward the region of the sacroiliac joint, and walked off the caudal border of the iliac wing in the region of the sacroiliac joint. To avoid the deeper neurovascular structures, the needle is not advanced once it is walked off the caudal aspect of the iliac wing. Diffusion of the injected material occurs within the middle or accessory gluteal muscles, near the caudomedial attachments of the ventral sacroiliac ligament. Care must be taken to avoid the adjacent and deeper neurovascular structures mentioned previously.

A second preferred approach involves needle placement at the cranial margin of the contralateral tuber sacrale (Fig. 53-8, *B*). This approach is slightly more technically challenging but has a lower risk of injury to the adjacent neurovascular structures. The needle is inserted along the cranial aspect of the contralateral tuber sacrale and directed toward the cranial edge of the ipsilateral greater trochanter. The needle is placed contralateral to the affected sacroiliac joint, because the needle has to be guided between the divergent sixth lumbar and first sacral dorsal spinous processes and directed into the space formed by the ventral aspect of the ilial wing and the dorsal portion of the sacrum. The needle is advanced

caudolaterally along the medial aspect of the affected ilial wing toward the caudomedial portion of the affected sacroiliac joint until contact is made with bone, near the sacroiliac joint margin. Diffusion of the injected material is expected to occur within the longissimus muscle or interosseous sacroiliac ligament. The dorsal nerve branches of the sacral nerves possibly may also be affected with this approach, as the nerves exit the first and second dorsal sacral foramina.

DIAGNOSTIC IMAGING

Ultrasonography has been used to evaluate the dorsal surface of the iliac wing and caudal margin of the sacroiliac articulation to identify dorsal cortex irregularities associated with incomplete and complete ilial wing fractures.[26,27] The dorsal sacroiliac ligament can be readily imaged on either side of the sacral dorsal spinous processes caudal to the tubera sacrale.[6] Abnormal ultrasound images of the dorsal sacroiliac ligament include enthesopathies at the tubera sacrale attachment sites, hypoechoic changes within the ligament, and modification of fiber orientation.[6,17] The interosseous sacroiliac ligament has not been imaged because of its inaccessible location underneath the ventral aspect of the ilial wing. In contrast, the ventral sacroiliac ligament and the ventral joint margins of the sacroiliac joint can be seen on transrectal ultrasonography.[6] Ventral periarticular remodeling of the sacroiliac joint has been observed with transrectal ultrasound approaches.

Primary indications for radiography of the pelvis include acute or severe pelvic asymmetries, upper hindlimb lameness, and pelvic crepitus or fractures.[28] Ilial wing overlap and the deep anatomical location of the sacroiliac joint make radiographic imaging difficult at best. The radiographic features of chronic sacroiliac joint disease are minimal and include nonspecific increases in the joint space and apparent enlargement of the caudomedial aspect of the sacroiliac joint.[4] Linear tomography has also been used to examine the lumbosacral and sacroiliac regions of horses, but limited access to equipment has restricted its clinical usefulness.[12] Positive findings include widening of the sacroiliac joint and irregular joint outlines, with osteophyte formation at the caudal aspect of the joint. Osseous changes are common bilaterally, but they may be more pronounced on the clinically affected side.[12]

Nuclear scintigraphy is considered by some authors to be an accurate and diagnostically useful technique for identifying acute and chronic sacroiliac joint injuries. Subjective evaluation or quantitative analysis of bone scans typically is able to identify asymmetric radiopharmaceutical uptake over the affected tuber sacrale. However, presumed normal horses, without a history of hindlimb or sacroiliac joint injuries, may also have asymmetrical uptake over the tubera sacrale. A dorsal view of the sacrum is considered the most diagnostic image for evaluating and comparing the sacroiliac joints.[7] However, overlap of uptake in the tubera sacrale and the sacroiliac joints on the dorsal view is usually extensive. Oblique views of the ilial wings are recommended to confirm left to right asymmetries in radiopharmaceutical uptake and to separate the tuber sacrale dorsally from the sacroiliac joint region ventrally.[29] However, oblique views may be difficult to interpret because of inconsistent camera positioning on the left and right sides. The thick overlying gluteal musculature may also attenuate radiopharmaceutical uptake from an affected sacroiliac joint. Stress fractures of the ilial wing may be difficult to differentiate from sacroiliac joint injuries because of the common location and extension of the incomplete fracture line into the sacroiliac joint.[7,22,30]

Thermography has been used to diagnose muscle strain, or inflammation in the region of the sacroiliac and croup regions. Horses with sacroiliac joint injuries are expected to have protective muscle spasms in the adjacent musculature. Palpation of muscle sensitivity has been correlated with abnormal thermographic images in most horses.[31]

DIFFERENTIAL DIAGNOSIS

Causes of sacroiliac joint pain or injury have been postulated to result from sacroiliac or lumbosacral arthrosis, sacroiliac desmitis or sprain, sacroiliac subluxation or luxation, pelvic stress fractures, complete ilial wing fractures, or sacral fractures.[25] Additional differential diagnoses include thrombosis of the caudal aorta or iliac arteries, exertional rhabdomyolysis, trochanteric bursitis, and impinged dorsal spinous processes in the lumbar vertebral region.[20] Horses with presumed thoracolumbar vertebral problems may also have concurrent chronic sacroiliac joint injuries. In a report on 443 horses with back problems, chronic sacroiliac joint problems were identified in 15%.[8] Clinical signs of lower hindlimb lameness may overlap and mimic signs of presumed pathological conditions of the sacroiliac joint. It is important that a thorough and complete lower limb lameness evaluation is completed before, or along with, an upper hindlimb or sacroiliac joint workup.

Based on a review of the literature, osteoarthritis of the sacroiliac joint is the most prevalent disease process affecting horses with sacroiliac joint pain or dysfunction.[1,4,8,14] Sacroiliac desmitis has been documented in horses and may be a significant cause of pathological conditions in horses with acute sacroiliac joint injuries.[6] Documented complete rupture of the sacroiliac ligaments has only been reported in the veterinary literature in a few horses with acute and chronic disease.[14,19] The presumed diagnosis of sacroiliac joint subluxation based solely on tubera sacrale height asymmetry is inappropriate.[24] Horses with chronic sacroiliac problems and presumed sacroiliac joint subluxation have not had identifiable changes in the sacroiliac ligaments at necropsy.[4] In addition, STB trotters with substantial tubera sacrale height asymmetries did not have significant increases in sacroiliac pain compared with horses with lesser degrees of asymmetry.[13] In my opinion, an antemortem diagnosis of sacroiliac joint luxation can only be supported if an acute change in tubera sacrale height caused by substantial trauma has been documented or if sacroiliac joint instability (i.e., crepitus or independent tubera sacrale movement) is evident during physical examination. Pelvic stress fractures also need to be ruled out in horses with sacroiliac pain or dysfunction.[32] A high prevalence of occult pelvic stress fractures has been reported in TB racehorses.[30] The incomplete fracture lines extend into the caudomedial aspect of sacroiliac joint, which could possibly produce concurrent sacroiliac joint inflammation and osteoarthritis. A diagnosis of sacroiliac joint injury is often based on a diagnosis of exclusion because of difficulties in clinical evaluation and diagnostic imaging. We are hopeful future investigations into sacroiliac joint problems in horses will produce a better and more comprehensive understanding of this often misdiagnosed clinical entity.

TREATMENT

Because definitive diagnosis of pathological conditions of the sacroiliac joint is difficult, treatment recommendations are usually symptomatic. Few studies on treatment efficacy have been reported in the equine literature. In general, rest and various forms of physical therapy are indicated for ligamentous injuries. Prolonged rest (6 to 12 months) and systemic anti-inflammatory medications have been prescribed for horses with acute and chronic sacroiliac joint injuries. Complete box stall rest for 30 to 45 days has been recommended to support ligamentous healing in horses with acute injuries.[20] The local injec-

tion of irritants or sclerosing agents have also been suggested to stimulate fibrosis and subsequent sacroiliac joint stability.[20] In my opinion, no scientific support or clinical indication exists for such proposed treatments of back or sacroiliac joint problems. With such haphazard and potentially injurious treatment modalities there appears to be a better chance of inducing further injury, rather than of stimulating any healing response. Treatment of chronic sacroiliac joint injury typically focuses on a gradual return to a low level of exercise to maintain muscle development of the back and gluteal regions to counteract the clinical signs of poor performance and reduced hindlimb impulsion.[23] Extended rest is contraindicated because reduced muscle tone may exacerbate the injury.

Because of the deep and inaccessible location of the sacroiliac joint, intra-articular injections of analgesic or anti-inflammatory medications for diagnostic or therapeutic purposes are impractical.[7] However, regional perfusion of the sacroiliac joint with local anesthetic solutions or anti-inflammatory drugs for diagnostic and therapeutic purposes is a viable alternative.[25] Concurrent lameness of the ipsilateral lower hindlimb has been reported in horses with signs of chronic sacroiliac joint injuries.[7] Therefore a detailed lameness examination coupled with appropriate treatment of the pathological conditions of the lower hindlimb is often indicated. Additional factors such as corrective shoeing and modifications in exercise or training programs also need to be addressed. Acupuncture and chiropractic are non-traditional approaches that have been used by some clinicians to assist in the diagnosis and symptomatic treatment of horses with presumed sacroiliac joint problems. Additional research into these modalities needs to be conducted before further recommendations can be made about their efficacy for horses with documented sacroiliac joint pathology.

PROGNOSIS

Long-term follow-up suggests that prognosis for horses with sacroiliac joint injury is poor for return to the previous level of activity.[7] Some horses may have an improvement in performance or lameness but will not be able to return to normal athletic activities because of recurring, low-grade lameness. Most horses will be pasture sound or able to function only at low levels of exercise. We are hopeful that improvements in providing a specific diagnosis of the type of sacroiliac joint injury present will provide affected horses with better and more specific treatment options in the future.

ACKNOWLEDGMENT

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CHAPTER • 54

Thoracolumbar Spine

Jean-Marie Denoix and Sue J. Dyson

Back problems are a major cause of altered gait or performance (see Chapters 52, 53, and 100). Identification and documentation of vertebral lesions is difficult in horses; therefore treating back pain is a real challenge for the equine clinician. The equine back is a large area covered by thick muscles. Assessment of the bony elements is therefore limited. Every joint in the thoracolumbar region has restricted mobility, and therefore detecting changes in restricted movement when the horse is working is difficult. Diagnostic imaging of the thoracolumbar region is also limited, and radiographic assessment requires special equipment. Specific treatment of back pain can only be performed after complete identification of the site and nature of the lesions.

ANATOMY AND FUNCTION

Bones

The thoracolumbar spine is basically composed of 18 thoracic vertebrae (T1 to T18), 6 lumbar vertebrae (L1 to L6), and 5 fused sacral vertebrae (S1 to S5). Some horses have individual (congenital) variations at the cervicothoracic, thoracolumbar, and lumbosacral junctions. The most common is the sacralization of the last lumbar vertebra (sacralization of L6), which can be seen by ultrasonography. Transitional vertebrae, with a rib on one side and a transverse process on the other side, can be found at the thoracolumbar junction. Intervertebral ankylosis alters the biomechanical behavior of the involved part of the spine, especially at the lumbosacral junction, which in normal horses is the most mobile joint between T2 and S1. Ankylosis of this joint puts more stresses on the caudal lumbar intervertebral joints and may predispose to osteoarthritis of the facet joints and intervertebral disk lesions. Fusion of the lumbar transverse processes (see the following discussion) is also sometimes seen, and this has similar consequences on adjacent intervertebral joints, but the consequences are fewer because little movement usually occurs in these joints. Transitional transverse processes, or ribs that do not involve the vertebral body, have less biomechanical significance.

The first 10 thoracic vertebrae have long spinous processes. These have a dorsocaudal orientation and provide insertion for the strong but elastic supraspinous ligament. The anticlinal vertebra, the vertebra with a spinous process perpendicular to the vertebral axis, is usually T15. Caudal to the anticlinal vertebra the spinous processes are orientated obliquely dorsocranially. The spinous processes are higher between L2 and L5. Therefore lumbar muscular atrophy results in a kyphotic appearance of the lumbar area.

In most horses the spinous processes of L6 and S1 are divergent, allowing a range of flexion and extension movements at the lumbosacral joint. The transverse processes from L5 (sometimes L4) to S1 articulate through inter-transverse synovial joints, which limit lateral flexion in this area. The lumbar vertebral bodies are bigger than the thoracic ones and have a ventral crest for the insertion of the diaphragm.

Joints

Stability of the thoracolumbar vertebrae is provided by the supraspinous and interspinous ligaments, the joints between the cranial and caudal articular processes (the facet joints), the joints between the vertebral bodies, and the dorsal and ventral longitudinal ligaments. Stability of the spinous processes is provided by the supraspinous and interspinous ligaments (Fig. 54-1). These ligaments are wider and more elastic in the cranial and middle thoracic areas, permitting more movement than in the caudal thoracic and lumbar regions.

The caudal and cranial articular processes articulate via synovial intervertebral articulations (the facet joints). At the base of the spinous processes these joints are symmetrically placed on each side of the median plane (Fig. 54-2). These are

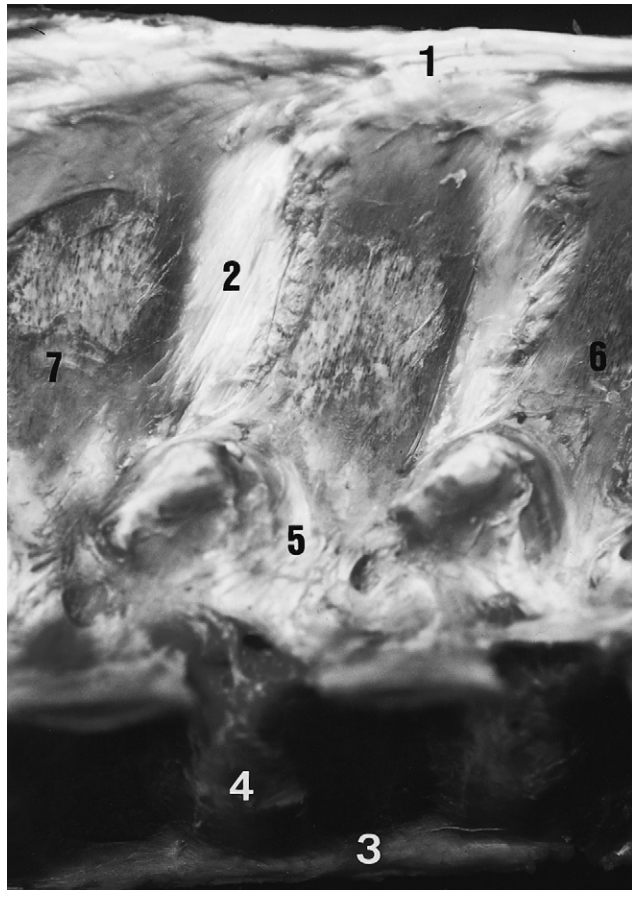

Fig. 54-1 Anatomical specimen showing the ligaments of the thoracolumbar spine. *1*, Supraspinous ligament; *2*, interspinous ligament; *3*, ventral longitudinal ligament; *4*, fibrous superficial part of the intervertebral disk; *5*, articular capsule of the synovial intervertebral joint; *6*, first lumbar vertebra; *7*, third lumbar vertebra.

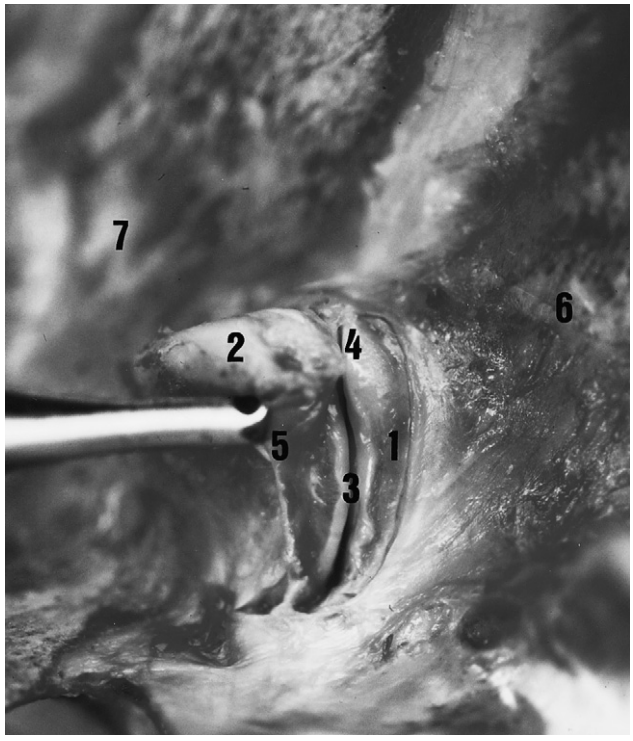

Fig. 54-2 Anatomical specimen showing a synovial intervertebral articulation. *1*, Caudal articular process; *2*, cranial articular process; *3*, joint space; *4*, articular margin; *5*, articular capsule and synovial membrane (reflected); *6*, first lumbar vertebra; *7*, second lumbar vertebra.

typical synovial (diarthrodial) joints with articular cartilage, a closed synovial cavity containing synovial fluid, a synovial membrane, and a fibrous capsule. They have a single flat articular facet in the cranial thoracic area (up to T12) and two angulated articular facets between T12 and T16. From T17 to S1 the articular facets are congruent, with a cylindrical shape aligned on a paramedian axis. These regional variations are correlated with the limited mobility of the lumbar spine and the wider range of movement in the thoracic region, including flexion and extension in the median plane, lateral flexion in the horizontal plane and rotation. The vertebral bodies are stabilized by joints composed of a fibrous intervertebral disk and two longitudinal ligaments. The ventral longitudinal ligament is replaced by the longissimus cervicis muscle in the cranial thoracic area. The dorsal longitudinal ligament is located in the vertebral canal and adherent to the dorsal border of each intervertebral disk.

Muscles

The vertebral column is moved by wide muscles (Fig. 54-3). The strong epaxial muscles, located dorsal to the vertebral axis, have an extensor effect when contracted bilaterally. Unilateral contraction induces lateral flexion and contributes to rotation of the vertebral column. Electromyographic studies show that the epaxial muscles limit flexion and stabilize the vertebral column during the suspension phase at the trot.[1,2]

The epaxial muscles include the following:

1. Spinosus muscle, inserting on the spinous processes
2. Longissimus dorsi muscle, which extends to the caudal cervical spine
3. Iliocostalis muscle

The longissimus dorsi is the strongest muscle. The iliocostalis muscles are small but have a greater role in lateral flexion because of their eccentric location. Caudally, these muscles

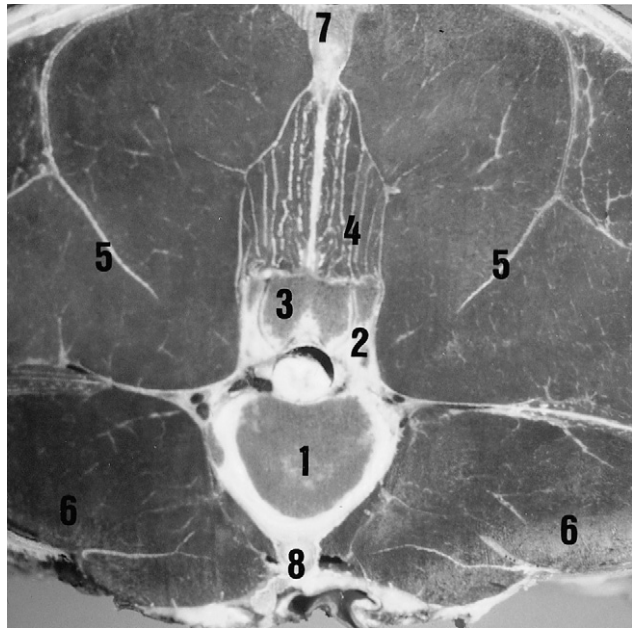

Fig. 54-3 Transverse anatomical section of the normal lumbar region of 6-year-old Selle Français mare. *1*, Vertebral head of third lumbar vertebra; *2*, cranial articular process of third lumbar vertebra; *3*, caudal articular process of second lumbar vertebra; *4*, multifidus muscle; *5*, erector spinae muscle; *6*, psaos muscles; *7*, supraspinous ligament; *8*, ventral longitudinal ligament.

fuse to form the erector spinae muscle. The multifidus muscle lies under the spinosus muscle and is in close contact with the vertebrae (juxtavertebral muscle). This muscle plays a major role in the stability of the vertebrae and in the proprioceptive adjustment of the spine.

The hypaxial muscles, located ventral to the vertebral axis, have a flexor effect on the spine when contracted bilaterally. Unilateral contraction induces lateral flexion and contributes to rotation of the vertebral column. The hypaxial muscles include psoas minor and major, rectus abdominis, and rectus oblique.

The psoas minor and major muscles insert on the ventral aspect of the lumbar and caudal three thoracic vertebrae (juxtavertebral muscles). They act mainly at the lumbosacral junction but are also able to flex the thoracolumbar junction and the lumbar spine. The rectus abdominis muscle is an effective flexor of the complete thoracolumbar spine because of its eccentric insertions on the pubis, sternum, and ventral part of the ribs. The oblique muscles can create lateral flexion and rotation of the thoracolumbar spine because of eccentric insertions on the tubera coxae and ribs. Electromyographic studies show that the rectus abdominis muscles act to limit extension and stabilize the vertebral column during each diagonal stance phase at the trot.[1,2]

Blood Vessels and Nerves

The vertebrae, intervertebral joints, and axial muscles are supplied by segmental thoracic and lumbar arteries and veins. Innervation is provided by thoracic and lumbar spinal nerves that pass through the intervertebral foramina.

DIAGNOSIS OF BACK PAIN

The objectives of clinical examination of the horse's back[3-5] are to determine if back pain is present, the site or sites of pain, and the potential lesions responsible for the pain. Acute back

pain can arise after traumatic injuries, such as a fall, or after an awkward jump. Acute pain and muscle spasm may be identified and may be the primary lesion, but a complete radiographic examination of the thoracolumbar spine may be indicated to assess for vertebral fractures or long-standing bony lesions that may be responsible for the accident. These lesions may become clinically significant because of over-stress on the intervertebral structures. For example, long-standing spondylosis lesion may cause acute pain on landing after a jump.

A poor rider or an ill-fitting saddle may cause back pain, but correction of these problems may not be easy. It is important to manage these causes of back pain before proceeding with more advanced imaging techniques.

Back pain may also be manifest by abnormal behavior, for example, bucking (see page 829).

Physical Examination

Physical examination is essential in diagnosing back pain. Only the main criteria for each step or procedure are discussed. It is obviously important to perform a comprehensive evaluation of the whole horse to identify any other potential problems that may be contributing to gait abnormalities or poor performance.

Inspection

The most commonly described abnormal curvatures of the back are lumbar kyphosis and thoracic lordosis. These can be seen in the same horse. Atrophy of the epaxial muscles in the lumbosacral regions results in prominence of the normal summits of the spinous processes and apparent kyphosis of the lumbar spine. Detection of atrophy of the epaxial muscles is a key finding in a horse with potential back pain, because atrophy reflects the reduction of movement in the painful areas. The finding can help provide information on the possible location of the lesions. However, one should bear in mind that muscular development also reflects the horse's previous work history, and if the horse has never worked properly through its back, the epaxial muscles will be poorly developed.

Thoracic lordosis may be seen in clinically normal horses and does not necessarily imply the presence of back pain. However, a short coupled horse with lordosis is more likely to have impinging spinous processes, a reflection of the horse's normal conformation.

Abnormal swellings in the saddle area or abnormal hair loss may reflect a poorly fitting saddle or the position and balance of the rider. A rider that sits crookedly predisposes to excessive movement of the saddle.

Palpation

Palpation of the thoracolumbar region should be performed with the horse standing quietly, bearing weight evenly on all limbs. Time should be spent getting the horse accustomed to the clinician's presence, especially with an apprehensive horse, so that the horse's true reactions to pain and pressure can be assessed. A thin-skinned Thoroughbred (TB)-type may give a false impression of guarding the back and having protective spasm of the epaxial muscles, unless approached quietly.

Palpation of the superficial structures of the thoracolumbar region helps to identify supraspinous desmopathy and deformation or malalignment of the spinous processes. Identification of localized muscle tension can be a key feature in establishing the presence of a significant lesion. The thickness of the epaxial muscles prohibits accurate assessment of deeper structures.

Pressure

Pressure on the superficial structures such as the supraspinous ligament and the epaxial muscles is useful to assess pain. Reliably assessing the response to pressure applied to deeper structures, such as the epaxial synovial intervertebral articulation complexes, is not possible.

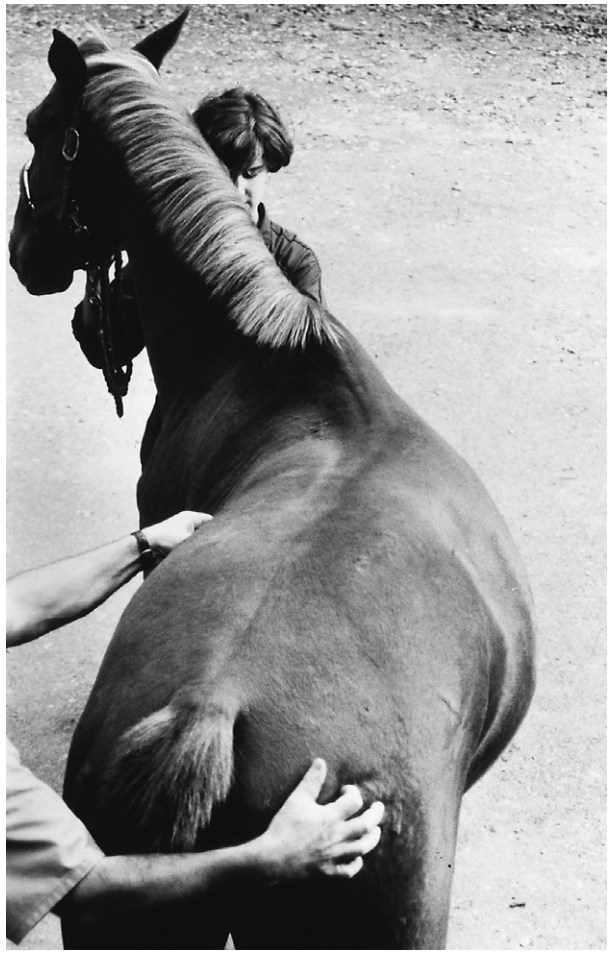

Fig. 54-4 Physical examination by left lateral flexion of the thoracolumbar spine. The two main criteria evaluated are the amount of movement and manifestations of pain.

Mobilization

Stimulation of movement of the thoracolumbar spine (Fig. 54-4) is important to assess the amount of movement tolerated by the patient and any signs of pain, such as flexion of the limbs, alteration of facial expression, tension of the back muscles, movement of the tail, and alteration of behavior (kicking, rearing, bucking, and grunting).

The following protocol is recommended:

1. Assessment of thoracic flexion, thoracic extension, thoracolumbar extension, lumbosacral extension, and complete thoracolumbar and lumbosacral flexion in the median (sagittal) plane
2. Assessment of right and left thoracolumbar lateral flexion (and rotation) (see Fig. 54-4)
3. Assessment of left and right cervical (and thoracic) lateral flexion (and rotation)

The clinician should try to determine which movements are restricted or not tolerated so as to determine potential sources of pain. These movements can be induced by skin stimulation of the dorsal and lateral aspects of the trunk and hindquarters. Although some horses respond to soft digital stimulation, in others a stronger stimulus is required, for example, using the tips of a pair of artery forceps. Firm stroking with a hard instrument displaced craniocaudally and inducing spectacular wide extension and flexion movements may lack sensitivity and specificity in determining the site of back pain, but firm stroking may be necessary in extremely stoical cob-type horses to induce any movement. A normal, relaxed horse is

able to flex and extend the thoracolumbar spine smoothly and repeatedly. The degree of movement reflects in part the type of horse. Cob-type animals naturally tend to have much more restricted movement than TBs or Warmbloods. The clinician will find it useful to keep one hand resting on the mid-back region during these maneuvers to detect induced muscle spasm or abnormal cracking of the muscles or ligaments (a crepitus-like feeling as the epaxial muscles or ligaments contract and relax).

Examination During Movement

Evaluation of the horse moving at walk, trot, and canter is essential to assess if pain is present and to identify functional disorders, such as limitation of regional intervertebral mobility (Fig. 54-5). The clinician should always bear in mind that impinging spinous processes can be present asymptomatically. Therefore the significance of impinging spinous processes should not be over-interpreted, unless clinical signs of back pain are evident. The horse should be assessed moving in straight lines and in small circles at a walk and trot on a hard surface and moving at a trot and canter on the lunge to determine if any reduction of back mobility is apparent (Table 54-1).

In vivo kinematic studies have quantified dorsoventral flexibility of the back in sound horses trotting[6,7] and at various gaits on the treadmill.[8] Horses with vertebral lesions showed a reduction of passive flexibility of the back at trot,[9] with reduced flexion and extension, lateral flexion, and rotation. Thus the horse may appear to hold its back rather stiffly. This can be caused by mechanical problems (partial or complete ankylosis) or pain. Back pain may also influence stride length and limb flight, resulting in a more restricted and less animated gait. On the lunge the horse may show loss of balance and a tendency to lean the body rather than bend the trunk toward the direction of circle.

Examination of the Horse Being Ridden or in Harness

The presence and the degree of back pain may be underestimated unless the horse is evaluated under its normal working conditions, that is, ridden or in harness. The clinician should watch carefully as the tack or harness is applied, particularly as the girth is tightened. However, the clinician should bear in mind that cold-back behavior (see page 829) is not necessarily a reflection of back pain, although it may be. The fit of the saddle for the horse and the rider should be evaluated. Back mobility,[3] the movements that the horse finds difficult, and the horse's attitude toward work should all be assessed (see page 830).The clinician should pay attention also to the observations of the rider, because the horse may feel considerably worse than it appears. The rider may describe lack of hindlimb power, lateral stiffness of the back to the left or right, unwillingness of the horse properly to take the bit, or loss of fluidity in the paces. The rider may complain of back pain induced by riding the horse.

Fig. 54-5 Dynamic examination by evaluating the active back movement at the canter on the lunge.

Table • **54-1**

Criteria Used for Evaluating Back Disorders*

GROUND	GAIT	DIRECTION OF MOVEMENT	CRITERIA
Hard	Walk	Straight line	Rotation (tuber coxae mobility) (lateral flexion)
		Figure eight (3 m diameter)	Lateral flexion
	Trot	Left circle (7 m diameter)	Lateral flexion
		Right circle (7 m diameter)	(Passive TL flexibility)
Soft	Trot	Right circle (10 m diameter)	Passive DV flexibility (TL>LS)
			Lateral flexion
			Hindlimb propulsion
	Canter	Right circle (10 m diameter)	Active flexion and extension movements (LS>TL)
			Hindlimb protraction/propulsion
			Hindlimb placement (rotary canter)
			Coordination and balance
	Trot Canter	Left circle (10 m diameter)	Same as above
Hard	Trot	Left circle (7 m diameter)	Lateral flexion
		Right circle (7 m diameter)	(Passive TL flexibility)
		Straight line	Passive DV flexibility (TL>LS): side aspect
			Rotation (tuber coxae mobility); lateral flexion: caudal aspect

TL, Thoracolumbar; *LS*, lumbosacral; *DV*, dorsoventral.
*Assessment involves a horse moving first on a hard surface, then on a soft surface, and then on a hard surface, in straight lines and circles, at walk, trot, and canter.

Examination of the horse while it is ridden also gives the clinician the opportunity to assess the rider, because back pain is readily induced by poor riding, a situation which may not be easy to handle diplomatically.

Local Analgesia

Diagnostic infiltration of local anesthetic solution may be useful to assess the clinical significance of impingement of spinous processes, where only the bones, ligaments, and adjacent muscles are affected by the analgesia. If impingement is severe, local anesthetic solution can only be deposited around the spinous processes and not between them. The volume of local anesthetic solution required depends on the number of spinous processes involved. Sixty to 80 ml of mepivacaine is injected at several sites using 4-cm needles if 4 to 5 impinging spinous processes exist. The response is assessed in 15 to 20 minutes and is most accurately evaluated by observing the horse while it is ridden and by the rider's feel. If kissing spines is the only significant lesion, then significant improvement should be anticipated, but if other lesions are contributing to pain, the response is limited.

If deeper injections are performed in the region of the epaxial synovial intervertebral articulations, interpretation is confounded, because the injections are effectively intramuscular injections, and the local anesthetic solution may readily diffuse to affect sites on the dorsal and ventral rami of the spinal nerves. Thus local analgesia has little value for the assessment of osteoarthritis of the synovial intervertebral articulation, intervertebral disk disease, and spondylosis.

IMAGING

Radiography and ultrasonography are essential to determine potential causes of pain or mechanical restriction. Nuclear scintigraphy is also a useful tool to detect abnormal bone activity and to help establish the clinical significance of radiographic findings.

Radiography

Indications for the radiographic examination of the thoracolumbar spine include owner complaints of a back problem in their horse, abnormal clinical findings, poor performance, or as part of the assessment of an obscure lameness.

The horse should stand squarely on all limbs.[10] Sedation may be necessary using detomidine (3 to 7 mg) or romifidine (12 to 25 mg). A high-output x-ray machine is necessary, with a rotating anode and an 80-kW power generator, for example.

Cassettes (20 × 40-cm format) with fast screens and sensitive or high-sensitive films are used to provide highly sensitive film-screen combinations. The cassettes should be placed in a vertically orientated ceiling or wall-mounted cassette-holder, which can be aligned with the x-ray machine. The cassette should be placed as close to the trunk of the horse as possible. A 10:1 ratio focused grid is used to reduce radiation scatter. The focus-film distance varies between 1.15 and 1.30 m. The x-ray beam is directed perpendicular to the vertebral column axis. Five exposures are required (Fig. 54-6) to obtain a complete evaluation of the thoracolumbar spine between T10 and L4. In some horses L5 may also be imaged. The exposure parameters are summarized in Table 54-2. To reduce motion, the exposure is performed during the last part of the expiration.

Ultrasonography

Ultrasonographic examination of all the epaxial structures is possible.[11,12] Imaging of the spinous processes and associated ligaments is performed with 7.5-MHz or 10-MHz probes. A standoff pad is used to improve detection of the superficial structures. Imaging of the articular and transverse processes

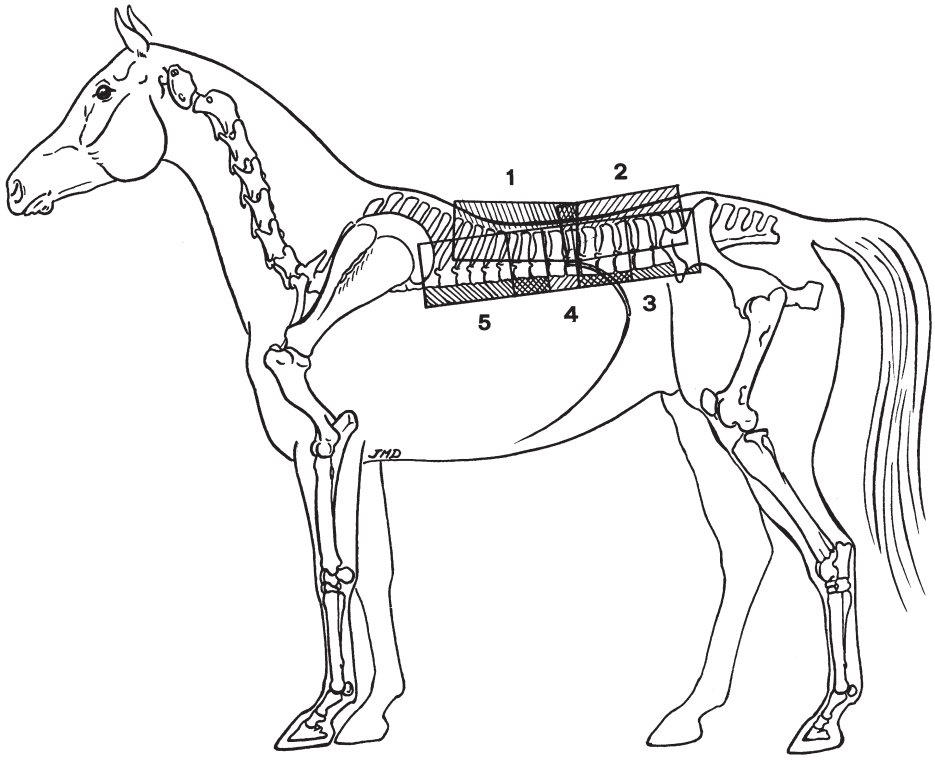

Fig. 54-6 Position of the cassettes for radiographic examination of the thoracolumbar spine: *1*, Thoracic spinous processes; *2*, lumbar spinous processes; *3*, lumbar vertebral bodies and synovial intervertebral articulation; *4*, thoracolumbar vertebral bodies and synovial intervertebral articulation; *5*, thoracic vertebral bodies and synovial intervertebral articulation.

Table • **54-2**

Technical Parameters Used for the Exposure of Different Regions of the Thoracolumbar Spine*

REGIONS	FILM	kV	MA	EXPOSURE DURATION (msec)
Thoracic dorsal spinous processes	Sensitive	73-77	80-110	71-107
Lumbar dorsal spinous processes	Sensitive	77-83	110-160	110-182
Mid-thoracic region, vertebral bodies, and facet joints (T10-T16)	High sensitive	70-77	100-140	86.5-141
Thoracolumbar junction, vertebral bodies, and facet joints (T15-L2)	High sensitive	81-85	160-250	177-344
Lumbar region, vertebral bodies, and facet joints (T18-L4)	High sensitive	85-93	280-320	406-580

*All examinations performed using a 10:1 grid and rare earth fast screen. Horses of 450 to 650 kg body weight.

can be performed with 5- to 2.5-MHz probes. Longitudinal median and paramedian scans and transverse scans are combined to image all dorsal vertebral structures. For ultrasonographic examination the hair on the median plane over the spinous processes is removed with a No. 40 clipper blade and the skin is cleaned. The skin is never shaved. When the hair is short (during the summer time) wetting the hair and skin with hot water for several minutes allows examination without clipping. Aqueous contact transmission gel is applied to the wet skin to couple the transducer to the skin to avoid air interference and artifacts.

Scintigraphy

Nuclear scintigraphy is used to detect lesions in the thoracolumbar spine. In a retrospective study of 50 horses the most common abnormal finding was abnormal radiopharmaceutical uptake in the spinous processes between T12 and L2, identified in 48 horses.[13] Three horses had abnormal radiopharmaceutical uptake in the vertebral bodies of L3 to L6 and T11 to T14.

Lateral or slightly oblique lateral images give the most information. Scattered radiation is a problem because of the large muscle mass overlying the vertebrae, so use of a high-resolution collimator is preferred. This requires longer acquisition times to obtain an adequate number of counts to give adequate image quality. Slight swaying movement of the horse during image acquisition and the movement of breathing detract from image quality unless motion-correction software is used. The gamma camera may need to be angled slightly to avoid the kidneys being superimposed over the caudal thoracic vertebrae. At least three images are required to evaluate the entire length of the thoracolumbar spine, and comparable images from the left and right sides can be useful. The summits of the spinous processes in the withers region, the caudoproximal aspect of the scapula, and the kidneys may all have greater radiopharmaceutical uptake than the rest of the thoracolumbar spine and may effectively conceal lesions (Fig. 54-7). Masking these areas out after image acquisition potentially results in more information. Using a high-frequency filter can also yield additional information. Image resolution in fat horses may be compromised by scattered radiation in the soft tissues.

Thermography

Thermography may be useful in identifying acute superficial muscle injuries or demonstrating to an owner the impact of a poorly fitting saddle or a rider sitting crookedly. However, in horses with chronic back pain, thermography does not usually add useful information and lacks sensitivity and specificity.

LESIONS

Spinal Processes and Associated Ligaments

Impingement of the Dorsal Spinous Processes: Kissing Spines

Impingement of the summits of the spinous processes, or kissing spines, is a well-known pathological entity of the horse's back,[14] but it is important to recognize that lesions may not be restricted to the summits. The most common location of these lesions is the vertebral segment between T10 and T18 (Fig. 54-8), although lesions do also occur between L1 and L6.[10] Remodeling of the dorsal aspect of a spinous process or an avulsion fracture reflects an insertional lesion of the supraspinous ligament. We grade impingement of the dorsal part of the spinous processes as follows:

- Grade 1: Narrowing of the interspinous space with mild sclerosis of the cortical margins of the spinous processes
- Grade 2: Loss of the interspinous space with moderate sclerosis of the cortical margins of the spinous processes
- Grade 3: Severe sclerosis of the cortical margins of the spinous processes, caused in part by transverse thickening, or radiolucent areas
- Grade 4: Severe sclerosis of the cortical margins, osteolysis, and change in shape of the spinous processes

Over-riding of spinous processes is a congenital condition with abnormal orientation of the spinous processes and similar changes in the margins of adjacent spinous processes. The contour of the middle part of a spinous process may be altered because of enthesophyte formation at the insertion of the interspinous ligaments. Kissing lesions may also be seen at the ventral part of a spinous process with type 4 lesions of the epaxial synovial intervertebral articulation (see page 518).

Ultrasonographic examination can easily demonstrate contact or remodeling between two adjacent spinous processes, transverse thickening of the processes, and abnormal alignment. However, ultrasonography is more useful to assess concomitant supraspinous ligament lesions, including insertion desmopathies (enthesopathies) on the summits of the spinous processes.

Nuclear scintigraphy can help identify evidence of active bone modeling of the spinous processes and other potential lesions. However, one must bear in mind that active bone modeling is not synonymous with pain. Increased modeling of the dorsal aspects of the spinous processes may reflect impingement or insertional desmopathy of the supraspinous ligament (Fig. 54-9). The degree of increased radiopharmaceutical uptake does not always appear to be well correlated

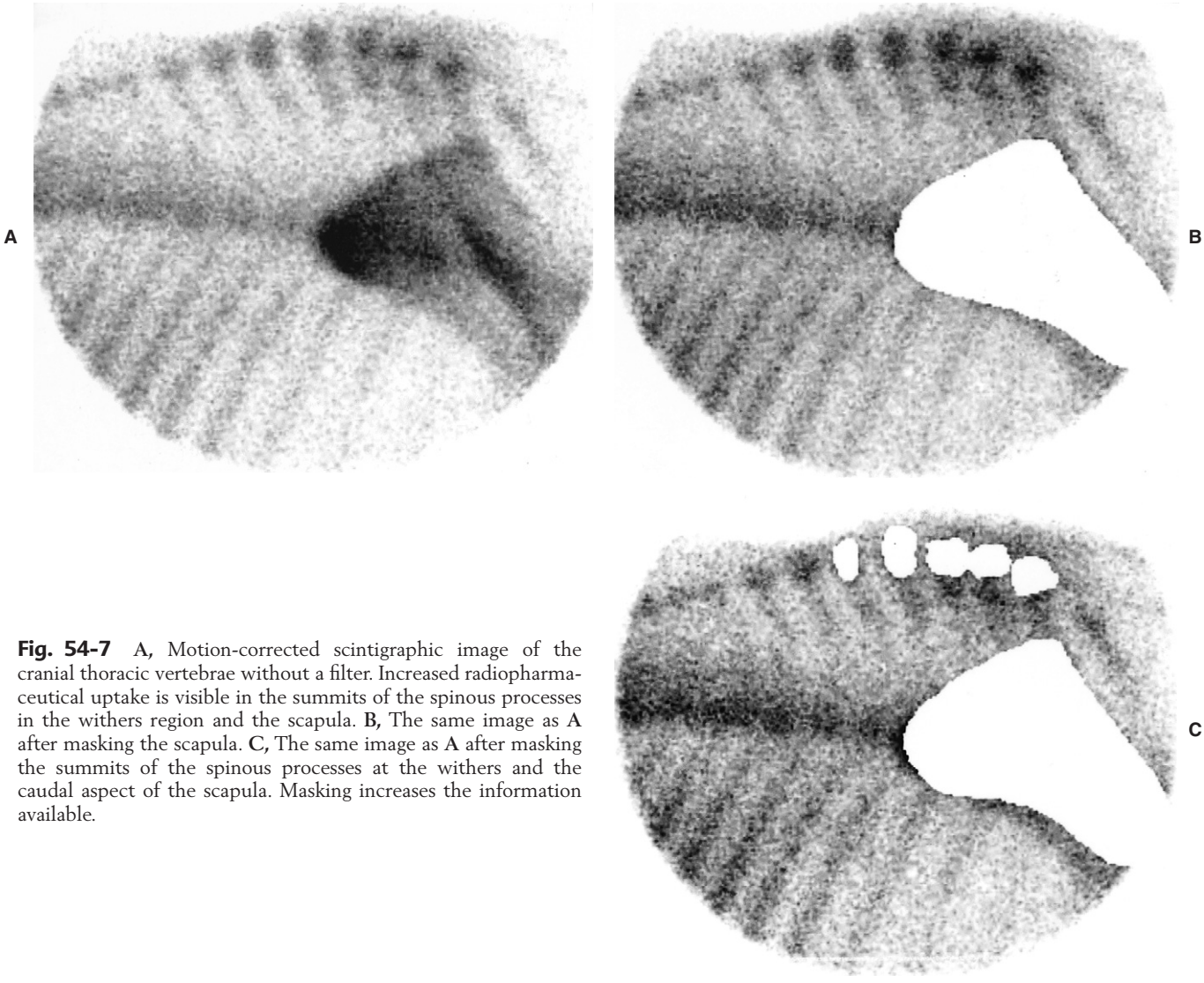

Fig. 54-7 **A,** Motion-corrected scintigraphic image of the cranial thoracic vertebrae without a filter. Increased radiopharmaceutical uptake is visible in the summits of the spinous processes in the withers region and the scapula. **B,** The same image as **A** after masking the scapula. **C,** The same image as **A** after masking the summits of the spinous processes at the withers and the caudal aspect of the scapula. Masking increases the information available.

with the severity of the clinical signs or the radiological abnormalities (see Fig. 54-9, *A*). Obvious focal increased radiopharmaceutical uptake (IRU) can be seen in horses with neither back pain nor radiological abnormality, whereas some horses with back pain associated with impinging spinous processes have only mild IRU. Mechanical limitations may contribute to back stiffness.

Kissing spines can be found in performance horses with no clinical manifestations of back pain. Therefore the clinical significance of kissing spines must be carefully assessed. Kissing spines may also be present with another vertebral lesion, which may have a greater influence on prognosis; therefore a comprehensive evaluation of all structures should be performed, even if kissing spines have been identified. Radiographic findings should be carefully correlated with the results of the physical examination and preferably with nuclear scintigraphic examination and the response to local analgesia.

Desmopathies: Supraspinous Ligament Injuries

Injuries of the supraspinous ligament occur most commonly between T15 and L3 and may be associated with palpable

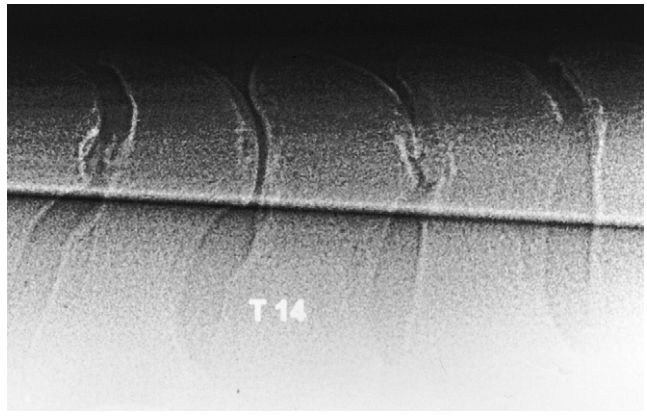

Fig. 54-8 Lateral radiographic view of the spinous processes of the twelfth to sixteenth thoracic vertebrae. Cranial is to the left. There are radiographic signs of impingement of the spinous processes between the eleventh and sixteenth thoracic vertebrae, with significant marginal bone remodeling. *T14,* Fourteenth thoracic vertebra.

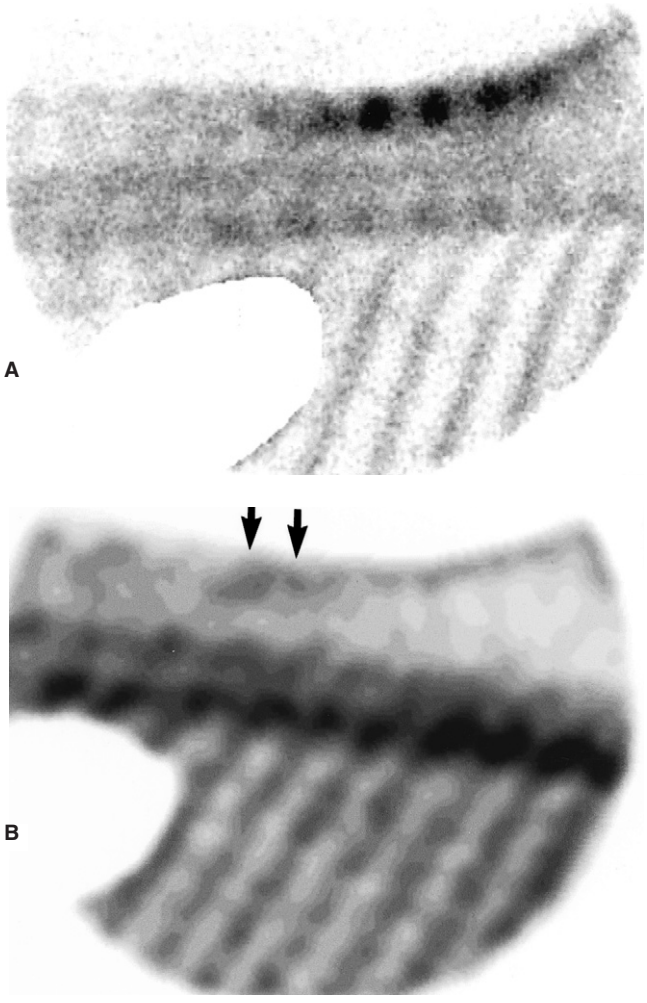

Fig. 54-9 **A,** Lateral nuclear scintigraphic image of the thoracolumbar region of 10-year-old Grand Prix dressage horse with loss of action. Increased radiopharmaceutical uptake is visible in the dorsal aspects of six adjacent spinous processes. The horse showed clinical signs of restricted mobility of the back. Radiographic evidence showed impinging spinous processes. The horse's gait was dramatically improved by infiltration of local anesthetic solution around the affected spinous processes. **B,** Lateral nuclear scintigraphic image of the thoracic region of 8-year-old show jumper gelding with stiffness of the thoracolumbar region and poor hindlimb impulsion. A high-resolution filter has been applied to the image. Only slight radiopharmaceutical uptake is visible in the summits of two spinous processes in the caudal thoracic region (*arrows*). However, five adjacent spinous processes were impinging, and infiltration of local anesthetic solution produced a profound clinical improvement. No soft tissue lesions were identified by ultrasonography.

localized thickening and pain. Lesions are identified best by ultrasonography.[11,12]

Thickening of the supraspinous ligament induces a local deformation (bump) of the dorsal profile of the thoracolumbar area, which can be measured by ultrasonography and compared with two adjacent equivalent locations. Lesions usually occur at a spinous process, sometimes extending between two adjacent spinous processes. The normal supraspinous ligament is uniformly echogenic (Fig. 54-10). Hypoechogenic lesions in the deep and intermediate part of the supraspinous ligament (Fig. 54-11) are compatible with recent or chronic desmopathies of the supraspinous ligament. Hyperechogenic foci,

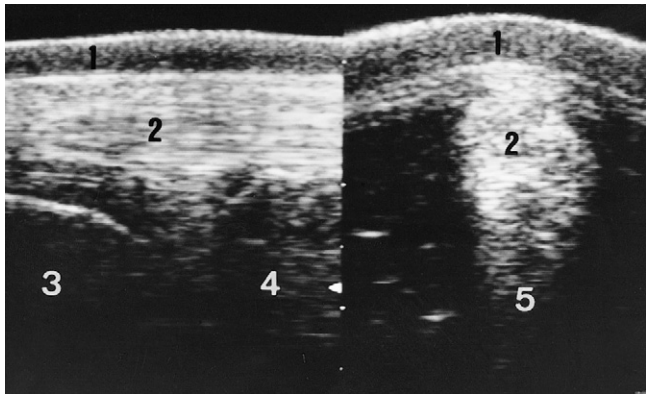

Fig. 54-10 Median (*left*) and transverse (*right*) ultrasound scans of the normal supraspinous ligament in the lumbar area. *1*, Skin; *2*, supraspinous ligament; *3*, second lumbar vertebra; *4*, third lumbar vertebra; *5*, interspinous ligament.

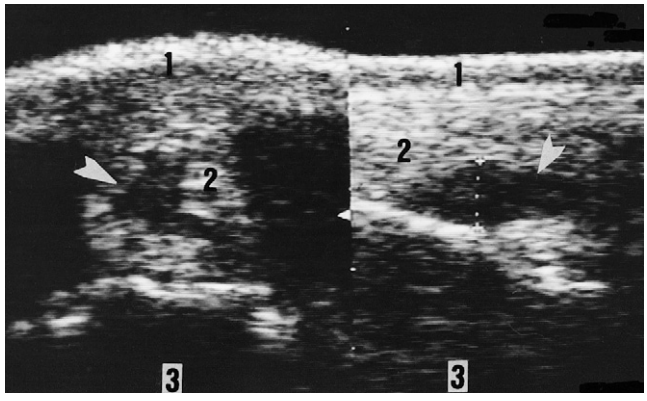

Fig. 54-11 Transverse (*left*) and median (*right*) ultrasound scans indicate supraspinous desmopathy. The ligament is thickened and presents a central hypoechoic zone (*arrowheads*). *1*, Skin; *2*, supraspinous ligament; *3*, seventeenth thoracic spinous process.

with or without acoustic shadows, may be seen in the supraspinous ligament. They reflect chronic or old desmopathy. Insertion desmopathy (enthesopathy) of the supraspinous ligament can be identified by irregularity of the surface of the spinous process, with a thicker appearance. Alteration of echogenicity and fiber orientation occurs in the supraspinous ligament.

With low-exposure radiographs, the soft tissue thickening and some focal radiopacities may be seen in long-standing injuries. Avulsion fractures or bone remodeling and sclerosis of the dorsal surface of the spinous processes may also be noted.[15]

Nuclear scintigraphic examination reveals increased radiopharmaceutical uptake in the summit of one or more spinous processes in association with insertional lesions of the supraspinous ligament.

Articular Processes: Synovial Intervertebral Articulations

Normal References

The articular processes synovial intervertebral articulation complex is located dorsal to the vertebral canal and is composed of the caudal articular process of one vertebra, the joint space, and the cranial articular process of the following vertebra (Figs. 54-12 to 54-14).[10]

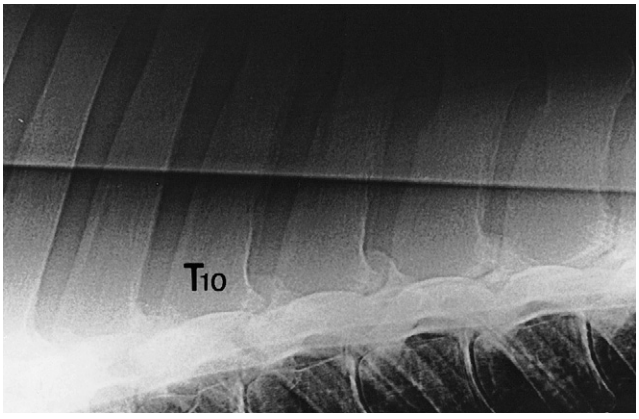

Fig. 54-12 Normal radiographic appearance of the synovial intervertebral articulations in the mid-thoracic area. Cranial is to the left. T_{10}, Tenth thoracic verterbra.

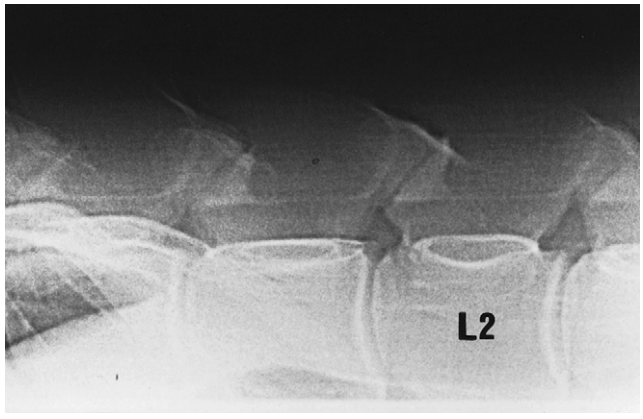

Fig. 54-14 Normal radiographic appearance of the synovial intervertebral articulations in the lumbar area. Cranial is to the left. *L2*, Second lumbar vertebra.

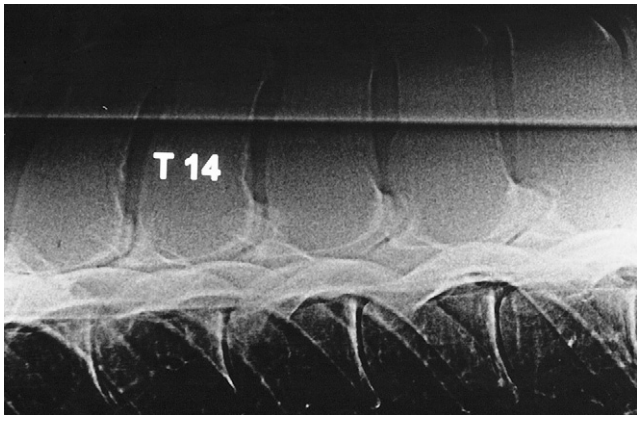

Fig. 54-13 Normal radiographic appearance of the synovial intervertebral articulations in the caudal thoracic area. Cranial is to the left. *T14*, Fourteenth thoracic vertebra.

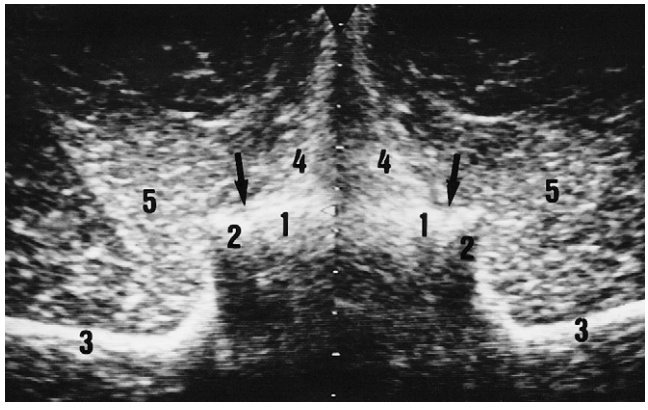

Fig. 54-15 Transverse ultrasound scan of the synovial intervertebral articulations between the first and second lumbar vertebrae shows normal appearance (compare with Fig. 54-3). *1*, Caudal articular process of first lumbar vertebra; *2*, cranial articular process of second lumbar vertebra; *3*, transverse process of second lumbar vertebra; *4*, multifidus muscle; *5*, longissimus muscle; *arrow*, joint space of the epaxial synovial intervertebral articulation.

On lateral radiographic views, in the middle of the film, the left and right synovial intervertebral articulations are superimposed. On the extremities of the film the images of the left and right synovial intervertebral articulations are progressively dissociated because of the divergent x-ray beam. In some horses the ribs are extremely convex dorsally and are superimposed over the thoracic and the first lumbar synovial intervertebral articulations, prohibiting assessment. The radiolucent cartilaginous joint space is thin and more clearly defined in the thoracic areas, where the articular facets are flat (Fig. 54-12), than in the lumbar area because of the condylar shape. Between T12 and T16 the joint space is a wide V shape, with a cranial oblique branch and a shorter vertical caudal branch (Fig. 54-13). At the thoracolumbar junction and in the lumbar region the joint space is less well defined. Usually, the joint space is linear and makes a 40° angle with the horizontal (Fig. 54-14). The caudal articular process is dorsal to the corresponding intervertebral foramen and is triangular with a caudal apex in the lumbar region. The thickness of the dense subchondral bone of the cranial articular process increases caudally. The cranial articular process is extended dorsocranially by a mamillary process, which is larger in the thoracic area than in the lumbar area.

On transverse ultrasonographic images obtained using 2.5- to 5-MHz probes, the synovial intervertebral articulations can be imaged (Fig. 54-15). On each side the joint space separating the cranial (lateral) and caudal (medial) articular process can be identified.[12] This joint space is limited dorsally by a thin articular capsule. Comparison of the left and right synovial intervertebral articulations of the same intervertebral joint is helpful to assess size and shape.

Abnormal Findings

Eight types of abnormal findings associated with osteoarthritis have been identified radiographically in the synovial intervertebral articulations of the equine thoracolumbar spine[10] (Table 54-3). These findings were mainly present at the thoracolumbar junction and in the lumbar area. Types 2, 4, and 6 (Figs. 54-16 to 54-19) were mainly found between T16 and L3. Types 5 and 7 have mainly been found in the lumbar area. Abnormalities have been found in mature riding horses and immature racehorses.

Dorsal peri-articular proliferation (types 4 and 6) can be imaged with ultrasonography, and with this procedure determining if the proliferation is symmetrical or, if not, which side is the most affected is possible (Fig. 54-20).

Nuclear scintigraphy can be helpful in identifying evidence of abnormal bone modeling in the region of synovial intervertebral articulations, especially in horses where radiographic appraisal is limited by the ribs. In our experience, synovial

Table • **54-3**

Types of Radiographic Lesions of the Thoracolumbar Synovial Intervertebral Articulations

TYPES	GENERAL CRITERIA	RADIOGRAPHIC SIGNS
1	Asymmetry	No clear joint space; double joint space
2	Modification of opacity of the articular process	Sclerosis of the subchondral bone Increased opacity of the synovial intervertebral articulations
3		Radiolucent areas in the subchondral bone Increased opacity of the articular process
4	Peri-articular proliferation	Dorsal peri-articular proliferation Increased size of the synovial intervertebral articulations Often associated with alteration in opacity of subchondral bone
5		Ventral peri-articular proliferation
6	Ankylosis	Dorsal bridge between two adjacent vertebrae
7		Osteolysis of the synovial intervertebral articulations No joint space
8	Fracture	Radiolucent line on the caudal (or cranial) articular process

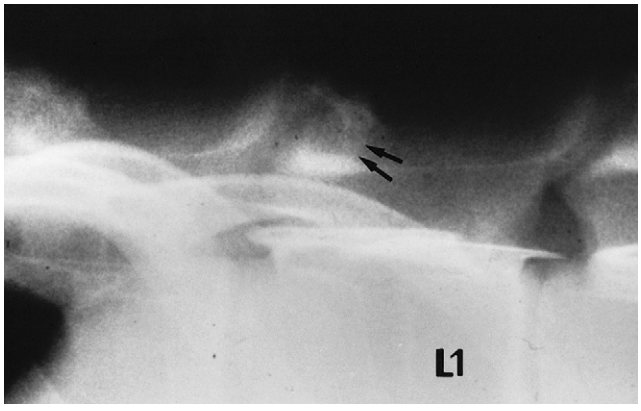

Fig. 54-16 Lateral radiographic view of the thoracolumbar junction. Cranial is to the left. There are type 2 lesions *(arrows)* of the synovial intervertebral articulations between the eighteenth thoracic and second lumbar vertebrae in this 5-year-old French trotter male. Compare with the opacity of the cranial articular processes in Fig. 54-14.

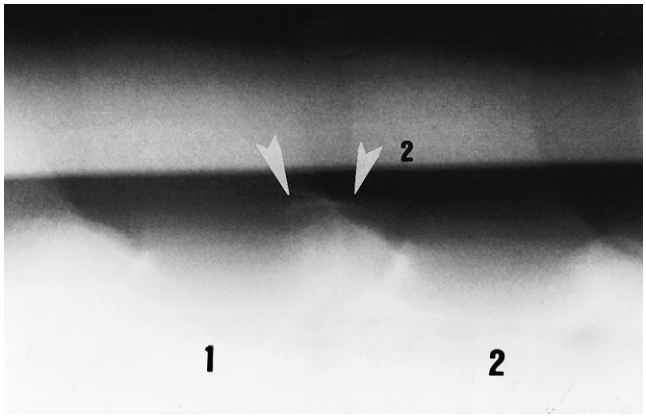

Fig. 54-17 Lateral radiographic view of the cranial lumbar vertebrae. Cranial is to the left. There is a mild (grade 1) type 4 lesion of the synovial intervertebral articulation complex between the first (*1*) and second (*2*) lumbar vertebrae in this 8-year-old mare.

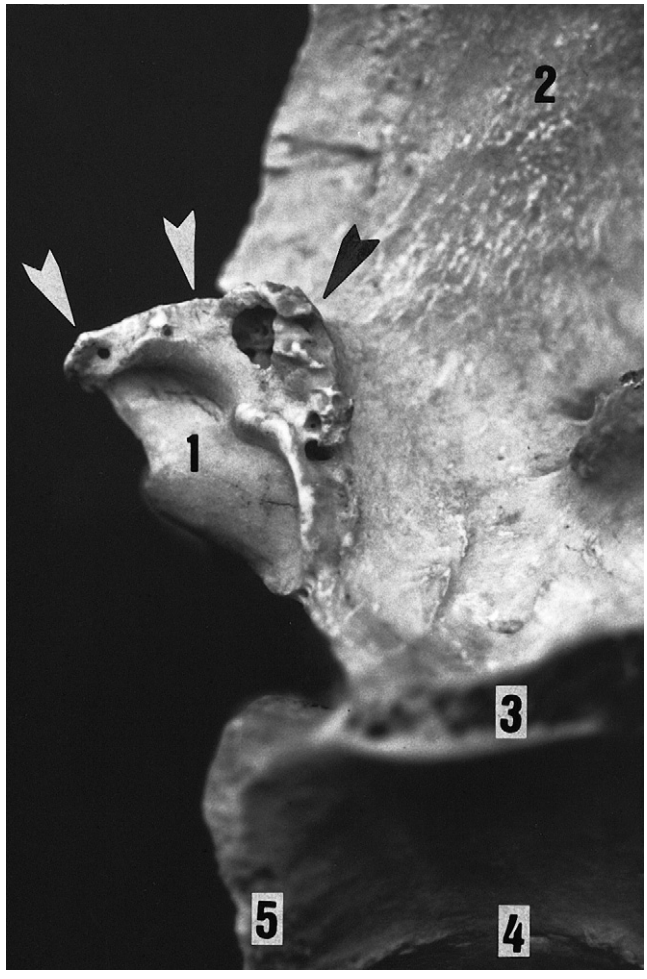

Fig. 54-18 Bone specimen showing a moderate (grade 2) type 4 lesion *(arrowheads)* of the caudal articular process of the third lumbar vertebra. *1*, Caudal articular process; *2*, dorsal spinous process; *3*, transverse process; *4*, vertebral body; *5*, vertebral fossa.

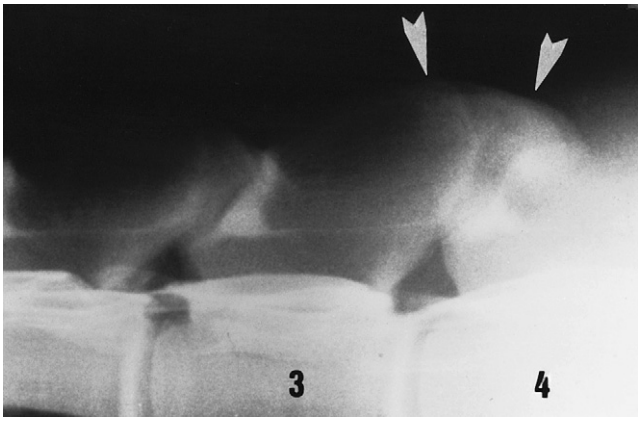

Fig. 54-19 Lateral radiographic view of the mid-lumbar vertebrae. Cranial is to the left. There is a severe (grade 3) type 4 lesion *(arrowheads)* of the synovial intervertebral articulation between the third *(3)* and fourth *(4)* lumbar vertebrae in this 12-year-old mare.

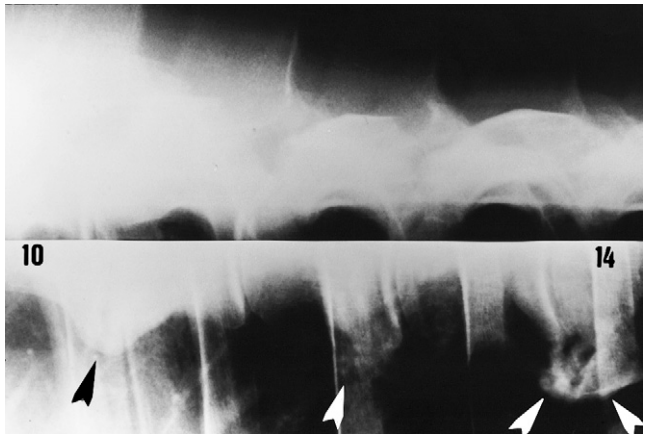

Fig. 54-21 Lateral radiographic view of the vertebral bodies of the caudal thoracic vertebrae. Cranial is to the left. There is extensive ventral spondylosis *(arrowheads)* between tenth *(10)* and fourteenth *(14)* thoracic vertebrae in this 13-year-old international show jumper stallion.

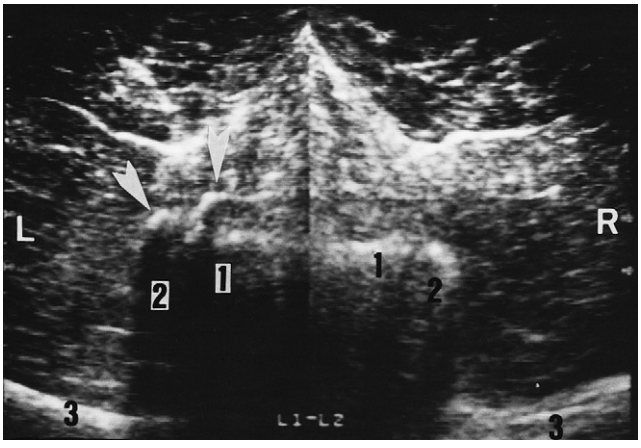

Fig. 54-20 Transverse ultrasound scan of the synovial intervertebral articulation between the first and second lumbar vertebrae in 5-year-old French trotter mare: on the left side (*L*) a dorsal peri-articular osteophyte is present *(arrowheads)*. *1*, Caudal articular process of first lumbar vertebra; *2*, cranial articular process of second lumbar vertebra; *3*, transverse process of second lumbar vertebra.

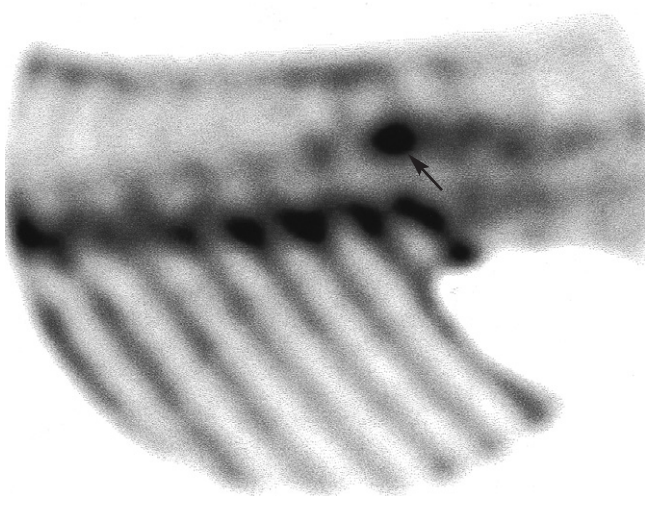

Fig. 54-22 Lateral scintigraphic image of the thoracolumbar region of 2-year-old Thoroughbred filly with back pain and loss of performance. A focal region of increased radiopharmaceutical uptake is visible at the thoracolumbar junction *(arrow)*, compatible with a laminar stress fracture.

intervertebral articulation lesions are much more consistently associated with back pain than are kissing spines.

Vertebral Bodies and Disks

Lesions of the vertebral bodies are less commonly found in horses[14] and cannot be imaged by ultrasonography in the thoracolumbar area (except caudal to L4 with a transrectal approach). These lesions include the following[10]:

1. Ventral, ventrolateral, or lateral bony proliferation (often called vertebral spondylosis). These lesions are mainly found in the mid-thoracic area (Fig. 54-21), but they can be seen in the lumbar area.
2. Vertebral body deformation (trapezoid shape) of the caudal thoracic vertebrae
3. Deformation of the adjacent vertebral head and fossa (disk enthesopathy) found in the caudal thoracic area
4. Ski jump deformation (dorsal bony extension of the vertebral fossa) found in the caudal thoracic area

These lesions have been found in horses with a history or clinical manifestations of back pain—acute, intermittent, and chronic—and are considered to be of likely clinical significance. However, spondylosis also is an incidental finding in low-level performance horses with no history or clinical manifestations of back pain. Nuclear scintigraphy may help to elucidate the significance of these findings in horses with back pain.

Fracture and subluxation of a lumbar vertebra normally results in neurological abnormalities, but occasionally horses have acute severe back pain, with rapid atrophy of the epaxial musculature, but no evidence of ataxia.

Vertebral laminar stress fractures have been identified using nuclear scintigraphy in young TB flat race horses with loss of performance and clinical evidence of back pain (Fig. 54-22). They have been seen most commonly at the thoracolumbar junction and in the lumbar vertebrae. In a post-mortem sample of TB race horses, 18 (50%) of specimens had evidence of vertebral stress factures.[16]

PROGNOSIS

The prognosis for horses with any back lesion depends on a number of factors, including the individual pain tolerance of the horse, the skill of the rider and trainer, the discipline in which the horse is involved, and the type, number, and severity of lesions. Vertebral lesions are tolerated better by flat race horses than by Three Day Event horses, show jumpers, and dressage horses. Racing trotters tolerate vertebral lesions poorly. The prognosis for each lesion is not influenced by the level of work but more by the rider's or trainer's ability to exploit the horse despite its problems. A horse with an acute isolated supraspinous ligament injury has a good long-term prognosis, whereas horses with restricted mobility of the back caused by extensive kissing spines or type 6 or 7 synovial intervertebral articulation lesions have a poor prognosis. Horses with grades 3 and 4 dorsal kissing spines have a guarded prognosis, as do those with ventral kissing spine lesions. The prognosis for horses with synovial intervertebral articulation lesions depends on severity and the number of joints affected. Patient tolerance of ventral thoracic spondylosis seems to vary more, but generally the larger the number of vertebrae affected, the poorer the prognosis.

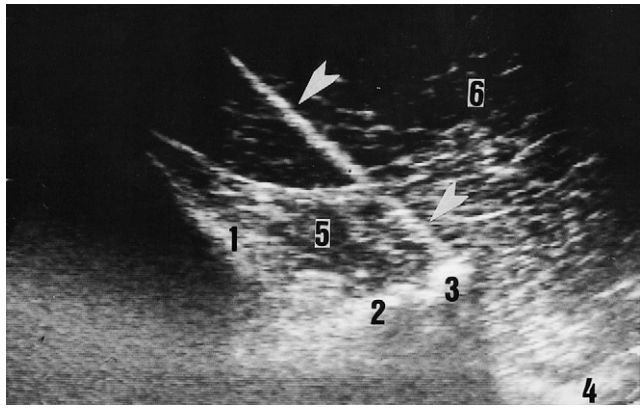

Fig. 54-23 Transverse ultrasound scan showing an ultrasound guided peri-articular injection for the treatment of synovial intervertebral articulation arthropathies. The probe is placed on a paramedian position, thus the image is slightly oblique. *1*, Spinous process; *2*, caudal articular process of first lumbar vertebra; *3*, cranial articular process of second lumbar vertebra; *4*, transverse process; *5*, multifidus muscle; *6*, longissimus muscle; *arrowheads*, needle.

SPECIFIC MANAGEMENT

The general aims of management of back problems are to remove pain and to make the horse as comfortable as possible, as soon as possible, to allow it to be exercised to avoid further muscle loss and to promote muscle function and strength. Medical treatment aims to remove pain and muscle spasm.

Systemic Treatment

Treatment of back pain with non-steroidal anti-inflammatory drugs (NSAIDs) is usually disappointing. Many horses are brought in for in-depth investigation of the cause of back pain because of lack of response to treatment with phenylbutazone or other NSAIDs. Treatment of muscle spasm with myorelaxants is indicated and sometimes useful; for example, thiocolchicoside (2 to 4 mg/100 kg twice weekly for 4 weeks) and methocarbamol (10 mg/kg IV). Repeated treatments may be necessary.

Local Injections

Local perispinal or interspinal injections of corticosteroids, sometimes in association with myorelaxants or Sarapin, are used to treat horses with kissing spines: flumethasone (0.5 to 1 mg per injection site with a maximum total dose of 4 mg); dexamethasone (isonicotinate, 1.5 to 2.5 mg per injection site, with a maximum total dose of 10 mg); methylprednisolone acetate (40 to 60 mg per injection site, with a maximum total dose of 200 mg).

Osteoarthritis of the synovial intervertebral articulations is treated by deep paramedian injections of corticosteroids (see the previous discussion) into the multifidus muscle on each side at the level of the lesion, 2 cm apart from the median plane, using 9- to 11-cm needles. These local injections are best performed using ultrasonographic guidance (Fig. 54-23). Injections are made into the multifidus muscles that provide proprioceptive information on intervertebral movements. We therefore suggest that the use of Sarapin is contraindicated in this situation. Local infiltrations can be performed alone or with mesotherapy.

Mesotherapy

Mesotherapy is a technique that has been used for more than 30 years in France and consists of intradermal injections with short and thin needles (5 mm long) in the dermatomes corresponding to the site(s) of the lesion(s).

The principle is based on the theory of the gait control of pain, which takes place in the dorsal horn of the spinal cord. According to this theory, which is currently controversial, types I and II nerve fibers coming from the skin have collateral fibers that can inhibit the conduction of information in the spinothalamic fasciculus, transmitting painful information from deep structures of the same spinal segment to the thalamus and cerebrum.

After aseptic preparation of the skin, mesotherapeutic injections are usually made using a local anesthetic solution (lidocaine, 140 mg), a short-acting corticosteroid (dexamethasone, 15 mg), and a myorelaxant (thiocolchicoside, 20 mg). Injection with only saline solution may also have beneficial effects. Because most horses are sensitive to the procedure, the patient is placed in stocks, and the use of a twitch and sedation is recommended. A multi-injector (Coveto, Montaigu, France) is used. These intradermal injections are performed at the level of the lesion and caudal to it, taking into account the caudal orientation of the segmental nerves. For example, if one is treating kissing spines between T10 and T15, the treated region extends from T10 to L1. If the lesions involve the synovial intervertebral articulation between T17 and L2, the treated area should extend between T17 and the lumbosacral junction. If lesions are at the lumbosacral junction, the treated area should extend caudally to the croup. Two to three rows of injections are made on each side of the median plane.

After local injections or mesotherapy, the horse is restricted to light work on the lunge, without a surcingle, for 3 days. Normal training is progressively resumed over 5 days. A substantial improvement is anticipated within 7 to 14 days. If improvement is limited, mesotherapy should be repeated 2 to 3 weeks after the first treatment. The expected duration of action varies between 3 and more than 12 months. In horses with chronic pain, ideally a maximum of two local infiltrations should be performed each year with, or alternately with, mesotherapy.

Acupuncture

Acupuncture can be useful for treating chronic pain in some horses (see Chapter 93).

TRAINING MANAGEMENT

With the medical treatment to reduce pain, modification of the training program is an essential part of the management of back problems.[17] The aims of the exercise management are to avoid further muscle atrophy and to develop the back proprioceptive control and intervertebral stability. Rest is contraindicated, except in untreatable horses that are not improved by any kind of treatment and management. The general recommendations include the following:

- Check the saddle fitting.
- In young horses, recognize that training must be progressive.
- Use a progressive warm-up program, first on the lunge without a saddle, then on the lunge with a saddle, then with the rider at walk, and so on.
- After the warm-up period at the walk, in sport horses, first work at the canter rather than at the trot. During cantering the vertebral column undergoes only one slow and active flexion/extension cycle per stride, whereas the vertebral column undergoes two passive flexion/extension movements per stride at the trot.
- Identify and then remove every exercise that induces discomfort. For example, do no short turns to the right for 2 weeks, then re-assess if the horse is more comfortable turning to the right.

Some physical exercises, such as lowering of the neck (flexion of the cervicothoracic junction), may have a therapeutic value because they induce an associated thoracic flexion, which provides more support to the weight of the rider. A separation of the spinous processes reduces the contact between them.[18,19] An elongation of the strong epaxial muscles reduces muscle contraction.[17]

ALTERNATIVE MEDICINES

Much interest exists in using alternative therapeutic techniques to manage back pain in horses. Once a clear diagnosis is established, alternative techniques, if appropriately applied and objectively assessed for each pathological entity, may be useful in managing back problems (see Chapters 94 through 99).

SURGERY

Surgical treatment of kissing spines by resection of the summit of one or more spinous processes has been successful in some horses that had not responded to conservative management. Case selection is important and should be restricted to horses with lesions only involving the dorsal aspect of the spinous processes. A post-operative convalescent period of 6 months is generally required. Twenty-seven of 50 Warmblood horses (54%) used for dressage or show jumping returned to full athletic function at the previous level of performance.[20] One hundred fifty of 209 horses (72%) used for various disciplines returned to full work after removal of the summits of 1 to 6 dorsal spinous processes, although the level of work was not specified.[21] There was no significant difference between the number of spines removed and the outcome.

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CHAPTER • 55

The Cervical Spine and Soft Tissues of the Neck

Sue J. Dyson

This chapter discusses disorders of the neck that may give rise to lameness or poor performance or result in an abnormal neck shape, abnormal neck posture at rest or while moving, or neck stiffness. Transient neurological conditions, or those caused by trauma resulting in other injuries, or gait abnormalities that may be confused with lameness are considered, but those associated with compression of the cervical spinal cord are discussed in Chapter 62.

ANATOMY

The neck comprises seven cervical vertebrae, which articulate both by intercentral articulations and synovial articulations, which have large joint capsules to accommodate the degree of movement between adjacent vertebrae. Interposed between the vertebral bodies are intervertebral fibrocartilages to which is attached the dorsal longitudinal ligament, which lies on the floor of the vertebral canal. The ligamentum flava connects the arches of adjacent vertebrae. The atlas (the first cervical vertebra) and the axis (the second cervical vertebra) have a unique shape and specialized joints. The atlanto-occipital joint is a ginglymus joint, which permits flexion and extension and also a small amount of lateral oblique movement. The atlantoaxial joint is a trochoid or pivot joint; the axis and head rotate on the axis. The ligament of the dens is strong and fan-shaped and extends from the dorsal surface of the odontoid peg (dens) to the ventral arch of the axis. The ligamentum nuchae extends from the occiput to the withers and comprises funicular and lamellar parts. The lamellar part separates the two lateral muscle groups. The atlantal bursa is interposed between the funicular part of the ligamentum nuchae and the dorsal arch of the atlas; a second bursa may exist between the ligament and the spine of the axis. The muscles of the neck can be divided into lateral and ventral groups. The neck has eight cervical nerves, the first of which emerges through the intervertebral foramen of the atlas, the second through that of the axis, and the eighth between the seventh cervical vertebra and the first thoracic vertebra. The sixth to eighth cervical nerves contribute to the brachial plexus.

REASONS FOR CLINICAL PRESENTATION

A horse may be presented for evaluation of the neck for a variety of reasons. The horse may have a history of a fall on the neck while jumping, having reared up and fallen over backward, or having collided with another horse or solid object, thus sustaining neck trauma. The horse may have neck pain from having pulled backward while being tied up. The horse may have no history of trauma but have abnormal neck posture, swelling, a stiff neck, neck pain, or difficulties in lowering and raising the head. The horse may have a performance-related problem such as unwillingness to work on the bit, an unsteady head carriage, or abnormal head posture. A neck lesion should also be considered in a horse with forelimb lameness, when pain cannot be localized to the limb. Subtle hindlimb gait abnormalities, such as a tendency to stumble, may be caused by a neurological deficit without overt ataxia, reflecting a compressive lesion of the cervical spinal cord.

CLINICAL EXAMINATION

It is important to recognize that head and neck carriage depends in part on conformation: the way in which the neck comes out of the shoulder and the shape of the neck. The shape of the neck is also influenced by the way in which the horse works. If a horse carries the head and neck high, with the head somewhat extended, the ventral strap muscles tend to be abnormally well developed, resulting in a ewe-neck conformation. Many horses naturally bend more easily to the right than to the left or vice versa, and the muscles on the side of the neck, especially dorsocranially, are developed asymmetrically. Such asymmetry is particularly obvious if the neck is viewed from above by the rider. If a horse is excessively thin, then the cervical vertebrae become prominent and the caudodorsal neck region becomes dorsally concave, whereas in a fit, well-muscled horse that works regularly on the bit, this region is dorsally convex. Most stallions and many native pony breeds have a prominent dorsal convexity to the neck region, resulting in a cresty appearance. A horse that is excessively fat tends to lay down plaques of fat throughout the body, including the neck region and this can be misinterpreted as abnormal neck swelling.

If a horse is particularly thick through the jaw, that is, has a large mandible, it is physically difficult to work on the bit (i.e., flexing at the poll so that the front of the head is in approximately the vertical position). Although neck pain can cause a reluctance to work on the bit, more common causes include rider-associated or training problems, mouth pain, forelimb or hindlimb lameness, or back pain. Some horses strongly resist the rider's aids to work on the bit, despite the absence of pain. The use of artificial aids such as draw or running reins, which give the rider a mechanical advantage, may help to break a vicious cycle and encourage the horse to become more submissive and compliant. Similarly, work on the lunge line using a chambon (a device that runs from the girth via a headpiece to the bit rings) can encourage the horse to work in a correct outline and develop fitness and strength of the appropriate musculature. Working the horse in trot over appropriately spaced trotting poles can also help to encourage a horse to work in a correct outline, with a round and supple back.

A rider may complain of neck stiffness or difficulties in getting a horse to bend correctly in a circle. Although this may be caused by neck pain, neck stiffness may be a protective mechanism by the horse to avoid pain associated with lameness, especially forelimb lameness. A horse with left forelimb lameness, for example, may be reluctant to bend properly to the left, and when unrestrained by a rider on the lunge, on the left rein may hold the neck and head slightly to the right,

giving the appearance of looking out of the circle. Thus load distribution is altered and lameness minimized. Such lameness actually may not be evident during riding, although this may be the only circumstance under which the rider recognizes the problem. The lameness may be more obvious on the lunge or even in hand in straight lines. When a horse has an abnormal neck and/or head posture, a comprehensive clinical evaluation of the entire horse should be performed. Neck pain or abnormal posture may reflect a primary lesion elsewhere (e.g., central or peripheral vestibular disease, fracture of the spinous processes of the cranial thoracic vertebrae, a mediastinal or thoracic abscess, or a systemic disease such as tetanus).

Detailed examination of the neck should include assessment of the neck conformation, shape and posture at rest, and the position of the head relative to the neck and trunk. The veterinarian should note any patchy sweating or change in hair color reflecting intermittent sweating that may suggest local nerve damage. Look carefully at the musculature to identify any localized atrophy. Palpate the right and left sides of the neck to assess symmetry and the presence of abnormal swellings or depressions and to identify any neck muscle pain, tension, or fasciculation. Deep palpation should be performed on the left and right sides of the neck to identify pain.

A series of nine equidistant acupoints (acupuncture points) exist along an arc on the crest of the neck.[1] The most cranial is in the depression just cranial to the wing of the atlas and just caudal to the ear. The most caudal point is a few centimeters dorsocranial to the dorsocranial aspect of the scapula. Six intervertebral acupoints also exist between the vertebrae. An abnormal response to firm palpation of these points may reflect neck pain.

Neck flexibility should be assessed from side to side and up and down. This can be done by manually manipulating the neck, but many normal horses resist this. Holding a bowl of food by the horse's shoulder to assess lateral flexibility is helpful. The clinician should try to differentiate between the horse properly flexing the neck or twisting the head on the neck. Compare flexibility to the left and to the right. To assess extension of the neck, the veterinarian should evaluate the ease with which the horse can stretch to eat from above head height. Observing the horse grazing is helpful to assess ventral mobility of the neck. Especially with lesions in the caudal neck region a horse may have to straddle the forelimbs excessively to lower the head to the ground to graze (Fig. 55-1, *A*).

Assessing skin sensation and local reflexes, such as the cervicofacial and the thoracolaryngeal reflexes, and comparing carefully the right and left sides may be useful. The consistency and patency of the jugular veins should be evaluated.

The horse should be observed moving in hand and on the lunge, and if necessary should be ridden, to assess neck posture and the presence of neurological gait abnormalities, restriction in forelimb gait or lameness. The clinician should note how any gait abnormality is influenced by the positions of the head and neck. Forelimb lameness occasionally is associated with a primary cervical lesion, usually, but not invariably, together with other clinical signs referable to the neck.[2]

IMAGING CONSIDERATIONS

Radiography

Comprehensive radiographic examination of the neck requires at least five exposures, assuming that large cassettes are used, including the poll, cranial, mid- and caudal neck regions, and the base of the neck to evaluate the first thoracic vertebra.[3] Lateral views are obtained easily in the standing position, but ventrodorsal views are best obtained with the horse in dorsal recumbency under general anesthesia, except in small ponies

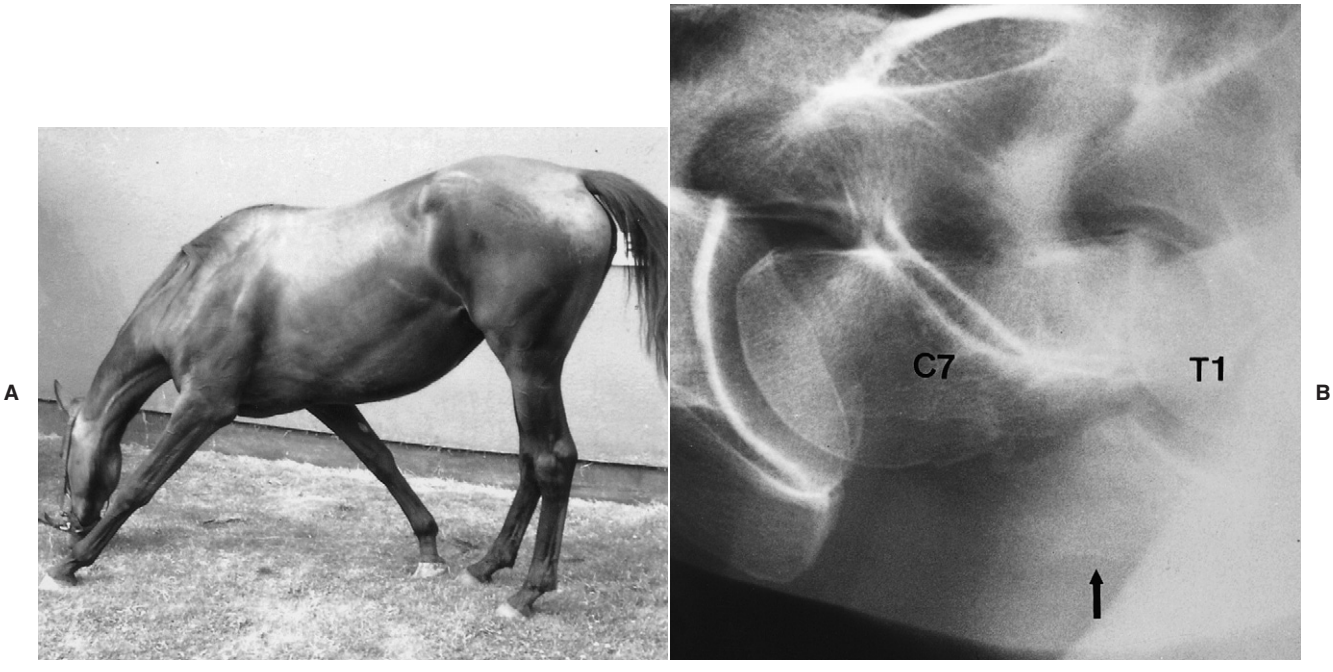

Fig. 55-1 **A,** A 3-year-old Thoroughbred filly with severe neck pain and restricted forelimb gait associated with a displaced articular fracture of the ventral processes of the seventh cervical vertebra. The horse must adopt the straddled forelimb stance to lower the head to graze. **B,** Lateral radiographic view of the caudal cervical vertebrae (sixth cervical to first thoracic vertebrae) of the same horse. Cranial is to the left. The ventral processes of the seventh cervical vertebra are displaced *(arrow)*. The intercentral joint space between the seventh cervical *(C7)* and first thoracic *(T1)* vertebrae is narrowed greatly, with abnormal orientation of the vertebral bodies.

and foals. Positioning of the neck is important, because any rotation of the head and neck makes evaluating the synovial articulations in particular difficult. Relatively large exposures are required for the more caudal neck regions, so radiation safety is important, and the cassette should be supported in a holder, not held by hand. A grid is useful, especially in the caudal neck region, to reduce scattered radiation. Obtaining exposures from left to right and right to left may be useful.

A number of variations of the normal radiographic appearance of the cervical vertebrae should not be confused as lesions. A spur on the dorsocaudal aspect of the second cervical vertebra may project into the vertebral canal. The ventral processes of the sixth cervical vertebra and occasionally other vertebrae have small separate centers of ossification. The ventral lamina on the sixth cervical vertebra may be transposed onto the ventral aspect of the seventh cervical vertebra, unilaterally or bilaterally. The seventh cervical vertebra has a small spinous process, which may be superimposed over the synovial articulation between the sixth and seventh cervical vertebrae and should not be confused with peri-articular new bone. In older horses small spondylitic spurs may be seen on the ventral aspect of the vertebral bodies. Modeling of the dorsal synovial articulations between the fifth and sixth and between the sixth and seventh cervical vertebrae is common in middle-aged and older horses[3,4] (Fig. 55-2).

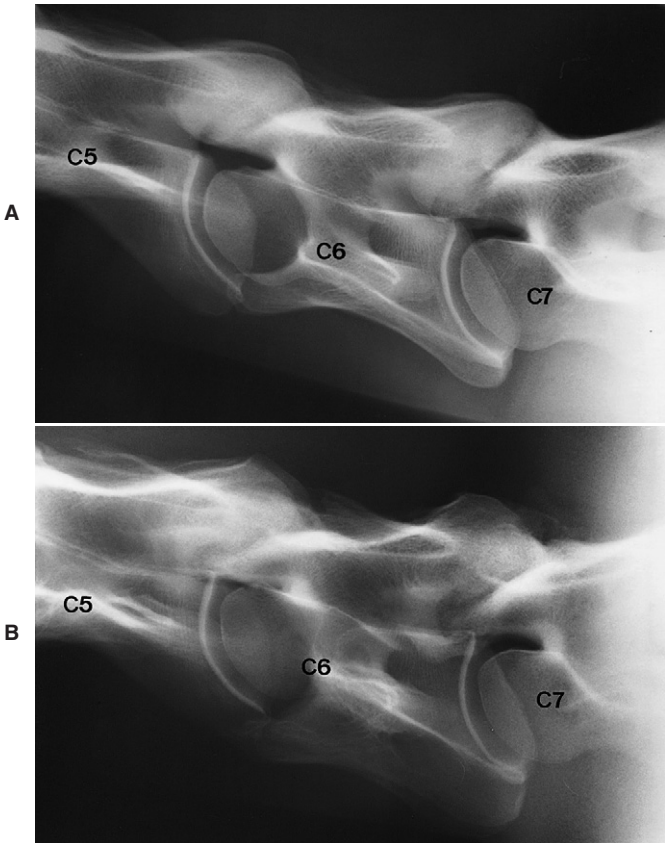

Fig. 55-2 **A,** Lateral radiographic view of the caudal cervical vertebrae of normal 4-year-old Thoroughbred. The synovial articulations between the fifth (C5) and sixth (C6) cervical and sixth and seventh (C7) cervical vertebrae are outlined smoothly. The intervertebral foramina are distinct. Compare with part **B** and Figure 55-8. **B,** Lateral radiographic view of the caudal cervical vertebrae of 9-year-old clinically normal horse. The synovial articulations are enlarged between the fifth (C5) and sixth (C6) cervical vertebrae and particularly between the sixth and seventh (C7) cervical vertebrae.

Major radiographic abnormalities such as fusion of two adjacent vertebrae can be present subclinically, in part because of the great mobility between adjacent vertebrae (Fig. 55-3, *A*). The significance of such lesions may also be determined by the athletic demands placed on the horse.

Nuclear Scintigraphy

Lateral and ventral scintigraphic images of the neck can be obtained. Ideally, images should be obtained from the left and right sides. In normal horses there is usually increased radiopharmaceutical uptake (IRU) in the synovial articulation between the sixth and seventh cervical vertebrae, compared with the more cranial articulations. There is often greater radiopharmaceutical uptake in the odontoid peg (dens) of the axis, compared with the surrounding vertebrae.

Abnormal IRU is not necessarily synonymous with a lesion that is clinically significant; therefore images must be interpreted with care (Fig. 55-3, *B*). The clinician should compare images obtained from the left and right sides carefully, because disparity in radiopharmaceutical uptake may be significant. The veterinarian should evaluate the actual conformation of the synovial articulations, because a change in shape, even

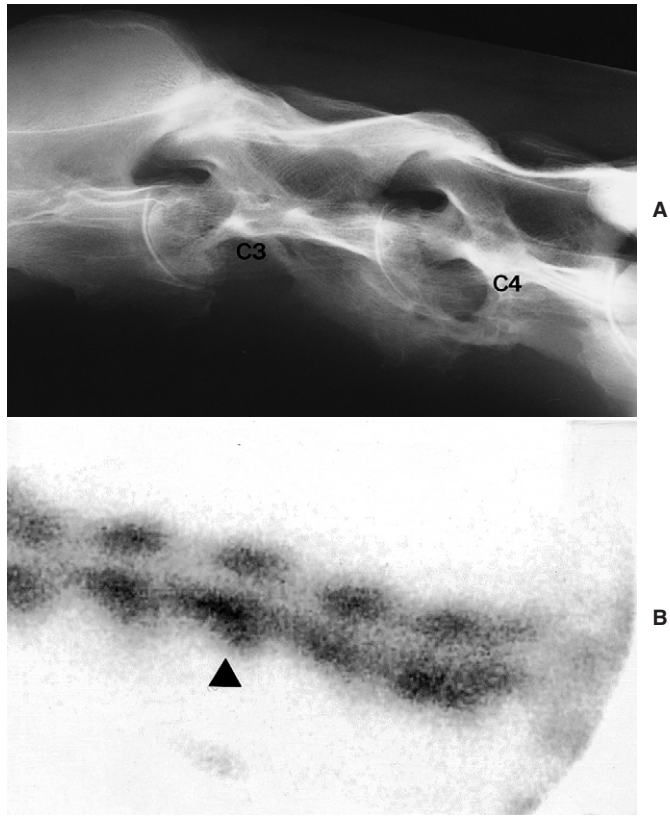

Fig. 55-3 **A,** Lateral radiographic view of a 15-year-old Arabian international-level endurance horse that had been competing successfully with no clinical signs referable to the neck. There is abnormal angulation between the third (C3) and fourth (C4) cervical vertebrae. The vertebral bodies of the third and fourth cervical vertebrae are osteoporotic. Extensive new bone has formed over the ventral aspect of the intercentral articulation between the third and fourth cervical vertebrae, with narrowing of the intercentral joint space and thinning of the caudal end plate of the third cervical vertebra. The ventral profile of the more cranial aspect of the third cervical vertebra is abnormal. **B,** Lateral scintigraphic image of the mid-neck region of the same horse. Note the increased radiopharmaceutical uptake in the vertebral body of the fourth cervical vertebra *(arrow)*.

without IRU may be important. Fractures are not always associated with a significant IRU, and lesions may be missed in the caudal neck region because of the overlying muscle mass and the scapulae.

Ultrasonography

The indications for ultrasonographic examination include evaluating swellings, assessing painful muscles and lesions of the ligamentum nuchae, and documenting jugular vein thrombophlebitis.[5]

Computed Tomography

Computed tomography (CT) has the potential to give three-dimensional information about the cervical vertebrae and with contrast-enhanced studies can give information about spinal cord and nerve compression. However, general anesthesia is required, and it is only practical to image the cranial cervical vertebrae.[6-8]

Thermography

Thermographic examination of the neck is discussed in detail elsewhere (see Chapters 25 and 98). However, I have found thermography of limited usefulness, except for identifying acute superficial muscle injuries.

OTHER DIAGNOSTIC TESTS

In selected horses with neck pain, valuable information may be obtained from hematological and serum biochemical tests. Measuring *Brucella* titers and tuberculosis testing are occasionally useful. Bone biopsy may be valuable for determining the cause of some bony lesions.

CLINICAL CONDITIONS

Occipito-Atlantoaxial Malformation

Occipito-atlantoaxial malformation (OAAM) is a congenital abnormality,[9] and although it can occur in any breed,[10] OAAM appears to be a heritable condition in Arabian horses[11] (see Chapter 124). Clinical signs are usually recognizable within the first few weeks of life and include an abnormal neck shape in the poll region, with prominence on the left or right sides or both, and/or scoliosis (Fig. 55-4, *A*). These signs are best appreciated when viewed from above. Usually no associated soft tissue swelling or pain exists, although an abnormal clicking sound may be audible because of subluxation of the atlantoaxial joint. The horse may have an abnormal limitation of movement in the poll region. The gait should be assessed carefully for neurological abnormalities; however, in many horses no neurological gait deficits are apparent.

Diagnosis is confirmed radiographically using lateral and ventrodorsal views. Bony abnormalities include fusion of the atlas to the occiput, atlantoaxial luxation, and abnormal shapes of the atlas and axis, often asymmetrical (Fig. 55-4, *B*).

No treatment is available for OAAM. Prognosis for athletic function is determined by the degree of neck stiffness. Because of the heritable nature of this condition in Arabian horses, breeding of affected horses, or the sire or the dam, is inadvisable.

Other Congenital Abnormalities

OAAM is the most common congenital abnormality of the cervical vertebrae, but congenital torticollis is seen occasionally, caused by malformation of more caudal cervical vertebrae. Vertebral body fusion usually is seen with a meningomyelocele, resulting in neurological abnormalities, and therefore is not considered further.

Subluxation of the First and Second Cervical Vertebrae

Subluxation of the first and second cervical vertebrae is an unusual condition, probably related to trauma, although the horse may have no recent history of such.[12,13] The condition is associated with damage of the ligament of the dens or the

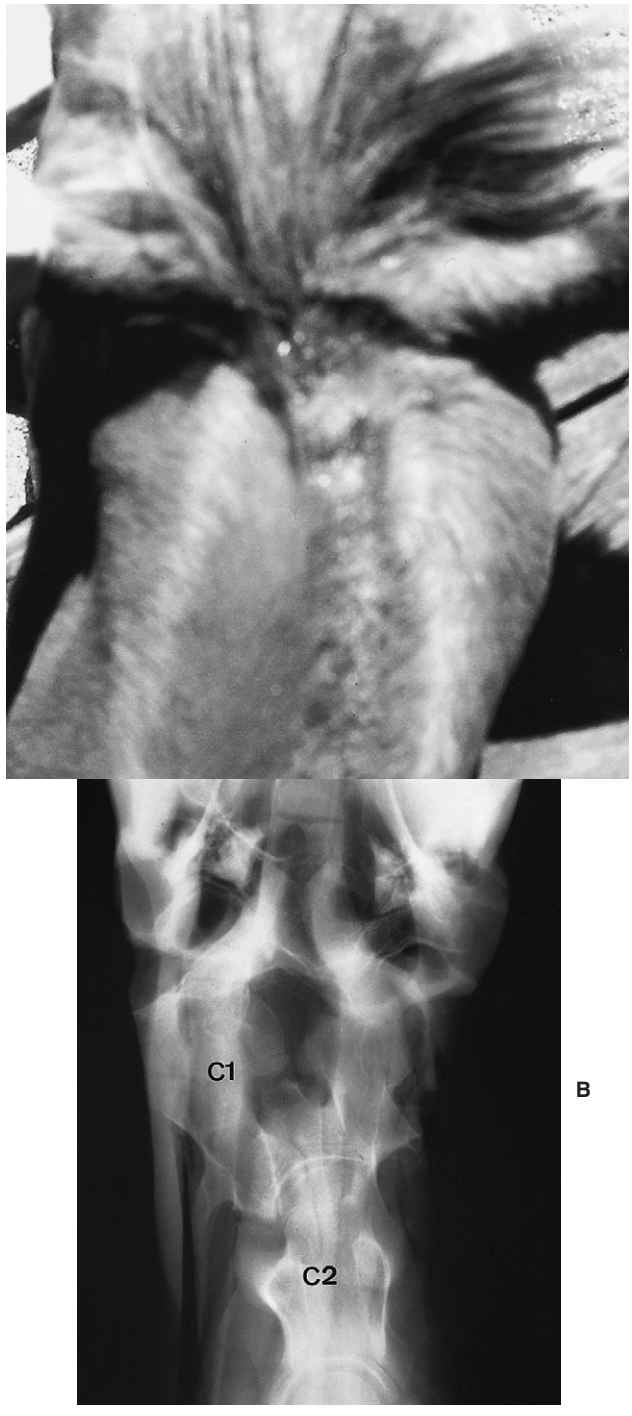

Fig. 55-4 **A,** Dorsal view of the poll region of a 6-month-old Thoroughbred filly with an abnormal asymmetrical shape and scoliosis of the cranial neck region and neck stiffness associated with occipito-atlantoaxial malformation. **B,** Ventrodorsal radiographic view of the same horse. Note the distorted shape of the atlas, its fusion with the occiput, and the abnormal orientation of the intercentral articulation between the first (*C1*) and second (*C2*) cervical vertebrae.

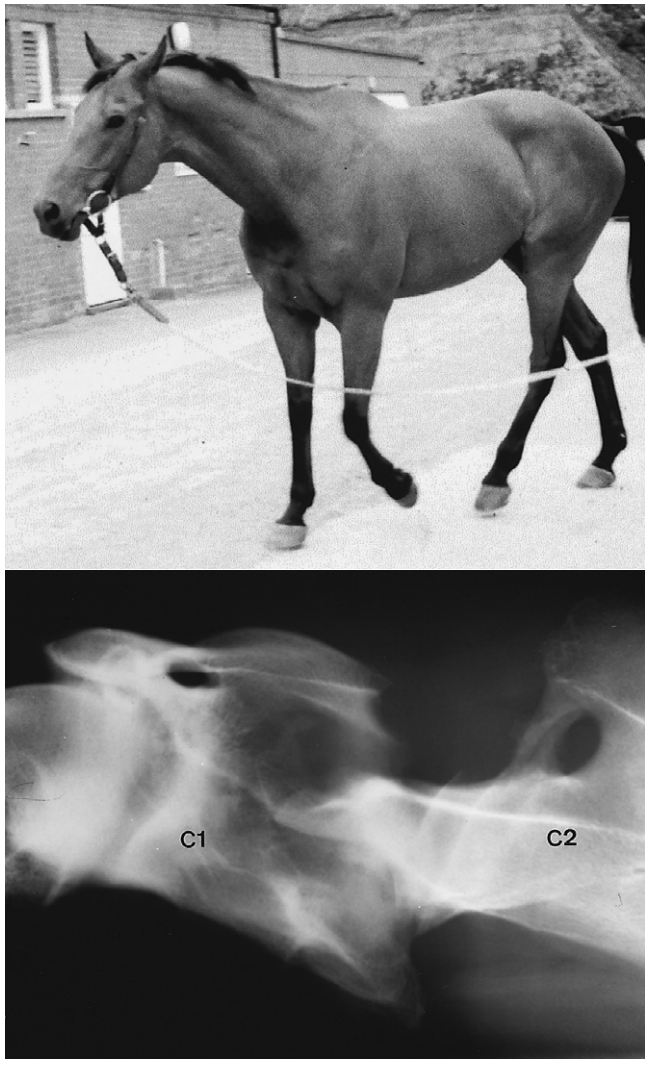

Fig. 55-5 **A,** A 9-year-old advanced event horse that had shown neck stiffness since traveling from America to Great Britain. The head is extended somewhat, and there was significant neck stiffness or guarding. A clicking sound emanated from the cranial neck region. **B,** Lateral radiographic view of the cranial neck region of the same horse. There is an abnormal orientation between the first *(C1)* and second *(C2)* cervical vertebrae. Space between the dorsal aspect of the dens and the ventral aspect of the dorsal lamina of the vertebral arch of the first cervical vertebra is reduced. The dorsal aspect of the dens is irregular because of new bone formation. Post-mortem examination confirmed partial disruption of the ligament of the dens.

ventral longitudinal ligament between the first and second cervical vertebrae or occurs secondary to a fracture of the dens.[14,15] An affected horse usually has a stiff neck and a tendency for the head and neck to be somewhat extended (Fig. 55-5, *A*). Differentiating between neck pain and stiffness may be difficult. An audible clicking noise may emanate from the region, and occasionally abnormal movement between the vertebrae can be appreciated. Because of the relatively wide sagittal diameter of the vertebral canal at this site, generally no associated compression of the cervical spinal cord occurs. Occasionally, neurological abnormalities are seen in horses with a displaced fracture of the dens.

Diagnosis is based on radiographic examination, using lateral views with the neck in natural (neutral) and extended positions. Radiological abnormalities may include abnormal orientation between the first and second cervical vertebrae (Fig. 55-5, *B*). The position of the dens may be abnormal, resulting in narrowing of the space between it and the dorsal lamina of the vertebral arch of the first cervical vertebra. In a study of yearling Thoroughbreds the mean minimum sagittal diameter was 34 mm and the minimum was 26 mm.[16] Narrowing of the distance between the vertebral arch of the first and second cervical vertebrae may occur in the extended versus neutral positions of the neck. The shape of the dens may be altered because of secondary new bone formation; occasionally the dens is fractured. The synovial facet joints between the first and second cervical vertebrae may be altered in shape.

Foals with fractures of the odontoid peg have been successfully treated by surgical stabilization, but limited information exists about long-term prognosis.[14,15,17] Foals with subluxation without neurological abnormalities have a good prognosis for life; prognosis for athletic function is fair, depending on the intended level of competition. No reports of successful management of subluxation in adult horses exist, and the prognosis for return to athletic function with conservative management is poor.

Insertional Desmopathy of the Nuchal Ligament and Injury to Semispinalis

The nuchal ligament is a bilobed structure, fans at its insertion on the occiput, and is surrounded by muscle, the semispinalis to the left and right and rectus capitis ventrally. New bone formation at the insertion on the occiput may be an incidental finding. Examination of 302 Warmbloods from 1 to 22 years of age revealed new bone in 85%. A post-mortem study of Warmbloods revealed a similar high proportion of horses with chondroid metaplasia at the insertion of the ligament and dystrophic mineralization.[18] A smaller radiographic study of Thoroughbreds revealed new bone on the caudal aspect of the occiput in only 5%.[19]

Horses with insertional desmopathy of the nuchal ligament or injury to the tendon of insertion of semispinalis often have a history of trauma to the region (e.g., pulling back when tied up) or an excessive amount of lunging exercise while restricted with side or draw reins.[18,20,21] Horses should be examined while being lunged, with and without side reins, and ridden. Clinical signs include permanent resistance against the reins, with difficulty or unwillingness to lower and flex the head and neck when ridden and poor flexion at the poll. In contrast to horses with back pain, hindlimb impulsion is usually good. The horse may have a tendency to rear or shake its head.

Pain cannot usually be elicited by palpation. Radiographic examination may reveal new bone on the caudal aspect of the occiput that may extend farther ventrally and dorsally than the actual insertion of the ligamentum nuchae (Fig. 55-6). Mineralization sometimes is seen dorsal to the first cervical vertebra as an incidental radiological finding, unassociated with clinical signs. Scintigraphic examination may be negative. Ultrasonographic examination is not easy and interpretation is difficult. Mineralization within the ligament may cause shadowing artifacts. CT offers the most sensitive means of detecting lesions in either the nuchal ligaments of the border of insertion of semispinalis.[21] Diagnosis depends on a positive response to infiltration of local anesthetic solution. Fifteen milliliters of mepivacaine is infiltrated on the left and right sides, and the response is assessed after 15 to 30 minutes.

Treatment comprises repeated infiltration of corticosteroids, Traumil (a homeopathic remedy), and local anesthetic solution and modification of the training program, with no work on the bit for 8 weeks.[21] The horse should be worked principally in straight lines. In the stable the horse should be encouraged to flex gently the poll region from side to side and up and down. The use of acupuncture or magnetic field therapy, laser, ultra-

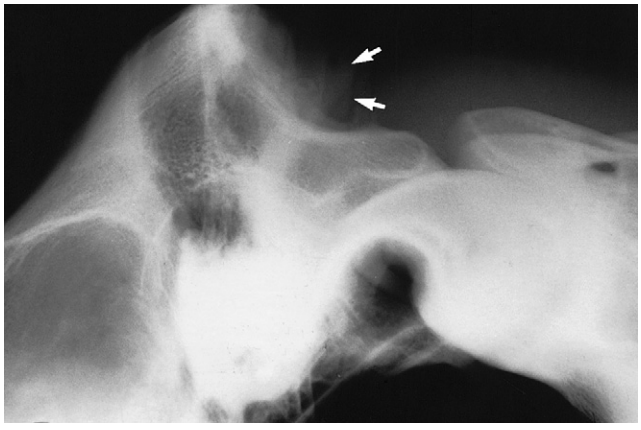

Fig. 55-6 Lateral view of the head of 8-year-old Warmblood show jumper with a history of reluctance to accept and go forward to the bit and stiffness. There is enthesophyte formation on the caudal aspect of the occiput at the insertion of the nuchal ligament (*arrows*). The horse responded well to local infiltration with corticosteroids and local anesthetic solution and modification of the training program.

sound, or shock wave therapy may help some horses. The results vary. Seventy percent of 26 horses return to full work, although not all were completely normal. Surgical treatment by transection of the nuchal ligament and the fascia of semispinalis has resulted in improvement in a small number of horses refractory to conservative management.[21]

Disorders of the Neck Musculature

The significance of localized muscle soreness and or tenseness is poorly understood and documented. I have had experience with a number of horses with subtle performance problems, including slight neck stiffness, reluctance to work properly on the bit and to accept an even contact, and intermittent, slight gait irregularities associated with significant soreness around and in front of the wings of the axis. Clinical improvement has been seen after relief of this pain by rapid and sudden rotation of the head about the axis.[22]

Many horses resent firm palpation of the braciocephalicus muscles at the base of the neck. This may be more obvious in horses with forelimb lameness, especially those with pain in the distal part of the limb. This muscle soreness is generally a secondary rather than a primary cause of lameness. Transient improvement in gait may be seen after local therapy using laser, H-wave, ultrasound, and/or massage.

Local muscle soreness also may be seen with a poorly fitting saddle or girth or with a rider who is unable to ride truly in balance with the horse. The primary problem must be addressed if treatment is to be successful. Some driving horses develop forelimb lameness that is seen only when the horse is pulling and may be associated with pressure from the harness. Adaptation of the harness may relieve the problem.

Some horses seem to need to learn how to use to maximum advantage the neck and forelimb musculature and have a restricted forelimb gait, without appearing overtly lame. The gait is not altered by distal limb nerve blocks. Some improvement may be achieved by daily massage of the muscles at the base of the neck and manual full protraction of the forelimbs. This is combined with exercise to encourage the horse to lengthen the forelimb stride and to round the back. Lunging in a chambon, trotting over appropriately placed trot poles, and repeated lengthening and shortening of the stride all may be beneficial. Trotting down the tramlines in a field of corn or rapeseed can also be of enormous help.

Occasionally as the result of a fall or pulling back when tied, acute severe neck muscle soreness develops. The horse is best treated initially with non-steroidal anti-inflammatory drugs (NSAIDs), rest, and local physiotherapy, followed by progressive remobilization, when the acute muscle soreness has subsided. The prognosis is good.

I have examined several horses that have had episodic transient attacks of profound neck pain and stiffness, holding the neck relatively low. In some horses a severe unilateral forelimb lameness occurs, often resulting in the limb being held in a semi-flexed position at rest. These attacks vary in duration (hours to days), and generally horses have been completely normal between episodes. To date, neither a definitive cause nor an effective treatment has been identified for this syndrome.

Dystrophic mineralization is seen sometimes as an incidental radiological finding in the neck musculature, secondary to previous intra-muscular injections.

Muscle Abscess

Horses sometimes develop localized muscle soreness and swelling at the site of intramuscular injection, especially equine influenza injections, which can result in neck stiffness and a restricted forelimb gait. Signs usually resolve within 24 to 48 hours. Treatment is generally unnecessary, although hot packing and analgesia may be beneficial. More irritant drugs, such as iron injections, may result in the development of a sterile abscess.

The development of a single or multi-loculated abscess cavity filled with malodorous material is usually a sequel to an intra-muscular injection of a variety of drugs administered within the past few weeks. Clinical signs include neck pain and stiffness, localized neck swelling with or without focal patchy sweating, and sometimes pyrexia. Diagnosis is based on the history and clinical signs and can be confirmed by ultrasonography. The abscess cavity is usually filled with anechogenic material, surrounded by a hyperechogenic abscess wall.

Treatment is by surgical drainage, which is easily performed in the standing horse. The abscess cavity should be thoroughly lavaged. Systemic anti-microbial drugs usually are not required, unless clostridial myositis is suspected based on the fulminant nature of the condition (see Chapter 84). The prognosis for most horses with muscle abscesses is good, provided that adequate drainage is established.

Osteoarthritis

Anatomical studies have shown that approximately 50% of normal mature horses have some unilateral or bilateral modeling of the synovial facet joints between the sixth and seventh cervical vertebrae.. The modeling often is accompanied by extension of fibrocartilage across the cranial border of the dorsal arch of the seventh cervical vertebra and irregular enlargement of the articular processes.[4] The spinous process of the seventh cervical vertebra may become flattened or fragmented by contact with the sixth cervical vertebra when the neck is extended. Radiographically these changes result in irregularity of the normally smooth outline of the synovial articulations. Similar modeling changes also occur in the synovial articulation between the sixth and seventh cervical vertebrae.[19] A bony knob may develop on the ventral aspect of one or both cranial articular processes at the articulations between the fifth and sixth cervical vertebrae and between the sixth and seventh cervical vertebrae. When well developed, this knob forms a buttress that impinges onto the body or the arch of the more cranial vertebra and forms a false joint. The buttress partially obliterates the intervertebral foramen, but it is often of no clinical significance.[3,4]

The potential exists for large amounts of new bone associated with osteoarthritis of the cervical synovial articulations

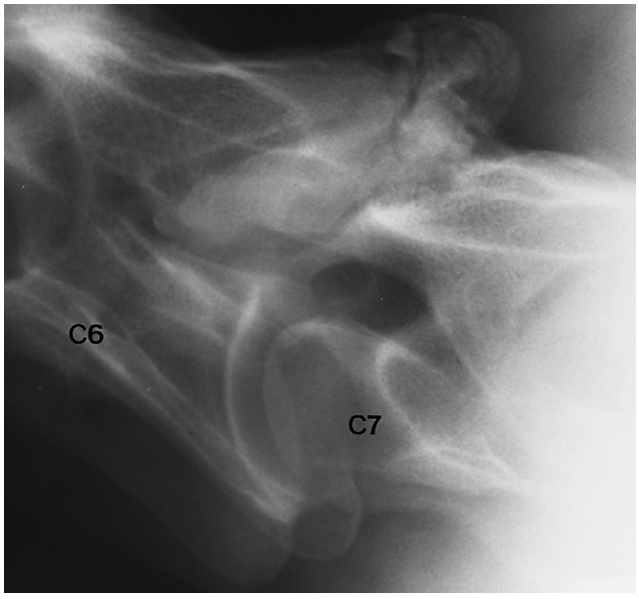

Fig. 55-7 Slightly oblique lateral view of the caudal cervical region of a 7-year-old riding horse with mild right forelimb lameness and an abnormally low head and neck carriage. Lameness was unaltered by comprehensive diagnostic analgesia of the right forelimb. There is extensive modeling of the articular facet joints between the sixth (C6) and seventh (C7) cervical vertebrae, with a large radiopaque mass with heterogeneous opacity extending dorsally. Obtaining a true lateromedial projection was not possible. The obliquity of the radiograph makes it difficult to assess the intervertebral foramen. Post-mortem examination revealed compression of the seventh cervical spinal nerve at the level of the foramen.

to encroach axially into the vertebral canal, resulting in compression of the spinal cord and hindlimb weakness and ataxia, or into the intervertebral foramen, resulting in nerve root compression, with local or referred pain and possibly lameness and patchy sweating. Severe osteoarthritic change may progress to partial or complete fusion and thus neck stiffness. With enlargement of a synovial articulation also generally comes enlargement of the joint capsule, and synovial outpouchings or cysts may develop, which may impinge on the spinal cord.

Radiographic abnormalities associated with osteoarthritis include enlargement of one or more of the articular facets and alteration in joint space width (Fig. 55-7). Widening of the joint space is usually associated with asymmetrical facet enlargement; narrowing is caused by articular cartilage degeneration. Pitted lucent zones may develop in the articular facets, with extension of the dorsal laminae between adjacent vertebrae and partial or complete obliteration of the adjacent intervertebral foramina. Sometimes fractures are seen dorsal to a joint. Abnormalities often develop on the left and right sides but are frequently asymmetrical. With substantial asymmetry of the synovial articulations the affected and immediately more cranial vertebrae may appear rotated on a lateral radiographic view, although the horse had appeared to be standing with its head and neck straight in the sagittal plane. Radiographic examination from left to right and from right to left can help to determine on which side a unilateral lesion is present. A lesion that is close to the cassette is clearer and magnification is less than if the lesion is on the opposite side of the neck.

The clinical significance of osteoarthritis of one or more synovial articulations can be difficult to determine by clinical and radiographic examinations alone, except by exclusion. The greater the degree of abnormality and the larger the number of articulations involved, the more likely the condition is clinically significant. In normal horses finding osteoarthritic change cranial to the articulation between the fifth and sixth cervical vertebrae is rare. Unilateral forelimb lameness has been seen with lesions between the fourth cervical and first thoracic vertebrae.[2] Nuclear scintigraphic examination may give further information in horses with neck stiffness or forelimb lameness apparently not referable to the limb itself.

Seven of eight horses with forelimb lameness associated with radiographic abnormalities of the cervical vertebrae also had subtle to obvious signs of neck pain.[2] Patterns of muscle atrophy in the neck and shoulder regions varied. The character of lameness varied. Radiographic abnormalities included significant modeling of the synovial articulations in the caudal neck region in three horses; a fourth had modeling and a fracture involving the synovial articulation between the fourth and fifth cervical vertebrae. One horse had abnormalities of the intercentral articulation between the seventh cervical and first thoracic vertebrae and a discrete mineralized fragment dorsal to it. Large lucent zones were identified in a vertebral body (the fourth and sixth cervical vertebrae) in two horses. A fracture of the vertebral body of the seventh cervical vertebra was seen in one horse.

Nerve root impingement in the caudal neck may cause radicular or referred pain and account for forelimb lameness. Neck pain itself can also cause forelimb lameness. Compression of the seventh cervical nerve was confirmed post mortem in a horse with osteoarthritis of the articulations between the sixth and seventh cervical vertebrae and between the seventh cervical and first thoracic vertebrae.[2] Nerve root compression with severe osteoarthritis has also been demonstrated using contrast-enhanced CT.[6]

In horses with neck stiffness or forelimb lameness associated with osteoarthritis of cervical synovial articulations, the response to rest and treatment with NSAIDs has been limited. Local peri-articular or intra-articular infiltration of corticosteroids, performed using ultrasonographic guidance, may bring temporary relief. Epidural injection of corticosteroids performed with the horse under general anesthesia resulted in relief of neck stiffness in a horse with osteoarthritis of the synovial articulation between the fourth and fifth cervical vertebrae and clinical evidence of nerve root compression.[23]

Osteoarthritis, especially in the caudal neck region, may result in associated enlargement of the joint capsule(s) and subsequent pressure on the spinal cord. In horses with mild osteoarthritis obvious ataxia may not be seen, but the history may include the tendency to stumble or to knuckle behind. Such signs often have been attributed to lameness but are invariably unaltered by diagnostic treatment with NSAIDs. Clinical signs may be subtle and intermittent. Such horses usually show abnormal weakness if pulled to one side by traction on the tail while the horse is walking, the sway test. A normal horse easily may be pulled off line once, but then strongly resists. A weak horse can be pulled off line repeatedly. Weakness may also be apparent as the horse decelerates from trot to walk, with exaggerated up and down movement of the hindquarters. This may result in irregular movement of each patella and may be confused with mild intermittent upward fixation or delayed release of the patella (see Chapter 47, page 459, and Chapter 49, page 475). At faster speeds the horse may look completely normal. In a young horse, advanced osteoarthritis of one or more caudal cervical synovial articulations is strong circumstantial evidence of cause and effect (Fig. 55-8). Definitive diagnosis in an older horse is much more difficult, because radiographic evidence of osteoarthritis may be present without associated clinical signs. Myelography may

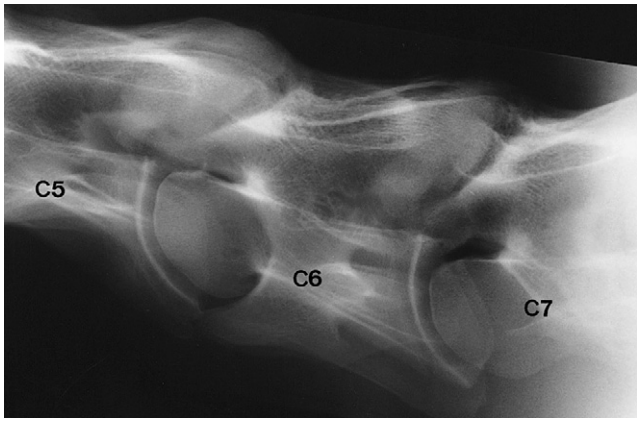

Fig. 55-8 Lateral radiographic view of the caudal cervical vertebrae of 4-year-old dressage horse with mild hindlimb weakness and a tendency to stumble behind, especially in downward transitions from trot to walk. The synovial articulations between the fifth (C5) and sixth (C6) cervical and sixth and seventh (C7) cervical vertebrae are enlarged greatly (compare with Fig. 55-2, *A*). The joint spaces are widened, reflecting asymmetrical modeling of the articular processes. There is a ventral buttress at the articulation of the sixth and seventh cervical vertebrae, with reduction in size of the intervertebral foramen.

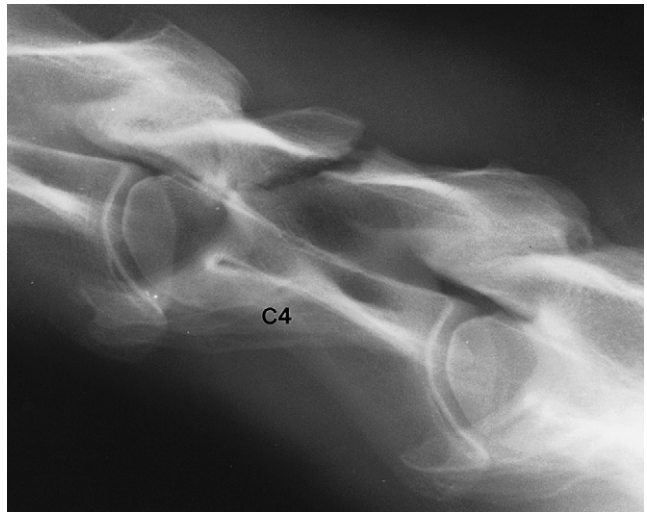

Fig. 55-9 Lateral radiographic view of the third to fifth cervical vertebrae of 9-year-old Dutch Warmblood dressage horse with severe neck pain, stiffness, and incoordination after a fall 12 days previously. The fourth cervical vertebra (C4) has a comminuted, slightly displaced fracture of the dorsal arch. Ataxia resolved within another 14 days, and the horse made a complete functional recovery.

help.[24] The prognosis is poor, and the horse may be potentially unsafe to ride.

Diskospondylitis

A survey of the cervical intervertebral disks of 103 horses from birth to 23 years of age confirmed that they consisted solely of fibrocartilage, with no nucleus pulposus.[25] Significant age-related degenerative changes were identified, but even with severe disintegration of the disks, no referable clinical signs had been apparent.

Diskospondylitis is a rare cause of neck pain, forelimb lameness, or ataxia. Although diskospondylitis is usually an infectious condition in dogs, no proven relationship occurs in the horse, and trauma may be an inciting cause. Lesions in the horse have been identified in the caudal neck region (the articulations between the sixth and seventh cervical vertebrae and the seventh cervical and first thoracic vertebrae) in association with severe neck pain and a bilaterally short, stiff forelimb lameness or episodic, unilateral forelimb lameness. High-quality radiographs of the caudal neck region are required for accurate diagnosis. Scintigraphic examination may help to localize the affected joint(s). The prognosis is guarded, although a broodmare that was presented with profound neck pain and periodic severe left forelimb lameness, associated with roughening of the end plates at the intercentral articulation between the sixth and seventh cervical vertebrae and narrowing of the intercentral space, after a collision with a fence, made a spontaneous recovery.[26]

Fracture

Fractures of the cervical vertebrae usually result from trauma: the horse rearing up and falling over backward or sideways, the horse pulling back when tied up, or falling while jumping, usually at speed. Clinical signs are sudden in onset and include holding the neck in an abnormally low position, stiffness, a focal or more diffuse area of pain, with or without localized or more diffuse soft tissue swelling, and muscle guarding. Audible or palpable crepitus is sometimes detected. The horse may be unable to lower its head to the ground or only able to do so by straddling of the forelimbs (see Fig. 55-1, *A*). Associated hindlimb and forelimb ataxia may be apparent, which can be transient and self-resolving, or persistent. Patchy sweating and localized muscle atrophy may develop. Occasionally an associated unilateral or bilateral forelimb lameness occurs.

Diagnosis is confirmed radiographically (Figs. 55-1, *A*, and 55-9). Most fractures are detectable on lateral views, although ventrodorsal views give additional information about the extent of the fractures, especially those involving the atlas or axis. Care should be taken not to confuse physes and separate centers of ossification with fractures.

The prognosis depends on the site and configuration of the fracture(s), the degree of displacement, and hence the likelihood of permanent compression of the spinal cord, either by a displaced fracture or by subsequent callus formation.

Fractures of the atlas and axis, especially through the physis of the separate center of ossification of the dens, are particularly common in foals. The prognosis for complete recovery is fair with conservative management, provided that no evidence of ataxia exists. Usually no treatment is required other than confinement to a box or small pen.

Fractures of the cervical vertebrae in adults more commonly involve the vertebral body or arch in the mid-neck region (the third to sixth cervical vertebrae) or the synovial articular facets of the more caudal vertebrae (the fifth to seventh cervical vertebrae). Local hemorrhage and edema may result in ataxia, which usually resolves within a few days. Persistence of ataxia warrants a guarded prognosis. Most fractures heal by callus formation, and this may subsequently impinge on the spinal cord, causing later ataxia. A fracture of a vertebral body also may result in damage to the adjacent intervertebral disk and associated ligaments, which subsequently may protrude into the vertebral canal and cause ataxia. Thus in the acute stage giving an accurate prognosis may be difficult. However, many fractures of the vertebral bodies and synovial articulations do heal, and horses may be able to return to athletic function although residual neck stiffness may be present.

In the acute stage the horse should be confined to box rest. Analgesics may be necessary to control severe pain, but they should be used judiciously to avoid encouraging excessive

movement of the neck. The position of the water bucket and manger should be adjusted so that the horse can drink and eat from normal head height. The hay should be fed at a height level with the head, preferably loose, or if in a hay net, a net with large holes, with the hay well shaken first. The horse should not be tied up during the convalescent period in case it pulls back. Re-appraising the horse clinically and radiographically every 6 to 8 weeks is helpful. Maximum clinical improvement may not be seen until 6 to 9 months after injury. Selected fractures may require surgical stabilization.[7]

Myeloma

Myeloma is a myeloproliferative disorder that can cause radiolucent lesions in any bone, including the cervical vertebrae, with associated bone pain.[19,27] Cervical vertebral myeloma was diagnosed in several horses of a wide range of breeds and ages.[19] Clinical signs included intermittent pyrexia, severe neck pain and stiffness, episodic forelimb lameness, weight loss, and a variety of other abnormalities. Diagnosis is based on hematological, radiographic, and bone biopsy examinations. Hematological abnormalities include anemia, leukocytosis, neutrophilia, and lymphocytosis. Total protein concentration is elevated greatly. Protein electrophoresis shows a significant monoclonal peak in the gamma region. Radiographic examination of affected bones reveals clearly demarcated lucent zones, usually without a sclerotic rim. Bone biopsy is useful to confirm the diagnosis, but currently no treatment is available and the prognosis is hopeless.

Other Cyst-like Lesions in Cervical Vertebrae

Occasionally, single or several well-demarcated radiolucent zones have been identified in one or more adjacent vertebrae, associated with profound neck pain, with or without forelimb lameness.[28] These lesions have not been proved to be caused by osteomyelitis or myeloma, although a definitive diagnosis has not always been possible by bone biopsy or post-mortem examination. One horse with extremely severe neck pain and forelimb lameness had radiolucent zones in the fifth and sixth cervical vertebrae, and bone biopsy revealed accumulation of abnormal plasma cells, but the horse made a most spectacular and complete recovery after exploratory surgery and returned to international show jumping. A show pony had a large cyst-like lesion in the fourth cervical vertebrae with profound neck pain and left forelimb lameness (Fig. 55-10). Post-mortem examination revealed a cavity filled with granulation tissue, surrounded by a large area of bone necrosis, but no suggestion of the underlying cause.

Vertebral Osteomyelitis

Cervical vertebral osteomyelitis usually occurs secondary to a systemic disease such as *Rhodococcus equi* infection in foals, *Streptococcus equi* infection (strangles), tuberculosis, or brucellosis; or as an extension of soft tissue infection[4]; or through hematogenous spread. Clinical signs may include pyrexia, neck stiffness or pain, and an abnormal neck posture, poor appetite, and weight loss. Usually leukocytosis, neutrophilia, and hyperfibrinogenemia are also present. Radiographic examination of the cervical vertebrae may reveal focal radiolucent zones, with or without surrounding sclerosis, in one or more vertebrae.[4] Useful diagnostic tests include bone biopsy, tuberculosis skin testing, and measurement of *Brucella* titers. Aggressive anti-microbial treatment may result in amelioration of clinical signs, but the prognosis is guarded.

Jugular Vein Thrombophlebitis

Thrombophlebitis of the jugular vein is a common condition associated with intravenous injection and chemical irritation, mechanical trauma to the vessel wall through catheterization, or a coagulation disorder. The vein feels abnormally hard, but unless the left and right sides are affected, the thrombus is infected, or thromboemboli settle elsewhere, usually no other clinical signs are apparent.

Occasionally a long length of the jugular vein is occluded by a thrombus in which bacteria are seeded, resulting in infectious thrombophlebitis. Clinical signs include neck stiffness and pain, localized heat and swelling, and pyrexia. If a long length of the vessel is involved, ipsilateral swelling of the head may occur. Diagnosis is based on clinical signs and ultrasonography.[5] A heterogeneous, cavitating echogenic thrombus can be seen in the jugular vein. Usually leucocytosis, neutrophilia, and hyperfibrinogenemia are present. A horse with a localized, small infected thrombus usually responds well to systemic anti-microbial therapy, but with more extensive lesions or associated toxemia or bacteremia, surgical ligation and removal of the vein may be required, combined with systemic anti-microbial treatment.

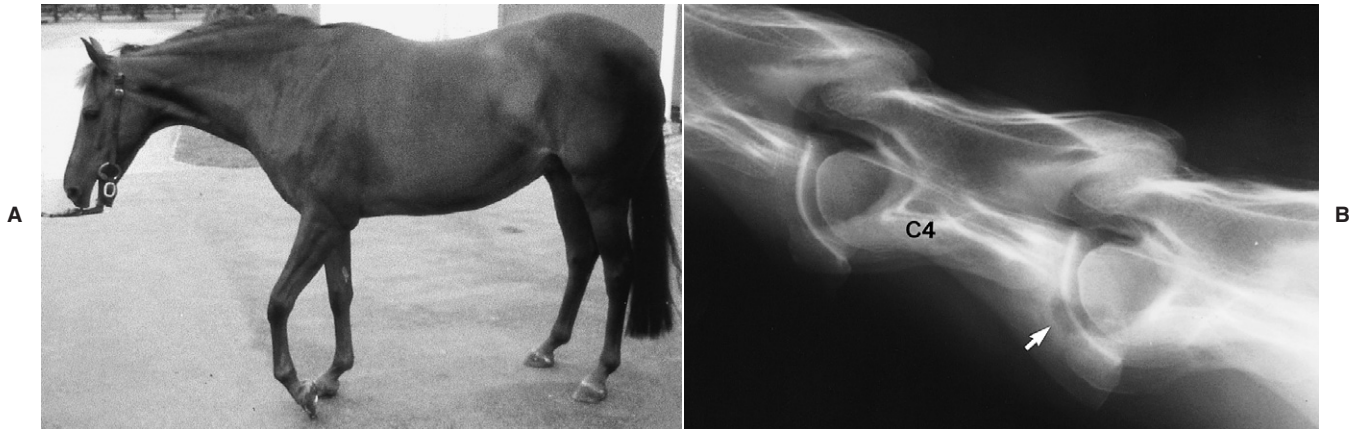

Fig. 55-10 **A,** A 13-year-old show pony with exquisite neck pain, worse on the left side, and episodic severe left forelimb lameness. The pony showed a shortened cranial phase of the stride at the walk, and periodically the pony stopped, holding the limb in a semi-flexed position, and was reluctant to move forward. There was significant hyperesthesia of the caudal half of the left side of the neck. **B,** Lateral radiographic view of the mid-neck region of the same pony. A large radiolucent zone (*arrow*) is visible in the caudal aspect of the vertebral body of the fourth cervical vertebra (*C4*). There is a loss of continuity of the caudal cortex.

Inadvertent perivascular injection of an irritant drug, such as phenylbutazone, may result in the rapid development of localized pain and swelling caused by chemical irritation. This may be followed by an aggressive inflammatory response, leading to sloughing of the skin. If perivascular injection is suspected, then the area should be treated by local infiltration with a balanced electrolyte solution (1 L) to dilute the drug, combined with local anesthetic solution (10 to 20 ml mepivacaine) to reduce pain. Periodic hot packing seems to ameliorate clinical signs. If initially untreated and local tissue necrosis supervenes, a skin slough is almost inevitable, and prophylactic antimicrobial therapy may be indicated.

Neck Stiffness and Cervical Vertebral Mobilization Under General Anesthesia

Assessment of neck flexibility has been described (see page 523). Restricted neck mobility associated with other clinical signs suggestive of neck pain, for example, reluctance to accept the bit, may be an indication for cervical vertebral mobilization under general anesthesia (see Chapter 98). The aim is to assist in restoring normal function to soft tissue components of joints. However, horses with pre-existing osteoarthritic changes are unlikely to respond. Repeated or maintained end-of-range passive joint movements may lower intra-articular pressure, inhibit reflex muscle contraction around a joint, and reduce muscle tension on the periarticular soft tissues and thus relieve pain. The manipulations are performed with the horse in left and right lateral recumbency under general anesthesia. A series of nine manipulations are performed in sequence to include extension of the head and neck, extension with rotation to the left and then to the right, rotation of the head to the left and to the right, flexion of the neck to the left and to the right, and flexion and rotation in each direction. For each maneuver the head and neck are moved to a position at the end of the resistance-free range. Constant pressure then is applied so that movement through the stiff and reduced range can be initiated. The pressure then is maintained until the joints and associated soft tissues move through the range of resistance. When movement ceases, a new end range is established and pressure is released. The maneuver may then be repeated. After treatment, horses are rested for 5 days and then start light work. Clinical improvement usually is appreciated within 2 weeks.[29] Repeated treatment may be necessary in selected horses. Occasionally, clinical signs deteriorate after treatment.

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CHAPTER • 56

Pathogenesis of Osteochondrosis

Janet Douglas

Osteochondrosis traditionally has been defined as a failure of endochondral ossification. The disease is caused by a disturbance of the normal differentiation of cells in growing cartilage and results in retention of growth cartilage (Fig. 56-1). This early lesion may be followed by necrosis of affected tissues and development of fissures that extend from the deepest layers of the lesion to the articular surface. Cartilaginous or osteochondral fragments may then detach from the parent bone, forming intra-articular fragments.[1] Once lesions extend to the articular surface, thereby causing inflammation of the joint, the condition may be referred to as *osteochondritis.*[2,3] The term *osteochondritis dissecans* usually is reserved for horses in which a dissecting flap of tissue is present[1] (Fig. 56-2).

Endochondral ossification is the process by which growing cartilage systematically is replaced by bone to form the growing skeleton. This process occurs at three main sites: the physis, the epiphysis, and the cuboidal bones of the carpus and tarsus. Chondrocytes in the physis can be divided into a series of layers or zones (Fig. 56-3). The zone farthest from the metaphysis is known as the resting or reserve zone. Adjacent to this is the proliferative zone, in which chondrocytes divide. These cells progress to the hypertrophic zone, in which they enlarge and form ordered columns. During this stage the chondrocytes become surrounded by an increasing amount of extracellular matrix that gradually becomes mineralized in the zone of provisional calcification. The chondrocyte columns then are invaded by metaphyseal blood vessels and bone forms on the residual columns of calcified cartilage. This mixture of calcified cartilage and immature bone (primary spongiosa) then gradually is remodeled to produce the mature bone of the metaphysis.[4] Early descriptions characterized osteochondrosis by a lack of chondrocyte differentiation that prevented provisional calcification of the matrix and invasion of the cartilage by blood vessels.[1] As a result, endochondral ossification is prevented and cartilage is retained. Endochondral ossification, which continues throughout the period of growth, also occurs in the articular-epiphyseal cartilage complex at the ends of long bones (Fig. 56-4).[5] The chondrocytes of the articular-epiphyseal cartilage complex that are closest to the articular

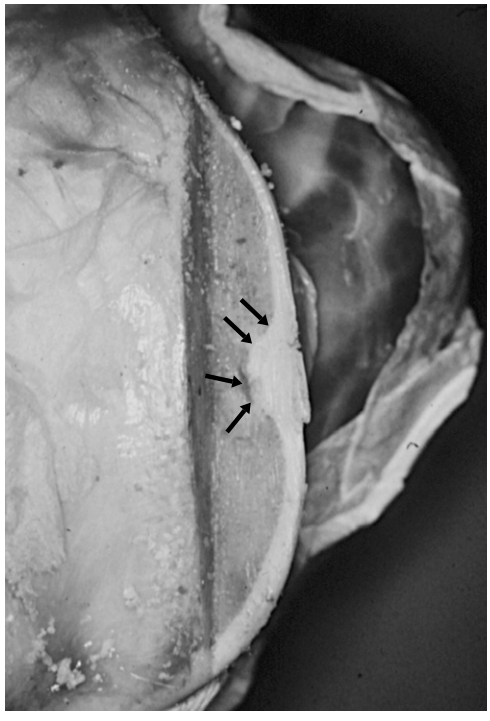

Fig. 56-1 Section through the articular-epiphyseal cartilage complex and epiphysis showing thickened retained cartilage *(arrows)*. (Courtesy Equine Orthopaedic Research Group at the Department of Clinical Veterinary Medicine, University of Cambridge.)

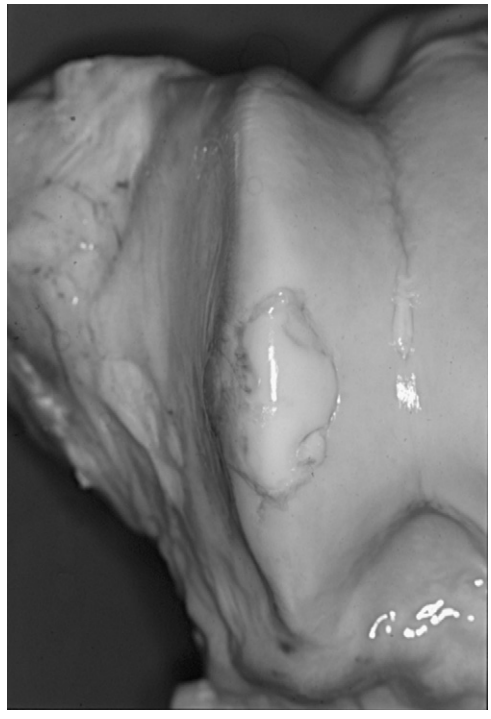

Fig. 56-2 Dissecting flap of articular cartilage (osteochondritis dissecans lesion) on the lateral trochlear ridge of the distal femur. (Courtesy Equine Orthopaedic Research Group at the Department of Clinical Veterinary Medicine, University of Cambridge.)

surface produce articular cartilage. Chondrocytes closer to the epiphysis participate in endochondral ossification in the same manner as occurs in the physis. The growth cartilages of the physis and the articular-epiphyseal cartilage complex are both susceptible to osteochondrosis.[1,6-8]

Because osteochondrosis is primarily a defect of cartilage, the suggestion was made that the condition be referred to as *dyschondroplasia*.[1] Dyschondroplasia may be preferable,[5,9] but the term osteochondrosis remains in widespread use and in this chapter refers to the primary lesion. Dyschondroplasia is used only when referring to work by authors who prefer this term.

A number of inconsistencies have been highlighted between the traditional definition of equine osteochondrosis and findings in horses with the disease.[10] For example, osteochondrosis has been identified in sites where endochondral ossification has ceased,[11] and osteochondral fragments that are thought to result from osteochondritis dissecans do not always show evidence of thickened cartilage.[12] In addition, defective endochondral ossification is not the only cause of cartilage thickening and hypertrophy.[13] As a result of these and other observations, it was suggested that a number of other mechanisms should be considered when an osteochondritic lesion is identified.[10] These include loss of blood supply (e.g., resulting from biomechanically induced shearing of cartilage canals or blunt trauma to cartilage caused by rapid locomotion or incoordination), congenital deformities leading to excessive biomechanical force at one site, and conditions such as copper deficiency that lead to connective tissue fragility.

CHARACTERISTICS OF OSTEOCHONDROSIS LESIONS

The lesions of equine osteochondritis dissecans were described as early as 1947.[6] Since then the gross and histological characteristics of the manifestations of osteochondrosis have been described and defined by numerous authors. The end result is a wide-ranging, heterogenous, and somewhat confusing body of literature, in which osteochondrosis has been ascribed to genetic, dietary, endocrine, biomechanical, traumatic, vascular, and toxic causes.[10,14-19] The confusion

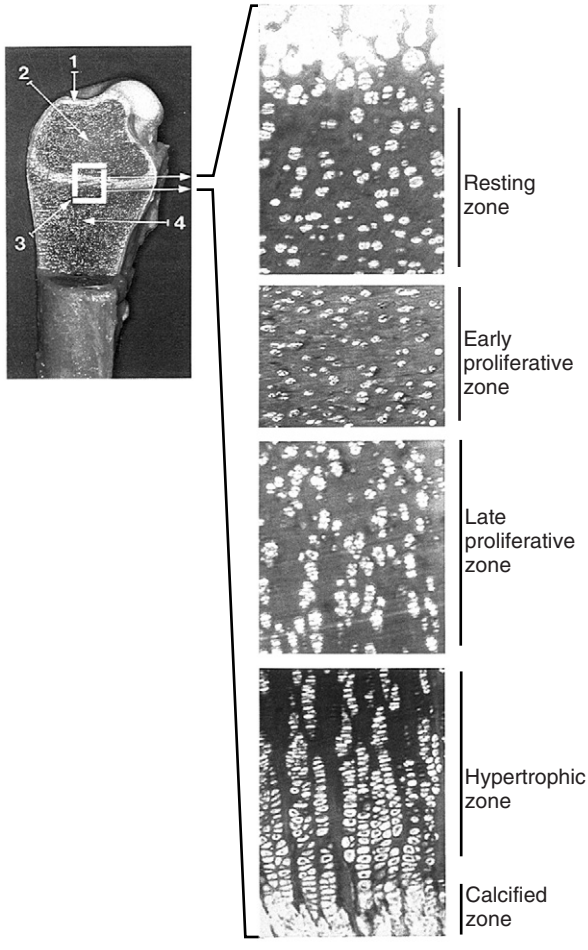

Fig. 56-3 Sagittal section of the epiphysis, metaphysis, and diaphysis of growing bone, with sequential histological sections showing the different zones of the physis (metaphyseal growth cartilage). *1*, Articular-epiphyseal growth cartilage complex; *2*, secondary ossification center in the epiphysis; *3*, metaphyseal growth cartilage; *4*, primary ossification center in the diaphysis. Toluidine blue stain. (Courtesy Equine Orthopaedic Research Group at the Department of Clinical Veterinary Medicine, University of Cambridge.)

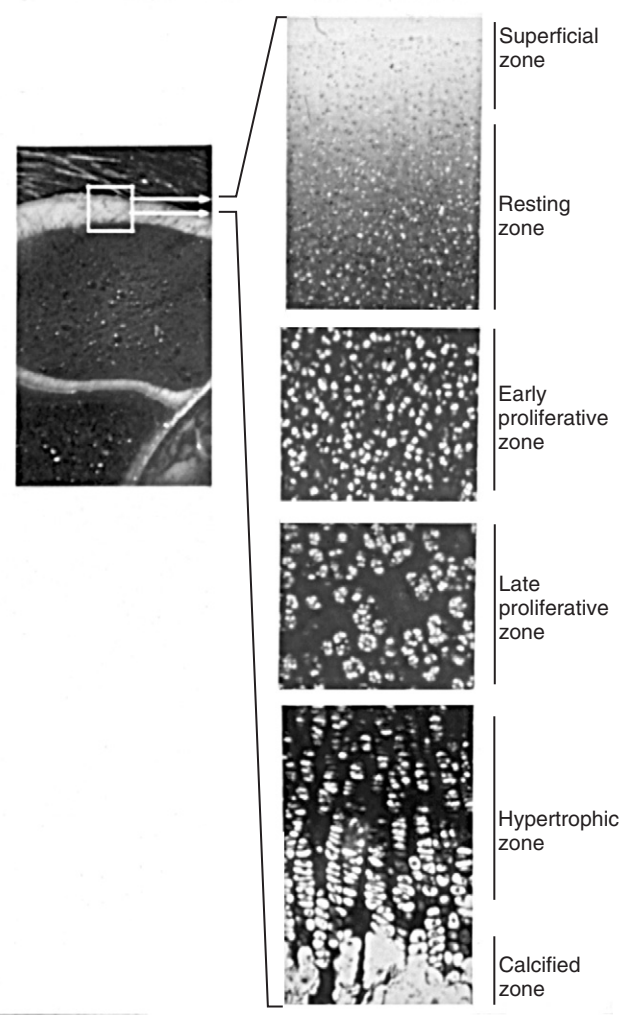

Fig. 56-4 Sagittal section of the epiphysis with sequential histological sections showing the different zones of the articular-epiphyseal cartilage complex. Toluidine blue stain. (Courtesy Equine Orthopaedic Research Group at the Department of Clinical Veterinary Medicine, University of Cambridge.)

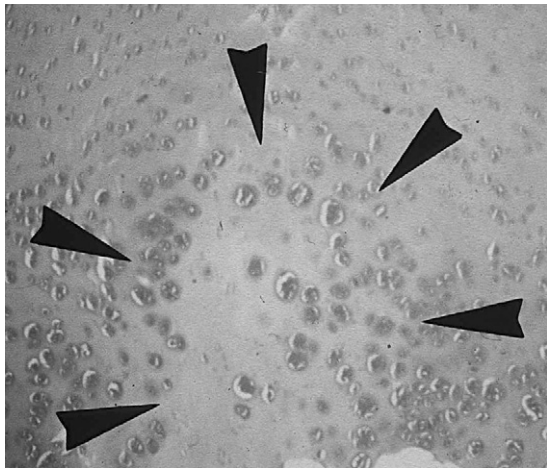

Fig. 56-5 Chondrocyte clusters *(arrows)* surrounding an area of necrotic cartilage. Toluidine blue stain. (Courtesy Equine Orthopaedic Research Group at the Department of Clinical Veterinary Medicine, University of Cambridge.)

stems from a number of sources. First, defining osteochondrosis on anything other than clinical, gross pathological, and histological criteria is currently not possible. Moreover, histological definitions and descriptions vary and accurate identification of lesions can be difficult.[20] Even chondrocyte clusters (Fig. 56-5), considered by many to be one of the most consistent findings in osteochondrosis lesions,[18,20] are not pathognomonic for the disease.[20] In addition, cartilage and bone have a limited repertoire of responses to injury and abnormalities of development,[10] and many lesions are not identified until they have reached the chronic stage. Differentiation among osteochondral lesions with different pathogeneses can be difficult to impossible.[10,21]

Henson et al.[20] attempted to define osteochondrosis lesions histologically by studying articular-epiphyseal cartilage complex samples from the lateral trochlear ridge of the femur of horses ranging in age from 270 days of gestation to 4 years. Their definition of dyschondroplasia was the presence of cartilage cores (cartilage extending into subchondral bone). Any joint exhibiting secondary pathological changes was excluded. Among the 27 lesions examined, two types were identified. Lesions in group A showed disruption of the normal sequential transition of chondrocytes through the stages of proliferation and maturation. These lesions were characterized by accumulation of large numbers of small, rounded chondrocytes, apparently arrested at the pre-hypertrophic stage. Lesions in group B, not previously described, showed alteration in the staining pattern of mineralized cartilage and adjacent subchondral bone and the complete absence of invading capillaries into newly formed bone.[20,22] The two lesion types were also characterized by differences in the degree of type VI collagen immunoreactivity. Retained blood vessels, chondrocyte clusters, chondronecrosis, and fissure formation were present in both types. The suggestion therefore was made that equine dyschondroplasia may result from more than one causative mechanism, and that lesions resulting from different pathogeneses may be differentiated on the basis of histological and immunological studies.[20]

From a clinical standpoint, equine osteochondrosis characteristically presents as one or two lesions manifesting in known predilection sites. In this so-called typical pattern of the disease, lesions are often bilaterally symmetrical, although only one lesion may cause clinical signs.[10] The femoropatellar (lateral and medial femoral trochlear ridges, lateral facet of patella), tarsocrural (distal intermediate ridge and medial malleolus of tibia, lateral and medial trochlear ridges of talus), scapulohumeral (glenoid fossa and humeral head), and metacarpophalangeal/metatarsophalangeal joints (mid-sagittal ridge and condyles of the third metacarpal or metatarsal bone) are affected most commonly. Osteochondrosis of the elbow, hip, and cervical vertebral joints also has been described,[10,23] but lesions in these sites are less common and etiology is more controversial.[10] This typical pattern of osteochondrosis contrasts with that of the atypical pattern in which animals show multiple articular (and sometimes physeal) lesions.[10,21] Predilection and non-predilection sites may be affected in these horses, and bilaterally symmetrical lesions are absent or infrequent. A third pattern of lesion distribution, the mixed pattern, describes horses in which typical and atypical lesions are present.[21]

RELATIONSHIP BETWEEN PHYSEAL DYSPLASIA (PHYSITIS), SUBCHONDRAL BONE CYSTS, AND OSTEOCHONDROSIS

The relationship between physeal dysplasia (physitis or epiphysitis) and osteochondrosis remains poorly defined. Osteochondrosis lesions of the physis have been described,[3,7] and retained cartilage long has been regarded as a possible cause of physeal dysplasia.[24,25] In foals with experimentally induced osteochondrosis, physes and articular-epiphyseal cartilage complexes may be affected.[18,26] Because both these sites contain growth cartilage, this accords with the definition of osteochondrosis as a generalized disturbance of endochondral ossification. Some have questioned whether articular and physeal lesions have the same cause, however, and proposed that physitis be regarded as a manifestation of developmental orthopedic disease, not osteochondrosis.[10] The relationship between the two conditions remains unclear. Physitis is covered in more detail in Chapter 59 and is not discussed further here.

The relationship between osteochondrosis, subchondral bone cysts, and osseous cyst-like lesions is also controversial. Originally interpreted as a manifestation of retained cartilage of the articular-epiphyseal cartilage complex,[7,27-29] many cysts now are thought to be mechanical in origin.[30-32] Subchondral bone cysts of the medial femoral condyle (a predilection site) can be induced by creating a linear slit in the articular cartilage and then allowing normal weight bearing.[31] Osteochondrosis thus may be only one of several possible causative mechanisms,[10,33,34] and cysts may result from a number of non-specific articular injuries sustained at a load-bearing site.[10]

In the past decade, as knowledge of pathogenesis has grown and more sophisticated techniques have been applied to study osteochondrosis, osteochondral lesions have been classified more carefully. Until the pathogenesis is understood fully and osteochondral lesions can be classified according to cause, some degree of confusion is probably inevitable. The following discussion summarizes major findings relating to the pathogenesis of equine osteochondrosis.

PROPOSED CAUSATIVE FACTORS

Body Size and Growth Rate

Until recently, little objective evidence existed to support the widespread belief that osteochondrosis is more prevalent in large horses and those with a rapid growth rate. This theory gained support based on the results of a survey of 77 Standardbred (STB) foals born and raised over two seasons on one Swedish farm. Skeletal growth and weight gain were measured from the time of birth to 16 months of age, and the hock and fetlock joints were monitored radiographically for osteo-

chondral abnormalities. A positive relationship existed between osteochondrosis of the tarsocrural joint and body weight at birth, body weight during the growth period, average daily weight gain, and skeletal frame size. Seven of the eight foals that developed tarsocrural osteochondrosis were sired by the same stallion (a total of 14 stallions sired the progeny in this study), but the relationship between osteochondrosis, body measurements, and growth rate was still present when affected and unaffected foals by this sire were considered in isolation. No relationship existed between body size or growth rate and osteochondral lesions in the fetlock joint (palmar/plantar osteochondral fragments or ununited palmar/plantar eminences). The conclusion was that tarsocrural and fetlock osteochondral lesions have different pathogeneses.[35] This supports the general consensus that palmar/plantar osteochondral fragments and ununited palmar/plantar eminences are not a manifestation of osteochondrosis.[10,21,36]

A relationship between osteochondrosis, body size, and growth rate has also been documented in Warmbloods. Weight and height from the time of birth to 5 or 11 months of age were measured in 43 Warmblood foals with a presumed genetic predisposition to osteochondrosis of the femoropatellar or tarsocrural joints.[37] The stifle and hock joints of the foals were evaluated radiographically, macroscopically, and histologically. Development of femoropatellar osteochondrosis was associated with a higher overall rate of weight gain and greater final body weight and height. The period during which weight gain of the osteochondrosis-positive foals was significantly higher than that of the osteochondrosis-negative foals coincided with the period during which femoropatellar joint lesions become visible radiographically.[38] In contrast to the previously described findings in STBs,[35] no relationship existed between tarsocrural osteochondrosis and body size or growth rate in this population of Dutch Warmbloods.

Nutrition

The incidence of metabolic bone disease on 19 breeding farms in Ohio and Kentucky was recorded and correlated with the quality of the foals' ration. The authors scored the ration based on their own experience. Epiphysitis, distended joints, lameness, contracted tendons, and spinal ataxia (suspected or radiographically confirmed) were included in the diagnosis of metabolic bone disease. Radiographs did not form a routine part of the examination. This study thus gives no information about nutritional factors associated with osteochondrosis per se, but it does provide information on nutrition and skeletal development. A negative correlation existed between the incidence of metabolic bone disease and the quality of the ration.[39] Diets of farms with low incidence of skeletal problems tended to have higher calcium, phosphorus, and copper contents. Two points should be noted about the relationship between dietary copper content and the incidence of skeletal problems:

1. The correlation was mainly driven by two outliers comprising farms with relatively high dietary copper levels (approximately 30 to 40 ppm) and a low incidence of problems.
2. Three farms with a negligible level of available dietary copper did not have an excessively high incidence of problems.

The relationship between dietary zinc and skeletal problems was described as U-shaped, with an increased incidence of problems at low and high dietary zinc levels. No valid conclusion could be drawn concerning the relationship between dietary energy and the incidence of skeletal problems because energy levels were similar on all farms.

In a follow-up study the nutritional balance was improved on 17 of these farms and the incidence of metabolic bone disease was recorded in the following season.[40] Highly significant improvements in the incidence of orthopedic abnormalities occurred with the improvements in ration. These data suggest that sub-optimal nutrition adversely affects skeletal development but provide no information on the pathogenesis of osteochondrosis.

Digestible Energy and Protein

A high plane of nutrition has been implicated in the pathogenesis of osteochondrosis for many years.[7] The effects on growth plate morphology of over-feeding and under-feeding were described in 1984.[41] Twelve Thoroughbred (TB) weanlings aged 6 to 8 months were randomly assigned by age to three groups and were fed diets containing 70%, 100%, or 130% of the National Research Council recommendations (1978) for digestible energy and protein.[42] All diets contained 100% of the recommended level of calcium and phosphorus. After 8 months of these diets, biopsies of the distal radial growth plate showed that growth plate thickness was directly proportional to diet level. The growth plates of the over-fed horses showed many features similar to those described as osteochondrosis: the reserve and hypertrophic zones were enlarged, the hypertrophic cartilage had lost its normal columnar organization, and metaphyseal capillaries appeared unable to penetrate this abnormal hypertrophic cartilage. The morphological characteristics of the articular-epiphyseal cartilage complex were not assessed. The conclusion was that the lesions associated with over-feeding were similar to those caused by hypothyroidism and that the link between dietary excess and osteochondrosis is mediated by endocrine factors.[41]

More recent work has shown that the greater the amount of carbohydrate ingested by a young horse during a meal, the greater the magnitude and the shorter the duration of the ensuing increases in serum insulin, triiodothyronine (T_3), and thyroxine (T_4).[43] Transient post-prandial hypothyroxemia may thus be induced by diets high in energy. These findings suggested that the effects of insulin, T_3, and T_4 on growing skeletal tissues might be desynchronized by diet-induced alterations in the secretion and metabolism of these hormones, resulting in abnormal cartilage development.[14] The relationship between abnormal insulin levels and osteochondral lesions is supported by the finding that young horses with osteochondrosis have an exaggerated insulin response when fed a meal high in digestible energy.[44]

The effects on skeletal development of feeding 129% of National Research Council (NRC, 1989)[45] recommendations for digestible energy, or 126% of NRC recommendations for crude protein to foals approximately 5 months of age was investigated.[18] A control group received a diet containing 100% of NRC recommendations for energy and protein. The foals were allowed only limited exercise. After 12 to 16 weeks of the experimental diets, a comprehensive postmortem examination was performed on all foals. Growth plates and the growth cartilage of the articular-epiphyseal cartilage complex were examined, and a definitive diagnosis of dyschondroplasia was made only when a retained core of cartilage (i.e., disturbed endochondral ossification) was identified histologically. Gross lesions suggestive of dyschondroplasia were found in 1 (8%) of 12 control foals, 1 (17%) of 6 foals fed the high-protein diet, and 11 (92%) of the 12 foals fed the high-energy diet. Histological confirmation of dyschondroplasia occurred in 2 (17%) control foals, in 4 (67%) foals on the high-protein diet, and in all 12 foals on the high-energy diet. The lesions in the foals in the high-protein group were minor and were mainly single lesions of the growth plates, with the articular-epiphyseal cartilage complex left unaffected. No significant difference in incidence occurred between the control and high-protein groups. In contrast, many of the foals in the high-energy group had lesions of the articular-epiphyseal cartilage complex and the

growth plates, and the difference in incidence between the control and high-energy groups was significant.

Recent in vitro work suggests that insulin may promote the survival or depress the differentiation of chondrocytes in equine growth cartilage, thereby reducing the rate at which cells enter the terminal phases of hypertrophy. It was suggested that the resulting accumulation of pre-hypertrophic chondrocytes could reduce the rate of endochondral ossification, predisposing the animal to development of focal lesions.[46] This work provides evidence of a potential mechanism for the demonstrated links between dietary energy intake, hyperinsulinemia, and osteochondrosis.

Calcium and Phosphorus

The effects of over-feeding phosphorus (388% of NRC recommendations), calcium (342% of NRC recommendations), and calcium and digestible energy (342% and 129% of NRC recommendations, respectively) to foals aged 2½ to 6½ months were assessed.[26] The diets containing excessive phosphorus and calcium provided 100% of NRC recommendations for digestible energy. The foals were allowed only limited exercise. A comprehensive post-mortem examination was performed after 16 to 18 weeks of these diets. Histologically confirmed dyschondroplasia lesions were found in multiple joints and growth plates in 2 (17%) control foals, 5 (83%) foals fed excess phosphorus, 2 (33%) foals fed excess calcium, and 6 (100%) foals fed excess digestible energy and calcium. The lesions were more numerous and severe in the foals fed high-phosphorus, or high–digestible energy/calcium diets than in the control foals, or those fed high-calcium diets only. These findings show that dyschondroplasia is not induced by diets high in calcium and is not alleviated by excessive calcium in foals fed excessive energy. No mechanism for the apparent association between excessive dietary phosphorus and abnormal endochondral ossification was proposed.

Copper

Copper is a necessary co-factor of lysyl oxidase, the enzyme that catalyzes the oxidative deamination of lysine and hydroxylysine residues to the corresponding aldehydes. This is a necessary step in the formation of pyridinoline cross-links in collagen and elastin. Low-copper diets have been associated with an increase in the soluble fraction of articular collagen, reduced collagen cross-linking of cartilage and bone, and an increased incidence of osteochondral lesions in growing foals.[47,48]

An association between copper deficiency and equine osteochondral lesions was recognized for many years. Low serum copper levels were found in seven of eight foals with severe, generalized osteochondral abnormalities.[49] In some horses there was evidence that the copper deficiency was secondary to zinc toxicosis. Affected foals showed separation of articular and physeal cartilage from adjacent bone in the hypertrophic zone at many sites. In some joints the subchondral bone was almost denuded of cartilage. The lesions were thus much more widespread and severe than in a typical horse with naturally occurring osteochondrosis. Similar lesions were induced by experimental diets containing 1.7 ppm copper.[47] Early lesions were characterized by intrachondral splitting through the hypertrophic zone, and retention of cartilage was not a major feature of the pathological condition. As in an earlier report, lesions were widespread and severe.[49] Similar lesions develop in foals with zinc-induced copper deficiency.[50]

The effect on equine skeletal development of copper supplementation was studied during late gestation and the early growing period.[16] Twenty-one pregnant mares were assigned to a control group (diet containing 13 ppm copper) or a supplemented group (32 ppm) during the last 3 to 6 months of pregnancy. The foals were subsequently fed diets containing 15 ppm copper (control) or 55 ppm copper (supplemented) for up to 6 months. The dietary copper content of the control foals was close to NRC recommendations. Thickening of cartilage within the physis and lesions of the articular-epiphyseal cartilage complex demonstrating cartilage thickening (with or without separation of cartilage from subchondral bone and subchondral fibrosis) were defined as osteochondrosis. Compared with the foals given copper supplements, 6-month-old control foals had osteochondrosis nearly twice as frequently in the physes and more than five times as frequently in the articular-epiphyseal cartilage complex. Also, more than four times as many thickenings of the articular-epiphyseal cartilage complex occurred per foal in the control group compared with the group given supplements. Separation of cartilage from bone was observed in 13 lesions in the control foals and in none of the foals given supplements. The most notable lesions in the control group were cartilage thickening and separation of the distal intermediate ridge of the tibia.

The effects of 8 and 25 ppm dietary copper fed to 3-month-old foals for 6 months were compared. Cartilage and bone lesions were rare in the foals fed 25 ppm copper. In contrast, the majority of the foals fed 8 ppm copper were severely affected with many lesions. Cartilaginous flaps, and cartilage thinning, erosion, and eburnation composed the majority of the lesions identified at post-mortem examination. Lesions were most commonly found on the dorsal articular facets of the cervical spine. Many of the lesions were associated with microfractures within the physes and primary spongiosa of the long bones and cervical vertebrae. Biochemical analysis of tissues from four low-copper diet foals with osteochondritis dissecans–like lesions showed that they had significantly fewer pyridinoline cross-links in articular cartilage, physeal cartilage, and bone than a group of six foals with no osteochondritis dissecans lesions. The latter group comprised foals given copper supplements and two foals on low-copper diets. This demonstrated a link between low dietary copper, inferior collagen quality, and osteochondritis dissecans–like lesions.[48]

In these two cited studies, foals fed the lower amount of dietary copper typically had multiple lesions, many of which were present in the cervical vertebrae.[16,48] This lesion distribution would thus be described as atypical.[10,21] Biochemically the pathological condition induced was almost certainly secondary to copper deficiency.[48] Lesions were not caused by a primary failure of endochondral ossification but by insufficient dietary copper. However, these studies have shown that dietary copper levels of 8 to 15 ppm can induce lesions grossly and radiographically similar to those of osteochondrosis. Clinically, these lesions frequently may be diagnosed as osteochondrosis. How many horses with naturally occurring osteochondrosis are induced partly or completely by inadequate dietary copper is unknown, but these investigations show that close attention should be directed not only to the levels of copper in the diets of growing foals but also to the levels of known copper antagonists, such as zinc and molybdenum. Dietary copper is not a panacea, however. Relatively high levels of dietary copper (25 and 55 ppm) do not completely prevent osteochondral lesions,[16,48] and relatively low levels (11 ppm) do not always result in foals with multiple osteochondral abnormalities.[18] Copper is but one factor to which attention should be paid when faced with an increased incidence of osteochondral abnormalities in foals.

Heredity

There is no doubt that at least some manifestations of osteochondrosis have a genetic component. Heritability studies of equine osteochondrosis are still relatively scarce, however, and the true extent of the contribution made by genetic factors remains unknown, particularly in breeds other than the STB. Among Scandinavian STB populations, the heritability of tarsocrural osteochondrosis ranged from 0.24 to 0.52[17,51,52] (Table 56-1). These heritability coefficients describe the proportion of the total variation in incidence attributed to genetic

Table • **56-1**

Incidence and Heritability of Osteochondrosis Lesions

LESION	BREED	RANGE OF INCIDENCE AMONG PROGENY GROUPS (%)	HERITABILITY COEFFICIENT*	REFERENCE
Hock (lesions not defined)	Standardbred	0 to 8	—	Hoppe[53]
Hock (lesions not defined)	Warmblood	0 to 8	—	Hoppe[53]
DIR	Standardbred	3.4 to 30	0.26	Schougaard et al.[51]
Hock (lesions not defined)	Standardbred	0 to 24	0.24 to 0.27	Philipsson et et al.[17]
POF	Standardbred	11 to 44	0.17 to 0.19	Philipsson et al.[17]
DIR or LTR	Standardbred	0 to 69	0.52	Grøndahl and Dolvik[52]
POF	Standardbred	0 to 41	0.21	Grøndahl and Dolvik[52]

DIR, Distal intermediate ridge of tibia; *LTR*, lateral trochlear ridge of talus; *POF*, palmar/plantar osteochondral fragments in metacarpophalangeal and metatarsophalangeal joints.
*The heritability coefficient may over-estimate the true heritability if a high incidence of lesions exists in one progeny group (as in the study of Grøndahl and Dolvik[52]).

factors. Thus even the low end of this range indicates a substantial heritable component to the disease. Table 56-1 also shows the heritability of palmar/plantar osteochondral fragments of the fetlock joints. As discussed elsewhere in this chapter, whether these lesions are true manifestations of osteochondrosis is controversial. Nevertheless, data on the heritability of these lesions are presented here, in part for comparison with the heritability of hock osteochondrosis. Although a traumatic cause for palmar/plantar osteochondral fragments has been proposed, it is possible that trauma causes existing osteochondrosis lesions in this site to become clinically manifest.

The studies summarized in Table 56-1 provide an overview of the genetic contribution to tarsocrural osteochondrosis in STBs of three Scandinavian countries. Most of the progeny of nine stallions in Denmark were radiographed over the course of two breeding seasons.[51] Of the 325 yearlings evaluated, no more than two were provided by any one stud farm. The hock and fetlock joints of 793 randomly selected progeny of 24 Swedish STB trotting stallions were radiographed, including at least 29 offspring of each stallion.[17] An analysis of 644 Norwegian STBs used progeny groups of five or more from 39 stallions.[52] In all these studies the offspring of each stallion were reared in many different environments, strengthening the conclusion that differences among progeny groups were mainly genetically determined. In two of these studies, tarsocrural osteochondrosis had a stronger genetic component than palmar/plantar osteochondral fragments, with no significant relationship between the expression of lesions in these two sites.[17,52]

The data presented suggest that genetic selection against tarsocrural osteochondrosis and palmar/plantar osteochondral fragments is feasible in horses, but the relatively high incidence of lesions in the offspring of stallions free of osteochondrosis or palmar/plantar osteochondral fragments indicates that any selective breeding program would have to be based on progeny testing.[17,51] In addition, tarsocrural osteochondrosis and fetlock palmar/plantar osteochondral fragments appear to be inherited independently of each other.[17,52] These lesions therefore would have to be regarded as independent traits in any selection scheme.[17]

Gender

Early reports suggested that the incidence of equine osteochondrosis was substantially higher in males than females.[6,7] Non-significant relationships between male gender and osteochondrosis also have been reported in epidemiological studies, in which male/female incidence ratios of 1.6:1 and 1.4:1 were found.[53,54] However, in a controlled study of Warmbloods, no gender predisposition for osteochondrosis of the tarsocrural or femoropatellar joints was found.[37] In a recent study of feral horses (49% male, 51% female), osteochondrosis lesions were found only in fillies.[55] The evidence for an association between osteochondrosis and male gender is thus inconsistent.

Involvement of Cartilage Canals

Cartilage canals are channels containing arterioles, venules, and capillaries that assist in the nutrition of epiphyseal cartilage. Two recent studies have provided evidence that these structures may be involved in the pathogenesis of equine osteochondrosis.[19,56] The mechanisms of involvement proposed by the two sets of authors are different, however.

Gross, microradiographic, and histological examinations of three osteochondrosis predilection sites (medial femoral condyle, lateral femoral trochlear ridge, and distal tibia) were performed in 35 horses younger than 18 months of age. The distal end of the proximal phalanx, a non-predilection site, was also examined. The aims were to determine the age range over which cartilage canals are viable at these four sites and whether any association existed between these structures and the lesions of osteochondrosis. Cartilage canals containing patent blood vessels were present at all sites examined in foals younger than 3 weeks of age and were absent from all sites by 7 months of age. Overall, 34% of the animals had lesions of osteochondrosis and the prevalence rose to 56% in animals between 3 weeks and 5 months of age. All the lesions seen in this age group (3 weeks to 5 months) were associated with necrotic cartilage canal blood vessels. Lesions found in animals 7 months of age and older had extensive involvement of subchondral bone and bone marrow and were considered to be chronic. The conclusion was that osteochondrosis lesions develop before 7 months of age and that ischemic necrosis of cartilage is involved in the pathogenesis of the condition.[19]

The second study to determine an association between cartilage canals and equine osteochondrosis examined articular-epiphyseal cartilage complexes from various sites in 30 normal horses and 6 horses with dyschondroplasia. Normal horses ranged in age from 130 days gestation to 2 years.[56] Horses with lesions were younger than 15 months of age. Cartilage canals were present by 130 days of gestation and were never identified in normal articular-epiphyseal cartilage complex samples after 6 months of age. In five of the six osteochondrosis lesions, cartilage canals were associated with the retained cartilage. The cartilage canals in the region of these lesions contained patent vessels and were surrounded by chondrocytes that were unusually small for the position within the articular-epiphyseal cartilage complex.[22,56] In contrast to the previous study,[19] necrosis

was not a feature of these lesions. The conclusion was that under some circumstances chondrocytes adjacent to cartilage canals fail to hypertrophy, thereby leading to failure of endochondral ossification.[56] The authors proposed that patent blood vessels within cartilage canals may expose growth cartilage in the vicinity of the canal to any imbalance of systemic hormones, such as that induced by a high-energy diet. The authors suggested that chronicity of lesions accounted for the difference between these findings[56] and those of the previous study,[19] in which necrosis was found. These two studies suggest involvement of cartilage canals in the pathogenesis of equine osteochondrosis, but the mechanism remains to be determined.

Exercise

Conflicting evidence exists regarding the role of exercise in the pathogenesis of osteochondrosis. Increased incidence of osteochondrosis has been found in foals born later in the season,[17,35,54] and the suggestion was that the greater amount of exercise that these foals receive early in life may be partly responsible for the increased lesion frequency.[35] This hypothesis has not been supported by controlled studies, however.

The incidence of osteochondrosis in foals subjected to low-exercise and high-exercise regimens between the ages of 3 and 24 months was compared.[57] All foals were housed in groups or kept in boxes and were allowed to exercise for 2 to 4 hours per day in a paddock. In addition, the low-exercise group received 15 to 45 minutes of walking exercise per day. The high-exercise group received 15 to 45 minutes of walking and trotting, plus 8 to 20 gallop sprints of short duration (10 to 15 seconds). At the end of the study, hock and stifle joints were examined clinically and radiologically. Osteochondrosis was detected in only 3 (6%) of the 50 foals in the high-exercise group, but in 13 (20%) of the 66 foals in the low-exercise group, demonstrating a significant protective effect of high-intensity, short-duration exercise on the incidence of osteochondrosis.

These findings were not supported by the results of a recent extensive investigation into the effects of exercise on musculoskeletal development. Forty-three foals with a presumed genetic predisposition to osteochondrosis of the femoropatellar or tarsocrural joints were subjected to one of three exercise regimens: pasture exercise only, confinement to a box stall, or confinement to a box stall with an increasing number of gallop sprints. These exercise regimens were imposed from 1 week of age to 5 months, and the incidence of osteochondrosis including subchondral bone cysts at 5 and 11 months of age was determined. At 5 months of age, lesions were found in all foals.[58] Lesion frequency was highest in the tarsocrural joints (1.9 lesions per foal), with a lower incidence in the femoropatellar/femorotibial (1.0), cervical intervertebral (1.0), and metatarsophalangeal joints (0.6). Exercise did not influence the number of lesions, although the tendency was for lesions in the foals subject to box rest to be more severe. Exercise also appeared to influence lesion type and distribution within the stifle joint. The foals subjected to box rest developed lesions mainly in the femoral condyles (subchondral bone cysts), and the lesions in trained foals were mainly on the lateral trochlear ridge of the femur (osteochondrosis or osteochondritis dissecans). This difference may suggest an effect of mechanical loading on lesion development, because the lateral trochlear ridge is loaded by the patella during exercise,[10] and subchondral bone cysts tend to develop at the point of maximum load bearing during the support phase of the stride. It was concluded that exercise has no role in the pathogenesis of osteochondrosis, although exercise may alter the appearance and distribution of lesions.[33,34,58] By 11 months of age the prevalence of lesions had decreased significantly. The decrease was most dramatic in the femoropatellar/femorotibial joints. This study thus showed that many osteochondrosis lesions repair as foals develop, never becoming clinically apparent,[58] and also suggests that most osteochondrosis lesions develop before 5 months of age.

Trauma and Biomechanical Force

The roles of trauma and biomechanical force in the pathogenesis of osteochondral lesions are not well established. It is generally agreed that biomechanical forces are responsible for converting an osteochondrosis lesion into a dissecting osteochondritis dissecans lesion,[3,6,7] although little solid evidence exists to support this assertion. More pertinent, perhaps, is the establishment of the roles of trauma and biomechanical force as primary causative factors. The consistent location of typical osteochondrosis lesions within joints does suggest involvement of physical factors in the pathogenesis.[12] One proposal is that physeal dysplasia could result from excessive force on normal tissues or from the superimposition of normal force on structurally deficient tissues.[25] A similar hypothesis has been proposed for the development of osteochondrosis.[21,59] Many of the factors discussed previously could generate abnormal skeletal tissues, and abnormally high forces could result from excessive or inappropriate exercise, excessive body weight, or poor conformation.[25] A possible relationship between conformation and tarsocrural osteochondrosis was noted in a study of growing STB foals.[35] Of the 77 foals in the study, 39 (51%) had moderate or severe outward rotation of the hindlimb. This conformational feature was present in 7 (88%) of the 8 foals that developed tarsocrural osteochondrosis. These data suggest that a correlation may exist between osteochondrosis and conformation, but outward rotation of the limb is common in unaffected horses. Proof of a causative effect requires further research.

Some authors believe that many lesions currently classified as osteochondrosis arise purely as a result of mechanical factors, with no underlying developmental abnormality.[8,10,21] One author proposed that many osteochondrosis lesions with a typical distribution (solitary or symmetrically paired lesions in typical sites) be included in this category.[21] Causative factors were classified as sufficient (factors that can cause osteochondrosis by themselves), necessary (factors that must be present for expression of the disease, but do not cause osteochondrosis alone), and contributory (factors that modify the expression or severity of lesions). Of the mechanical factors considered, direct trauma and shearing of cartilage canals were deemed sufficient factors, biomechanical loading was classified as a necessary factor and conformational faults were considered contributory factors.[10] However, distinguishing between a developmental lesion and a traumatic lesion on gross and histological examination can be difficult, if not impossible,[2,21] especially if lesions are chronic and if the horses have undergone intense exercise before evaluation. Because a mechanical model of osteochondrosis induction is not available, determining the true role of biomechanics in the pathogenesis of osteochondrosis is difficult.

Transforming Growth Factor-β1

Transforming growth factor-β (TGF-β) plays an important role in controlling chondrocyte differentiation and hypertrophy.[60-62] Among the different isoforms of TGF-β, TGF-β1 is particularly important in controlling mammalian endochondral ossification.[63,64] The distribution of TGF-β1 messenger ribonucleic acid and protein in the articular-epiphyseal cartilage complex of the lateral trochlear ridge of the distal femur of horses with and without dyschondroplasia was compared. TGF-β1 expression was reduced at the site of dyschondroplastic lesions, and it was suggested that this may result in cessation of chondrocyte hypertrophy and an accumulation of pre-hypertrophic chondrocytes. The clinical significance of these findings has not yet been established, but this work does suggest that TGF-β1 may be involved in the pathogenesis of osteochondrosis.

Toxic Causes of Osteochondral Lesions

Foals exposed to excessive amounts of zinc and zinc and cadmium in combination have been shown to develop generalized, severe osteochondral lesions.[15,65-67] The gross lesions described in these reports were characterized by separation of articular cartilage from subchondral bone. Excessive dietary zinc can cause secondary copper deficiency.[68] The role of cadmium in the pathogenesis of these lesions is less clear.[15,65,67] These environmental contaminants are clearly toxic causes of osteochondral lesions and are not considered to be factors in the pathogenesis of osteochondrosis. However, they should be considered in the investigation of any unexplained outbreak of severe, generalized osteochondral lesions in foals, which in most instances do not resemble those seen in horses with naturally occurring osteochondrosis.

Osteochondral Lesions No Longer Considered as Osteochondrosis

Despite the controversies discussed previously, equine osteochondrosis is still most commonly regarded as a developmental condition.[22,32,55,58] There is general consensus that a number of lesions originally referred to as osteochondrosis have no developmental component, however, and should not be included under the osteochondrosis umbrella. These include juvenile spavin[10,21] and erosions of the palmar third metacarpal bone in racing TBs.[3,10,21] Debate continues concerning palmar/plantar osteochondral fragments and ununited palmar/plantar eminences of the fetlock joint, because although trauma may play a role in development, the high incidence in STBs suggests a possible developmental cause.[10,21,36]

SUMMARY

A range of conditions result in failure of endochondral ossification and retention of cartilage cores at the cartilage-bone interface, but the relative contribution to naturally occurring equine osteochondrosis is unknown. The strong heritable component[17,52,62] suggests that at least some manifestations of osteochondrosis may result from a complex interaction of environmental influences and genetic susceptibility. In support of this theory, osteochondrosis was more likely to develop in large, fast-growing offspring of a particular stallion than in the smaller, slower-growing siblings, suggesting that environmental influences can modify the expression of genetic factors.[35]

Recent studies have provided information on the age range at which lesions develop in different sites.[19,38,69] Early tarsocrural osteochondrosis lesions can be detected radiographically by 3 months of age. Lesions of the distal intermediate ridge of the tibia could be detected radiographically by 1 month of age in a population of foals genetically predisposed to osteochondrosis. Many of these lesions resolved, but osteochondrosis lesions of the hock (distal intermediate ridge and lateral trochlear ridge of the talus) that were still present at 5 months of age never regressed. Five months was designated as the age of no return for tarsocrural osteochondrosis lesions. In the same study, lesions of the lateral trochlear ridge of the femur became radiographically obvious from 3 to 4 months of age. As with tarsocrural lesions the majority of these lesions resolved, but any lesions remaining at 8 months of age appeared to be permanent. Eight months was designated as the age of no return for lateral trochlear ridge lesions.[38] A separate study found no evidence of new lesion development in the tarsocrural or fetlock joints after the age of 8 months,[69] and a third study concluded that osteochondrosis lesions of the medial femoral condyle, lateral femoral trochlear ridge, and distal tibia all develop before 7 months of age.[19] Researchers aiming to determine the pathogenesis of osteochondrosis can now focus their attention on the period during which lesions are known to develop.

It is generally agreed that equine osteochondrosis is a multifactorial condition.[3,5,22] This conclusion may be refined in the future, however, as the application of increasingly sophisticated investigational techniques leads to a more definitive definition of the condition and narrows the spectrum of conditions classified as osteochondrosis. Such a change is already occurring. Over the years, osteochondral lesions in numerous sites and with various pathogeneses have been described as osteochondrosis. It is now agreed that some of these lesions are not associated with a primary failure of endochondral ossification but are in fact traumatic or toxic in origin.[10,15,21,65] New techniques are allowing researchers to describe osteochondral lesions more fully and categorize lesions by cause. A recent example is the work in which osteochondral lesions associated with a low-copper diet were examined using histological, morphometric, and biochemical techniques.[48] These investigations strongly suggested that the lesions were caused by a copper deficiency rather than by a primary defect in the process of endochondral ossification. A second example is recent work in which histological and immunohistochemical techniques were applied to lesions with the gross appearance of osteochondrosis. The methods used allowed the lesions to be subdivided into two types, and it was proposed that these lesion types had different pathogeneses.[70]

It is obvious from this summary that opinions still differ widely on the major factors involved in the pathogenesis of equine osteochondrosis.[10,22] The last decade has brought a substantial amount of progress, however, and a firm foundation of knowledge involving many disciplines now exists on which future work can be built. However, until osteochondrosis is better defined and the pathogeneses of osteochondral lesions are more fully determined, the equine industry must work to reduce the inherent susceptibility of growing horses to osteochondrosis and control the environmental and husbandry influences that appear to predispose horses to osteochondral lesions.

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CHAPTER • 57

The Role of Nutrition in Developmental Orthopedic Disease: Nutritional Management

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Nutrition may play an important role in the pathogenesis of developmental orthopedic disease in horses. Deficiencies, excesses, and imbalances of nutrients may result in an increase in the incidence and severity of physitis, angular limb deformity, wobbler syndrome (wobbles), and osteochondrosis.

NUTRITIONAL FACTORS AS A CAUSE OF DEVELOPMENTAL ORTHOPEDIC DISEASE

Mineral Deficiencies

A deficiency of minerals, including calcium, phosphorus, copper, and zinc, may lead to developmental orthopedic disease. Most commonly fed cereal grains and forages contain insufficient quantities of several minerals. A ration of grass hay and oats supplies only 40% and 70% of a weanling's calcium and phosphorus requirement, respectively, and less than 40% of its requirement for copper and zinc (Table 57-1). The best method of diagnosing mineral deficiencies is through ration evaluation. Blood, hair, and hoof analysis is of limited usefulness.

Mineral Excesses

Horses can tolerate fairly high levels of mineral intake, but excesses of calcium, phosphorus, zinc, iodine, fluoride, and heavy metals, such as lead and cadmium, may lead to developmental orthopedic disease (Table 57-2).

Mineral excesses occur because of overfortification or environmental contamination. Massive oversupplementation of calcium (>300% of required amount) may lead to a secondary

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CHAPTER • 57

The Role of Nutrition in Developmental Orthopedic Disease: Nutritional Management

Joe D. Pagan

Nutrition may play an important role in the pathogenesis of developmental orthopedic disease in horses. Deficiencies, excesses, and imbalances of nutrients may result in an increase in the incidence and severity of physitis, angular limb deformity, wobbler syndrome (wobbles), and osteochondrosis.

NUTRITIONAL FACTORS AS A CAUSE OF DEVELOPMENTAL ORTHOPEDIC DISEASE

Mineral Deficiencies

A deficiency of minerals, including calcium, phosphorus, copper, and zinc, may lead to developmental orthopedic disease. Most commonly fed cereal grains and forages contain insufficient quantities of several minerals. A ration of grass hay and oats supplies only 40% and 70% of a weanling's calcium and phosphorus requirement, respectively, and less than 40% of its requirement for copper and zinc (Table 57-1). The best method of diagnosing mineral deficiencies is through ration evaluation. Blood, hair, and hoof analysis is of limited usefulness.

Mineral Excesses

Horses can tolerate fairly high levels of mineral intake, but excesses of calcium, phosphorus, zinc, iodine, fluoride, and heavy metals, such as lead and cadmium, may lead to developmental orthopedic disease (Table 57-2).

Mineral excesses occur because of overfortification or environmental contamination. Massive oversupplementation of calcium (>300% of required amount) may lead to a secondary

Table • **57-1**

Mineral Requirements for Weanlings

MINERAL	NUTRIENT CONCENTRATION REQUIRED IN TOTAL DIET (90% DRY BASIS)		GRASS HAY	ALFALFA HAY	OATS	CORN	BARLEY
	MODERATE GROWTH	RAPID GROWTH					
Calcium (%)	0.62	0.70	0.35	1.25	0.08	0.05	0.05
Phosphorus (%)	0.40	0.45	0.20	0.22	0.34	0.27	0.34
Zinc (ppm)	65	65	9	16	6	4	8
Copper (ppm)	22	22	17	28	35	19	17

Table • **57-2**

*Toxic Mineral Levels**

MINERAL	MINERAL LEVEL NEEDED BY YOUNG HORSE (PPM)	TOXIC LEVEL (PPM)
Zinc	60-70	9000
Iodine	0.2-0.3	5
Fluoride	—	50
Lead	—	80
Selenium	0.2-0.3	5
Manganese	60-70	4000
Copper	20-30	300-500
Cobalt	0.1	400
Iron	125	5000

*Adapted from Cunha[1] and 1989 National Research Council.[2]

mineral deficiency by interfering with the absorption of other minerals such as phosphorus, zinc, and iodine. Excessive calcium intake may be compounded by using legume hays as the primary forage source. Iodine and selenium oversupplementation occurs if supplements are fed at inappropriate levels. A ration evaluation is the best way to identify this type of mineral imbalance.

Environmental contamination is a more likely cause of developmental orthopedic disease, because contamination may result in extremely high intakes of potentially toxic minerals. If a farm is experiencing an unusually high incidence of developmental orthopedic disease or if the location and severity of skeletal lesions are abnormal, environmental contamination should be investigated. Blood, feed, and water analysis should be performed. Chemical analysis of hoof and hair samples may reveal valuable information. Farms that are located near factories or smelters are most at risk, although osteochondrosis caused by zinc-induced copper deficiency has been reported on farms using fence paint containing zinc or galvanized water pipes.

Mineral Imbalances

The ratio of minerals may be as important as the actual amount of individual minerals in the ration. High levels of phosphorus inhibit the absorption of calcium and lead to a deficiency, even if the amount of calcium present is normally adequate. The calcium/phosphorus ratio in the rations of young horses should never be below 1:1 and ideally should be 1.5:1. Too much calcium may affect phosphorus status, particularly if the level of phosphorus is marginal. Calcium/phosphorus ratios greater than 2.5:1 should be avoided if possible. Forage diets with high calcium levels should be supplemented with phosphorus. The zinc/copper ratio should be 3:1 to 4:1.

Dietary Energy Excesses

Excessive energy intake can lead to rapid growth and increased body fat, which may predispose young horses to developmental orthopedic disease. A recent Kentucky study showed that growth rate and body size might increase the incidence of certain types of developmental orthopedic diseases in Thoroughbred (TB) foals.[3] Yearlings that showed osteochondrosis of the hock and stifle were large at birth, grew rapidly from 3 to 8 months of age, and were heavier than the average population as weanlings.

The source of energy for young horses also may be important, because hyperglycemia or hyperinsulinemia have been implicated in the pathogenesis of osteochondrosis.[4,5] Foals that experience an exaggerated and sustained increase in circulating glucose or insulin in response to a carbohydrate (grain) meal may be predisposed to develop osteochondrosis.[6]

In a large field trial, 218 TB weanlings (average age 300 ± 40 days, average body weight 300 ± 43 kg) were studied.[6] A glycemic response test was conducted by feeding a meal that consisted of the weanling's normal concentrate at a level of intake equal to 1.4 g non-structural carbohydrate per kilogram of body mass. A single blood sample was taken 120 minutes after feeding to determine glucose and insulin levels. A high glucose and insulin response to a concentrate meal was associated with an increased incidence of osteochondrosis. More research is needed to determine whether the incidence of osteochondrosis can be reduced through feeding foals concentrates that produce low glycemic responses.

RATION EVALUATIONS

The best way to determine whether nutrition is a contributing factor to developmental orthopedic disease is to perform a ration evaluation, which compares the intake of several essential nutrients with the requirements of the horse. Gross deficiencies or excesses of key nutrients then can be identified and corrected. In the past, ration evaluations were time consuming and cumbersome, because much of the mathematical calculation was done by hand. Fortunately computer programs are now available that make ration evaluations quick and easy to interpret. Kentucky Equine Research Inc. (Versailles, Ky.) has developed an equine ration evaluation program called MicroSteed (www.ker.com).

Types of Evaluations

Ration evaluations can be approached in two ways. One way is to add up what is being fed and compare it with the horse's requirements. This may not be easy because most horsemen do not know exactly what their horses are eating. Alternatively, a new ration may be developed.

Protocol

Every nutrition evaluation should include a description of the horse, definition of nutrient requirements, determination of nutrients in feedstuffs, determination of intake of feedstuffs, calculation of nutrient intake, comparison of intake with requirements, and adjustment of the ration to correct deficiencies or excesses.

Describing the Horse

Different classes of horses have different nutrient requirements and each class may eat different amounts of forage and grain. Within each class of horse, it is important to know the horse's current body weight, its age and mature body weight if growing, and its rate of body weight gain or loss.

Defining Nutrient Requirements

Ration evaluations are intended to compare a horse's daily nutrient intake with a set of requirements to determine how well the feeding program meets the horse's nutritional needs. The National Research Council (NRC) publishes a set of requirements for horses, but NRC values generally represent *minimum* requirements for most nutrients. These are the levels of intake that are required to prevent frank deficiency symptoms. No allowances are included to account for factors that may increase the requirement of a nutrient. The bioavailability of nutrients may be different and other substances within a ration may interfere with the digestibility or use of a nutrient.

MicroSteed includes two different sets of nutrient requirements, the NRC and Kentucky Equine Research requirements, based on a combination of NRC numbers, research conducted since publication of the most recent NRC recommendations, and experience in the field. The user has the option of selecting NRC or Kentucky Equine Research requirements or adding a custom set of requirements.

NRC values for digestible energy and protein fairly accurately describe the needs of most horses. These two requirements were primarily developed from direct measurements of growth response and energy balance. Other requirements, such as those for calcium and phosphorus, were developed using more theoretical calculations, involving estimates of endogenous losses and digestibility. Still others were based on values developed for other species or from single experiments that were far from conclusive. Kentucky Equine Research requirements use values ranging from 1.25 to 3 times those recommended by the NRC for most vitamins and minerals. These nutrient requirements are not absolute, but it is assumed that they adequately reflect horses' needs under a wide range of conditions.

Determining Nutrients in Feedstuffs

The accuracy of evaluating the diet depends on proper sampling of feedstuffs. The feeds should be thoroughly mixed and a representative sample taken. Pelleted feeds are fairly uniform, but sampling is more critical for textured feeds and home mixes. If an odd nutrient value is encountered, the clinician should look to sampling error as a likely cause.

A hay core can be used to obtain a representative hay sample for analysis. Pasture analysis is more difficult. Should the entire pasture be systematically sampled or only those areas heavily grazed? Horses tend to be spot grazers; therefore sampling the heavily grazed areas is probably best.

When expressing feed intakes and nutrient composition, air dry values for hay and grain and 100% dry matter values for pasture are used, because hay and grain intakes actually are measured as fed, and pasture intakes tend to be estimated. The moisture content of the pasture is not relevant to the evaluation and only complicates intake calculations.

A number of commercial laboratories analyze forages and feeds. For a typical ration evaluation for young growing horses, the following nutrients should be analyzed or calculated for each forage and concentrate: digestible energy (megacalories [Mcal] or megajoule [MJ], typically estimated), crude protein (percent), lysine (percent, typically estimated), acid or neutral detergent fiber (percent), calcium (percent), phosphorus (percent), zinc (percent), copper (percent), and manganese (percent).

These nutrients usually are included on a standard panel analysis at a reasonable cost. Other minerals, such as selenium and iodine, usually are analyzed separately, and analysis can be expensive. Selenium and iodine are not essential for evaluations that focus on identifying nutritional causes of developmental orthopedic disease.

Determining Intake of Feedstuffs

A common flaw in many ration evaluations is measuring intake inaccurately. A weighing scale should be used to measure the amount of grain and hay offered. A certain degree of hay wastage usually occurs and this should be taken into account when calculating intake. The amount of forage and grain consumed by young horses varies tremendously, depending on its geographical location and forage availability. Typically, horses that are raised in tropical environments depend heavily on grain in the ration. Yearlings raised in temperate areas with abundant forage may eat rations that contain 80% forage.

Calculating Nutrient Intake

Determining pasture intake is the most difficult part of conducting a ration evaluation. Two methods usually are employed to estimate pasture intake. The simplest method is arbitrarily to estimate intake at about 1% to 1.5% of a young horse's body weight. The obtained value is approximate, but it is representative of most young horses at pasture for most of the day. A second and more accurate method is to calculate pasture intake energetically, by subtracting the digestible energy intake from all other feedstuffs from the horse's daily energy requirement. Dividing this number by the calculated energy density of the pasture yields daily dry matter intake. For example, a yearling that weighs 330 kg with an average daily gain of 0.55 kg/day should require 20.4 Mcal of digestible energy per day. If that yearling is eating 3.65 kg of sweet feed (10.8 Mcal of digestible energy) and 2 kg of mature alfalfa hay (3.6 Mcal digestible energy), then the yearling must be consuming around 6 Mcal of digestible energy from pasture. Most grass pastures contain about 2.2 Mcal of digestible energy per kilogram, so this yearling must consume about 2.73 kg of pasture dry matter per day. These intakes can then be used to evaluate the adequacy of the ration for other nutrients. In MicroSteed, pasture intake can be estimated automatically by first entering the other feedstuffs into the ration and then using an estimate key to perform the calculation just described. This method of calculating pasture energy intake works well, provided the horse actually is consuming the intakes of other feedstuffs and the correct energy requirements were selected.

Using the method described previously for estimating pasture intake often yields a negative number. If this occurs, then the digestible energy intake of the other feeds is too high, or the calculated energy consumption is too low. Sometimes horse owners report higher intakes of feeds than actually are eaten, which is particularly true for forages, because hay rarely is weighed and large quantities often are wasted. Grain intake can also be overestimated because the coffee can that is used to measure grain does not hold nearly as much grain as it does coffee. At other times the hay and grain intake may be correct, but the horse may be consuming more energy than calculated. Increased energy intake can occur if the horse is expending extra energy to work or keep warm in cold weather, or a young horse may be growing faster than assumed. For example, a yearling needs about 5 kg of additional grain (16.1 Mcal of digestible energy) per kilogram of gain. If average daily gain is higher than assumed, then the horse may be eating significantly more digestible energy than calculated.

Comparing Intake with Requirements

Rarely will the nutrients supplied by a ration exactly match a horse's requirements, and balancing rations with this type of precision is unnecessary. Instead the key to interpreting a ration evaluation is to identify deficiencies, excesses, or imbalances of nutrients that may affect growth and skeletal soundness. For most nutrients a level of intake in excess of 90% of required is not considered deficient. What is interpreted as excessive varies tremendously among nutrients. For instance, potassium plays only a minor role in skeletal development; a young horse at pasture may consume greater than 300% of its potassium requirement. Most of this potassium comes from the pasture and is perfectly harmless. Even small excesses of other nutrients, such as energy, may play a significant role in the development of skeletal disease. Energy intakes that are 115% of required might trigger mild developmental orthopedic disease, and levels above 130% almost certainly will cause problems in rapidly growing horses.

FEEDING PRACTICES THAT CONTRIBUTE TO DEVELOPMENTAL ORTHOPEDIC DISEASE

Several feeding scenarios may contribute to developmental orthopedic disease. Once identified, most can be corrected easily through adjustments in feed type and intake. Several of the most common mistakes made in feeding young growing horses are explained.

Overfeeding

One of the most common problems of feeding young horses is excessive intake that results in accelerated growth rate or fattening. Both conditions may contribute to developmental orthopedic disease. Unfortunately, there are no simple rules about how much grain is too much, because total intake of forage and grain determines energy consumption. Large intakes of grain are appropriate if the forage is sparse or poor quality, as often is the case in tropical environments. For example, grain intakes as high as 2% to 2.5% of body weight may be necessary to sustain reasonable growth in weanlings that have access to no forage other than tropical pasture. Conversely, grain intakes higher than 1% of body weight may be considered excessive when weanlings are raised on lush temperate pasture or have access to high-quality alfalfa hay.

The surest way to document excessive intake is by weighing and using condition scoring in the growing horse. Based on a system developed by Henneke et al.,[7] condition scoring measures fat deposition. Horses are scored from 1 to 9 (1 denoting extreme thinness and 9 indicating obesity). In a Kentucky study, fillies tended to have higher condition scores than colts, and the difference was greatest at 4 months of age (fillies 6.48; colts 6.0). These condition scores are considered moderate to fleshy according to the Henneke scoring system. By 12 months of age the condition scores of the colts and fillies had dropped to 5.3 and 5.4, respectively. Both sexes increased condition score slightly from 14 to 18 months.

If growth rate cannot be measured, excessive intake can often be assessed by ration evaluation. For example, a 6-month-old TB weanling (250 kg body mass; 500 kg mature body mass) was being fed 4 kg/day of a 16% protein sweet feed and 2 kg of alfalfa hay, with access to high-quality fall Kentucky pasture. To support a reasonable rate of growth (0.80 kg/day), this weanling required about 17 Mcal of digestible energy per day. The hay and grain intake of this foal alone would supply about 17.5 Mcal of digestible energy, which is slightly above the weanling's requirement. If a reasonable level of pasture intake were included (1% body mass or 2.5 kg dry matter), this weanling would be consuming 135% of its digestible energy requirement, a level likely to cause problems.

To reduce intake, the alfalfa hay should be eliminated, if the pasture is indeed adequate. If hay were needed when the weanling was stalled, grass hay would be more appropriate. Grain intake should be reduced to a level of about 3 kg/day. At this level of grain intake the weanling would need to consume about 3.3 kg of pasture dry matter to support a growth rate of 0.80 kg/day, and the ration would be nicely balanced.

Inappropriate Grain for Forage Provided

Occasionally the concentrate offered to a growing horse is incorrectly fortified to complement the forage that is being fed. The problem occurs particularly when the forage is mostly alfalfa or clover. Most concentrates for young horses are formulated with levels of minerals and protein needed to balance grass forage.

For example, a 12-month-old yearling (315 kg body mass; 500 kg mature body mass; 0.50 kg/day average daily growth) is raised without access to pasture, and the only forage available is alfalfa hay, which is fed at a level of intake equal to 1.5% of the yearling's body mass (4.72 kg/day). At this level of forage intake, the yearling would only require about 2.5 kg of grain per day. If a typical 14% protein sweet feed that was formulated to balance grass forage were used, the ration would be inappropriate for a number of reasons. Calcium would be 183% of the yearling's requirement, with a calcium/phosphorus ratio of 2.9:1. This would not be a problem except that phosphorus and zinc are marginal in the ration. Because calcium may interfere with the absorption of these minerals, the yearling may be at risk of developmental orthopedic disease from a zinc or phosphorus deficiency. The solution is to feed a concentrate that is more appropriately balanced for legume hay. For example, a 12% protein feed with 0.4% calcium, 0.9% phosphorus, and 180 ppm zinc would be more suitable.

Inadequate Fortification in Grain

The most common reasons for inadequate fortification are using unfortified or underfortified grain mixes, using correctly fortified feeds at levels of intake that are below the manufacturer's recommendation, or using fortified feeds diluted with straight cereal grains. These errors in feeding can be corrected by incorporating a highly fortified grain balancer supplement.

For example, a 6-month-old weanling (200 kg body mass; 400 kg mature body mass; 0.60 kg/day average daily growth) is fed 3 kg/day of a 10% protein sweet feed that is intended for adult horses. To compound matters, the weanling is also fed grass hay, with an estimated intake of 2.3 kg/day. This ration is deficient in protein, calcium, phosphorus, zinc, and copper. The foal would be prone to a rough hair coat and physitis. There are two ways to correct this problem. A properly formulated 14% to 16% protein grain mix with adequate mineral fortification could be used, or 1 kg of a grain balancer pellet could be substituted for 1 kg of the 10% sweet feed. This type of supplement is typically fortified with 25% to 30% protein, 2.5% to 3.0% calcium, 1.75% to 2.0% phosphorus, 125 to 175 ppm copper, and 375 to 475 ppm zinc. This is an extremely useful type of supplement to correct underfortified rations.

FEEDING SYSTEMS TO PREVENT DEVELOPMENTAL ORTHOPEDIC DISEASE

The nutritional requirements of a broodmare can be divided into three stages. Stage one is early pregnancy, from conception through the first 7 months of gestation. Barren mares and pregnant mares without suckling foals fit into this nutritional category. Stage two encompasses the last trimester of pregnancy, from around 7 months of pregnancy through foaling. Stage three is lactation, which generally lasts 5 to 6 months after

foaling. The most common mistakes are overfeeding during early pregnancy and underfeeding during lactation.

Early Pregnancy

Proper feeding of a mare during pregnancy requires an understanding of how the fetus develops during gestation. Contrary to popular belief, the fetus does not grow at a constant rate throughout the entire 11 months of pregnancy. The fetus is small during the first 5 months of pregnancy. At 7 months of pregnancy the fetus equals only about 20% of its weight at birth. At this stage in pregnancy the fetus equals less than 2% of the mare's weight, and its nutrient requirements are minuscule compared with the mare's own maintenance requirements (Table 57-3). Therefore the mare can be fed essentially the same as if she were not pregnant. Mare owners often greatly increase feed intake after a mare is pronounced in foal, reasoning that she is now eating for two. Increased feeding is unnecessary and may lead to obesity and foaling difficulties, especially if the mare has access to high-quality pasture.

Late Pregnancy

The fetus begins to develop rapidly after 7 months of pregnancy, and its nutrient requirements become significantly greater than the mare's maintenance requirements; therefore adjustments should be made to the mare's diet. Digestible energy requirements increase about 15% over early pregnancy. Protein and mineral requirements increase to a greater extent, because the fetal tissue being synthesized during this time is high in protein, calcium, and phosphorus. During the last 4 months of pregnancy the fetus and placenta retain about 77 g of protein, 7.5 g of calcium, and 4 g of phosphorus per day. Trace mineral supplementation is also important, because the fetus stores iron, zinc, copper, and manganese in its liver for use during the first few months after birth because mare's milk is low in these elements.

New Zealand researchers studied the effect of copper supplementation on the incidence of developmental orthopedic disease in TB foals.[8] Pregnant TB mares were divided into copper-supplemented and control groups. Live foals born to each group of mares were also divided into copper-supplemented and control groups. Copper supplementation of mares was associated with a significant reduction in the physitis scores of the foals at 150 days of age. Foals from mares that received no supplementation had a mean physitis score of 6, whereas foals from supplemented mares had a mean score of 3.7. A lower score means less physitis. Copper supplementation of the foals had no significant effect on physitis scores. A significantly lower incidence of articular cartilage lesions occurred in foals from supplemented mares. However, copper supplementation of the foals had no significant effect on articular and physeal cartilage lesions.[6] Mares in late pregnancy often are overfed energy in an attempt to supply adequate protein and minerals to the developing foal. If a pregnant mare becomes fat during late pregnancy, she should be switched to a feed that is more concentrated in protein and minerals, so that less can be fed per day. This will restrict her energy intake, while ensuring that she receives adequate quantities of other key nutrients.

Lactation

A mare's nutrient requirements increase significantly after foaling. During the first 3 months of lactation, mares produce milk at a rate equal to about 3% of body weight per day. This milk is rich in energy, protein, calcium, phosphorus, and vitamins. Therefore the mare should be fed enough grain to meet its greatly increased nutrient requirements. Mares in early lactation usually require from 4.5 to 6.5 kg of grain per day, depending on the type and quality of forage they are consuming. This grain mix should be fortified with additional protein, minerals, and vitamins to meet the lactating mare's needs. Trace mineral fortification is not as important for lactating mares, because milk contains low levels of these nutrients, and adding more to the lactating mare's diet does not increase the trace mineral content of the milk. Calcium and phosphorus are the minerals that should be of primary concern during lactation. Grain intake should be increased gradually during the last few weeks of pregnancy so that the mare is consuming nearly the amount that it will require for milk production at foaling. A rapid increase in grain should be avoided at foaling, because this may lead to colic or laminitis. Milk production begins to decline after about 3 months of lactation, and grain intake can be reduced to maintain the mare in desirable condition.

Sucklings

If a broodmare has been fed properly during late pregnancy, giving mineral supplements to a suckling is unnecessary until it reaches 90 days of age. At 90 days, moderate amounts of a well-fortified foal feed can be introduced and gradually increased until the suckling is consuming around 0.5 kg of feed per month of age. It is critical that the suckling be accustomed to eating grain before weaning. If the suckling does not become accustomed to eating grain, there is a good chance that its growth rate will decrease dramatically at weaning. When the weanling finally starts eating grain, a compensatory growth spurt occurs that may result in developmental orthopedic disease.

Weanlings

The most critical stage of growth for preventing developmental orthopedic disease is from weaning to 12 months of age, when the skeleton is most vulnerable to disease and nutrient intake and balance is most important. Weanlings should grow at a moderate rate with adequate mineral supplementation. In temperate regions the contribution of pasture to the diet

Table • **57-3**

Expected Feed Consumption by Horses

HORSE	% OF BODY WEIGHT		% OF DIET	
	FORAGE	CONCENTRATE	FORAGE	CONCENTRATE
Maintenance	1-2	0-1	50-100	0-50
Pregnant mare	1-2	0.3-10	50-85	15-50
Lactating mare (early)	1-2.5	0.5-2	33-85	15-66
Lactating mare (late)	1-2	0.5-1.5	40-80	20-60
Weanling	0.5-1.8	1-2.5	30-65	35-70
Yearling	1-2.5	0.5-2	33-80	20-66
Performance horse	1-2	0.5-2	33-80	20-66

often is underestimated, leading to excessive growth rates and developmental orthopedic disease.

Yearlings

Once a horse reaches 12 months of age, it is much less likely to develop many forms of developmental orthopedic disease than a younger horse. Many of the lesions that become clinically relevant after this age probably developed at a younger age. Proper nutrient balance remains important for the yearling, and delaying as long as possible the increased energy intakes that are required for sales preparation is best, because the skeleton is less vulnerable to developmental orthopedic disease as a yearling ages. Normally, increasing energy intake 90 days before a sale is enough time to add the extra body condition that often is expected in a sales yearling. Physitis in the carpus is often a major concern for the sales yearling. The level of trace mineral supplementation should remain high to reduce the incidence of physitis in sales yearlings, and a significant portion of the energy normally supplied from grain should be replaced with fat and fermentable fiber. Sales preparation grain mixes can contain as much as 10% fat. Good sources of fermentable fiber include beet pulp and soy hulls.

NUTRITIONAL MANAGEMENT OF DEVELOPMENTAL ORTHOPEDIC DISEASE

The goal of a feeding program for young horses is to reduce or eliminate the incidence of developmental orthopedic disease. Unfortunately, developmental orthopedic disease still occurs in some foals. Nutritional intervention can help reduce the severity of many forms of developmental orthopedic disease, but not all of the damage resulting from the disease is reversible. However, it is important to alter the feeding programs of foals with developmental orthopedic disease. The type of alteration follows a similar pattern but depends on the foal's age and the type of developmental orthopedic disease. In almost every instance, energy intake should be reduced while maintaining adequate levels of protein and minerals. The rationale for this type of modification is that skeletal growth should be slowed, but adequate substrate should be available to promote healthy bone development.

Physitis

Grain intake should be restricted to a level supplying around 75% of the foal's normal energy requirement. This restriction, however, should not compromise protein and mineral intake, so a different type of feed formulation may be required. For instance, a 6-month-old weanling (250 kg body mass; 500 kg mature body mass; 0.8 kg average daily growth) on a decent fall pasture would normally consume around 3.5 kg of a 16% protein foal feed. If this foal developed physitis, it should be confined and fed grass hay (3 kg/day). Reducing the grain intake to 75% of the foal's normal digestible energy would result in shortages of protein, lysine, calcium, and phosphorus. These shortfalls could be overcome by replacing 1 kg of the 16% sweet feed with a grain balancer pellet. This ration would supply 90% of the foal's normal protein requirement along with a good supply of minerals. As the physitis resolves, intake of the 16% grain mix can be slowly increased and the supplement pellet intake slowly decreased, until the foal returns to its normal ration.

Cervical Vertebral Malformation

A feeding program like the one described previously is also appropriate for a horse with cervical vertebral malformation, except that the degree of exercise and energy restriction may be more severe. In this case a feeding program that combines grass hay (2 kg) with a moderate amount of alfalfa hay (2 kg/day) and 1 kg of balancer pellet would result in a reduction in energy intake equal to 65% of normal intake while maintaining adequate levels of protein and mineral intake.

Osteochondrosis

Once a foal develops osteochondrosis that is severe enough to produce clinical signs, the effect of diet is minimal in solving the existing lesion, but reducing energy intake and body weight while maintaining adequate protein and mineral intake is advised.

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CHAPTER • 58

Diagnosis and Management of Osteochondrosis and Osseous Cyst-like Lesions

Dean W. Richardson

The pathogenesis of osteochondrosis, palmar/plantar osteochondral fragments of the proximal phalanx, ununited palmar/plantar eminences of the proximal phalanx, subchondral bone cysts, and other osseous cyst-like lesions is discussed in Chapter 56. In this book the term *subchondral bone cyst* is used for large radiolucent areas in the subchondral bone of the medial femoral condyle; these cysts have a consistent pathological structure with a true fibrous cyst lining. Osseous cyst-like lesions in other locations vary more in structure. This chapter discusses the diagnosis and management of these lesions and other osseous fragments believed to be developmental in origin. The joints most commonly affected include the hock, stifle, fetlock, and shoulder. Although lesions often occur bilaterally in the equivalent contralateral joint, for joints in different anatomical locations to be clinically affected simultaneously is unusual.

Clinical signs of osteochondrosis range from none to severe lameness. Often moderate to severe joint effusion occurs, with minimal or mild lameness. The clinical signs are not necessarily coincident with the development of the lesion. Most osteochondrosis lesions are probably formed before 6 months of age, yet most are diagnosed at a later age. Some radiographic lesions heal, or they at least become stable enough never to cause a clinically apparent lameness. Lesions in racing Thoroughbreds (TBs) and Standardbreds (STBs) are usually recognized by 2 years of age, but in Warmbloods (WBLs) that are older when they begin training, clinical signs may not be seen until the horse is 5 to 6 years of age or even older. Acute, more severe lameness in older horses occurs occasionally when osteochondritic fragments become loose.

Diagnosis is based on typical clinical signs and radiography. Most osteochondritis dissecans flaps have an osseous component and so can be identified using plain radiography. Defects in subchondral bone contours are also easily identified. Radiographic defects in subchondral bone must be interpreted with caution in foals, especially in areas where endochondral ossification occurs later, such as the femoral or talar trochlear ridges. An irregularity in the contour of subchondral bone does *not* mean that an articular defect is necessarily present; the lesion still possibly may heal. Therefore surgical intervention should be delayed until it is clear that either a loose fragment is present or the healing is progressing inadequately. Radiographically apparent lesions must always be interpreted with the clinical signs, because some do not cause clinical signs. If a lesion is identified, the contralateral joint should be examined radiographically, because bilateral involvement (quadrilateral in the fetlock) is common. The presence of bilateral radiographically apparent lesions in a horse that is unilaterally lame is not unusual and further emphasizes the need to evaluate the individual horse fully.

Decisions concerning treatment should consider the age of the horse, its intended use, the severity of the lesions and anatomical location, if the horse is intended for sale through public auction, and if so, the timing of the sale and the conditions of sale. Prophylactic removal of osteochondrosis lesions for economic reasons may be justified in all horses intended for future sale. Most buyers are more willing to purchase horses after an osteochondrosis lesion has been removed and the horse has returned to work.

LESIONS IN THE METACARPOPHALANGEAL AND METATARSOPHALANGEAL JOINTS

Osteochondrosis of the Sagittal Ridge of the Third Metacarpal and Metatarsal Bones

Most foals with osteochondrosis lesions of the sagittal ridge of the third metacarpal (McIII) or third metatarsal (MtIII) bones are not recognized as lame. A variable degree of joint effusion occurs, and the lesions are usually first identified on pre-sale radiographs. Unstable lesions cause lameness and persistent effusion when a horse starts work. Lameness is eliminated by intra-articular or perineural analgesia.

Lesions are more common in MtIII than McIII, but all fetlocks should be examined radiographically if a lesion is recognized in one, using perfectly exposed (or slightly underexposed) flexed lateromedial views (Fig. 58-1). Lesions on the dorsoproximal aspect of the sagittal ridge are easiest to recognize, but lesions may occur farther distally or, less commonly, on the palmar/plantar aspect of the sagittal ridge. The latter can also be seen radiographically on the dorsopalmar (plantar) projection. The larger fragments extend abaxially along the proximal margin of the articular cartilage under the dorsal synovial fold.

Conservative management should always be considered in horses younger than 18 months old, because lesions can show improvement radiographically and presumably heal. Surgery is indicated in young horses with fetlock effusion and lameness. Young horses treated surgically often do well clinically,

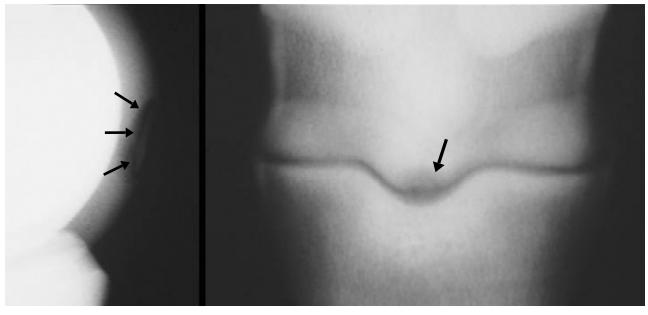

Fig. 58-1 A typical distal sagittal ridge osteochondritis dissecans lesion *(arrows)* of the third metacarpal bone is seen best on a slightly underexposed flexed lateromedial radiographic view *(left)* and well-exposed dorsopalmar view *(right)* of a metacarpophalngeal joint.

but the long-term radiographic appearance is inferior to spontaneous healing. Older horses of training age with unstable fragments are treated by arthroscopic debridement. Proximal lesions are technically easy to approach and debride, but lesions that are more distal can only be exposed by slight or moderate flexion of the joint. The arthroscope portal therefore should be made more distal than usual, and a needle should be used to identify a suitable instrument portal. Debriding a sagittal lesion with a portal placed through the extensor tendon directly over the lesion is easiest. Long reaches across a flexed joint result in undesirable iatrogenic trauma.

The prognosis depends on the size and location of the flap. Probably 90% of horses with typical lesions of the more proximal portion of the sagittal ridge go on to athletic function. Horses with distal lesions in a more weight-bearing location do not do as well as those with flaps at the proximal articular margin, and the radiographic appearance does not improve spontaneously as often.

Osseous Cyst-like Lesions

Osseous cyst-like lesions occur on the weight-bearing surface of the condyles of McIII and MtIII. Most occur in the medial condyle and are diagnosed in horses 1 to 2 years of age. The lesions are easy to recognize radiographically and result in obvious lameness, exacerbated by distal limb flexion. Lameness is improved by perineural or intra-articular analgesia. Lameness associated with an osseous cyst-like lesion sometimes occurs in foals. At the time of onset of lameness no lesion may be identifiable radiographically, or only a subtle defect in the outline of the subchondral bone may be apparent. However, sequential radiographs obtained over the following 6 to 8 weeks may reveal development of an osseous cyst-like lesion. Surgical debridement may be successful,[1] but corticosteroids administered intra-articularly combined with several months of rest also may result in significant improvement in young horses. Although simple debridement of McIII and MtIII osseous cyst-like lesions has been reasonably successful, the intensity of loading on distal McIII/MtIII bones in athletic horses makes these lesions good candidates for future improvements in joint resurfacing techniques.

Palmar/Plantar Osteochondral Fragments of the Proximal Phalanx

Osteochondral fragments arising from the proximal palmar or plantar aspect of the proximal phalanx are common radiographic findings in WBLs, STBs, and TBs, especially in hindlimbs. Palmar/plantar osteochondral fragments and ununited palmar/plantar eminences may not cause clinical signs or merely cause mild discomfort at high levels of performance. Assessment of clinical significance and management are discussed in detail in Chapter 43.

Osteochondral Fragments on the Dorsoproximal Aspect of the Proximal Phalanx

Small, well-rounded osseous opacities on the dorsoproximal aspect of the proximal phalanx are common radiographic findings. Most probably occur as small chip fractures of the developing dorsal rim of the proximal phalanx in foals that subsequently ossify. Whether a true underlying developmental defect in this location exists is unclear. Although fragments in this location do not always cause overt lameness, they often are removed in valuable prospective athletic horses and sales horses. Many of these fragments are loosely tethered along the dorsal rim of the proximal phalanx and can cause damage to the distal McIII/MtIII bones in an intensively exercised horse. Arthroscopic removal is quick and easy, and the prognosis is close to 100% for full function. Horses can be returned to full work within 4 to 6 weeks after removal of these fragments.

LESIONS OF THE PROXIMAL INTERPHALANGEAL JOINT

Lesions in the proximal interphalangeal joint usually cause lameness at a young age, often when the horse is a weanling. Single, discrete, well-defined osseous cyst-like lesions or multiple poorly defined radiolucent lesions may be identified, occurring most commonly in the distal condyles of the proximal phalanx (Fig. 58-2). Surgical access to most pastern osseous cyst-like lesions is difficult, and simple debridement is not usually feasible. Lesions may be advanced when lameness is first recognized, and although occasionally horses spontaneously improve without treatment, horses that are obviously lame usually develop osteoarthritis. Therefore horses that do not respond adequately to corticosteroids administered intra-articularly probably should have an early arthrodesis, especially if a hindlimb is affected. Arthrodesis in young or small horses is readily accomplished with one of the well-described techniques using two or three transarticular 5.5-mm screws placed in lag fashion.[2-5] In larger horses, more stability is afforded by a dorsal four-hole narrow dynamic compression plate combined with two abaxial transarticular 5.5-mm screws. The prognosis for athletic function after pastern arthrodesis appears to depend highly on the desired level of activity. Little doubt exists, however, that the prognosis is far superior in the

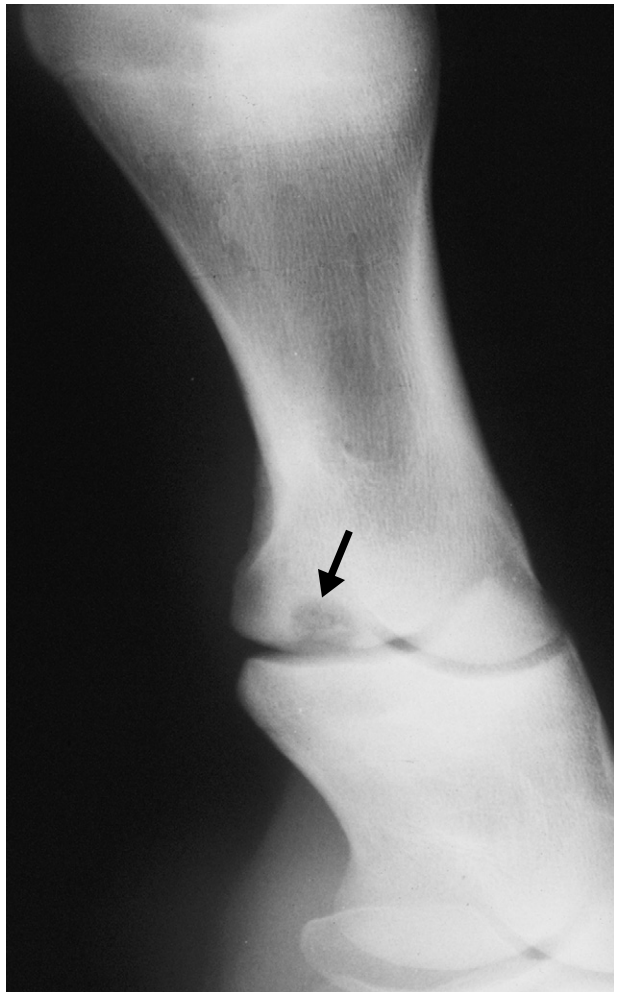

Fig. 58-2 Dorsolateral-palmaromedial radiographic view of a proximal interphalangeal joint. The most common manifestation of osteochondrosis in the pastern is an osseous cyst-like lesion *(arrow)* in the distal condyle of the proximal phalanx.

hindlimb than in the forelimb. Most retrospective studies have reported greater than 80% return to athletic function after hind pastern arthrodesis, but forelimb results may be only around 50%. The intensity of the exercise is probably critical. Few successful elite-level athletic horses (racehorses, upper-level dressage, jumping) have fused front pasterns.

LESIONS OF THE DISTAL INTERPHALANGEAL JOINT

Well-rounded osteochondral fragments on the dorsoproximal aspect of the extensor process of the distal phalanx are also believed to be developmental in origin. Small fragments are frequently incidental radiographic findings, and treatment is not necessary unless associated lameness exists. Lameness should be improved substantially by intra-articular analgesia. Nuclear scintigraphy may help definitive diagnosis. Treatment of horses with lesions associated with lameness is arthroscopic removal of the fragment. The prognosis is excellent (>90%) if the fragment is removed before osteoarthritis of the distal interphalangeal joint develops. These fragments are often well imbedded in the extensor tendon insertion, so not all fragments adversely affect joint function. As with many other small osteochondral fragments, removal is sometimes done prophylactically.

Osseous cyst-like lesions of the distal phalanx can be large and associated with dramatic lameness (Fig. 58-3). Apparently the pathogenesis may be similar to that of subchondral bone cysts of the distal femur. An attempt usually is made to manage such lesions with medications administered intra-articularly. If that is unsuccessful, surgical debridement can be attempted, but the lesions are difficult to access, and the outcome after surgery appears to be less than 50% for a return to adequate function.

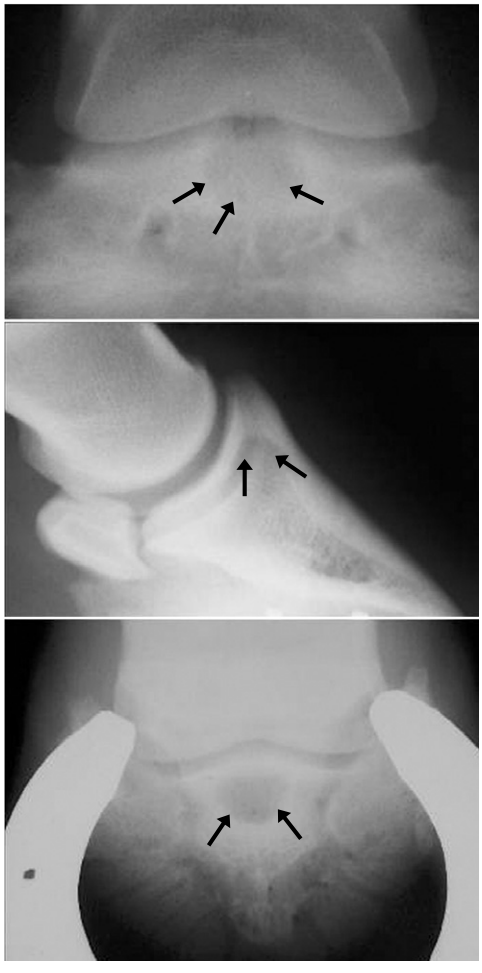

Fig. 58-3 Dorsopalmar *(top)*, lateromedial *(middle)*, and dorsoproximal-palmarodistal oblique *(bottom)* radiographic views of a foal. There is a large osseous cyst-like lesion in the distal phalanx *(arrows)*. Large osseous cyst-like lesions of the distal phalanx are difficult management problems because they are relatively inaccessible to surgical debridement.

LESIONS OF THE SCAPULOHUMERAL JOINT

Osteochondrosis of the scapulohumeral joint may involve the glenoid of the scapula or the humeral head. Humeral head lesions can be solitary, but scapular lesions usually appear with substantial pathological abnormalities of the humeral head in young horses (<2 years old). Lameness can develop insidiously or acutely, presumably after an acute disruption of an unstable osteochondral flap. Recognizing a developing flexural deformity or clubfoot may confuse diagnosis of shoulder lameness in a chronically lame young horse. Occasionally mild lesions are not recognized until a horse starts training. Lameness is improved by intra-articular analgesia but often is not eliminated. Radiographic abnormalities may include poorly defined subchondral radiolucent areas, with or without sclerosis, flattening of the articular surfaces, and modeling of the articular margins, especially the ventral angle of the scapula (see Chapter 41).

Lameness in some young horses improves with rest, and early surgery should not be encouraged in foals younger than 8 to 10 months old unless they are intractably lame. Affected foals should be confined to a stall or small paddock and re-examined clinically and radiographically at 60-day intervals. Intra-articular medication has not been consistently helpful in weanlings and yearlings. The outcome after arthroscopic treatment of osteochondrosis of the scapulohumeral joint is unpredictable and lameness can dramatically worsen after surgery. Radiography may underestimate the severity of lesions, especially those involving the humeral head. It is important to emphasize to an owner before surgery that considerable uncertainty exists about the clinical outcome in any given horse with osteochondrosis in the shoulder. Solitary osseous cyst-like lesions also occur, most commonly in the middle of the distal scapula but sometimes in the humeral head (see Chapter 41).

OSTEOCHONDROSIS OF THE TARSOCRURAL JOINT

Osteochondrosis of the tarsocrural joint is common in many breeds. Obvious joint capsule distention (bog spavin) may precede recognition of any gait abnormality. Effusion may not be recognized in racehorses examined for poor performance associated with osteochondrosis, but in show horses with lameness, effusion almost invariably occurs. Radiographic lesions are not always associated with lameness; therefore diagnosis should be confirmed by intra-articular analgesia or by response to intra-articular medication with corticosteroids. At least four radiographic views should be obtained, because multiple lesions may be present. If osteochondrosis is suspected clinically, but no lesion is identified radiographically, repeating the dorsoplantar view with slightly different angles of projection is worthwhile, because medial malleolar lesions can be difficult to see (Fig. 58-4). Flexed lateromedial views may be necessary to identify lesions involving the plantar aspect of the tarsocrural joint.

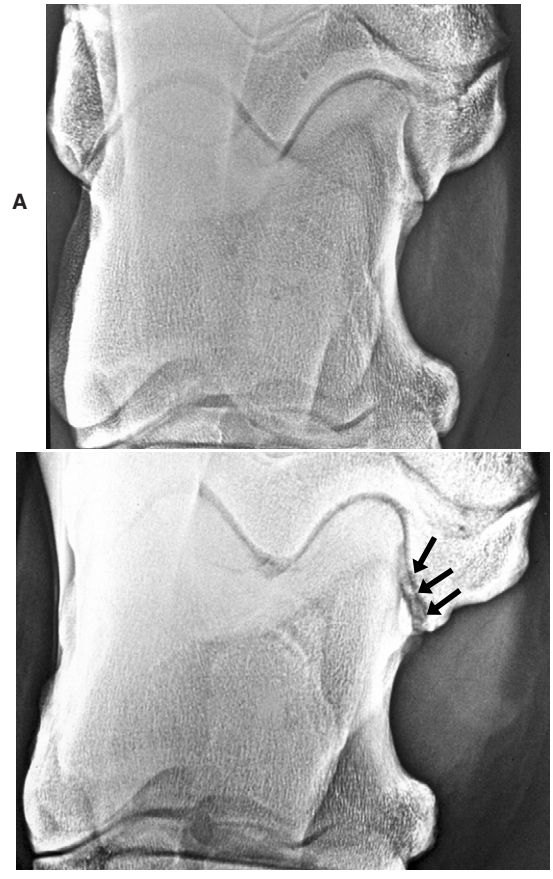

Fig. 58-4 **A,** Dorsoplantar and **B,** dorsal 10° lateral-plantaromedial oblique radiographic views of a hock. Identification of osteochondritis dissecans lesions of the medial malleolus requires well-positioned films. Identifying the lesion on the dorsoplantar xeroradiograph **(A)** is difficult, but a slight alteration in the obliquity **(B)** reveals a large defect *(arrows)*.

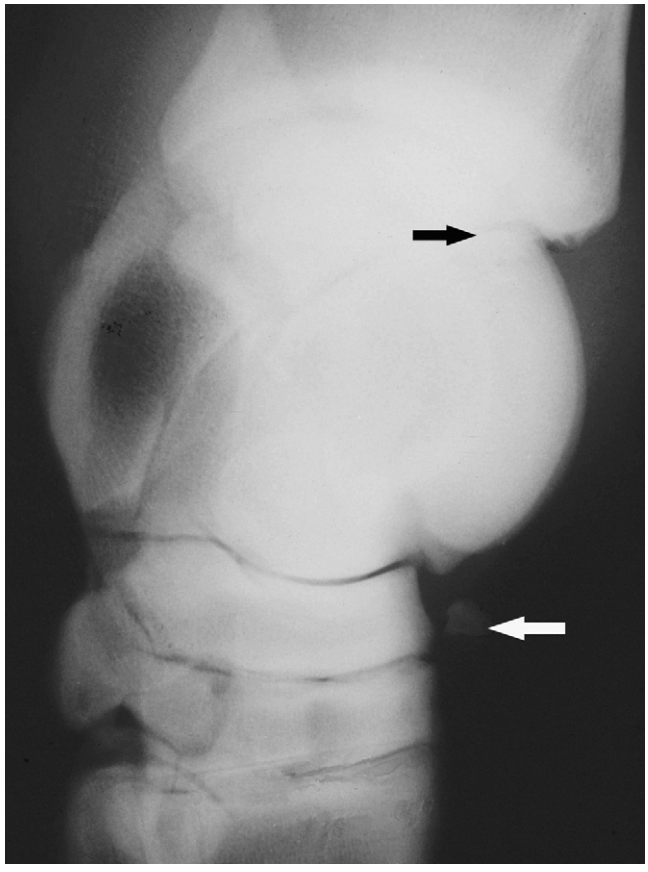

Fig. 58-5 Lateromedial radiographic view of a hock. Osteochondritis dissecans lesions that originate anywhere in the tarsocrural joint and become loose *(white arrow)* frequently lodge in the dorsal aspect of the proximal intertarsal joint. The likely origin of this fragment was the cranial aspect of the intermediate ridge of the distal tibia *(black arrow)*.

Osteochondrosis of the tarsocrural joint is recognized as a common radiographic finding in STBs, TBs, and WBLs, but it is not always associated with lameness. Few of the common osteochondrosis lesions in the hock heal spontaneously after 5 months of age, so surgical removal in young horses, especially sales yearlings, has become commonplace. These lesions are almost always in peripheral, less than completely weightbearing locations, and removal does not seriously compromise joint function. Prognosis is excellent. However, fragments that cause lameness at a later age are a greater problem because the loose fragments and debris often cause extensive cartilage damage. Older show jumpers in particular may dislodge distal tibial osteochondritis dissecans lesions and develop acute clinical signs of effusion and lameness (Fig. 58-5).

The cranial intermediate ridge of the distal tibia is the most common site of lesions (Fig. 58-6). These lesions rarely cause overt lameness unless significant effusion occurs. Arthroscopic removal of the fragment is straightforward and nearly always results in improvement of the lameness and effusion. Lesions may be identified radiographically bilaterally, although only one limb is chronically affected. Loose fragments may become detached in the tarsocrural joint and can become lodged in the communicating proximal intertarsal joint. The opening from the tarsocrural joint into the proximal intertarsal joint at the distal margin of the talar trochlear groove is not patent or is small in young foals or weanlings, but as the opening enlarges in older horses, large fragments can move into the proximal intertarsal joint pouches.[6] If a fragment of bone is found dorsal to (or even on the distal edge of) the central tarsal bone, the fragment is probably one loose in the proximal intertarsal joint that gravitated into that pouch. Fragments associated with lameness should be removed. It is important to recognize that the fragments in the proximal intertarsal joint usually are not easily seen with the arthroscope and often have to be removed blindly with a rongeur guided arthroscopically into the joint pouch.

Lesions of the distal aspect of the lateral trochlear ridge of the talus can be enormous in size and, if loose, may cause acute severe lameness and substantial effusion (Fig. 58-7). This lesion seems to be more common in heavy breeds and STBs. Horses with small, well-attached lesions can be managed conservatively, but any displaced fragment should be removed. The prognosis generally depends on how proximal and axial the lesion extends, but even large lesions can be removed with a good prognosis.

Osteochondritis dissecans lesions of the medial malleolus of the tibia are easily missed, because they are always on the axial rather than the distal (more visible) part of the malleolus (Fig. 58-4). Medial malleolar osteochondritis dissecans lesions usually cause more lameness and more effusion than the more common intermediate ridge lesions. Unless the lesion is unusually large, the prognosis after arthroscopic removal is excellent. Early removal is recommended, because erosive lesions on the medial trochlea of the talus can become severe over time.

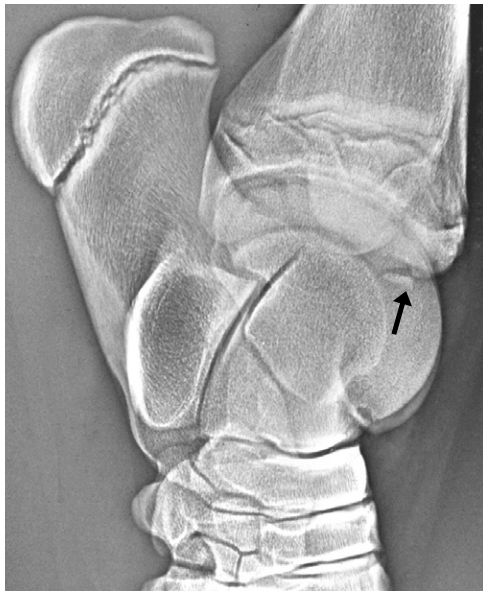

Fig. 58-6 Lateromedial radiographic view of a hock. The most common location for osteochondritis dissecans in the tarsocrural joint is the cranial intermediate ridge of the distal tibia *(arrow)*.

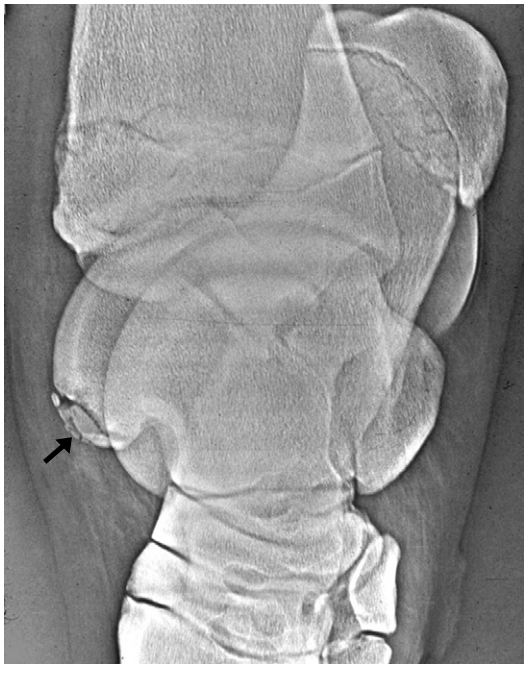

Fig. 58-7 Dorsomedial-plantarolateral radiographic view of a hock. Osteochondritis dissecans fragments *(arrow)* arising from the distal lateral trochlear ridge of the talus are often large, but after surgical removal horses have a good prognosis regardless of size of the lesion.

Medial trochlear ridge lesions should be interpreted with caution; most distal medial trochlear ridge lesions are incidental findings and do not require treatment. Unstable lesions may cause lameness (Fig. 58-8). Distal medial trochlear ridge lesions may be on the proximal or distal side of the capsular attachment separating the tarsocrural and proximal intertarsal joints. The fragments wholly within the tarsocrural joint appear to be less stable and more likely to cause lameness. In

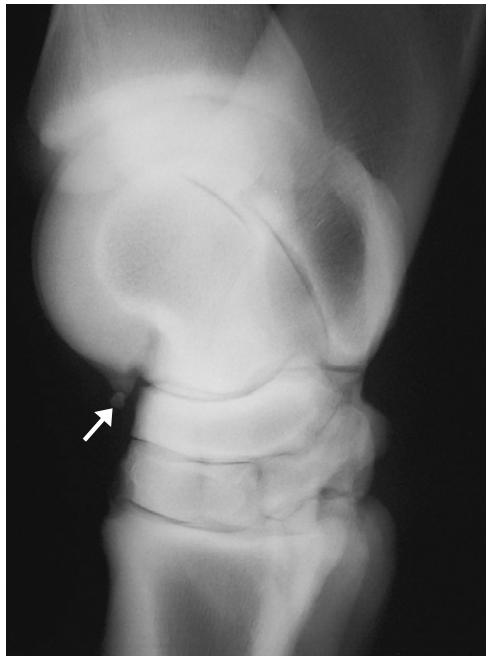

Fig. 58-8 Lateromedial radiographic view of the hock. Small fragments *(arrow)* along the distal dorsal aspect of the medial trochlear ridge of the talus usually do not cause lameness, but some are unstable and induce effusion and lameness, particularly fragments on the proximal side of the proximal intertarsal joint capsule.

all horses the decision for surgical removal should be based on a definitive response to intra-articular analgesia. The arthroscopic removal of the tarsocrural lesions is easy and can be done with minimal dissection. Lesions under the capsular attachment often require sharp dissection of the attachment to expose the fragment. Care should always be taken to dissect the minimal amount of soft tissue, especially because the attachments course abaxially.

More centrally positioned, smoothly outlined depressions in the surface of the medial trochlear ridge of the talus are common in large WBLs, Draft, and Draft-cross horses. The subchondral bone architecture is usually normal, and no associated clinical signs are apparent. These lesions rarely have an unstable chondral or osteochondral flap that can be removed.

Lateral malleolar fragments are usually traumatic (see Chapter 45) and are only rarely (about 1%) osteochondrosis lesions. Unlike the traumatic fractures, lateral malleolar osteochondritis dissecans lesions have irregular diffuse radiolucency within the malleolus instead of a distinctly separated fragment. Arthroscopic debridement of lateral malleolar osteochondritis dissecans lesions is difficult because of the extensive collateral ligament attachments that obscure the structure.

Arthroscopic removal of osteochondritis dissecans lesions from the hock usually can be followed by a short convalescence and rapid return to work. Typically a horse is walked by hand for 4 to 6 weeks and then re-evaluated. If the effusion has resolved and the horse is jogging soundly, the level of exercise can be increased steadily. Intra-articularly administered hyaluronan is given at about the time of suture removal.

Most horses with osteochondritis dissecans of the hock have an excellent prognosis, especially if lesions are removed early, before substantial damage to the joint occurs. The specific location of the osteochondritis dissecans probably makes less difference than the size, because most hock osteochondritis dissecans lesions occur on the peripheral, less than

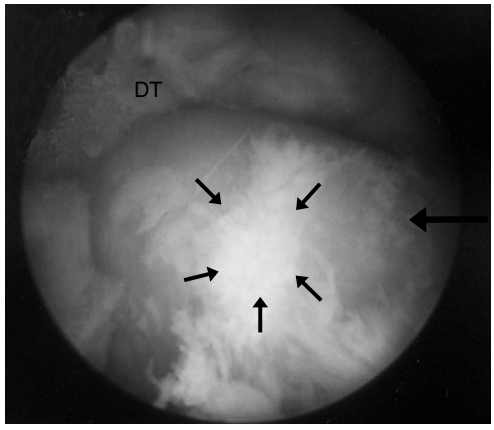

Fig. 58-9 Unlike in most other joints in the horse, full-thickness cartilage erosion of the talus (medial trochlear ridge, *large arrow*) and distal tibia *(DT)* occurs with surprisingly few bony changes such as subchondral lysis or marginal osteophytes. Arthroscopy affords the best means of making the diagnosis. The medial trochlear ridge is nearly devoid of cartilage except for loose and fibrillated cartilage on its summit *(small arrows)*.

fully weight-bearing surfaces of the bones. Controversy remains about the need to remove all hock osteochondritis dissecans lesions, but clearly horses have an excellent (>80%) chance of performing at the expected level after arthroscopic removal of typical lesions.[7-9]

The tarsocrural joint appears to be particularly vulnerable to a form of erosive arthritis that can result in extensive, full-thickness loss of articular cartilage (Fig. 58-9). This occurs in the absence of known trauma or radiographically evident lesions, but most horses with the lesion have a history of intra-articularly administered corticosteroids used to manage an effusion. The exact mechanism of this process in the tarsocrural joint is not known, but corticosteroids should be used cautiously to manage effusions of this joint.

LESIONS OF THE STIFLE

Diagnosis and management osteochondrosis of the femoropatellar joint, subchondral bone cysts in the medial femoral condyle, and other osseous cyst-like lesions are discussed in detail in Chapter 47.

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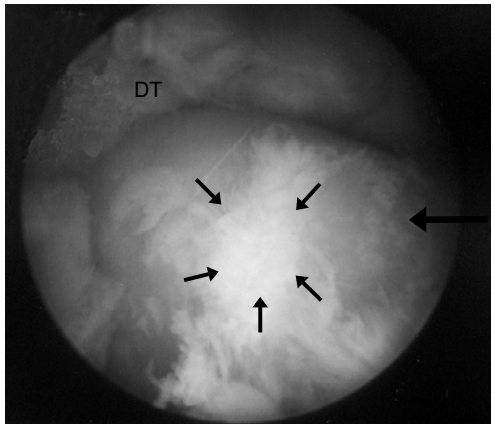
CHAPTER • 59

Physitis

David R. Ellis

Physitis occurs in the horse in three principal forms: infectious physitis and type V and type VI Salter-Harris growth plate injuries.[1] Type V growth plate injury can arise secondary to congenital but persistent angular limb deformities in foals, but these, infections, and type VI injuries are not considered in this chapter. Type V Salter-Harris injuries are considered a manifestation of acquired physitis, one of the group of disorders known as *developmental orthopedic disease*.[2] The inclusion of acquired physitis under the umbrella of osteochondrosis may be more debatable. Jeffcott[3] considered that the condition was better defined as physeal dysplasia, implying a disturbance of endochondral ossification rather than an inflammatory condition. Bramlage[4] outlined two forms of physitis based on radiographic interpretation of the site at which changes were seen. The two forms were those that were seen at the periphery, usually on the medial aspect, and those that were seen in the axial or central section of the growth plate.

Physitis is largely confined to the lighter-boned, faster-growing breeds of horse, particularly Thoroughbreds and Thoroughbred crossbreeds. Milder physitis is relatively common, and rarely does experienced stud management



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CHAPTER • 59

Physitis

David R. Ellis

Physitis occurs in the horse in three principal forms: infectious physitis and type V and type VI Salter-Harris growth plate injuries.[1] Type V growth plate injury can arise secondary to congenital but persistent angular limb deformities in foals, but these, infections, and type VI injuries are not considered in this chapter. Type V Salter-Harris injuries are considered a manifestation of acquired physitis, one of the group of disorders known as *developmental orthopedic disease*.[2] The inclusion of acquired physitis under the umbrella of osteochondrosis may be more debatable. Jeffcott[3] considered that the condition was better defined as physeal dysplasia, implying a disturbance of endochondral ossification rather than an inflammatory condition. Bramlage[4] outlined two forms of physitis based on radiographic interpretation of the site at which changes were seen. The two forms were those that were seen at the periphery, usually on the medial aspect, and those that were seen in the axial or central section of the growth plate.

Physitis is largely confined to the lighter-boned, faster-growing breeds of horse, particularly Thoroughbreds and Thoroughbred crossbreeds. Milder physitis is relatively common, and rarely does experienced stud management

allow the condition to develop far enough for severe lameness and conformational changes to occur. Only in horses with more extreme or persistent physitis would radiographic examination be undertaken in the United Kingdom. Clinical recognition of enlargement of the growth plate or slight change in conformation usually presumes a diagnosis of physitis and indicates an immediate change of the horse's management as the first line of treatment.

PATHOGENESIS

I consider the pathogenesis of physitis to be consistent with a type V Salter-Harris growth plate injury. This implies a compression lesion that arises medially as a consequence of the greater weight being borne on the medial aspect of the forelimb. Although the medial aspect of the forelimb is the most frequent site, physitis also occurs laterally in a foal with a carpal valgus deformity. Physitis arises at a time in the most active growing phases of young horses, when endochondral ossification is at its peak in the affected physis. It also occurs in the contralateral limb after a chronic lameness, at the fetlock in the younger foal, or the carpus in older foals and yearlings. For example, I have encountered physitis in the contralateral forelimbs of foals with severe acquired flexural deformity of the distal interphalangeal joint (Fig. 59-1). Although physitis may result in an upright conformation of the fetlock joint, probably from off-loading the limb to relieve pain, physitis does not cause other flexural deformity.

Physitis is more common on farms that use rigorous or heavy feeding programs. Owners whose stock live on lush pasture and are supplemented with concentrate feed have a higher incidence of physitis among their horses than those who keep their horses on leaner management. A higher incidence of developmental orthopedic disease is noted in foals and yearlings raised on young (<7 years old) pastures.

I have seen balanced mineral supplementation and withdrawal of concentrates dramatically reduce the incidence of physitis on some farms. Diet and forage analysis can be helpful where high incidence of developmental orthopedic disease is persistent (see Chapter 57), but mineral clearance ratios (creatinine phosphate in particular) are rarely necessary to adjust supplementation. Certain mares may also produce more foals with physitis compared with similarly managed mares, which implies a hereditary predisposition.

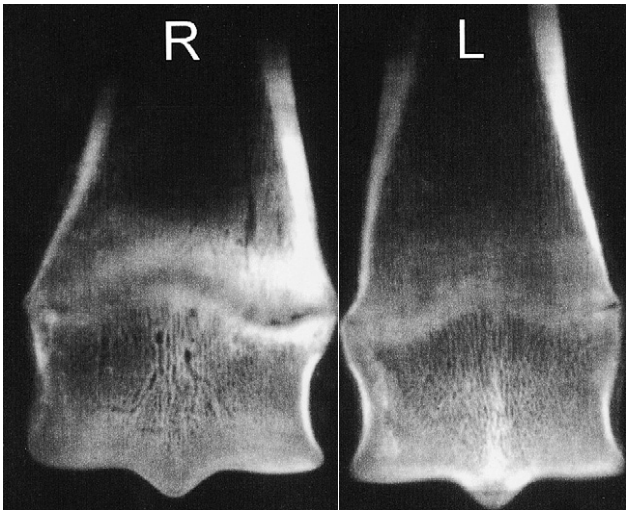

Fig. 59-1 Dorsopalmar radiographic view of frontal sections of the distal third metacarpal bones of a 5-month-old foal with chronic and severe flexural deformity of the left *(L)* distal interphalangeal joint and physitis in the contralateral distal metacarpal physis *(R)*. Note the more intense calcification of the growth plate cartilage and bridging at the medial aspect of the right forelimb, typical of Salter-Harris type V growth plate injury.

CLINICAL SIGNS

Clinical physitis is recognized at three main sites: the distal cannon (metacarpal or metatarsal growth plates), the distal radius, and, less frequently, the distal tibia. During lameness investigations, physitis has been noticed at other sites, such as the distal femur and cervical vertebrae.

Physitis is seen in the fetlock region between the ages of 3 and 6 months and occurs at the distal radius from 8 months to 2 years of age. Clinical signs are characterized by firm, warm, and painful enlargement of the growth plate, most commonly on its medial aspect. The proximal physis of the proximal phalanx also may be enlarged at the fetlock, giving the fetlocks an hourglass shape when viewed from the cranial aspect (Fig. 59-2). Early signs at the distal radial or tibial growth plates include a convex appearance in the medial contour of the metaphysis just proximal to the physis: this should be a straight or slightly concave outline. Swelling also is located around the dorsomedial aspect of the distal radius (Fig. 59-3). This dorsal component can lead the clinician to suspect that the condition has arisen from external trauma. As with the fetlock joint, there is warmth on palpation and tenderness when pressure is applied across the growth plate while the leg is held partially flexed. Full and forced flexion of the fetlock, carpus, or tarsus also may be resented. Most affected horses have changes at the distal medial radius, but a few with pre-existing carpus valgus conformation may develop the condition laterally. Bilateral lameness may be seen as a stilted gait. The conformation may become more upright through the pastern, and fetlock varus deformity may develop

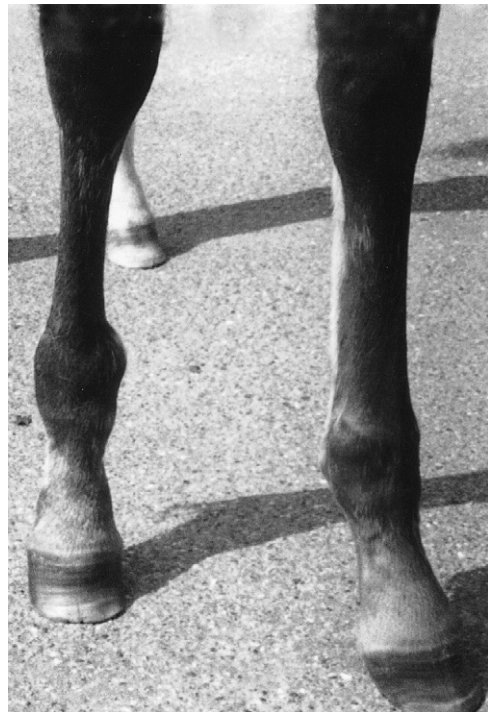

Fig. 59-2 Physitis of the right distal third metacarpal bone (growth plate), with some involvement of the proximal physis of the proximal phalanx, giving the fetlock an hourglass shape.

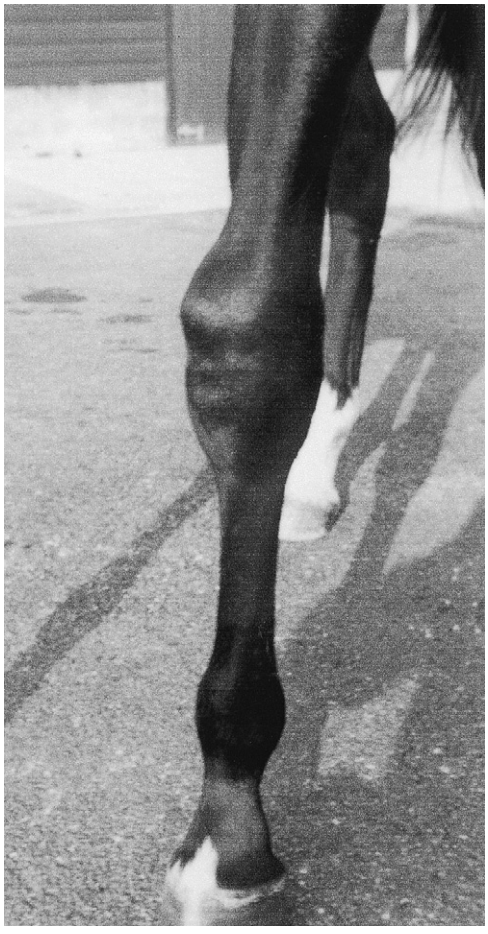

Fig. 59-3 Dorsal view of a left forelimb; lateral is to the right. Physitis of the left distal radial growth plate with characteristic dorsomedial swelling and concavity of the outline of the distal medial metaphysis.

because the horse walks on the outside of the hoof to alleviate pain from pressure on the medial physis. I suspect that foals and yearlings that develop a carpus varus conformation (start to go bandy) are likely to have a subclinical form of physitis that encourages them to bear more weight on the lateral aspect of the forelimbs. If physitis continues unchecked, growth ceases on the medial side, exacerbating the carpus varus deformity, and ultimately, premature closure of the growth plate fixes the abnormal conformation. This chain of events also occurs in horses that develop physitis in the lateral aspect of the distal radius, resulting in a permanent carpus valgus conformation.

TREATMENT

Treatment centers on stable rest. Non-steroidal anti-inflammatory drugs can be helpful, particularly in horses that are very lame or have inflamed and painful growth plates. I have no experience of using corticosteroids or anabolic steroids in horses with physitis. Although body condition and conformation can vary, it is important to assess the nutrition of affected foals and other horses. Reduction of body weight and particularly a reduction in energy content in the diet must be made. Starving these horses completely is unwise, and it is important that mineral intake includes the correct balance of calcium and phosphorus and adequate copper and zinc. The length of rest required varies with the severity of the physitis and is between 2 weeks and 2 months. Corrective hoof trimming must also be undertaken, especially if the horse has developed an angular limb deformity (see Chapter 60). For varus deformity the unworn medial hoof wall must be removed (inside is lowered), and if the angulation is severe, fitting a lateral hoof extension is necessary.

Surgery usually is unsuccessful in horses that develop severe varus limb deformity resulting from physitis. This experience, coupled with the disappointing results obtained when operating on yearlings with congenital angular deformities, has led me to stop recommending periosteal elevation or physeal retardation techniques in these groups of affected horses.

The prognosis for racing or other extreme athletic activities is good, provided the condition was not so severe that the conformation is permanently affected. If carpus varus and bench-knee conformation is significant, then the animal is likely to suffer lameness from splints, carpitis, sesamoiditis, or suspensory branch desmitis when more serious training is undertaken.

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CHAPTER • 60

Angular Limb Deformities

DIAGNOSIS AND CONSERVATIVE MANAGEMENT

• Eric J. Parente

Angular limb deformities are considered lateral or medial deviations to the long axis of the limb in the frontal plane. A lateral deviation distal to the carpus, termed *carpus valgus*, is the most common. Tarsus valgus and fetlock varus (a medial deviation) are the next most common, respectively. The toed-in or toed-out appearance that often accompanies an angular deformity is a concurrent rotational deformity and should not be confused with the angular deformity. An outward (external) rotation is usually observed concurrently with a valgus deformity and an inward (internal) rotation is seen with a varus deformity. No confirmed breed or sex predilection is known, but subjectively there may be some genetic predisposition. For a foal to be born with a mild bilateral carpal valgus and toed-out appearance is considered normal by many clinicians, and as the foal grows and the chest widens, the limbs straighten progressively.

Angular limb deformities usually are congenital, but they can be developmental, and have numerous causes. An acquired angular deformity is defined as worsening or failure of correction of the normal slight carpus valgus conformation in the neonatal foal. The cause and the severity and progression of the deformity are vital pieces of information that should be acquired before formulating a treatment plan. Treatment is predicated by these factors. Foals are like molding plastic. The conformation changes slowly with growth. Genetics, nutrition, amount of exercise or weight bearing, and veterinary interventions all influence the conformation of the adult horse. Some of these factors can be influenced as the animal matures and often small adjustments need to be made to the treatment plan as the animal grows. The foal should always be evaluated with the amount of remaining growth in mind and not just its present status. Because the greatest impact on conformation is made during periods of rapid growth, early recognition and regular re-evaluations are extremely important to achieve a positive outcome.

Growth rates are most rapid in the neonate and slow considerably within the first year. Most of the growth from the distal radial and tibial physes is within the first 6 months of age. Most of the growth from the distal third metacarpal (McIII) and metatarsal (MtIII) bones is within the first 3 months of age. Minimal changes take place beyond these times. Radiography alone cannot be used to determine the end of bone growth, because the physis is radiographically apparent long after clinically relevant growth has abated. A normal foal should correct a carpus valgus to within 5° to 7° of normal by 4 months of age and should be almost straight by 8 to 10 months of age.

EXAMINATION OF THE FOAL

It is critical to determine the extent and cause of the deformity before developing a management plan. Foals should be examined while standing and walking and also by radiography. Occasionally foals are assessed in lateral recumbency.

Deformities can be assessed subjectively by visual examination. The foal should stand as squarely as possible, with the foot directly below the proximal part of the limb. Deviations from this stance exacerbate any deformities that truly exist. Because most foals only stand still transiently, repositioning the foal several times to evaluate each limb independently is often necessary. This allows observation of how the foal stands most frequently in a relaxed position. The clinician stands directly in front of the dorsum of the long bones for evaluation of the forelimbs, not necessarily at the front of the toe. The orientation of the toe may be affected by a concurrent rotational deformity, which confounds interpretation. Hindlimbs should be evaluated similarly but directly from behind. The forelimbs can also be evaluated by standing shoulder to shoulder with the foal, looking down the limb toward the ground.

All limbs also should be evaluated with the foal walking away from and toward the clinician. Breakover is determined for each foot, which may be helpful in deciding the most appropriate way to manage the foal. The entire assessment of a foal should be graded and recorded on video or on paper for future reference.

Radiography provides an objective assessment of angular deformity, but sequential radiography may be unreliable if the obliquity varies. Differences in radiographic projection can result in a misinterpretation of worsening or improvement, which is particularly true when trying to quantify small differences in the angle. Long, narrow (18 × 43 cm) cassettes should be used to measure the angle of the deformity, by evaluating the intersection of a line representing the long axis of the proximal and distal long bones from the joint in question. This is more accurate in the carpus and fetlock than the tarsus. Radiography is essential to identify cuboidal injury or malformation. Such a deformity dramatically worsens the foal's prognosis. Foals with angular deformity resulting from cuboidal bone abnormalities usually have compromised range of motion, but this often is detected best with the foal in lateral recumbency.

PERI-ARTICULAR LAXITY

Peri-articular laxity is the major cause of congenital angular limb deformities and often improves dramatically within the first 4 weeks of life, without any intervention, as the peri-articular tissues become less elastic. The improvement is most dramatic in a windswept foal, which has a tarsus valgus of one limb and a concurrent varus of the other. Limited exercise is all that is required for these foals to become normal.

Infrequently the deformity can be so severe, particularly in the fetlock, that the foal is unable to bear weight on the sole of its foot. Immediate treatment is required to establish normal weight bearing. Custom-made glue-on shoes are particularly useful to prevent abnormal breakover and to keep the foot flat on the ground. If the foal has excessive laxity of the lateral collateral ligaments and a tendency to break over on the lateral side of the foot, a lateral extension shoe is used

to maintain appropriate alignment of the limb. The foal should initially be restricted to a stall before turnout in a small paddock or round pen with just the mare. Soft tissues become progressively stronger, and normal activity can be permitted within a relatively short time. Allowing premature excessive exercise can lead to proximal sesamoid bone fractures (see Chapter 37) and other injuries. Glue-on shoes are usually required for several weeks, but they then should be removed to avoid contracture of the foot. External coaptation also should be avoided if possible. Splints are used only to maintain joint alignment if absolutely necessary. Splints are contraindicated to try to pull or push a limb straight. Rigid support from a splint or cast usually leads to greater soft tissue laxity. Trying to support a limb results in continued laxity and soft tissue wounds from bandaging. Every foal must be managed on an individual basis with the goal of achieving normal weight bearing and function while providing the minimal amount of support necessary.

ASYMMETRICAL PHYSEAL GROWTH

Asymmetrical growth of a distal physis is a cause of angular limb deformity. Greater growth from the distal physis of the radius medially compared with growth laterally results in carpus valgus. Continued asymmetrical growth precludes the normal correction anticipated with resolution of peri-articular laxity. Greater growth from the lateral distal physes of the McIII or MtIII bones results in fetlock varus. With time and limited exercise (stall or small paddock turnout, alone with the mare) substantial self-correction occurs for most foals with angular deformities. Radiography can be used to evaluate objectively the degree of deformity and the difference in physeal growth, but it is not always required.

Fetlock varus is often overlooked and affects the left hind fetlock most commonly, possibly because of in utero positioning. Because growth at the distal physis of McIII and MtIII bones terminates within a few months, early recognition of this deformity is critical.

TRAUMA-RELATED DEFORMITIES

Developmental causes of angular deformities are likely secondary to excessive or asymmetrical weight bearing and can be more serious than congenital abnormalities. Incomplete ossification of the cuboidal bones in a foal can result in cuboidal bone crush and a secondary angular limb deformity. The lateral carpal bones most commonly are affected, resulting in carpus valgus. Although the deformity is clearly apparent, the health and function of the joint is most critical. The carpi and tarsi of any premature or dysmature foal should be examined radiographically. Because ossification is incomplete, abnormal cuboidal bones appear round or in other shapes and, if crushing exists, the bones are wedge shaped, overlap opposing bones, or are fractured. If the foal's activity can be managed strictly in a hospital situation, no further support is recommended. Limited, strictly controlled exercise encourages appropriate ossification. If the foal's activity cannot be strictly managed and the foal has moderate strength, sleeve casts are recommended to prevent cuboidal bone crush. Sleeve casts should be changed or removed in 10 to 14 days in a growing foal. Radiographic re-evaluation every 2 weeks helps determine the length of time a cast is required.

Foals can also develop angular limb deformities secondary to lameness in the contralateral limb. If foals become non–weight bearing on a single limb, they adopt a tripod-like stance by bringing the contralateral limb more medial and toeing in. Over several weeks this results in asymmetrical bony growth of the non-lame limb and varus deformity of all joints, with internal rotation of the distal limb. A dorsolateral toe extension can be placed on the foot of the non-lame limb to try to encourage a more abducted stance, but resolution of the primary lameness is the best way to prevent the contralateral limb deformity from occurring. Once the deformity has occurred, correction is improbable.

CONSERVATIVE MANAGEMENT

In most situations, judicious minimal intervention is all that is required to correct angular limb deformities. This includes limited small paddock exercise and careful attention to the foot. Trimming the foot and using shoe extensions can influence a foal's ability to self-correct an angular limb deformity by affecting the weight bearing and tracking of the limb. A foal younger than 4 months of age with mild carpus valgus likely requires no specific intervention. A foal with a moderate carpus valgus, toed-out appearance would benefit from shoes with dorsomedial extensions. This encourages the foal to adduct the limb and turn in more when breaking over. The result is more physiologically normal strain on the limbs, which should result in a straighter limb. A similar external appearance can be achieved by over-trimming the lateral hoof wall, but the effect is not the same. Although trimming the foot in this manner may give the appearance of an immediate benefit, it may have a negative long-term effect of an unbalanced foot, compensating for a limb with a deformity. This method has, however, been used for years with apparent success.

More aggressive intervention is infrequently required. Surgical intervention should be reserved for those foals that are not improving fast enough for the amount of growth potential remaining. Therefore it is critical for frequent re-evaluation of a foal with an angular limb deformity to monitor progress. Surgical intervention has a greater effect on a young foal because of the more rapid growth; thus early surgical intervention should be considered for a foal with a severe deformity.

SURGICAL MANAGEMENT

• José García-López

A high proportion of foals with angular limb deformities are treated successfully conservatively, but surgical intervention is warranted if a deviation is severe, or if deformity persists despite adequate management, including restriction of exercise and corrective farriery. A variety of surgical techniques aimed at accelerating or decreasing the growth on a particular side of the growth plate have been described.[1-6] Surgical technique depends on the age of the horse, the degree of angular limb deformity, the anatomical site, and whether the deformity is varus or valgus (Figs. 60-1 and 60-2).

Before surgery, all the limbs should be assessed from the front, back, and while standing next to the limbs. Good-quality radiographs, which include a substantial length of the bones proximal and distal to the deviation, should be obtained to assess bone structure (Fig. 60-3) and to determine the pivot point and pivot point angle of the deviation. The pivot point is the intersection of two lines drawn parallel to the long axis of the bones proximal and distal to the articulation in question (Fig. 60-4). The pivot point indicates the origin of the deviation and helps to determine whether the cuboidal bones, in the case of the carpus and tarsus, are involved in the deviation, or if deviation is caused by disproportionate physeal growth only. Abnormalities in the structure of these bones can

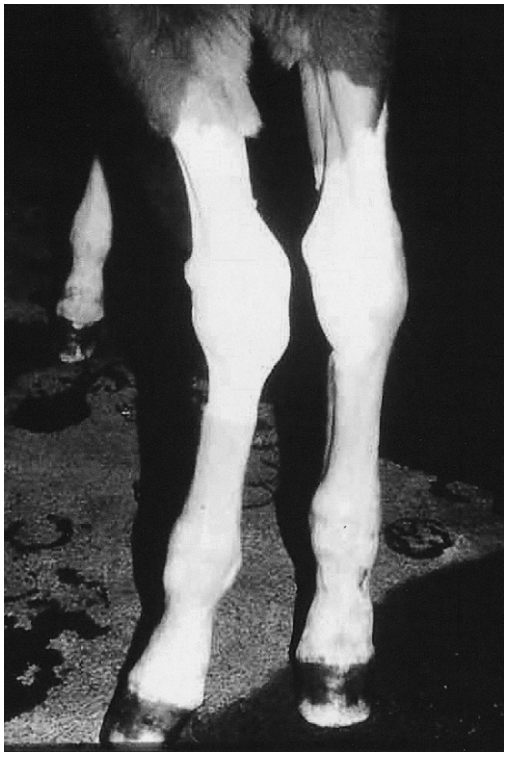

Fig. 60-1 Frontal view of a foal with right carpus valgus.

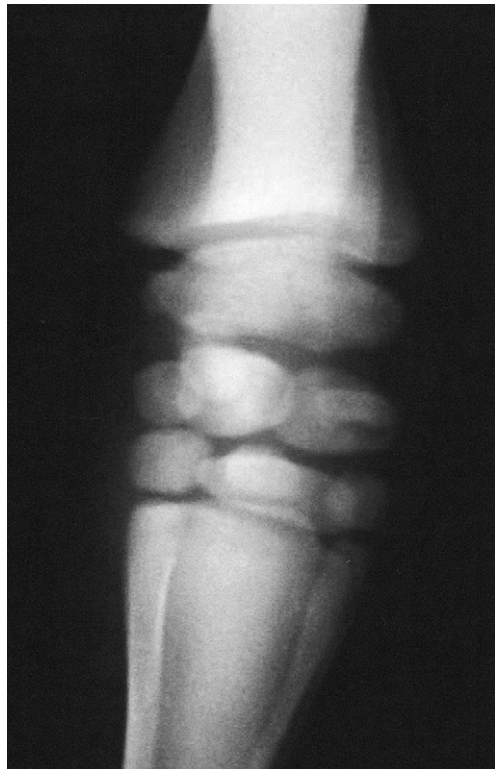

Fig. 60-3 Dorsopalmar radiographic view of a carpus with mild incomplete ossification of the carpal bones and valgus deformity. Medial is to the right.

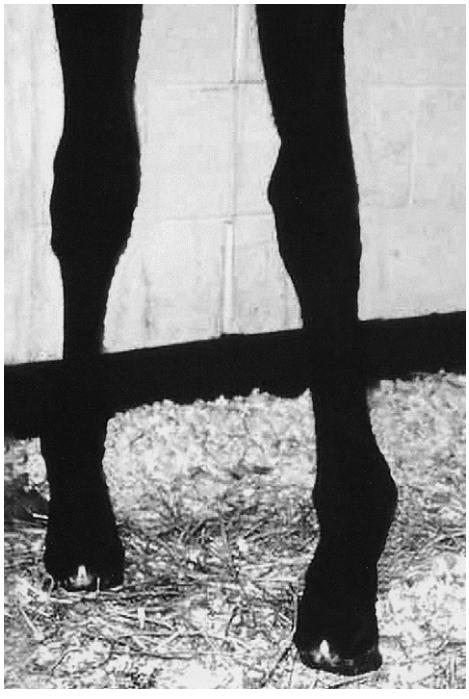

Fig. 60-2 Cranial view of a foal with varus deviation of the left metacarpophalangeal (fetlock) joint.

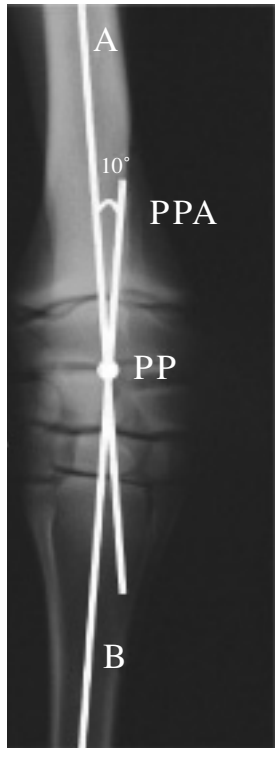

Fig. 60-4 Dorsopalmar radiographic view of a carpus with valgus deformity. Medial is to the right. Lines are drawn parallel to the long axis of the radius (*A*) and third metacarpal bone (*B*). The point where these lines intersect is known as the pivot point (*PP*), and the angle between them is the pivot point angle (*PPA*).

have a substantial influence on the effect of the procedure and the future athletic potential of the horse. The pivot point angle is the angle formed by the intersection of these two lines and indicates the severity of the condition.[7]

Hemicircumferential periosteal transection and elevation, or periosteal stripping, aims to accelerate growth on the concave side of the limb, laterally for valgus and medially for varus deformities.[1] Previous work on chicken radii had shown that a circumferential division of the periosteum, rather than a longitudinal one, resulted in increased bone growth. The proposal was that the periosteum functioned as a fibroelastic tube, which spanned the diaphysis and provided an even tension between both epiphyses, that was responsible for the regulation of growth.[1,8] A horizontal or circumferential division of the periosteum would result in a release of tension at the level of the growth plate, resulting in the induction of new bone production on the side of the division.

The procedure has been thoroughly described.[4-6] Hemicircumferential periosteal transection and elevation is performed with the foal in lateral recumbency under general anesthesia, with the concave side of the affected limb uppermost. If the procedure is going to be performed bilaterally, dorsal recumbency is recommended. The position of the physis is identified using a 20-gauge needle. For a carpus valgus deformity, a 4- to 6-cm longitudinal incision is made between the common and lateral digital extensor tendons starting just proximal to the physis. The incision is extended through to the periosteum. Using a curved scalpel blade (No. 12), a horizontal incision is made 1 to 2 cm proximal and parallel to the physis, at the distal end of the initial incision (parallel to the skin incision) forming an inverted T. The periosteal flaps are elevated with the aid of a periosteal elevator and then allowed to return to the normal position. It is important to transect the remnant of the ulna, or if the ulna is ossified, it should be removed with the aid of rongeurs. In foals with hindlimb tarsus valgus the veterinarian should bear in mind that a fibular remnant may be present. The incision is closed routinely and the area is bandaged for 10 to 14 days. The foal is kept in a large stall or small paddock until the deformity has been corrected.

Hemicircumferential periosteal transection and elevation has been reported to exert its effects for up to 2 months, but the procedure can be repeated if further correction is needed.[4] There are no reports of over-correction of the deformity. Early reports suggested approximately an 80% success rate[1,3]; however, more recent work indicates less favorable results.[9-11] A large retrospective study in Thoroughbred racehorses investigated racing performance after hemicircumferential periosteal transection and elevation.[9] A lower percentage of treated horses were able to start a race and had a lower starts percentile ranking number compared with half-siblings. Most of the foals appeared to respond favorably to hemicircumferential periosteal transection and elevation based on external appearance of the limbs, but pre-existing conditions, such as abnormal cuboidal bone formation from incomplete ossification or osteoarthritis secondary to abnormal loading of the limb before correction, may have influenced subsequent performance.

Angular limb deformities are probably the most common orthopedic problem affecting Thoroughbred foals.[9,12] Early surgical intervention was previously recommended to take maximum advantage of the growth potential of the physis, to try to provide foals with excellent conformation, and to enhance sale value and possibly potential performance. Based on current knowledge, it now seems likely that a large number of foals with mild angular limb deformities underwent unnecessary surgery. The reported success for correction of angular limb deformities, particularly in the carpus, after hemicircumferential periosteal transection and elevation has been recently challenged. Foals suffering from carpus valgus that underwent hemicircumferential periosteal transection and elevation were no more likely to improve compared with those managed with stall rest and corrective farriery.[10,11] Although the efficacy or need to perform hemicircumferential periosteal transection and elevation when treating foals with mild to moderate carpus valgus deviations is a matter of current debate, the same cannot be said necessarily in other regions of the limb, such as the tarsus and fetlocks, without further investigation.

Transphyseal bridging is performed on the convex side of a deformity to decrease the growth rate on that side of the physis. Traditionally, transphyseal bridging has been performed in young foals with severe deformities or in foals in which most of the growth in a particular physis has subsided. Transphyseal bridging has been performed successfully using cortical screws with cerclage wire in a figure of eight, with a small dynamic compression plate, or with orthopedic staples.[4-6]

The foal is positioned in lateral recumbency under general anesthesia, with the convex side of the affected limb uppermost, or in dorsal recumbency. The position of the physis is identified and checked radiographically using a 20-gauge needle inserted into the physis. Stab incisions are made for placement of cortical screws and cerclage wire; one incision is made in the center of the epiphysis and the other approximately 2 cm proximal to the physis. Once the subcutaneous tissues between both incisions have been bluntly dissected, a 4.5- or 3.5-mm cortical screw is placed through each incision, slightly angled toward the physis. Screw size depends on the size of the foal, but 4.5-mm screws are preferred. Before complete tightening of the screws, a loop of 18-gauge wire is fed through the proximal incision and positioned around the distal screw head. The wire is then tightened over the proximal screw head, forming a figure of eight. Once the wire is tightened, the screws are tightened and the incisions are closed routinely. When performing this procedure in the distal tibia, the clinician should be aware of the contour of the tarsocrural joint, because placing the epiphyseal screw into the joint is possible. The screw should be angled more sharply toward the physis than in the distal radius. Post-operatively the foal should be kept in a large stall and monitored closely. Follow-up radiographs are strongly recommended to assess the amount of correction. Prompt removal of the implants is essential, because over-correction of the deformity is possible.

In foals and young horses with an angular limb deformity that persists after physeal closure, corrective ostectomy or osteotomy can be performed. A step ostectomy (sagittal plane) or step osteotomy (frontal plane) are preferred to closing wedge ostectomy, because limb length is preserved and interfragmentary compression between the bone pieces is better.[4] These techniques are generally considered to be a salvage procedure.

Tarsus valgus deformities frequently go unrecognized by both owners and veterinarians, possibly from lack of observation of the foal from behind and the inherent offset position of the tarsus.[13,14] Early recognition and sometimes more aggressive surgical management of tarsal angular limb deformities are critical to achieve satisfactory results. Although the distal tibial physis has a tremendous growth rate until 4 months of age, foals younger than 2 months of age responded more favorably to hemicircumferential periosteal transection and elevation than older foals. Transphyseal bridging was more effective than hemicircumferential periosteal transection and elevation, especially in foals older than 2 months of age.[13] This is a significant change from the previous perception that hemicircumferential periosteal transection and elevation alone was adequate when managing most tarsus valgus deformities in foals 4 to 6 months of age. Early recognition of incomplete ossification of the tarsal bones (Fig. 60-5) is crucial, because the condition, if unrecognized, leads to collapse of the third or central tarsal bones,

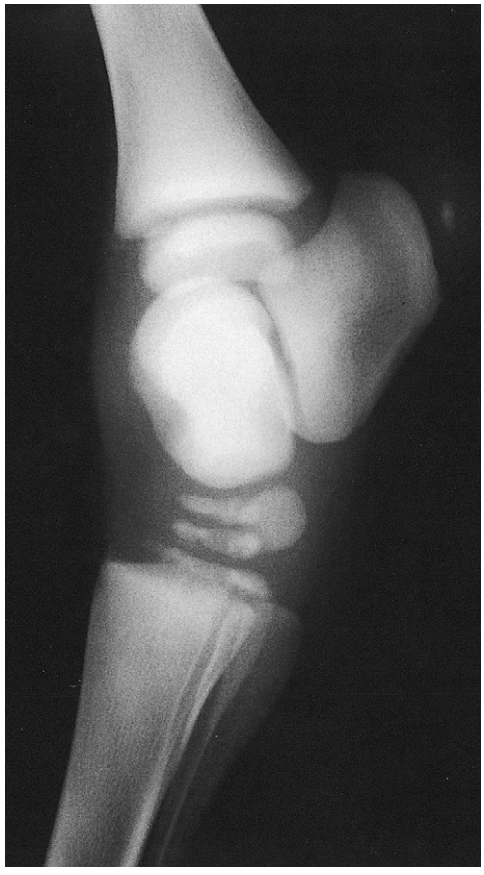

Fig. 60-5 Lateromedial radiographic view of neonatal tarsus with incomplete ossification of the tarsal bones.

resulting in osteoarthritis.[13,14] Of 22 foals with incomplete ossification of the tarsal bones, 73% had tarsus valgus deformities. Only 32% of the foals were able to reach the intended use.[14]

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Suggested Readings

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Dutton DM, Watkins JP, Honnas CM, et al: Treatment response and athletic outcome of foals with tarsal valgus deformities: 39 cases (1988-1997), *J Am Vet Med Assoc* 215:1481, 1999.

CHAPTER • 61

Flexural Limb Deformity in Foals

Robert J. Hunt

Flexural limb deformity is the inability to extend a limb fully. Generally hyperflexion of a region results from a disparity in the length of the musculotendonous unit relative to the length of the bone. The deformities are categorized as congenital or acquired and may occur during in utero development or at any time after birth. Different anatomical structures may be involved including the flexor tendons, suspensory apparatus, joint capsule, and surrounding fascia, skin, and bone.[1-6]

CONGENITAL FLEXURAL DEFORMITY

Congenital deformities are present at birth and the cause is often unknown. Although uterine malposition of the fetus is often discussed, because of the voluminous nature of the uterus and the ability of the foal to move, uterine malposition is an improbable cause of flexural deformity in most instances. Other documented causes include exposure of the mare to influenza or ingestion of Sudan grass, locoweed, or other teratogenic agents during development of the fetus.[2,3,7] Equine goiter, neuromuscular disorders, and defects in elastin formation or collagen cross-linking may be involved in the pathogenesis.

Congenital flexural deformities most commonly involve the carpus, tarsus, metacarpophalangeal and metatarsophalangeal joints singularly or in combination.[2] Both forelimbs are usually involved and occasionally other variations of skeletal deformities are evident, such as hindlimb deformity (windswept), spinal deformity, rye nose, and cleft palate. Sporadically occurring deformities involving the scapulohumeral, cubital, coxofemoral, or femorotibial joints may occur in combination with other congenital skeletal anomalies. Congenital flexural deformities are a common cause of dystocia in the broodmare, resulting in loss of the foal and subsequent reproductive loss of the mare.

Treatment for congenital flexural deformities varies with the anatomical location involved and severity of the condition. Therapeutic intervention for severe flexural deformities associated with arthrogryposis or gross spinal deformities should be discouraged, and humane destruction is recommended. Surgical transection of the flexor tendons and palmar carpal fascia generally fails to alleviate this deformity. Splinting and casting is also futile, although rare foals have survived with limited athletic potential.

Mild flexural deformity of the carpus or metacarpophalangeal/metatarsophalangeal joints resolves spontaneously if the foal has the ability to stand, nurse, and ambulate on its own. Most foals with mild and moderate deformities respond favorably to physiotherapy, by manually extending the limbs every 4 to 6 hours for 15-minute sessions, or forcing the foal to ambulate (Fig. 61-1). Heavy bandaging, splinting or casting, and the administration of oxytetracycline can be helpful.[2,8] Foals with deformities advanced enough to require assistance to stand respond more rapidly after application of cast, which should be changed every 2 to 3 days (Fig. 61-2). If a carpal deformity severe enough to prevent standing is recognized at parturition, the foal should be heavily sedated, within 30 to 45 minutes, and full-leg casts should be applied with the limbs placed in extension. These should be changed within 24 hours and reset. Generally a rapid response is seen after the first or second application of casts. If the foal incurs any complications with the casts or becomes distressed, casts should be removed. Commercially available articulating braces (Almanza Corrective Boot; Agustin-Almanza@usa.net; Equine Bracing Solutions, Trumansburg, NY) are available, which allow adjusting the degree of extension in a specific region of the limb (Fig. 61-3). As with all splints, caution must be exercised to prevent pressure sores. It is important to assist the foal nursing and to provide supportive care in an intensive neonatal facility, because these are high-risk patients for developing complications with other body systems.

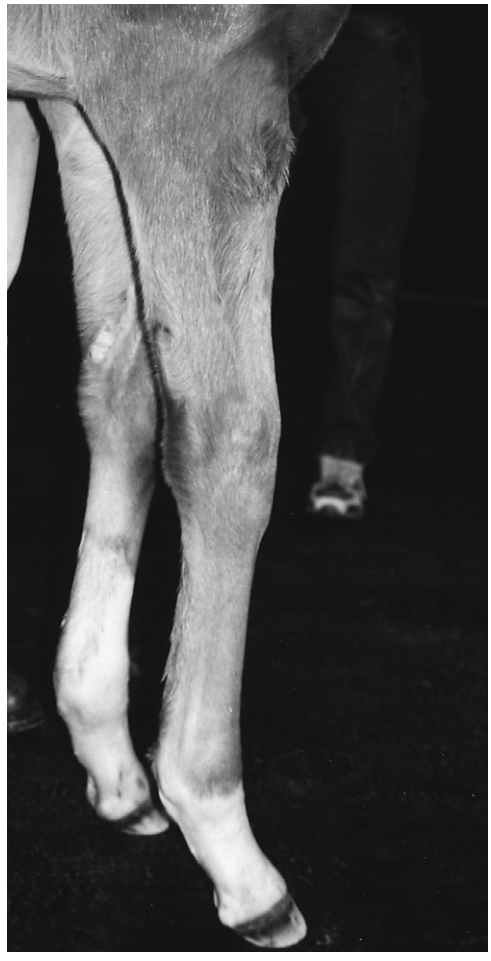

Fig. 61-1 Mild bilateral flexural deformity of the carpus. This degree of deformity is self-limiting.

Fig. 61-2 Moderate to severe bilateral flexural deformity of the carpus. This foal required assistance to stand, external coaptation, and oxytetracycline therapy.

Oxytetracycline (2 to 4 g) administered slowly intravenously is also beneficial, but it should not be used in the neonate until serum creatinine is within normal limits.[8] Oxytetracycline may be given daily or every other day for 3 to 4 treatments. Complications of oxytetracycline administration include renal failure, diarrhea, and, most commonly, excessive laxity of other normal joints. The precise mechanism of action for tendon relaxation remains speculative.

Flexural deformity of the distal interphalangeal joint occasionally is seen at birth but is most often self-limiting if the foal can stand without buckling forward. Bandaging of the lower limb and administration of oxytetracycline hastens recovery. If the foot angle is beyond 90° (i.e., the dorsal aspect of the foot is in front of the vertical), casting or splinting may be necessary.

When splints are used, the clinician must exercise extreme caution to prevent rub sores or pressure sores. Splints should only be applied for brief periods, in a schedule such as 4 to 6 hours on, followed by 4 to 6 hours off. Properly applied casts are more effective, require less maintenance, and result in fewer pressure sores than splints.

Prognosis for future performance as an athlete is good for foals with congenital flexural deformities that progressively respond favorably in the first 2 weeks of life. In general, if the foal is able to stand, nurse, and ambulate without assistance, most congenital flexural deformities are self-limiting and foals warrant a good prognosis. Foals may require several months to stop buckling dorsally at the carpus. Foals with flexural deformity of the metacarpophalangeal or metatarsophalangeal joints typically respond within the first or second week of life. Prognosis for future performance is poor for foals with severe flexural deformity of the carpus, tarsus, or metacarpophalangeal/metatarsophalangeal joints that show no improvement after several days of treatment and have difficulty ambulating without assistance after 1 to 2 weeks of treatment. Foals with congenital flexural deformities involving the scapulohumeral, cubital, coxofemoral, or femorotibial joints have a poor prognosis for future athletic performance.

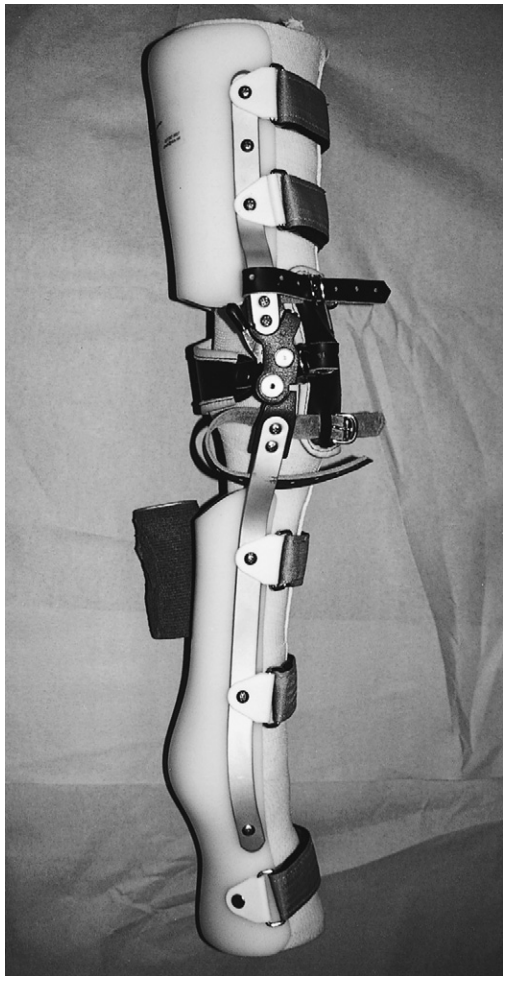

Fig. 61-3 Articulating brace used in foals with flexural deformity of the carpus and the metacarpophalangeal joint.

ACQUIRED FLEXURAL DEFORMITIES

Acquired flexural deformities develop after birth until the second year of life. Commonly involved areas include the distal interphalangeal joint, metacarpophalangeal joint, and carpus. Acquired flexural deformity of the distal interphalangeal joint is recognized between 3 and 6 months of age, carpal flexural deformity is seen between 1 and 6 months of age, and flexural deformity of the metacarpophalangeal joint is recognized later during the yearling or early 2-year-old year (9 to 18 months).[9]

Common causes for flexural deformity of the metacarpophalangeal joint and carpus are believed to include a genetic propensity for rapid growth, over-nutrition, and pain. Relative excess feeding or over-assimilation of nutrients in foals that have an inherent potential for rapid physical development is a frequent finding in foals with acquired flexural deformity, especially involving the carpus and the metacarpophalangeal joint. During periods of rapid bone lengthening the potential for passive elongation of the tendonous unit is limited because of the accessory ligaments and a discrepancy in the length of the bone to tendon unit may result. Another theory is that a pain-mediated response during physeal dysplasia results in altered load bearing on the limbs. This is believed to initiate secondary contraction and shortening of the musculotendonous unit, resulting in limited extension of a region. Any cause of pain resulting in prolonged reduced weight bearing in a limb may result in this syndrome.

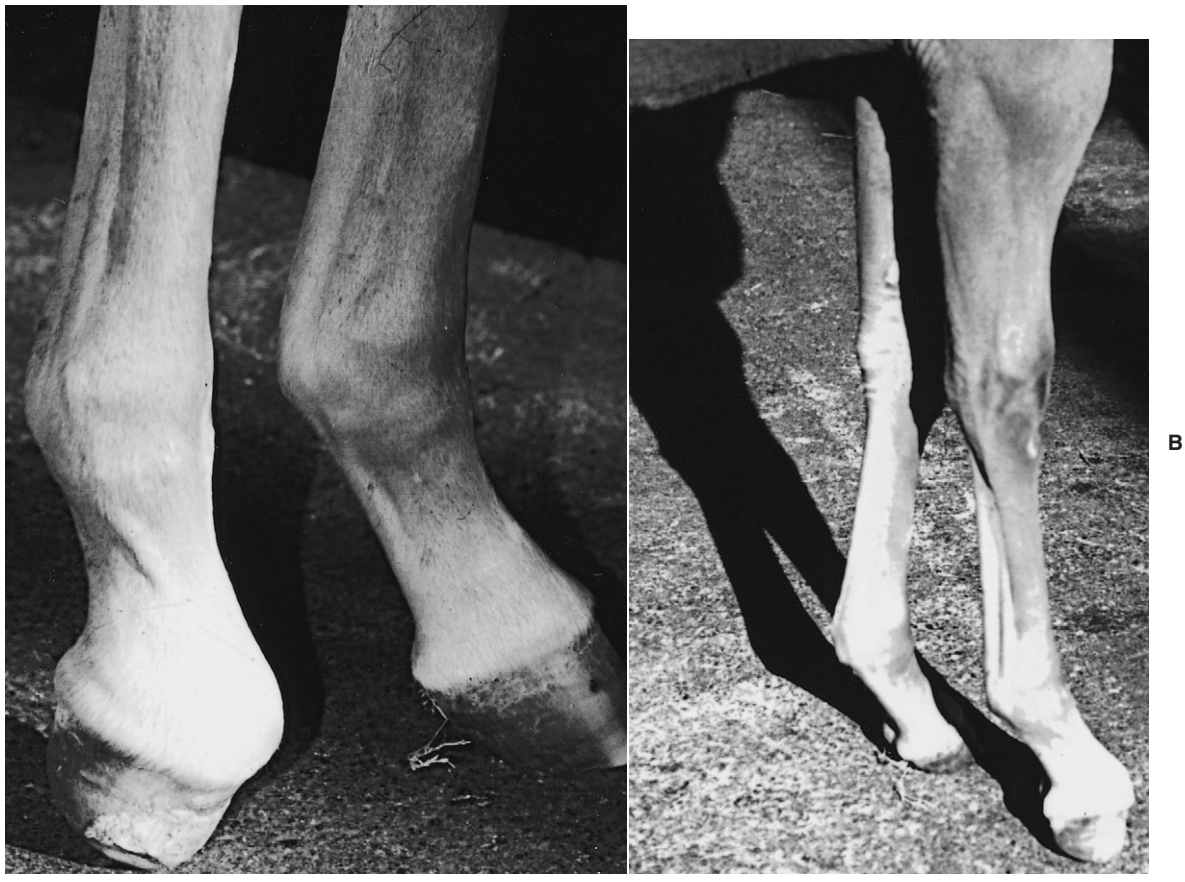

Fig. 61-4 **A,** Advanced flexural deformity of the distal interphalangeal joint. The foal's heels do not contact the ground, and the dorsal hoof wall is dish shaped. **B,** Calf-kneed conformation is associated with a clubfoot.

Genetics may be involved with the development of acquired flexural deformity of the distal interphalangeal joints (clubfeet). Other factors include diet and exercise. Protracted lameness from another cause such as osteochondrosis of the upper limb also predisposes to development of acquired distal interphalangeal joint deformity. It is therefore important to isolate an associated source of lameness.

Early clinical signs of acquired flexural deformity of the distal interphalangeal joint consist of a prominent bulge at the coronary band, increase in length of the heel relative to the toe, and failure of the heels to contact the ground after trimming (Fig. 61-4, *A*). Eventually the foot develops a boxy shape with a dish shape along the dorsal hoof wall. This deformity is commonly accompanied by a back-at-the-knee (calf-knee) conformation (Fig. 61-4, *B*).

Treatment for foals with acquired flexural deformity of the distal interphalangeal joint initially entails exercise restriction, frequent lowering of the heels, dietary restrictions, and pain control with non-steroidal anti-inflammatory drugs such as flunixin meglumine. Weaning the foal may be advantageous. If the toe becomes excessively worn, constructing a cap over the toe with one of the commercially available hoof composites may aid in protecting the toe and dorsal sole from bruising and may temporarily serve as a lever arm. Although elevating the heel with a wedge shoe or pad provides comfort for the foal, the deformity gradually worsens and eventually obtaining a normal angle and shape to the hoof is difficult. Glue-on shoes should likewise be used with caution, because they tend to promote a box shape to the foot. Oxytetracycline may be used but exacerbates the back-at-the-knee conformation.

If no improvement is achieved within 1 to 2 months of conservative treatment, surgical intervention with desmotomy of the accessory ligament of the deep digital flexor tendon (ALDDFT) is recommended. When combined with a trimming program, hoof conformation and function will be much improved. The owner should be made aware of the potential for a blemish at the surgical site.

Foals in which the dorsal aspect of the hoof is in front of the vertical (hoof angle >90°) are candidates for deep digital flexor tenotomy, although desmotomy of the ALDDFT has been helpful in some foals when combined with farriery, oxytetracycline, and phenylbutazone therapy. With deformities of this severity, contracture of the joint capsule and surrounding soft tissues often precludes successful management for an athletic future.

Early treatment of foals with flexural deformity involving the metacarpophalangeal and metatarsophalangeal joints consists of eliminating pain through the use of analgesics, exercise restriction, and correction of any underlying nutritional problems. Any extremes in hoof angle should be corrected and the foot should be trimmed at an angle suitable for that individual. If no improvement is achieved with conservative treatment, desmotomy of the accessory ligament of the superficial digital flexor tendon or of the ALDDFT may be helpful in select instances of moderate deformity. If the underlying cause is not addressed, the benefits of surgery will only be transient. Foals with severe deformity resulting in the limb persistently buckled forward rarely respond favorably to conservative or surgical treatment. Transection of both accessory ligaments or of the superficial digital flexor tendon may provide some improvement in horses with advanced deformity.

Treatment for acquired flexural deformity of the carpus is aimed at eliminating the underlying cause, and if the deformity is recognized early, conservative management is generally effective. The nutritional program (see Chapter 57) and growth

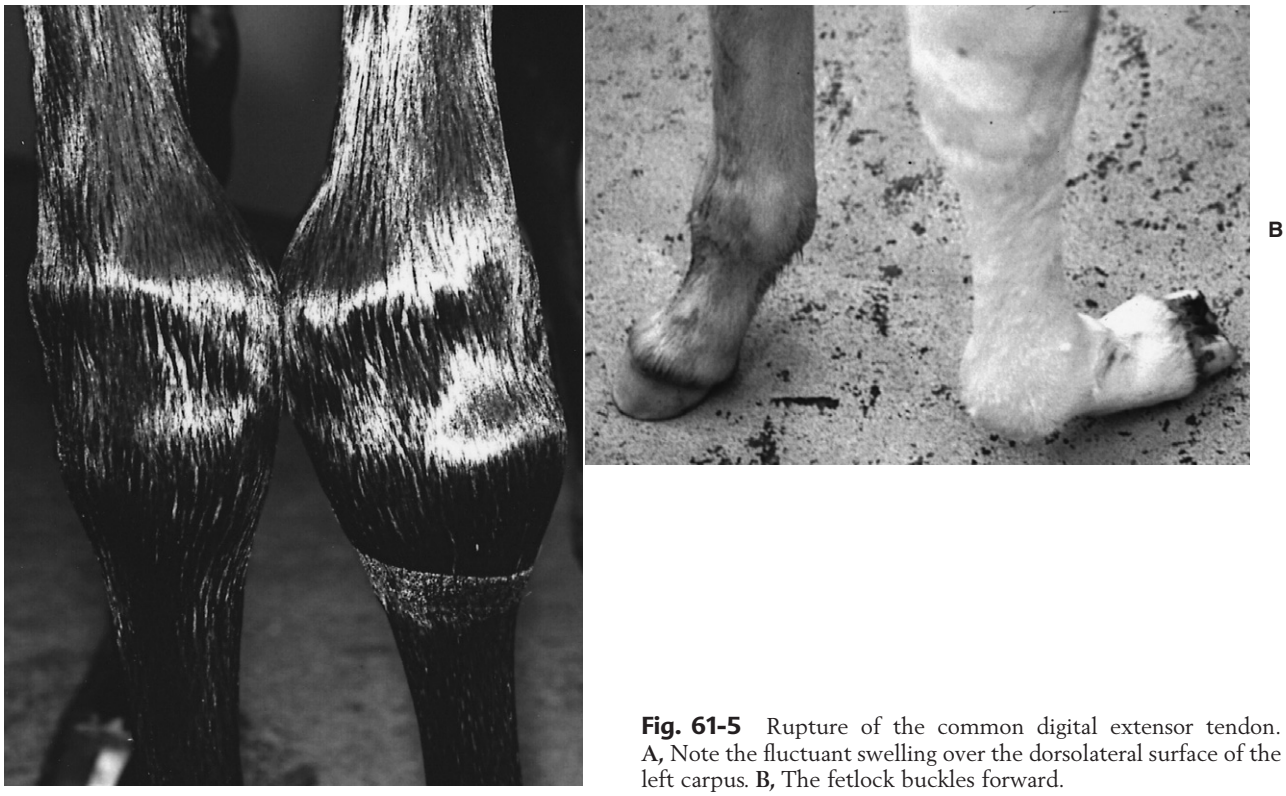

Fig. 61-5 Rupture of the common digital extensor tendon. **A,** Note the fluctuant swelling over the dorsolateral surface of the left carpus. **B,** The fetlock buckles forward.

chart for the foal should be reviewed and errors corrected if found. The nutritional program is often already balanced in some of these foals and treatment is empirical and focused on reducing the energy intake and slowing the growth. Dietary restriction and limitation of turnout schedule and exercise and treatment with non-steroidal anti-inflammatory drugs are currently accepted practice. Grain and other sources high in energy and protein to the mare should be restricted in an attempt to reduce the nutritional quality of the milk and to prevent the foal from ingesting grain while eating with the mare. In foals with severe deformity, limiting access to milk through muzzling the foal and stripping the mare periodically throughout the day may be attempted. However, weaning the foal from the mare to control the diet is generally easier. Acquired carpal flexural deformities may require weeks to months to correct. Splints, bandaging, and oxytetracycline may provide some benefit. These benefits are often short lived if the underlying cause is not addressed.

Rupture of the common or lateral digital extensor tendon is often mistaken for flexural deformity of the metacarpophalangeal joint or carpus because the foal knuckles forward when walking. The disorder may be recognized shortly after birth or generally by 3 to 4 days of life; rarely it may occur in foals up to 3 weeks of age. Diagnosis is based on the characteristic gait of knuckling forward at the fetlock and the presence of a fluctuant swelling over the dorsolateral surface of the carpus (Fig. 61-5). There is usually palpable laxity in the extensor tendons. The limb can be placed in a normal position when the foal is standing. There may be a greater tendency to rupture the extensor tendons in foals with flexural deformities, especially premature contracted foals.

Treatment consists of applying a heavily padded splint over the dorsal or palmar aspect of the lower limb to prevent knuckling forward and traumatizing the dorsum of the pastern and fetlock. Dorsally placed splints are most effective if they do not slip or rotate. Casting may be used but is generally not necessary. Stall confinement is important until the foal develops the ability to use the limb. Foals generally require between 1 week and 2 months of splinting and bandaging before they learn to ambulate properly. The prognosis is invariably good.

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CHAPTER • 62

Cervical Stenotic Myelopathy

Bonnie R. Rush

Cervical stenotic myelopathy (wobbler syndrome) is one of the most common spinal cord diseases in young horses. This developmental disorder is characterized by malformation of the cervical vertebrae, stenosis of the vertebral canal, and spinal cord compression.[1] The age of onset is typically 6 months to 3 years, although mature horses (4 to 20 years) are sometimes identified with cervical stenotic myelopathy. Affected horses often demonstrate rapid growth and are more likely to have developmental orthopedic disease of the appendicular skeleton than peers.[2] Male horses are more frequently affected than females by a ratio of 3:1. Cervical stenotic myelopathy has been reported in most light and Draft horse breeds, although Thoroughbreds appear to be particularly predisposed.

CLINICAL SIGNS

Horses with cervical stenotic myelopathy demonstrate upper motor neuron deficits to all limbs, characterized by symmetrical weakness, ataxia, and spasticity.[3] At rest, affected horses may have a base-wide stance and demonstrate delayed responses to proprioceptive positioning. At a walk, weakness is manifested by stumbling and toe dragging. Horses with prolonged clinical signs of cervical stenotic myelopathy may have hooves or shoes that are chipped, worn, or squared at the toe. Ataxia (proprioceptive loss) appears as truncal sway at a walk and is manifested by circumduction and posting (pivoting on the inside limb) of the hindlimbs during circling. Moderate to severely affected horses have lacerations on the heel bulbs and medial aspects of the forelimbs from overreaching and interference. Spasticity, characterized by a stiff-legged gait and decreased joint flexion, is often observed in moderately affected horses. When prompted to back, horses may stand base-wide, lean backward, and drag the forelimbs. In most instances, the hindlimbs are more severely affected than the forelimbs. Occasionally, forelimb ataxia may be more severe in horses with stenosis of the sixth and seventh cervical vertebrae because of compression of the cervical intumescence. The clinical signs of spinal cord compression often progress for a brief period and then stabilize. Owners often report a traumatic incident with the onset of clinical signs of cervical stenotic myelopathy. The traumatic incident may result from mild neurological deficits with the injury exacerbating the clinical signs of spinal cord compression.

Asymmetrical ataxia and paresis may be observed occasionally in horses with dorsolateral compression of the spinal cord caused by proliferative, degenerative articular processes and peri-articular soft tissue structures.[4] Infrequently, clinical signs of nerve root compression are seen, such as cervical pain, atrophy of the cervical musculature, cutaneous hypalgesia, and hyporeflexia of cervical reflexes adjacent to the site of spinal cord compression. These signs are more commonly observed in horses older than 4 years of age with moderate to severe arthropathy of the fifth to seventh cervical vertebrae and result from peripheral nerve compression by proliferative articular processes as the nerve root exits the vertebral canal through the intervertebral foramen.[5]

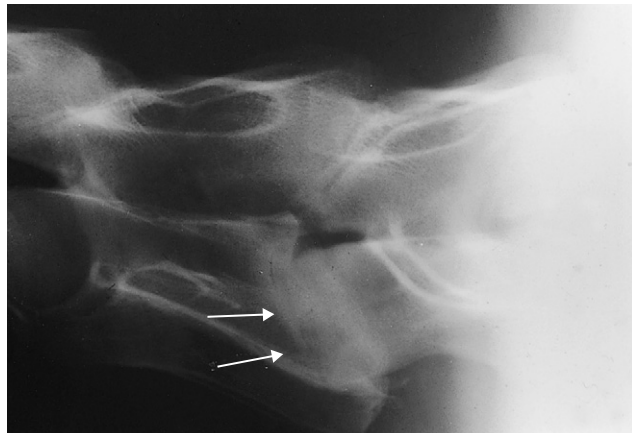

Fig. 62-1 Lateral radiographic view of the caudal neck region of a 4-year-old Paint gelding with cervical pain and spinal ataxia. Cranial is to the left. Note the abnormal intercentral articulation between the sixth and seventh cervical vertebrae *(arrows)*. This represents end-stage diskospondylosis.

In some instances arthropathy of the caudal cervical vertebrae may produce cervical pain and forelimb lameness, caused by peripheral nerve compression, without producing clinical signs of spinal cord compression.[6] Affected horses typically have a short cranial phase of the stride and a low forelimb foot arc and may stand or travel with the head and neck extended (see Chapter 55). Rarely, diskospondylosis of the cervical vertebrae produces a short-strided gait and cervical pain, with or without spinal ataxia (Fig. 62-1). Horses with diskospondylosis or arthropathy of the caudal vertebrae may demonstrate increased rate and depth of respiration with cervical manipulation because of pain.

DIAGNOSIS

Radiographic examination of the cervical vertebrae and cerebrospinal fluid analysis are indicated in horses with symmetrical spinal ataxia. The following neurological disorders should be considered potential differential diagnoses and may produce clinical signs similar to or indistinguishable from cervical stenotic myelopathy: equine protozoal myeloencephalitis (EPM), equine degenerative myeloencephalopathy (EDM), equine herpes virus I myelitis, occipito-atlantoaxial malformation, spinal cord trauma, vertebral fracture, vertebral abscess, and verminous myelitis. Horses with traumatic or infectious cervical vertebral disorders usually demonstrate pain during manipulation of the neck, and the disorders can be differentiated from cervical stenotic myelopathy by standing radiographic examination. Occipito-atlantoaxial malfor-

mation (see Chapter 55) occurs primarily in Arabian horses and is diagnosed definitively by radiographic examination (see Fig. 55-4, *B*). Equine degenerative myeloencephalopathy is a diagnosis of exclusion (unremarkable cerebrospinal fluid [CSF] cytological examination, negative immunoblot analysis for *Sarcocystis neurona*, and negative radiographic and myelographic examination). The veterinarian may suspect EDM during neurological examination (hyporeflexia; forelimbs equally affected to hindlimbs). However, the definitive diagnosis is determined by post-mortem examination. Although most breeds have been reported as having the disease, EDM appears to have a familial predisposition in Standardbred and Appaloosa horses. Horses with equine herpes virus I myelitis may demonstrate urinary incontinence, poor tail tone, and hindlimb weakness. Cytological evaluation of CSF typically reveals xanthochromia and albuminocytological dissociation (high protein, normal cell count) and rising serum antibody titer against equine herpes virus I provides supportive evidence of herpes virus I myelitis. EPM is the most difficult disease to differentiate from cervical stenotic myelopathy. The geographic distribution of EPM is limited to North and South America and to horses exported from these regions. Asymmetrical ataxia, focal sweating, and focal muscle mass loss should direct diagnostic efforts toward EPM. Symmetrical spinal ataxia does not preclude a diagnosis of EPM. EPM-affected horses with symmetrical ataxia are differentiated from those with cervical stenotic myelopathy on the basis of standing radiographic evaluation, CSF immunoblot analysis for *S. neurona*, and myelographic evaluation. It is important to recognize that immunoblot analysis of CSF is frequently positive in horses affected by cervical stenotic myelopathy if they are in a geographic area with a high seroprevalence of EPM. Therefore differentiation of these two conditions should not be determined on the basis of CSF analysis alone. Cytological analysis of CSF is usually unremarkable in horses with cervical stenotic myelopathy, although mild xanthochromia or increases in protein concentration may be observed in horses affected by cervical stenotic myelopathy, with acute spinal cord compression. Myelographic examination is required to confirm a definitive diagnosis of cervical stenotic myelopathy.

Assessment of plain film radiographs of the cervical vertebrae can determine the likelihood of cervical stenotic myelopathy in horses with spinal ataxia.[7] Cervical radiographs should be evaluated for subjective evidence of vertebral malformation and objective determination of vertebral canal diameter. The five characteristic malformations of the cervical vertebrae in horses with cervical stenotic myelopathy are flare of the caudal epiphysis of the vertebral body, abnormal ossification of the articular processes, subluxation between adjacent vertebrae, extension of the dorsal laminae, and osteoarthritis of the articular processes (Fig. 62-2). Osteoarthritis of the caudal cervical vertebrae is the most frequent and severe malformation observed in horses affected by cervical stenotic myelopathy. Nonetheless, degenerative arthropathy occurs in 10% to 50% of non-ataxic horses and is the most frequent and severe vertebral malformation in horses without cervical stenotic myelopathy.[8] Therefore subjective evaluation of osteoarthritis of the articular processes leads to false-positive diagnosis of cervical stenotic myelopathy. Although the presence of characteristic vertebral malformations supports the diagnosis of cervical stenotic myelopathy, subjective evaluation of cervical radiographs does not reliably discriminate between horses affected or unaffected by cervical stenotic myelopathy.[9] Objective assessment of the vertebral canal diameter is more accurate than subjective evaluation of vertebral malformation for identifying horses affected by cervical stenotic myelopathy.

The sagittal ratio technique has been developed for objective determination of vertebral canal diameter in horses.[7] The sensitivity and specificity of the sagittal ratio for identifying

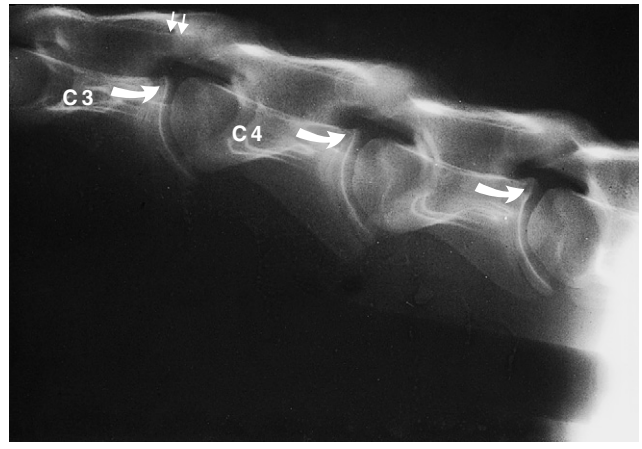

Fig. 62-2 Lateral radiographic view of the mid-neck region of an 8-month-old Quarter Horse colt with spinal ataxia caused by cervical stenotic myelopathy. Bony malformations consistent with cervical stenotic myelopathy include flare of the caudal epiphyses (*curved arrows*), caudal extension of the third cervical vertebra dorsal lamina (*arrows*), and malalignment of the articulation of the third *(C3)* and fourth *(C4)* cervical vertebrae.

horses affected by cervical stenotic myelopathy is approximately 89% for vertebral sites between the fourth and seventh cervical vertebrae.[7] Accuracy of the sagittal ratio technique for identifying horses affected by cervical stenotic myelopathy, without consideration of other bony malformations of the cervical vertebrae, suggests that generalized stenosis of the vertebral canal may be the most important factor in the development of cervical stenotic myelopathy. The sagittal ratio is calculated by dividing the minimum sagittal diameter of the vertebral canal by the width of the vertebral body. The minimum sagittal diameter of the vertebral canal is obtained by determining the narrowest diameter measured from the dorsal aspect of the vertebral body to the ventral border of the dorsal laminae (Fig. 62-3). The vertebral body width is measured perpendicular to the vertebral canal at the widest point of the cranial aspect of the vertebral body. Because the vertebral body is located within the same anatomical plane as the vertebral canal, determination of this proportion negates the effects of magnification resulting from variability in object-film distance. The sagittal ratio should exceed 52% from the fourth to sixth cervical vertebrae and 56% at the seventh cervical vertebrae in horses greater than 320 kg. The clinician should classify the patient into one of the following categories:

1. Low sagittal ratio (<48% for the fourth to sixth cervical vertebrae) and moderate to severe bony malformation: Perform myelographic examination to identify sites of spinal cord compression and to classify lesions as static or dynamic.
2. Marginal sagittal ratio (48% to 56%) and mild to moderate bony malformation: Perform myelographic examination to confirm or exclude cervical stenotic myelopathy.
3. High sagittal ratio (>56%) and minimal bony malformation: Pursue alternative differential diagnoses.

The semi-quantitative scoring system developed by Mayhew et al.[10] should be used in foals younger than 1 year of age to assess cervical radiographs for diagnosis of cervical stenotic myelopathy. The scoring system combines objective measurement of vertebral canal diameter and subjective evaluation of vertebral malformation. Stenosis of the vertebral canal is assessed by determination of the intervertebral and intravertebral minimum sagittal diameter (Fig. 62-3) The interverterbral and intravertebral minimum sagittal diameters

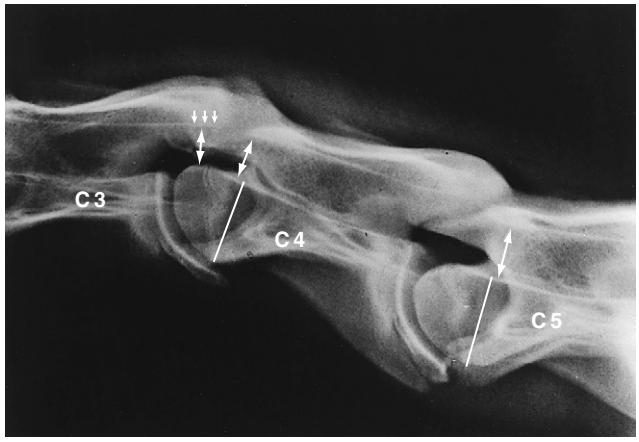

Fig. 62-3 Lateral radiographic view of the third *(C3)*, fourth *(C4)*, and fifth *(C5)* cervical vertebrae of a 2-year-old Thoroughbred colt with cervical stenotic myelopathy. The sagittal ratio is determined by dividing the intravertebral minimum sagittal diameter (*double arrow*) by the width of the vertebral body (*line*). The intervertebral minimum sagittal diameter is measured from the caudoventral aspect of the dorsal lamina of the third cervical vertebra to the craniodorsal aspect of the vertebral body of fourth cervical vertebra (*double arrow below the smaller arrows*). There is caudal extension of the dorsal lamina of the third cervical vertebra (*smaller arrows*), and there is malalignment at the articulation of the third and fourth cervical vertebrae.

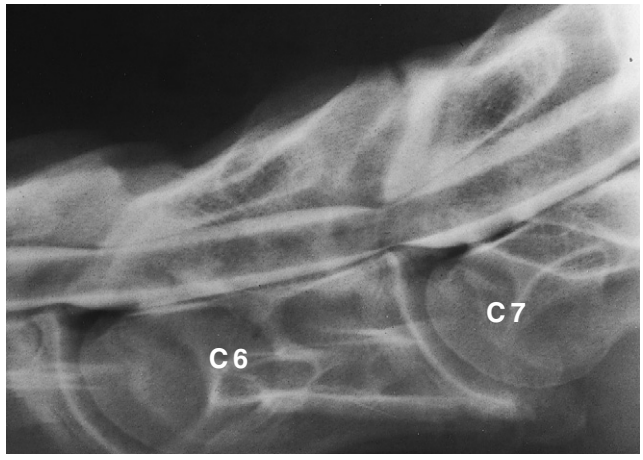

Fig. 62-4 Lateral radiographic view during myelographic examination of the fifth to seventh *(C7)* cervical vertebrae of a 3-year-old Thoroughbred colt with static spinal cord compression. The dorsal and ventral contrast columns are attenuated by more than 50% at the fifth and sixth *(C6)* and sixth and seventh cervical vertebral articulations.

are corrected for radiographic magnification by dividing these values by the length of the vertebral body. The maximum score for cervical vertebral stenosis is 10 points. Cervical vertebral malformation is determined by subjective assessment of five categories: (1) encroachment of the caudal epiphysis of the vertebral body dorsally into the vertebral canal, (2) caudal extension of the dorsal lamina to the cranial physis of the adjacent vertebra, (3) angulation between adjacent vertebral bodies, (4) abnormal ossification of the physis, and (5) osteoarthritis of the articular processes. The maximum score allotted for each category of bony malformation is 5 points. A total score of 12 or higher (maximum total score 35) confirms the radiographic diagnosis of cervical stenotic myelopathy. Stenosis of the vertebral canal and malalignment between adjacent vertebrae are the most discriminating parameters in this semi-quantitative scoring system to differentiate normal foals from those affected by cervical stenotic myelopathy.

Accurate assessment of cervical radiographs requires a precise lateral radiograph of the cervical vertebrae.[11] Obliquity of the cervical vertebrae results in indistinct margins of the ventral aspect of the vertebral canal, producing erroneous values for minimum sagittal diameter and vertebral body width. Obtaining precise lateral radiographs of the cervical vertebrae in recumbent horses is difficult. Plain film radiographs should be obtained in the standing, sedated horse whenever possible.

Although plain film radiography can determine the likelihood of cervical stenotic myelopathy, radiographs of the cervical vertebrae cannot replace myelographic examination for identifying the location and number of affected sites and for classification of spinal cord compressive lesions in horses with cervical stenotic myelopathy. The definitive diagnosis of cervical stenotic myelopathy is defined myelographically by a 50% or greater decrease in the sagittal diameter of the dorsal and ventral contrast columns.[12] The decrease in the contrast column is quantified by comparing it with the mid-vertebral site, cranial or caudal to the compressed site. The ventral column is often obliterated at the intervertebral space in normal studies, particularly in the flexed position. A 50% or greater decrease in opposing dorsal and ventral columns, compared with the adjacent mid-vertebral region, must be present for definitive diagnosis of cervical stenotic myelopathy. I prefer to use less than 2 mm dorsal contrast column as the diagnostic criterion for cervical stenotic myelopathy to reduce the number of false-positive myelographic studies.

In addition to providing the definitive diagnosis of cervical stenotic myelopathy, myelographic examination can differentiate between dynamic and static spinal cord compression.[13] Horses with dynamic spinal cord compression (type I) have obliteration of the dorsal and ventral contrast columns during flexion of the neck, whereas spinal cord compression is not be apparent with the neck in the neutral position. Dynamic spinal cord compression usually occurs in younger horses (<2 years of age) and is associated with instability of the cervical vertebrae, particularly between the third and sixth cervical vertebrae. Dorsal laminar extension, caudal epiphyseal flare, and abnormal ossification patterns often occur in horses with dynamic spinal cord compression. Static vertebral canal stenosis (type II) is characterized by constant spinal cord compression, regardless of neck position (Fig. 62-4). Static compression usually occurs in slightly older horses (2 to 4 years of age) and may occur in considerably older horses. It results from osteoarthritis of the articular processes and proliferation of peri-articular soft tissue structures. A synovial cyst, associated with osteoarthritis of the articular processes, may produce waxing and waning asymmetrical neurological signs. In some horses with static compression, flexion of the neck stretches the ligamentum flavum and relieves spinal cord compression, whereas extension exacerbates compression.

A complete myelographic study should include neutral and stressed (flexed and extended) views of the cervical vertebrae.[12] In horses with obvious sites of spinal cord compression on neutral myelographic views, excessive flexion and extension of the neck should be avoided while obtaining dynamic views to prevent exacerbation of spinal cord injury. Horses should be monitored for 24 hours after the myelographic procedure for depression, fever, seizure, and worsening in neurological status. Worsening of neurological status after myelography may result from spinal cord trauma during hyperflexion, iatrogenic puncture of the spinal cord, or chemical meningitis. Administration of phenylbutazone (4.4 mg/kg PO q24 h) 1 day before through

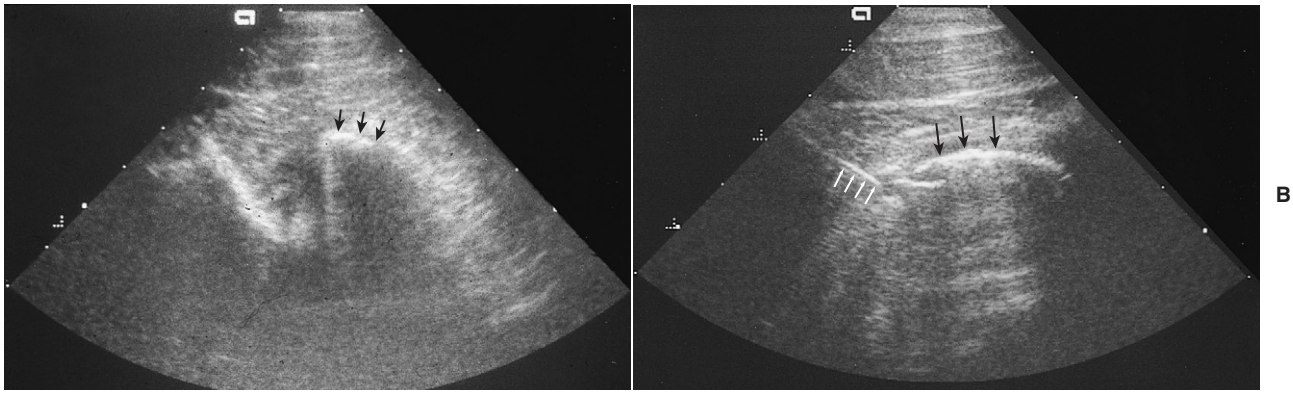

Fig. 62-5 **A,** Ultrasonographic examination of the articulation of the fifth and sixth cervical vertebrae (coronal view). The cranial articular process of the sixth cervical vertebra (*arrows*) is superficial to the caudal articular process of the fifth cervical vertebra. **B,** Ultrasonographic examination during arthocentesis of the articulation of the fifth and sixth cervical vertebrae (transverse view). A 15-cm spinal needle is entering the ventrolateral aspect of the joint (*small arrows*) deep to the cranial articular process of the sixth cervical vertebra (*arrows*).

1 day after myelographic examination attenuates fever and depression associated with chemical meningitis.

CONSERVATIVE MANAGEMENT

Successful conservative management of foals younger than 1 year of age with cervical stenotic myelopathy has been achieved using the paced diet program.[14] The goal of this dietary program is to retard bone growth, enhance bone metabolism, and allow the vertebral canal diameter to enlarge to relieve spinal cord compression. This dietary program is restricted in energy and protein (65% to 75% of National Research Council [NRC] recommendations) but maintains balanced vitamin and mineral intake (minimum 100% NRC recommendations). Vitamins A and E are provided at three times NRC recommendations, and selenium is supplemented to 0.3 ppm. Roughage is provided by pasture or low-quality (6% to 9% crude protein) timothy hay. Dietary regimens are individually formulated according to the age and weight of the foal. Solitary stall confinement is recommended to minimize repetitive spinal cord compression caused by dynamic instability. This program of dietary management and restricted exercise has been successful for preventing neurological signs in foals with radiographic evidence of cervical stenotic myelopathy and treating foals demonstrating clinical signs of cervical stenotic myelopathy.[10]

Anti-inflammatory therapy is indicated in all horses with cervical stenotic myelopathy. Administration of glucocorticoids, dimethylsulfoxide, and non-steroidal anti-inflammatory drugs may reduce edema and provide transient improvement in neurological signs. Spontaneous recovery from cervical stenotic myelopathy without dietary management or surgical intervention is not reported.

Horses with cervical pain and forelimb lameness caused by cervical vertebral arthropathy may benefit from intra-articular administration of corticosteroids or chondroprotective agents. Arthrocentesis of the cervical vertebral articulations (facets) is performed with ultrasound guidance using a 15-cm (6-inch), 18-gauge spinal needle in a standing, sedated horse or a recumbent horse. The cranial facet of the caudal vertebra appears superficial to the caudal facet of the cranial vertebra (Fig. 62-5, *A*). The articular space is accessed at the cranioventral opening of the articular facet, which is angled approximately 60° from the ultrasound beam. The needle should be introduced 5 cm cranial to the facet and inserted at a 30° angle to the skin surface (Fig. 62-5, *B*). Joint penetration should be confirmed by aspiration of synovial fluid. If the neck is extended, the transverse process of the cranial vertebrae may obscure the path to the articulation. Intra-articularly administered triamcinolone (6 mg per joint) or methylprednisolone (100 mg per joint) has produced a reduction in cervical pain in more than 50% of horses with arthrosis of the articular processes.[15] The goal of intra-articular anti-inflammatory therapy should be to improve cervical mobility, reduce cervical pain, and eliminate forelimb lameness. It is unlikely that intra-articular therapy will significantly improve clinical signs of spinal ataxia.

SURGICAL TREATMENT

Surgical intervention is the most widely reported therapeutic approach for horses with cervical stenotic myelopathy. Cervical vertebral interbody fusion was first described in 1979 for horses with dynamic spinal cord compression, and nearly 1000 surgeries have been performed since that time.[16] This procedure fuses adjacent vertebrae in the extended position, which provides immediate relief of spinal cord compression and prevents repetitive spinal cord trauma. Dorsal laminectomy (subtotal Funkquist type-B) is described for horses with static cervical stenotic myelopathy and provides immediate decompression of the spinal cord.[17] Portions of the dorsal lamina, ligamentum flavum, and joint capsule overlying the site of spinal cord compression are removed during dorsal laminectomy. This procedure effectively decompresses the spinal cord. However, it has been associated with significant post-operative complications.[18] Interbody fusion is an alternative to dorsal laminectomy for horses with static compressive lesions. After cervical vertebral fusion, the articular processes remodel and soft tissue structures atrophy, resulting in delayed decompression of the spinal cord over a period of weeks to months.[19] Decompression is immediate with subtotal laminectomy. However, because of its relative safety, interbody fusion is selected by some surgeons as the technique of choice for horses with dynamic and static compressive lesions.

Cervical vertebral fusion improves the neurological status of 44% to 90% of horses with cervical stenotic myelopathy, and 12% to 62% of horses return to athletic function.[18,20] Of the horses that return to athletic function, approximately 60% are

able to perform at the level of intended use, including racing, jumping, and pleasure performance activities. The anticipated magnitude of improvement is one to two neurological grades after cervical fusion. Occasionally, three grades of improvement in neurological status have been achieved. However, it is unusual for a horse with grade IV neurological deficits to become neurologically normal after cervical vertebral fusion. Rarely, a domino effect can occur in horses after vertebral fusion, wherein spinal cord compression develops at the intervertebral site adjacent to the site of fusion. This may result from added forces at the adjacent site or natural progression of the disease.[13] Subtotal laminectomy improves the neurological status of 40% to 75% of horses with static compression.[17]

Fatal post-operative complications have been reported with surgical repair of static compressive lesions of the caudal cervical vertebrae (subtotal laminectomy and interbody fusion). Complications directly related to the surgical procedure include vertebral body fracture, spinal cord edema, and implant failure. Seroma formation is common after cervical vertebral interbody fusion. A pressure bandage is maintained over the surgical site for 2 to 3 weeks to minimize this post-operative complication.[18]

The most important patient factor for determining post-operative prognosis is duration of clinical signs before surgical intervention. Horses with neurological gait deficits present for less than 1 month before surgery are more likely to return to athletic function than are horses with signs of greater than 3 months' duration.[18] The number of spinal cord compressive sites and patient age does not appear to affect the long-term outcome of horses with cervical vertebral interbody fusion. Horses with dynamic compressive lesions appear to have a better post-operative result than those with static compressive lesions. Horses with lesions involving the sixth and seventh cervical vertebrae have a less favorable prognosis than those with lesions affecting the third to fifth cervical vertebrae.

The duration of convalescence and rehabilitation after cervical vertebral interbody fusion is approximately 6 to 12 months. An individualized exercise program dependent on capability, projected use, and neurological status of the horse should be designed to promote muscular strength. Extended exercise at slow speed, including ponying (being led from another horse) and lunging on inclines, is recommended during rehabilitation to build muscular strength. The point at which the horse is competent to return to athletic function after cervical vertebral interbody fusion should be determined by neurological examination. Significant improvement in neurological status occurring after 1 year post-operatively is unlikely.

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CHAPTER • 63

Osteoarthritis

John P. Caron

Joints are highly differentiated structures composed of a number of connective tissues including bone, articular cartilage, and peri-articular soft tissues, all of which contribute to normal joint function and undergo changes in structure and metabolism in disease.[1,2] From the point of view of joint diseases, perhaps the most important of these components is articular or hyaline cartilage, composed principally of a precisely organized arrangement of collagens and proteoglycans. This tissue is responsible for the load-distributing functions of the joint and, in health, cartilaginous surfaces glide over one another in a virtually frictionless manner, even when under substantial load. In joint trauma or osteoarthritis the normal structure and function of articular cartilage are deranged, leading to biochemical, structural, and biomechanical abnormalities in all joint tissues. If the trauma is uncorrected, the result is progressive joint destruction, a process likened to organ failure in other body systems. Joint disease is a particularly prevalent cause of lameness and as such is an expensive equine health problem.[3-5] An understanding of the biology and pathobiology of joints enables the clinician better to diagnose joint disease and to provide appropriate treatment and prevention recommendations.

STRUCTURE AND FUNCTION OF NORMAL JOINTS

Synovium and Synovial Fluid

The synovium is a vascular connective tissue lining the inner surface of the joint and consists of the cells of the synovial intima and a subsynovial stroma, the latter comprised of various amounts of fibrous, areolar, and fatty tissues. The synovium covers all articular surfaces, excluding articular cartilage and localized areas of bone. However, synovium is not uniform throughout the joint, and dense connective tissue may be found in its place in areas predisposed to trauma. Because the synovial lining bears neither true epithelium nor conventional basement membrane separating the joint cavity from the synovial vasculature, no true synovial membrane exists. Rather the intima and subsynovial tissues comprise a structural and functional continuum that acts as a macromolecular sieve.

The synovial intima is lined by a diverse population of synoviocytes, which have been classified according to their ultrastructure and with the use of specific antisera.[6-8] The three cell types are type A cells, of macrophage origin; fibroblast-derived type B cells; and type C cells, which appear to be an intermediate between A and B forms.[7,9-11] The most abundant are the type B cells, which synthesize a variety of important macromolecules, including collagen and hyaluronan.[12,13] The viscosity of synovial fluid is largely due to the concentration and degree of polymerization of hyaluronan, which serves a vital function in soft tissue lubrication. Type A cells, comprising only 10% to 20% of the lining cells, are predominately phagocytic. However, apparently some overlap in function between the two principal cell types exists.[14,15] Importantly, synoviocytes synthesize a variety of soluble mediators implicated in the pathogenetic events of osteoarthritis, including cytokines (e.g., interleukin-1),[16-18] eicosanoids (e.g., prostaglandin E_2),[19,20] and proteinases.[21] That the synovial lining is capable of expressing these substances supports a role for the synovium in the pathogenesis of osteoarthritis.

Deep to the synovial lining the subsynovial region possesses a rich blood supply that is essential to generating synovial fluid, facilitates the exchange of nutrients and metabolic wastes of the synovium, and provides the sole source of nutrition to adult articular cartilage. Because of the specialized structure and functions of the synovial lining and subsynovial stroma, synovial blood flow is subject to a complex regulatory system involving extrinsic control and locally produced factors such as angiotensin II, endothelin-1, and nitric oxide.[2]

Peri-Articular Soft Tissues

The peri-articular soft tissues include muscles, tendons, ligaments, and joint capsule. Muscles effect movement and, via complex reflexes, are vital to providing joint stability and protecting the joint from supraphysiological excursion. Muscle mass is more abundant near joints with a wide range of movement, such as the shoulder and the hip, and less so around joints that move in a single plane. Tendons serve as a bridge between muscle and bone, and ligaments provide stability between bones composing a joint. Tendons and ligaments are of similar though not identical composition, consisting mainly of water, an organized array of collagen bundles (predominantly type I), and a sparse population of fibroblasts. Ligaments contain more elastin fibers and have greater elasticity than tendons.[22] Importantly, the molecular composition of these tissues responds to physical stimuli and immobilization elicits catabolic events leading to tissue weakening.[23] The composition of capsular structures parallels that of ligaments. Indeed some ligaments can be recognized only as hypertrophied portions of the joint capsule. The fundamental role of the capsule is to provide stability; however, its specific nature varies with anatomical location and joint position. For example, the caudal capsule of the human knee is lax in flexion but exerts an important stabilizing force when the joint is in extension.[24]

Subchondral Bone

Although subchondral bone is histologically and biochemically similar to bone in other locations, the organization of the subchondral plate is specific. The plate is thinner than cortical bone found at other locations and its haversian systems are oriented parallel to the joint surface rather than parallel to the long axis of the bone.[25] Similarly the organization of subchondral cancellous bone varies between joints, reflecting predominant biomechanical forces and adaptation to exercise.[26,27] The deformability of the subchondral cortical and epiphyseal trabecular bone exceeds that of the diaphyseal cortical shaft by many times and has the important function of force attenuation. As such, possibly the bone stiffening (sclerosis) observed in osteoarthritis contributes to disease progression.[28,29]

Articular Cartilage

Cartilage is the principal working tissue of the joint and allows simultaneous motion and weight-bearing with negligible friction. Cartilage covers the subchondral plate of bones composing the joint, to which the cartilage is firmly attached. Its thickness varies between joints and at different locations within them. Cartilage is composed of water, collagen, and proteoglycans that are present in respective proportions of 65% to 80%, 10% to 30%, and 5% to 10% of its wet weight. Chondrocytes account for less than 2% of its volume in most species. In adults, cartilage is avascular, alymphatic, and aneural, thus cartilage is nourished mainly via the synovial fluid (see the following discussion). Because articular cartilage is aneural, lesions restricted to cartilage are non-painful, and the innervation of the underlying bone and adjacent peri-articular soft tissues is responsible for providing information on joint position.

Cartilage possesses a number of zones or layers including the following:

- The superficial (tangential or gliding) zone, in which the cells are elongated and oriented parallel to the joint surface
- A middle (transitional) zone, in which the cells are rounded and appear randomly distributed
- A deep (radial) zone, containing cells arranged in columns oriented perpendicular to the surface
- A calcified zone, in which cells are heavily encrusted with hydroxyapatite crystals (Fig. 63-1)

The latter two zones are separated by an irregular line, visible on standard histological preparations, called the tidemark, the specific function of which is unclear.[30] The density of chondrocytes in the matrix varies with depth from the articular surface, as does the macromolecular composition of the matrix surrounding the chondrocytes. These regional differences can be identified histologically and have been designated as the pericellular, territorial, and interterritorial regions.

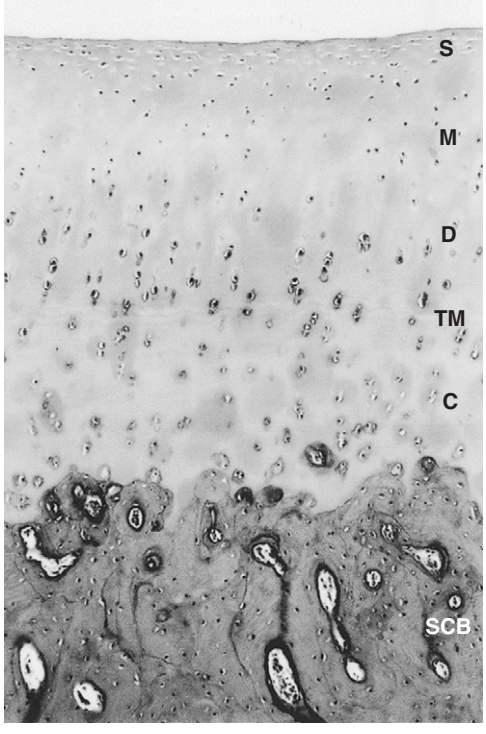

Fig. 63-1 Photomicrograph illustrating regional organization of mammalian cartilage. The four main zones from the articular surface to the subchondral plate include the superficial (tangential) (*S*), the middle (transitional) (*M*), the deep (radial) (*D*), and the calcified (C) zones. The deep and calcified zones are separated by the tidemark (*TM*). Pericellular matrix regions, distinguishable by their histological and ultrastructural differences, and located at progressively greater distances from chondrocyte lacunae, are the pericellular, territorial, and interterritorial regions. (×50.)

The unique functional properties of articular cartilage are reflected in its biochemistry. Articular cartilage is comprised of an abundant, specialized extracellular matrix maintained by the aforementioned sparse population of chondrocytes (Fig. 63-2). Its water content varies with age but may be as high as 80%.[31] This water is freely exchangeable with that in the synovial fluid and is maintained in the matrix in the form of a gel, with matrix collagens and proteoglycans. Water movement is believed to be pivotal to the capacity of cartilage to absorb and distribute compressive load and for its lubrication.

Collagens

The collagens of articular cartilage differ from those found in most other locations in the body. Several collagens, fibrillar and non-fibrillar, are present in this tissue and are thought to provide cartilage with structural support. These proteins also interact with other matrix components to contribute to cartilage architecture and function.[32-34] Collagen fibrils are oriented parallel to the joint surface in the superficial zone and act as a protective layer, whereas larger, radially oriented fibrils in the deeper layers anchor the cartilage to the underlying articular end plate.

Type II collagen is the most abundant in cartilage, accounting for about 90% of the fibrillar network and half of the dry weight of cartilage.[2,35] Type II collagen consists of three identical amino acid chains arranged in a triple helix, is less soluble, possesses a higher proportion of hydroxylysine residues, and is more richly glycosylated than type I collagen.[36,37] Unlike type I, which typically forms fibers, type II collagen is organized in the form of fibrils that are composed of molecules aligned with a 25% overlap or quarter stagger (Fig. 63-3). This structure is stabilized by chemical bonds between specific amino acids in each chain, called hydroxypyridinium cross-links.[38] Fibrils are not uniform in size throughout the matrix; they tend to be larger in the middle and deep zones of the matrix, which reflects regional biomechanical demands.[39] This protein is arranged in arcades, which form the three-dimensional network or skeleton of the cartilage matrix. Type II collagen is produced by the chondrocytes, and whereas significant degradation and resynthesis of fibrils occurs during growth and development, limited turnover occurs in adults.[40,41]

Minor collagens are present in modest amounts in cartilage and the specific roles of these collagens in its structure and function have yet to be defined fully. Type XI is a fibrillar collagen that is found within type II fibrils. Its function is unclear, but likely it plays a role in type II collagen fibril assembly and organization, because a mutation in the type XI gene in mice leads to a disorganized matrix, with abnormally thick collagen fibrils.[42] Type VI is a microfibrillar collagen that may act as a bridge between fibrillar collagen and other matrix components.[43,44] So-called fibril-associated small collagens include collagens IX, XI, and XIV. Type IX collagen molecules bound covalently to the surface of type II fibrils may serve to stabilize the latter.[45] Types XII and XIV collagen also are associated with fibrillar collagen, but the specific functions have yet to be identified.

Proteoglycans

By definition, proteoglycans are composite molecules consisting of protein and glycosaminoglycan (polysaccharide) components. This definition is broad, because some of the aforementioned minor collagens (e.g., type IX) have a single glycosaminoglycan side chain and thus can be designated as proteoglycans. A number of proteoglycans are found in articular cartilage. Aggrecan, the largest and most abundant, has a

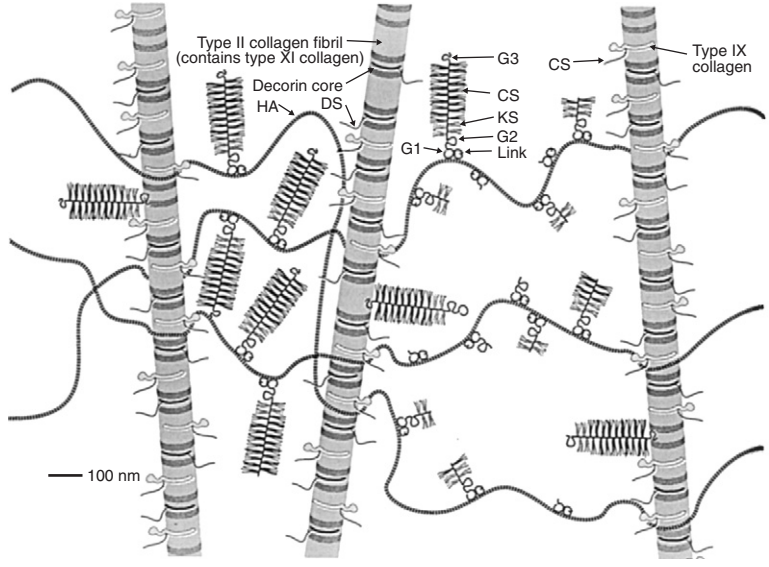

Fig. 63-2 Organization of the major extracellular matrix components in articular cartilage. The principal collagen of cartilage is type II and a network of these fibrils provides much of the tensile strength of the tissue. Aggrecan is composed of a linear protein with three globular domains (*G1* to *G3*) to which are attached numerous glycosaminoglycan chains of chondroitin sulfate (*CS*) and keratan sulfate (*KS*) (see also Fig. 63-4). Supramolecular aggregates are formed by the noncovalent interaction of aggrecan with hyaluronan (*HA*) and stabilized by link protein (*Link*). The negatively charged glycosaminoglycans (*CS* and *KS*) attract several times their weight in water, and this proteoglycan-water composite is responsible for the compressive stiffness of cartilage. Cartilage also has a number of minor proteoglycans and collagens (e.g., decorin and dermatan sulfate [*DS*], the functions of which are not fully characterized). Fragments of aggrecan, remaining bound to hyaluronan, are depicted to illustrate the effects of proteolytic activity in cartilage. (From Koopman WJ, editor: *Arthritis and allied conditions: a textbook of rheumatology,* ed 13, vol 1, Baltimore, 1997, Williams & Wilkins.)

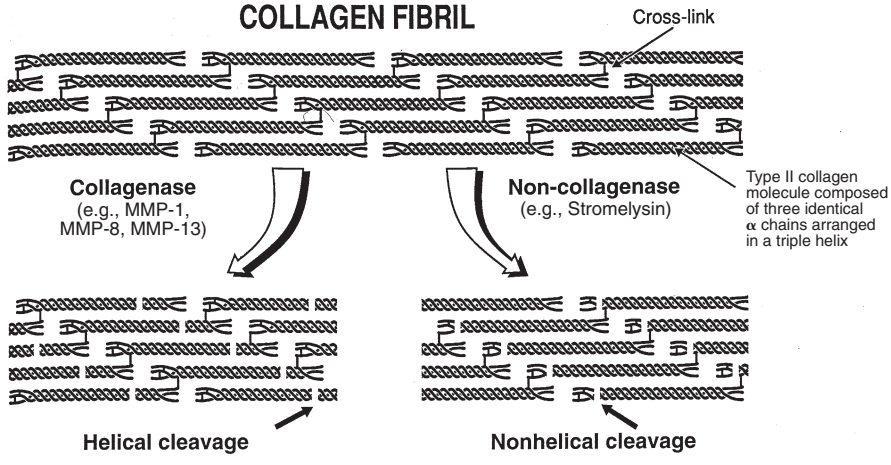

Fig. 63-3 Schematic representation of collagen fibril organization and its proteolytic degradation in cartilage. Cartilage collagen is arranged in fibrils of cross-linked, triple-helical molecules overlapping at regular intervals. Collagenases cleave intact helical collagen and produce characteristic "¾-¼" fragments. Other proteases (e.g., stromelysin) degrade collagen in non-helical regions. (From Koopman WJ, editor: *Arthritis and allied conditions: a textbook of rheumatology,* ed 13, vol 1, Baltimore, 1997, Williams & Wilkins.)

well-defined function in the extracellular matrix; however, the specific roles of the smaller proteoglycans remain to be characterized fully.

Aggrecan is the primary proteoglycan of articular cartilage that interacts with hyaluronan to form aggregates (Figs. 63-2 to 63-4). The individual or monomeric form of this molecule consists of a linear core protein interrupted by three globular domains. The first of these globular domains is designated G_1, exists at the amino-terminal portion of the molecule, and is the site at which the proteoglycan attaches to hyaluronan. As many as 100 aggrecan monomers may be attached to the same hyaluronan chain to form supramolecular aggregates of

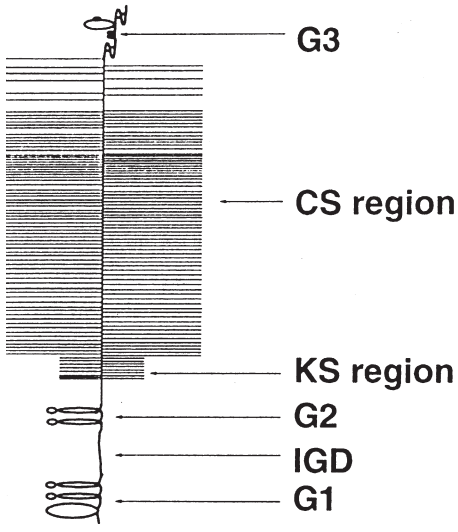

Fig. 63-4 Schematic representation of an aggrecan monomer. Proteolytic cleavage of the molecule in vivo usually first occurs in the G_1 to G_2 interglobular domain (*IGD*). The specific sites involved vary among the enzymes implicated in the process. Keratan sulfate (*KS*) and chondroitin sulfate (*CS*) regions are positioned on the periphery of the moleculte. (From Koopman WJ, editor: *Arthritis and allied conditions: a textbook of rheumatology*, ed 13, vol 1, Baltimore, 1997, Williams & Wilkins.)

micrometer dimensions (see Fig. 63-2).[46] The interaction of aggrecan with hyaluronan is non-covalent but is stabilized by a link protein that binds to the G_1 domain and hyaluronan with equal affinity.[47] Equine link protein recently has been characterized and is similar to that found in human cartilage.[48] The specific functions of the G_2 and G_3 domains are unclear; however, because the G_3 domain is present in only about one third of the aggrecan monomers in adult cartilage, it is unlikely that it plays a pivotal role in the extracellular matrix.[49]

In the region between the second and third globular domains, glycosaminoglycan chains of variable length and composition are attached radially to the protein core (Fig. 63-4). Immediately adjacent to the G_2 domain is a region rich in keratan sulfate and this portion of the proteoglycan, detectable by monoclonal antibodies, has served as a tissue marker of matrix turnover.[50] Farther peripherally on the core protein is the chondroitin sulfate–rich region, where up to 100 chondroitin sulfate chains may be found attached radially to the core protein. These chondroitin sulfate chains vary in length, which is the main reason for heterogeneity in the size of aggrecan. Importantly, these glycosaminoglycan chains contain numerous carboxyl and sulfate groups, so that aggrecan is highly negatively charged and can bind up to 50 times its weight in water.[46,51,52] This highly hydrated matrix gives cartilage its compressive stiffness and ability to dissipate load.

Matrix Proteins

Cartilage, like other connective tissues, contains a number of non-collagenous proteins, many of which are proteoglycans. Among the best characterized of the small proteoglycans are decorin, biglycan, lumican, and fibromodulin, all of which are similar in molecular organization. These proteins have been shown to interact with a number of matrix constituents, including cartilage collagens, and in many cases these interactions involve a number of different collagens and appear to regulate a variety of metabolic processes.[46,52] For example, decorin and fibromodulin inhibit fibrillogenesis of type II collagen, a process that may regulate the size of collagen fibrils in the matrix.[53] Some of these small matrix proteins also may contribute to the anti-adhesive properties of articular cartilage.[54,55]

Cartilage also contains a number of small proteins that are neither collagens nor proteoglycans,[56,57] and most are involved in interactions with a variety of matrix molecules and chondrocytes. For example, anchorin is found on the surface of chondrocytes and within the cell membrane and has a high affinity for type II collagen fibrils. These properties suggest that anchorin may act as a mechanoreceptor, providing chondrocytes with information on changes in stresses experienced by the matrix. Fibronectin is a minor component of cartilage that is thought to contribute to matrix assembly, via interactions with chondrocytes and elements of the extracellular matrix. Fibronectin fragments are present in elevated quantities in osteoarthritis and may contribute to catabolic events in affected cartilage.[58,59] Cartilage oligomeric matrix protein (COMP), also known as thrombospondin-5, is abundant in articular cartilage and is formed by the association of five identical subunits. COMP is most abundant in the proliferative cell layer of growth cartilage where it is thought to regulate cell growth.

Chondrocytes

Studies of cartilage metabolism contradict the seemingly inert histological appearance of this relatively acellular tissue. Despite the fact that chondrocytes represent a small percentage of the volume of cartilage, they are responsible for extracellular matrix synthesis, including all the collagens and proteoglycans. They are also capable of elaborating a variety of proteolytic enzymes effecting degradation of matrix macromolecules. The rate of turnover of the various matrix components is not uniform. At least a portion of the proteoglycan pool is renewed at a relatively rapid rate, whereas the rate of collagen turnover is minimal.[60-62] Chondrocyte metabolism is influenced by intrinsic and extrinsic mechanical influences. For example, cyclic loading and alterations in matrix pressure, as a result of changes in solute content, materially influence proteoglycan synthetic rates.[63,64] Thus the maintenance of the cartilage matrix involves the chondrocyte-mediated processes of synthesis and degradation, and the cartilage loss in osteoarthritis appears to be attributable to a disequilibrium in favor of matrix degradation.

Nutrition

Unlike the cartilage of growing animals, in which articular cartilage receives some blood supply from subchondral vasculature, adult articular cartilage contains no blood vessels. As a result the chondrocyte exists under relatively hypoxic and acidic conditions, with an extracellular pH typically 7.1 to 7.2.[65] Nutrients migrate from subsynovial vessels to the synovial fluid and subsequently penetrate the dense connective tissue matrix of the cartilage, while metabolic wastes are simultaneously cleared in the opposite direction. The density of the matrix appears not to hamper diffusion, because molecules as large as hemoglobin (65 kD) can penetrate normal articular cartilage.[66] The highly charged proteoglycans contained in the matrix do not inhibit the diffusion of small, uncharged molecules.[67] Entry of solutes into the matrix occurs by simple diffusion or may be facilitated by compression-relaxation cycles. Intermittent loading of cartilage is vital to its health, as is evidenced by the deleterious effects of immobilization.[68]

Joint Lubrication

Although several mechanisms for cartilage-on-cartilage lubrication have been hypothesized, two main systems are accepted: a hydrostatic or weeping system that functions at high loads; and a boundary system that functions at low loads.[69] Hydrostatic lubrication of opposing cartilaginous surfaces is effected by a thin film of water liberated from the matrix during cartilage compression. Because little movement of water can occur from cartilage to the subchondral bone, most is squeezed from the opposing cartilages onto the

surface, immediately peripheral to the zone of impending contact.[70] With the release of compressive force, the cartilage expands and water is drawn back into the matrix.

Whereas hydrostatic mechanisms function well under relatively heavy loads, boundary lubrication occurs under low-load conditions. Boundary lubrication is accomplished by specialized materials including lubricin[71] (a glycoprotein of synovial origin) and hyaluronan. These molecules bind to opposing articular cartilage surfaces and prevent the direct contact of these surfaces under low loads. Coefficients of friction were unchanged after hyaluronidase treatment of synovial fluid, suggesting that hyaluronan has no place in cartilage-on-cartilage lubrication.[72,73] However, more recent information indicates that hyaluronan actually does function as a boundary lubricant.[74,75]

Articular soft tissues require lubrication because they contribute most of the frictional resistance to joint movement. Indeed, the energy requirement for the stretching of articular soft tissues is 100 times that of the frictional resistance of opposing cartilage surfaces.[76] The synovium is lubricated by a thin film of synovial fluid, rich in hyaluronan, its principal boundary lubricant.[77]

Intra-Articular Volume and Pressure

Intra-articular volume varies and is influenced by joint position. Specifically, volume and pressure are respectively minimal and maximal near the extremes of flexion and extension.[78-81] This effect is exacerbated in horses with synovial effusion, providing a physiological rationale for diagnostic flexion tests in equine lameness examinations. Moreover, the pointing of an equine limb with a joint effusion likely parallels the observation that in the human knee there is a maximum of intra-articular volume (and minimum of intra-articular pressure and pain) at 30° of flexion.[82,83]

Whereas intra-articular pressure varies during movement, pressure within a normal joint is sub-atmospheric at rest.[84,85] As a result the normal synovial cavity is merely a potential space, the surfaces of which are coated with a thin film of synovial fluid to reduce friction during movement. Although the mechanisms by which this negative pressure occurs remain unclear, the phenomenon contributes measurably to joint stability.[86] Lack of familiarity with the physiological concept of negative intra-articular pressure in normal joints results in the common misconception that the sound of air being aspirated into a joint during arthrocentesis heralds a dry or diseased joint.

Biomechanical Considerations

Articular cartilage remains healthy despite being regularly subjected to considerable normal and shear forces during normal activities. A number of mechanisms exist to facilitate this phenomenon, including the transmission of forces to surrounding tissues by peri-articular soft tissues, the incongruity of cartilage surfaces, and the inherent compliance of cartilage and subchondral bone. Indeed, the capacity for considerable elastic deformation permits normal cartilage to withstand compressive stresses considerably greater than those of body weight alone.[87,88] Nonetheless, cartilage is subject to mechanical breakdown after supraphysiological stresses, and loads exceeding 25 kg/cm^2 are reported to result in matrix damage.[89] Apparently these loads occur in specific areas of cartilage under a variety of clinical circumstances, such as the cartilage degeneration that accompanies the incongruent articular surfaces of a poorly reduced or unstable intra-articular fracture.

At the tissue level the ability of cartilage matrix to resist compression and shear is a function of the interaction of collagen, aggrecan, and tissue fluid. Aggrecan can absorb many times its weight in water, but its complete hydration is restricted by the collagen network. Thus a balance exists between the internal swelling pressure exerted by the association of water with aggrecan (Gibbs-Donnan ionic equilibrium) and the tensile forces of the collagen fibrils. Cartilage under load undergoes a two-phase (viscoelastic) deformation.[90,91] Initially rapid bulk movement of water from the matrix and compression of collagen occur. Subsequently a time-dependent compression occurs, known as the creep phase, in which water flows through the matrix at a slower rate.

These mechanical phenomena have been studied in experiments evaluating the mechanical properties of cartilage after the selective depletion of specific matrix components. The tensile strength of cartilage is a function of its type II collagen content, because strength is reduced in collagen-depleted tissues but is unaffected by proteoglycan removal.[92,93] Proteoglycans (mainly aggrecan) provide the matrix with its compressive stiffness and protect the collagen network from mechanical damage. Trypsin-treated (proteoglycan-depleted) specimens lose the ability to rebound from compressive load and have reduced stiffness.[94]

At rest, opposing articular surfaces are not completely congruent, but when loaded, articular cartilage contact increases, which serves to distribute stress and increase joint stability. This may be a physiological reason why cartilage tends to be somewhat thicker in less congruous joints, such as the hip and stifle.[95] Although cartilage is designed to withstand compressive stress, its ability to act as a shock absorber is finite, largely because it receives its nutrition by diffusion and as a result is of limited thickness. Because its ability to absorb load is limited, cartilage must transmit load to the underlying subchondral bone. As such the articular ends of most bones are flared (less force per unit area) and deform under physiological load to absorb stress.[96] Noteworthy is that the stiffness of the subchondral bone is attributable not only to the cancellous trabeculae, but also to the extracellular fluid content. This was demonstrated in an experiment where subchondral stiffness of canine femoral heads was reduced by 30% after fluid decompression by drilling.[97] When the subchondral bone is unable to accommodate loading, so-called adaptive remodeling failure occurs, in which repetitive subchondral deformation causes trabecular microfractures, which may or may not be accompanied by changes in articular cartilage. Fortunately, when occurring at an acceptable rate, trabecular microfractures undergo a reparative response leading to an orientation of subchondral bone that provides improved strength and shock absorption capacity.[98] Articular surfaces are protected by stress distribution mechanisms beyond those of cartilage and bone. For example, muscles absorb a large proportion of the force experienced during impact loading, leaving the remainder to be cushioned by cartilage and bone. Fine-tuned neuromuscular reflexes are required for this system to work effectively and small failures in these reflex arcs lead to insufficient attenuation of impact loading, which may lead to degenerative changes in cartilage and subchondral bone.[99]

OSTEOARTHRITIS

Etiopathogenesis

Osteoarthritis has been defined as an essentially non-inflammatory disorder of movable joints, characterized by degeneration and loss of articular cartilage and the development of new bone on joint surfaces and margins.[36,100] As in people, equine osteoarthritis is probably not a single disease but reflects a common response of joint tissues to a number of potential causes. Unfortunately the specific contributions and interactions of various mechanical and biological factors contributing to development of osteoarthritis lesions remain unclear.

Three pathogenetic mechanisms are hypothesized for osteoarthritis.[100] The first involves a fundamentally defective carti-

lage, with abnormal biomechanical properties. In this pathway a biomechanically flawed matrix fails under normal loading. In people a recently identified type II collagen defect exemplifies this primary form of osteoarthritis.[101,102] Osteoarthritis attributed to inherently defective cartilage matrix components has not yet been identified in the horse.

A second proposed pathogenetic pathway of osteoarthritis involves physical changes in the subchondral bone.[28,29] Because articular cartilage is too thin to be an effective shock absorber, impact loading must be attenuated by peri-articular soft tissues, muscles, and subchondral bone. Although substantially stiffer than cartilage or joint capsule, cancellous subchondral bone is considered an important shock attenuator. Thus in this hypothesis of osteoarthritis pathogenesis, normal mechanical stresses result in microfractures of the subchondral and epiphyseal trabecular bone. However, when occurring at an excessive frequency, these fractures exceed the rate at which optimal healing and remodeling of the subchondral trabeculae can occur. Bone accretion with healing of these microfractures increases the density of the subchondral plate and adjacent trabeculae, with a concomitant reduction in the ability to absorb repetitive physiological loads. The resulting increase in bone stiffness leads to a state in which the bone-cartilage unit fails to deform normally under load, and the cartilage experiences supraphysiological stresses, resulting in mechanical damage. Subsequent events are those outlined in the following discussion of the third pathogenetic mechanism of osteoarthritis.

To date, a cause-and-effect relationship between subchondral bone plate thickening and cartilage degeneration remains to be established. The hypothesis that subchondral bone and cartilage degeneration are related is supported by the demonstration of microfractures of the subchondral plate and more distant trabeculae in arthritic specimens.[103] Moreover, in mice with osteoarthritis, cartilage degenerates over areas of sclerotic bone but remains intact over areas of normal bone density.[104] However, mathematical models predict that even with considerable increases in subchondral bone stiffness, cartilage stresses are only modestly increased.[105] Collectively these data indicate that subchondral sclerosis contributes to the osteoarthritic process but is probably not a prerequisite to initiate articular cartilage destruction.[106]

The third and most popular hypothesis of the pathogenesis of osteoarthritis is based on the concept of mechanical forces causing damage to healthy cartilage.[36,52,100,107] Matricial or cellular injury by these forces results in metabolic alterations of chondrocytes, leading to the release of proteolytic enzymes that cause cartilage fibrillation and breakdown of the proteoglycan network. Cartilage is remarkably resistant to shear forces but is relatively susceptible to repetitive impact trauma. In people, repetitive trauma is an acknowledged predisposing factor to osteoarthritis in athletes (e.g., metacarpophalangeal joints of boxers) and certain occupations (e.g., shoulder joints of jack-hammer operators). Of many potential causes, repeated microtrauma (use trauma) is probably the most common pathogenetic factor in equine osteoarthritis and the correlation of lesions at defined sites in horses participating in specific sports supports this hypothesis.

Role of the Synovium

Although conventional concepts of osteoarthritis emphasize the direct and predominant involvement of cartilage and bone in osteoarthritis development, it is increasingly recognized that the synovium contributes to the central pathophysiological event of cartilage matrix depletion increasingly. Recent investigations in several species have shown that synoviocytes are a rich source of a variety of inflammatory mediators and degradative enzymes implicated in cartilaginous degeneration, including prostaglandins,[19,20,108-111] cytokines,[112-114] and matrix metalloproteinases.[113-116] These laboratory data are supplemented by the identification of increased levels of these and other inflammatory mediators in the synovial fluid of horses with naturally occurring or experimentally induced synovitis.[117-124] Experiments using synovially conditioned culture media, or co-incubation of synovial tissues with cartilage, support a role of the synovial membrane in cartilage degradation.[125,126] However, determining the specific role of the synovium in the disease process is hampered by the fact that both chondrocytes and synoviocytes are a rich source of the pertinent mediators and enzymes. Thus precise characterization of the relative quantitative and temporal contributions of cartilage and synovium to lesion development has not yet been accomplished.

Role of the Chondrocyte

Of all joint tissues, articular cartilage shows the greatest aberration from normal during disease development, and it is generally considered that metabolic changes in chondrocytes play a primary role in the pathophysiological events of cartilage loss. In normal joints chondrocytes are responsible for maintaining a balance between matrix degradation and repair, and this equilibrium is maintained by a complex interaction between chondrocytes, cytokines, and mechanical stimuli.* In osteoarthritis a disruption of this homeostatic state develops, in which catabolic processes predominate. Though proteoglycan synthesis is greater than normal early in the disease, the rate of matrix digestion is sufficient that the result is a net loss of matrix. With this imbalance toward matrix depletion, cartilage mass is progressively lost and the viscoelastic properties of the remaining tissue become insufficient to withstand normal loads. Subsequently, cartilage fissuring and separation occur (Fig. 63-5). The ultimate result is generalized cartilage loss and secondary remodeling of bone and articular soft tissues (Fig. 63-6).

A number of studies indicate that an important initial biochemical change in osteoarthritis is the loss of aggregating proteoglycans. Up-regulation of chondrocyte proteoglycan synthesis is insufficient to offset enhanced degradation, so that the concentration in the matrix progressively diminishes. In addition to a reduced quantity of proteoglycan, the quality of molecules remaining in the matrix, and newly synthesized replacements, appears to be altered.[128,129] Collagen degradation accompanies proteoglycan loss and is manifested by surface fibrillation (Fig. 63-7). The loss of collagen and changes in collagen fibril size contribute to weakening of the matrix and may account for the increased water content in early cartilage lesions.[130-132]

Whereas the degradation of articular cartilage may occur by the action of a number of mediators, including oxygen-derived free radicals,[133-136] proteolytic enzymes synthesized by chondrocytes are thought to be the major mediators of matrix depletion. Proteinases are classified according to the catalytic mechanism into four main groups, including aspartic proteinases, cysteine proteinases, serine proteinases, and metalloproteinases. Members of each class are synthesized by chondrocytes or synoviocytes and may contribute to cartilage degradation. However, the matrix metalloproteinases and related enzymes apparently are the most active in osteoarthritis.[36,52,137-139]

Matrix-Degrading Enzymes

Matrix metalloproteinases (MMPs) are considered to play a major role in cartilage matrix degradation in osteoarthritis, because this group of proteinases is capable of digesting all major components of the extracellular matrix. The relative contributions of these proteolytic enzymes to the overall process remain to be firmly established; however, a wealth of evidence implicates them in cartilage loss. Specifically, MMPs

*References 1, 2, 36, 52, 57, 64, 68, 127.

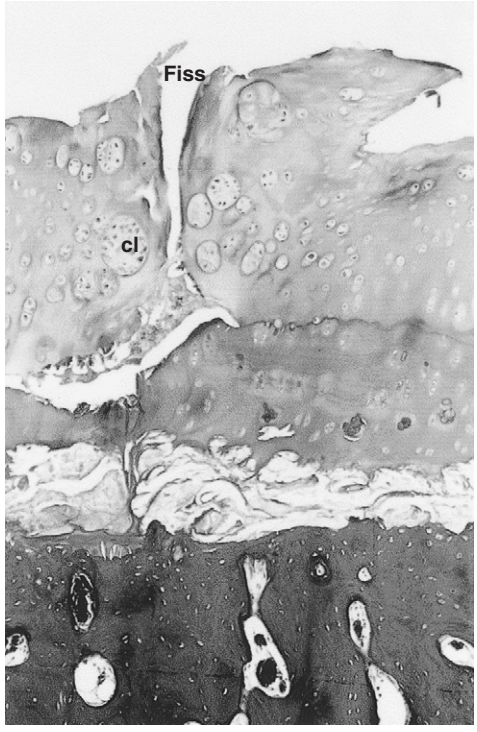

Fig. 63-5 Photomicrograph illustrating the pathological changes associated with cartilage matrix degeneration. Loss of matrix proteoglycans alters the biomechanical properties and ultimately leads to fissures (*Fiss*) in the cartilage, in this case a full-thickness fissure. Chondrocyte clones (*cl*) represent the abortive healing attempts of chondrocytes. (×50.)

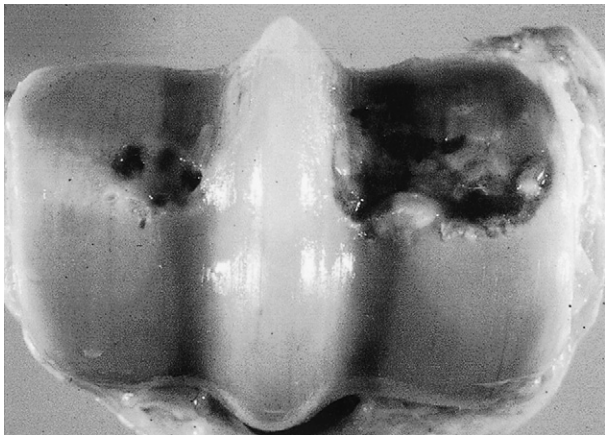

Fig. 63-6 Post-mortem specimen of the distal aspect of an equine third metacarpal bone illustrating partial and full-thickness cartilage loss. Although osteoarthritis affects all articular tissues, degeneration of extracellular matrix of cartilage and its subsequent loss is considered the central event in the disease. Note also the wear lines on the articular cartilage.

are synthesized by synoviocytes and chondrocytes[21,52,140-142] and are present in increased concentrations in diseased cartilage,[143,144] and the topographical distribution and concentration of MMPs in cartilage is correlated with the histological severity of lesions.[144-146] Several types of MMPs are expressed by articular tissues and these are classified as collagenases, stromelysins, gelatinases, membrane-type metalloproteinases, and other MMPs.[142] In addition to other substrates, collagenases degrade intact, helical type II collagen. Stromelysins cleave partially degraded collagen, proteoglycans, and other minor proteins in cartilage. The gelatinases have a diverse range of substrates, including partially degraded type II collagen and types X and XI collagen and elastin. Like collagenases, membrane-type 1-MMP is also capable of digesting fibrillar collagen and a number of other matrix components. MMPs are secreted in an inactive or latent form and require activation through proteolytic cleavage. A variety of enzymes including trypsin, chymotrypsin, plasmin, kallikrein, cathepsin B, and certain MMPs themselves are capable of such cleavage.[52,137,146] The classification and general properties of MMPs are summarized in Table 63-1.

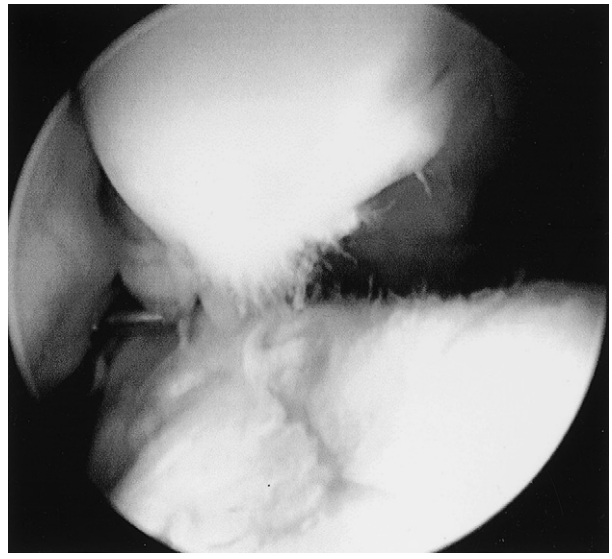

Fig. 63-7 Arthroscopic endophotograph of the middle carpal joint of a Standardbred racehorse with a large osteochondral fragment of the third carpal bone (*top*) and concomitant lesions of the intermediate carpal bone (*bottom*). Cartilage fibrillation is substantial, and its evaluation is greatly facilitated by fluid distention of the joint. Fibrillation indicates damage to the collagenous network of the extracellular matrix.

Recently several members of the ADAM (*a d*isintegrin *a*nd *m*etalloproteinase) family of enzymes have been shown to be expressed by chondrocytes.[147,148] Many of the ADAM enzymes are proteinases and are structurally and functionally related to MMPs. Importantly, certain members of this group cleave aggrecan at a specific site in the interglobular (G_1 to G_2) domain, resulting in aggrecan fragments identical to those found in the tissues and synovial fluids of osteoarthritic animals. These proteinases have been termed *aggrecanase* and share many similarities to typical MMPs, including the inactivation with conventional MMP inhibitors and being inhibited by tissue inhibitor of matrix metalloproteinase-1 (TIMP-1).[149] Recent characterization of aggrecanase has revealed that at least two forms exist and that both are ADAMTS enzymes (ADAM with thrombospondin type 1 motifs).[150,151] The thrombospondin subunits of the protein appear to be critical for the binding and digestion of aggrecan.[152] Aggrecanase is considered pivotal in proteoglycan degradation. Indeed some studies suggest aggrecanase is the principal mediator of proteoglycan depletion in osteoarthritis.[139]

In healthy cartilage the activity of proteolytic enzymes is controlled by a number of mechanisms, one of which is naturally occurring inhibitory proteins. The most important of these inhibitors are the TIMPs.[52,137,142,153] Synthesized by synoviocytes, chondrocytes, and endothelial cells, TIMPs inactivate MMPs by binding to them in a 1:1 non-covalent complex,[154]

Table • 63-1

Matrix Metalloproteinases Implicated in Cartilage Matrix Degradation

PROTEINASE*	MMP†	CARTILAGE SUBSTRATES
Collagenases		
Interstitial collagenase (collagenase 1)‡	MMP-1§	Collagens II and X (not IX and XI), denatured type II, aggrecan, link protein
Neutrophil collagenase‡	MMP-8	Collagen II, aggrecan, link protein
Collagenase 3‡‖	MMP-13§	Collagens II, IV, IX, X; aggrecan; fibronectin
Stromelysins		
Stromelysin 1‡	MMP-3§	Aggrecan, fibronectin; denatured collagen II; collagens IV, IX, X, XI; procollagens; link protein; decorin; elastin; laminin
Stromelysin 2‡	MMP-10	Same as for stromelysin 1
Gelatinases		
Gelatinase A (72 kD)‡	MMP-2§	Denatured collagen II, collagens X and XI, elastin
Gelatinase B (92 kD)‡	MMP-9§	Aggrecan, fibronectin, collagens IX and XI, procollagens, link protein, decorin, elastin
MT-MMPs		
MT1-MMP‡	MMP-14	Aggrecan, collagen II, denatured collagen II, fibronectin, laminin
Others		
Matrilysin (PUMP)‡	MMP-7	Aggrecan
Stromelysin-3	MMP-11	Proteoglycan, denatured collagen II, fibronectin, laminin
Macrophage metalloelastase	MMP-12	Elastin
Novel MMP	MMP-19	Denatured collagen II, collagen IV, aggrecan, fibronectin, laminin

*All except membrane-type 1 MMP are inhibited by some or all of the tissue inhibitors of metalloproteinases 1 to 3.
†*MMP,* matrix metalloproteinase; *MT(1)-MMP,* membrane-type (1) matrix metalloproteinase; *PUMP,* putative metalloproteinase.
‡Expressed by chondrocytes. All are expressed in synovium.
§MMPs characterized in the horse.
‖MMP-13 expression is relatively weak in equine synovium.

Table • 63-2

General Classification of Cytokines and Their Actions on Cartilage Metabolism

CATEGORY OF CYTOKINE	EXAMPLES	ACTIONS
Catabolic (pro-inflammatory) cytokines	IL-1, TNFα*	Promote MMP synthesis Promote nitric oxide and PGE_2 production Inhibit collagen II and aggrecan synthesis
Modulatory (regulatory) cytokines†	IL-4, IL-6, IL-10, IL-13	Stimulate TIMP synthesis Promote IRAP synthesis Inhibit IL-1 synthesis
Anabolic cytokines (growth factors)	IGF-1, TGF-β, bFGF	Promote collagen II synthesis Promote proteoglycan synthesis

**IL,* interleukin; *TNF,* tumor necrosis factor; *MMP,* matrix metalloprotein; *NO,* nitric oxide; *PGE_2,* prostaglandin E_2; *TIMP,* tissue inhibitor of matrix metalloproteinase; *IRAP,* interleukin-1 receptor antagonist protein; *IGF,* insulin-like growth factor; *bFGF,* basic fibroblast growth factor.
†Regulatory cytokines can have mixed actions (e.g., IL-6 amplifies IL-1 effects on MMP synthesis but induces TIMP synthesis).

and these inhibitors are hypothesized to be critical to the longevity of the extracellular matrix of cartilage.[146,155] TIMP exists in at least four forms, the first three of which (TIMP-1, TIMP-2, TIMP-3) are expressed by chondrocytes. Interestingly, each is subject to somewhat different regulatory mechanisms.[156,157] The important role of TIMPs in cartilage matrix health is supported by the observation that imbalances in the ratio of MMP to TIMP synthesis in cartilage is an important determinant of the rate of matrix degradation.[146,158]

Cytokines

Cytokine is a general term to describe a broad array of small regulatory proteins produced by a variety of cells in the body. In joints these mediators exist in a complex balance of activities that regulate the metabolism of the synovial membrane, bone, and articular cartilage in health and disease (Table 63-2).[159,160] Numerous cytokines are involved in articular metabolism and they possess one or more pro-inflammatory (catabolic), anti-inflammatory (regulatory), or anabolic functions. Important in osteoarthritis are the pro-inflammatory cytokines, such as interleukin-1 and tumor necrosis factor-α (TNFα). Chondrocyte receptors for interleukin-1 and TNFα are up-regulated in osteoarthritic cartilage and the activation of these receptors has several deleterious effects on chondrocyte metabolism.[161,162]

A wealth of recent research suggests that interleukin-1 is the most important of the pro-inflammatory cytokines in

osteoarthritis. Early studies using cartilage organ culture provided data supporting a role for interleukin-1 in cartilage matrix degradation,[163] which were supplemented by the identification of elevated levels of this cytokine in synovial fluids of affected patients, including horses.[119,120] Interleukin-1 is involved in the destruction of the extracellular matrix and formation of the functionally inadequate repair tissue in arthritic cartilage. Interleukin-1 decreases the synthesis of proteoglycans and type II collagen and induces the synthesis and secretion of proteolytic enzymes that degrade these matrix macromolecules.[164-174] Decreased synthesis of matrix macromolecules occurs in cartilage exposed to interleukin-1 concentrations substantially less than those required to stimulate matrix degradation.[166] Catabolism is further promoted by inhibition of the synthesis of matrix metalloproteinase inhibitors such as TIMP.[175] In addition, interleukin-1 stimulates the synthesis of prostaglandin E_2 and nitric oxide, the effects of which are outlined in the following discussion.[176-180] Interleukin-1 may also contribute to the proliferative events in the disease. Osteophytosis may be caused, at least in part, by the stimulation of osteoblast-like cells by interleukin-1.[181] Perhaps the most compelling evidence supporting the involvement of interleukin-1 in osteoarthritis is the protective effect of interleukin-1 receptor antagonist protein, which blocks many of the catabolic events typical of interleukin-1 in vitro. This naturally occurring competitive antagonist of interleukin-1 has also been shown to be protective for osteoarthritis-like lesions in arthritis models.[182-184]

TNFα is another pro-inflammatory cytokine that has been implicated in the development of osteoarthritic lesions and is found in elevated concentrations in inflamed and arthritic joints.[185-188] Like interleukin-1, this cytokine stimulates the synthesis of matrix degrading enzymes[172] and inhibits chondrocyte synthesis of proteoglycan and collagen.[173] TNFα appears to be less potent than interleukin-1[189]; however, the effects of interleukin-1 and TNFα are potentiated when combined.[190] TNF appears to stimulate the synthesis of interleukin-1.[191]

The degradative effects of certain cytokines, including interleukin-1 and TNFα, are balanced by inhibitory cytokines (e.g., interleukin-4, interleukin-10, and interleukin-13). Moreover, opposing effects on matrix synthesis are induced by other cytokines, also known as growth factors (e.g., insulin-like growth factor and basic fibroblast growth factor) (see Table 63-2). Using these anti-inflammatory and inhibitory cytokines to control the osteoarthritic process is an active area of research.

Nitric Oxide

Nitric oxide is another mediator of the pathophysiological events in osteoarthritis. This highly reactive, cytotoxic free radical is a by-product of the oxidation of L-arginine to citrulline, catalyzed by a group of enzymes called nitric oxide synthases, which produce large amounts of the mediator when cells expressing this enzyme are activated by mediators such as endotoxins and cytokines.[179,180,192] Early evidence for the involvement of nitric oxide in rheumatic diseases was the observation that nitrite, a stable end product of nitric oxide, was found in elevated concentrations in the synovial fluid and serum of rheumatoid arthritis patients.[193] Subsequently, it has been shown that osteoarthritic cartilage spontaneously produces nitric oxide.[194-196] Nitric oxide may mediate the inhibition of chondrocyte synthetic activities that occur in osteoarthritis. Proteoglycan and type 2 collagen synthesis are inhibited under conditions conducive to nitric oxide formation.[197-199]

Nitric oxide also is hypothesized to mediate in part the augmented expression and activation of MMPs,[200,201] and reduced synthesis of the natural interleukin-1 receptor antagonist protein.[195] However, the specific role of nitric oxide in interleukin-1–induced cartilage matrix depletion is controversial. Early laboratory studies revealed that the MMP activity in interleukin-1 and TNFα-stimulated cartilage cultures was enhanced by adding substrates favoring nitric oxide formation (nitric oxide donors) and this effect was blocked by nitric oxide synthase inhibitors.[200,201] Conversely, cytokine-mediated induction of MMP expression can occur independently of stimulation by nitric oxide,[202] and cytokine-stimulated cartilage explants cultured in the presence of nitric oxide inhibitors had rates of proteoglycan depletion comparable with controls.[203] Nonetheless nitric oxide remains an important area of study, given that in animal models of inflammatory arthritis and osteoarthritis, using compounds that directly or indirectly inhibit nitric oxide synthase activity reduces the severity of lesions.[204-207]

Prostaglandins

Prostaglandins are found in elevated concentrations in inflamed joints,[185,208] and although the specific effects of prostaglandins on joint metabolism are unclear, it is widely held that prostaglandin E_2 contributes to the lesions of osteoarthritis. Prostaglandin E_2 causes synovial inflammation and may contribute to cartilage matrix depletion[209,210] and the erosion of cartilage and bone.[211] Certain data indicate that prostaglandins may actually modulate the release of metalloproteases, such as collagenases and stromelysins.[212,213] Conversely, increasing evidence suggests that cytokine and matrix metalloprotease expression in articular cells is regulated by E-series prostaglandins.[20,214] The net effect of this regulation is unclear, because like corticosteroids, prostaglandin E_2 appears to inhibit TIMP synthesis and MMP synthesis.[215] Moreover, some of the effects of prostanoids may be indirect, acting by promoting the synthesis of other proteins that have unique influences on cartilage metabolism.[216] Thus, although prostanoids are a factor in the signs and certain of the pathophysiological processes of osteoarthritis, the specific role in regulating cartilage depletion and the interactions with other mediators of cartilage lesion development requires elucidation.

Clinical Evaluation of Joint Disease

Joint Pain

Traumatic arthritis and osteoarthritis may be the most common cause of lameness in equine athletes of all types. Unfortunately there is a weak correlation between the magnitude of pain and the severity of articular damage observed.[217-219] The hallmark of osteoarthritis is articular cartilage degeneration, a process occurring in a tissue devoid of sensory innervation. As a result, lameness is typically attributed to involvement of peri-articular soft tissues and bone, the former being relatively richly innervated. In capsular and ligamentous tissues, unmyelinated sensory nerve fibers conduct painful sensations from widely distributed free nerve endings.[220,221] With joint inflammation, these receptors exhibit increased sensitivity. Specifically the threshold for these receptors is reduced by inflammatory mediators such as prostaglandins, and increased receptor activity accompanies physiological joint excursions.[222] Although the severity of soft tissue changes and lameness are related,[223,224] horses with significant peri-articular fibrosis occasionally demonstrate less than the expected degree of lameness. Studies of joint capsule innervation in arthritic specimens have revealed that with time degeneration of neurons is common, which provides a potential reason for the less than expected magnitude of pain in some patients having clearly demonstrable changes in peri-articular soft tissues.

Bone and periosteum also contribute to osteoarthritis pain. The periosteum is well innervated, and the periosteal disruption that accompanies the development of peri-articular osteophytes is a source of joint pain.[225] The subchondral plate and epiphyseal trabecular bone make variable contributions to clinical signs. For example, many, but not all, horses with subchondral cystic lesions demonstrate lameness.[226-228] Inconsistent lameness among horses with similar radiographic signs parallels the weak correlation between pain and radiographic findings of

early osteoarthritis in people.[229,230] Osseous receptor stimulation often accompanies joint movements that cause elevations in intramedullary pressure. People with osteoarthritis of the hip have elevated intra-osseous pressure,[231] which in some people responds favorably to cortical fenestration. Elevation in intramedullary pressure occurs with significant flexion or extension of equine joints and is a likely source of articular pain in some horses. For example, both simulated effusion and metatarsophalangeal joint flexion increase intramedullary pressure in the third metatarsal bone.[232] The concept also is supported by the clinical observation of a favorable response to transcortical decompression in horses with lameness related to osseous cyst-like lesions.

Local Signs

Limited range of motion is a common feature of equine joint disease and is probably caused by a combination of factors, including guarding from pain, synovial effusion and edema, and progressive peri-articular fibrosis. Synovial edema and proliferation and pain are probably the main causes of reduced range of motion in horses with early osteoarthritis, whereas fibrosis is important in chronically affected horses. The specific mechanisms causing peri-articular fibrosis are unclear. However, cytokines and neuropeptides are likely to contribute to fibrosis, given the mitogenic effects on fibroblasts of these substances.[233-235]

Effusion is a common feature of joint disease and is manifested in distal joints as visible or palpable distention of joint pouches. Leakage of protein into the synovial space, because of increased permeability in the capillary endothelium and intercellular spaces of the synovium, which is not matched by compensatory increases in lymphatic clearance, leads to a progressively increased colloid osmotic pressure and augmented synovial fluid volume. Although mild effusion enhances nutrient exchange in the joint,[236] significant effusion results in progressively elevated intra-articular pressure that ultimately destabilizes the joint and causes pain, stiffness, and a reduced range of motion. Increased permeability of the synovium to cells and proteins varies with the degree of synovial inflammation and is reflected in cytological findings in synovial fluid samples.

Synovial Fluid Changes

Reduced viscosity of synovial fluid is a frequent finding in horses with joint disease, particularly in horses with active synovitis. Reduced viscosity has been attributed to a reduced concentration, or depolymerization, of synovial fluid hyaluronan. Substantial reductions in hyaluronan concentration have been documented in the synovia of horses with chronic traumatic arthritis[237]; however, considerable variability exists. In a study comparing the hyaluronan concentration in normal horses with those having lameness that could be eliminated by intra-articular analgesia, normal horses had a mean hyaluronan concentration approximately 50% higher than that of horses with synovitis. However, the variability between horses was sufficient that this difference was not statistically significant.[238] Clinical determinations of hyaluronan concentration are not routine because of this variability and the technically involved procedures required for the quantitative determination of hyaluronan. The mucin clot test is a relatively simple, semi-quantitative test of hyaluronan quantity and quality, but it is not particularly sensitive. Therefore the quality of hyaluronan is often determined clinically by assessing viscosity on gross inspection of synovial fluid obtained during arthrocentesis.

Because increases in cell numbers and protein concentration in osteoarthritis are not dramatic, cytological evaluation of synovial fluid is not used routinely diagnostically. Cytological analysis of synovial fluid is most useful in identifying and monitoring infection and untoward post-injection reactions. Approximate values for cell count and total protein concentrations under a variety of clinical situations are given in Table 63-3. Total protein concentration varies between joints, tending to be considerably higher in the larger, more proximal joints, such as the scapulohumeral joint.

Role of Radiography

Radiography has long been the traditional means of assessing the structural changes of osteoarthritis (see Chapter 15). Radiography has the advantages of availability, convenience, relative safety, and economy. Indeed, it is standard practice for many veterinarians to localize lameness to a particular area of the limb and subsequently to obtain radiographs in an attempt to identify changes to support a diagnosis of osteoarthritis. Unfortunately, although radiography has some merit in characterizing changes in bone that accompany established osteoarthritis, the technique lacks sensitivity and is of dubious value in identifying patients with incipient or focal lesions. It is well known that radiographically undetectable performance-limiting lesions occur in horses.[247,248]

Radiographic findings tend to witness past events in the pathological process and do not consistently reflect ongoing processes. Additionally, in horses, as has been long accepted in people, a lack of correlation exists between lameness or reduced performance and specific osseous structural changes evident on radiographs.[249-253] A lack of correlation with arthroscopically evident degeneration and radiographic findings is also common.[254-257] In addition to this underlying fundamental biological dichotomy, precise quantification of radiographic findings is hampered by difficulties in precisely duplicating conditions from one radiographic examination to the next. Positioning, degree of weight bearing, and radiographic technique contribute measurably to results. Nonetheless, largely because of its aforementioned advantages, radiography remains a principal method of evaluating horses with joint disease.

The radiological features of osteoarthritis mirror the pathological changes occurring in the affected joint (Fig. 63-8). Initially, joint space narrowing, subchondral sclerosis, and osteophytosis occur. With time subchondral lysis, osteochondral fragmentation, and eventually ankylosis may develop (Box 63-1).

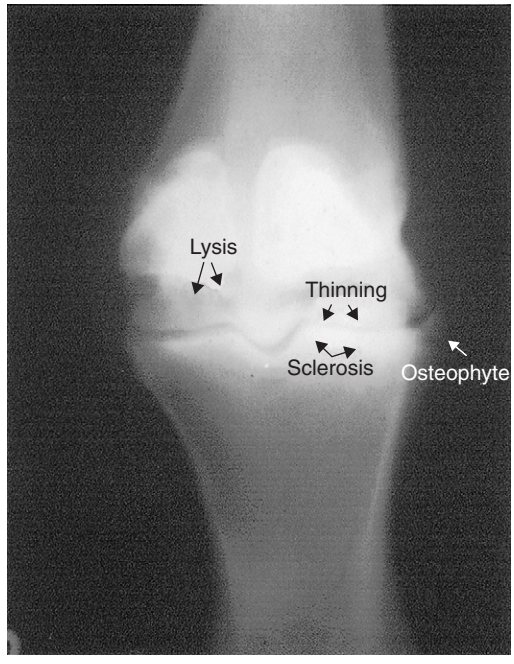

Fig. 63-8 Dorsopalmar radiographic projection of the metacarpophalangeal joint of a horse demonstrating radiographic changes of advanced osteoarthritis. Peri-articular osteophytosis, joint-space thinning, and subchondral sclerosis and subchondral lysis are indicated.

Table • **63-3**

Synovial Fluid Cytology for Various Clinical Conditions and Diagnostic or Therapeutic Manipulations[239-246]*

PARAMETER	NORMAL	MILD SYNOVITIS (e.g., OCD)	OSTEOARTHRITIS	INFECTIOUS ARTHRITIS†	ARTHROCENTESIS‡	BALANCED ELECTROLYTE SOLUTION‡	LOCAL ANESTHETICS‡§	GENTAMICIN‡	DMSO‡ (10% SOLUTION)
Total leucocytes (per µl)	50-500	20-250	≤1 × 10^3	20-200 × 10^3	1-4 × 10^3	6-45 × 10^3 (typically 20 × 10^3)	2-10 × 10^3		8-40 × 10^3
Neutrophils (%)	<10	<10	<15	>90 (variable toxic changes)	50	80	60	50	>50
Mononuclear cells (%)	>90	>90	>85	<10	50	20	40	50	<50
Total protein (g/dl)	0.8-2.5	0.8-3.0	0.8-3.5	4.0-8.0+	1.5-2.5	3.0-4.0	2.5-4.0	4.5-6.0	2.5-4.0

* Listed ranges are approximate. Considerable variability exists in published reports.
† Significant elevations in leukocyte counts and total protein concentration occur within the first 12 hours in experimentally inoculated joints. Values shown represent those observed at 24 hours.
‡ Leukocyte counts and total protein concentrations correspond to the maximum values that typically occur within the first 24 hours.
§ Synovial response to lidocaine and mepivacaine are comparable.

Box • **63-1**

Radiographic Features of Osteoarthritis

Radiographic feature	*Pathogenetic mechanism**
Peri-articular osteophytosis	Endochondral ossification occurring at bony margins of unknown cause. Possible repair attempt modulated by altered cytokine milieu.
(Asymmetrical) Joint space thinning	Cartilage degeneration and loss.† Usually at areas of weight bearing or high stress. May be absent when focal cartilage loss occurs.
Subchondral sclerosis	Deposition of new bone as a response to changes in force transmission and from healing of trabecular microfractures. Corresponds to areas of maximum stress. Significant sclerosis often corresponds to full-thickness cartilage loss.
Subchondral lysis	Less common change of uncertain pathogenesis. Possibly pressure necrosis from synovial fluid gaining access to subchondral plate via fissures, or related to pressure necrosis from trauma to bone.
Osteochondral bodies	Disintegration of joint surfaces or fractured osteophytes. May represent inciting lesions (e.g., osteochondral fracture).
Advanced remodeling/ankylosis	Articular response to advanced degeneration. Environment more consistent with fracture than synovial joint.

*Specific pathophysiological mechanisms and reasons for disproportionate representation of changes among and between joints remain unclear.
†Seldom used as a marker of disease progression because of problems with technical aspects of radiographic positioning and focal-film distance.

In horses substantial differences appear between specific joints respecting the relative degree of these changes. For example, radiographically evident changes tend to be less dramatic and appear later in the disease process in the metacarpophalangeal and metatarsophalangeal joints than in many other articulations.

Other Imaging Modalities

Increasingly available are other imaging modalities for assessing joint diseases. Among them are nuclear scintigraphy, magnetic resonance imaging (MRI), and ultrasonography.

Nuclear scintigraphy. Although more commonly used to detect non-displaced, incomplete fractures and to identify stress-related bone damage in performance horses, nuclear scintigraphy can provide useful information in selected horses with joint disease (see Chapter 19). Whereas radiography (and related techniques, including computed tomography) provide considerable anatomical detail of osseous changes in affected joints, nuclear scintigraphy yields current physiological information on bone metabolism. The main disadvantages of scintigraphy are its relatively poor resolution (isotopes produce much fewer gamma rays than the number of x-rays generated by a cathode tube) and a lack of specificity, because bone responds to most insults by increasing turnover. Owing to the unavailability of a cartilage specific agent, the most common approach to the scintigraphic study of osteoarthritis is using the delayed bone scan images, obtained 3 to 4 hours after injection of the radiopharmaceutical. Currently used agents are the biphosphonates, which bind to the microcrystals of hydroxyapatite in bone. Areas of increased bone modeling have enhanced localization of the radiopharmaceutical, as long as an adequate blood supply exists.

It has long been known that bone-seeking isotopes accumulate rapidly in the bone of osteoarthritis joints.[258] The most intense radiopharmaceutical uptake typically occurs in the subchondral bone and at the osteochondral junctions of osteophytes, although temporal variation in the anatomical distribution of uptake may occur, as has been illustrated in an animal osteoarthritis model.[259] Evidence exists that scintigraphy is useful in predicting the progression of osteoarthritis in the human knee,[260] and scintigraphy apparently may prove useful in diagnosing preclinical joint disease, that is, before the appearance of radiographic abnormalities.[261] Conversely, bone-phase scans may be normal in chronic osteoarthritis joints if the rate of bone turnover returns to normal.

In the equine athlete, scintigraphy has proved useful to document a number of sport-specific lesions among different types of equine athletes.[251,262,263] Unlike the human digit, the size of the bones in the equine skeleton is sufficient that relatively precise anatomical localization of lesions is often possible. Moreover, nuclear scintigraphy has the advantage of allowing a survey of all joints in a single examination. Importantly, as for radiography, close correlation between lameness and scintigraphic findings does not always exist.[251,264]

Magnetic resonance imaging. This imaging method involves detecting alternating electrical current produced when hydrogen protons, predominately found in fat and water, are subjected to pulsed electromagnetic fields applied in a specific manner (see Chapter 21). The rate at which protons change orientation varies among soft tissues and fluids of different composition, which allows the considerable tissue discrimination possible with MRI.

Although to date MRI has played a minor role in assessing joint disease in animals and people, MRI has a number of compelling advantages over many of the existing modalities. Specifically, MRI has the capacity for high-resolution, three-dimensional (tomographic) images of all joint components, obtained in a non-invasive manner. Moreover, advances in equipment and improved imaging sequences provide much greater contrast between articular cartilage and surrounding soft tissues than was previously possible. Indeed, variations in the orientation of collagen in the cartilage matrix provide for images with four laminae corresponding to the histological zones of the tissue. Changes at the tissue level that accompany osteoarthritis include increased water content and matrix swelling, which can be detected by this imaging method.[265] Using specialized techniques to optimize resolution and reduce interference from adjacent fat and fluid, evaluating moderately sized articular cartilage defects with considerable sensitivity and specificity is possible.[266,267] Beyond cartilage

changes, MRI also can be used to characterize the amount and nature of synovial fluid and is a sensitive means of delineating osteophytes and subchondral bone changes.[265] Importantly the identification of subtle lesions requires relatively sophisticated equipment and considerable experience for proper image acquisition and interpretation.

To date, MRI in the horse has been largely limited to anatomical and correlative studies of cadaver specimens.[268-271] In one of the few published reports of using the technique to investigate osteoarthritis in the horse, postmortem magnetic resonance images of the metacarpophalangeal joint correlated well with arthroscopic and necropsy findings.[272] Widespread use of MRI in horses is limited by the expense and the tunnel configuration of currently available equipment. Some time may pass before equipment and software will be available that allows routine use of the technology in equine athletes. Developments in low-field magnets adaptable to the standing horse represent a potential means by which magnetic resonance technology soon may be more readily applicable to horses.[273]

Ultrasonography. Ultrasonographic evaluation of diseased joints represents an additional diagnostic tool that has enjoyed increased use lately (see Chapter 17). Ultrasonography initially was used to assess chronic proliferative synovitis in the metacarpophalangeal joint.[274,275] More recently its use has expanded to include most appendicular (and some axial) skeletal joints.

The principal benefit of ultrasonographic examination over conventional radiography is its superiority in demonstrating soft tissue abnormalities. With experience and an appropriate knowledge of the regional anatomy and acoustic principles, it is possible to identify and localize accumulations of synovial or other fluids, thickened synovial and capsular tissues, damaged intra-articular and peri-articular ligaments, peri-articular osteophytes and entheseophytes, osteochondral fragments, and irregularities in cartilage and subchondral bone. Inherent limitations of ultrasonography preclude its use in areas where the volume of tissue overlying the joint of interest is too voluminous and for structures having a shape or orientation that is not conducive to ultrasonographic evaluation. The principles and techniques of ultrasonographic examination of equine joints were recently the subject of an excellent review.[276]

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CHAPTER • 64

Markers of Osteoarthritis: Implications for Early Diagnosis and Monitoring of Pathological Course and Effects of Therapy

David Frisbie

Although joint disease can be diagnosed using routine clinical methods, more accurate and earlier diagnosis may lead to identification of osteoarthritis before irreversible changes occur within joint tissues. Measuring levels of molecular products of tissue turnover, that is, biomarkers, from healthy and diseased cartilage and bone has the potential to achieve early diagnosis and allow a better understanding of osteoarthritis pathophysiology. The potential also exists to monitor disease, especially in response to novel therapeutic agents. Work with biomarkers of articular cartilage and bone in human and equine osteoarthritis patients has yielded promising results. This chapter discusses some of the markers that currently are being evaluated in synovial fluid and serum samples, with a focus on those of potential benefit to the equine industry.

STRUCTURE AND METABOLISM OF ARTICULAR CARTILAGE AND BONE IN HEALTH AND DISEASE

Articular cartilage is a complex tissue with an extensive extracellular matrix. The two main components that define the cartilage matrix are type II collagen and aggrecan (see Chapter 63). A balance of synthesis and degradation orchestrated by the chondrocytes maintains normal populations of these molecules within the cartilage matrix. Osteocytes, osteoblasts, and osteoclasts maintain the structural and functional integrity of bone matrix by regulating synthetic and degradative pathways. Synoviocytes also influence homeostasis in cartilage and bone. Osteoarthritis often is characterized by degradative changes within articular cartilage, bone, and synovium. Direct and indirect factors assault the matrix molecules of these tissues, resulting in degeneration and loss of some macromolecules.

What are direct and indirect molecular markers? Fibrillar collagens, such as types I and II, are synthesized as immature procollagens that undergo proteolytic changes before conversion to mature collagen fibrils. Peptides at either end of the procollagen molecule are cleaved before the procollagen is incorporated into a mature collagen fibril. Estimations of type II collagen synthesis have been obtained from synovial fluid and serum samples by using a specific antibody that recognizes the propeptides cleaved from the carboxy termini.[1] This direct molecular marker specifically identified a known molecular process within a given tissue. Conversely, an indirect molecular marker reflects more general change that is not clearly definable and may represent contributions from several events and tissues. Indirect markers are cytokines, growth factors, and matrix metalloproteinases (MMPs). Osteoarthritis involves changes in subchondral bone and synovium, so assessment of molecular markers from these tissues is relevant.

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INDIVIDUAL SKELETAL BIOMARKERS OF ARTICULAR CARTILAGE METABOLISM IN OSTEOARTHRITIS

Anabolic Processes

The carboxy propeptide of type II collagen is a useful measure of the anabolic process of type II collagen synthesis. Levels of carboxy propeptide of type II collagen were significantly higher in synovial fluid from people with osteoarthritis compared with those without osteoarthritis. Levels peaked early in the radiographic progression of the disease and declined in patients with severe radiographic changes.[2,3] This biomarker also has been shown to change significantly in serum samples from people with osteoarthritis and rheumatoid arthritis.[4,5]

Chondroitin sulfate (CS) is a major glycosaminoglycan (GAG) of aggrecan, and measuring specific CS epitopes on newly synthesized proteoglycan (PG) molecules is a useful biomarker for aggrecan synthesis. An epitope called CS-846 that normally is found in fetal, but is almost absent in healthy adult articular cartilage has been measured in many species. Levels of CS-846 epitope are increased in synovial fluid in people after injury or primary osteoarthritis compared with levels in synovial fluid from normal joints. Serum levels also are elevated in joint disease but to a lesser extent than synovial fluid levels.[6,7] Other CS epitopes such as 3B3 and 7D4 were shown to be useful in assessing cartilage injury in animal models and in people with clinical disease.[8] Using arthroscopic evaluation, a negative correlation was found between synovial fluid 3B3 concentrations and gross articular damage that was thought to be caused by decreased normal cartilage volume, or inhibition of synthesis, with increasingly severe lesions. Conversely, in people, increased levels of synovial 7D4 epitope were found in diseased knees compared with contralateral normal knees.[9]

Catabolic Processes

Measuring the degradation of type II collagen is of potential benefit in monitoring osteoarthritis. Antibodies have been developed to identify exposed but previously inaccessible cleaved or denatured type II collagen fragments. Significant elevations in levels of degraded type II collagen were demonstrated in synovial fluid and serum samples from horses, dogs, and rabbits with experimental osteoarthritis.[10,11] Significant increases were detected in the serum of people with osteoarthritis, with a correlation to disease activity.[10]

Keratan sulfate (KS), one of the GAGs found on proteoglycan molecules of aggrecan, has been evaluated extensively. In people, elevations in serum levels of KS were associated with osteoarthritis in some, but not all, studies.[7,12,13] Lack of correlation of serum[14] and synovial fluid[8] KS levels with cartilage damage leads me to question the value of serum KS as a biomarker of joint disease in people. In dogs a specific KS epitope (5D4) was of limited value in experimentally induced and naturally occurring cruciate injury.[15,16] The usefulness of KS in serum and synovial fluid of horses with osteochondral fragmentation is also questionable.[17]

In an initial equine study of molecular markers, carboxy propeptide of type II collagen and the GAG epitopes CS-846 and KS were measured in synovial fluid and serum of horses with and without carpal osteochondral fragments.[17] Synovial fluid and serum CS-846 epitope concentrations were significantly higher in joints with osteochondral fragments compared with normal joints and showed good correlation with grades of cartilage damage. Serum concentrations of carboxy propeptide of type II collagen were elevated in horses with osteochondral fragments, and good correlation between carboxy propeptide of type II collagen concentration and arthroscopic lesion grade was found. A single blood sample assayed for CS-846 and carboxy propeptide of type II collagen levels resulted in 79% accuracy for prediction of an osteochondral fragment.

CS-846, KS, and carboxy propeptide of type II collagen concentrations were measured in synovial fluid from normal joints and those with osteochondrosis.[18] Significantly higher levels of carboxy propeptide of type II collagen and lower amounts of CS-846 and KS epitopes were found in affected joints compared with normal joints.

Cartilage oligomeric protein (COMP) is an abundant non-collagenous protein constituent of cartilage. COMP was once thought to be cartilage-specific, but recently it also has been localized in tendons and synovium. Serum and synovial fluid concentrations of COMP are increased in people with osteoarthritis.[19,20] A positive correlation exists between COMP levels and radiographic grading of osteoarthritis, progression of radiograhpic changes,[21] and results of nuclear scintigraphy.[22] Gene expression of COMP in synoviocytes is up-regulated in osteoarthritis, suggesting that this marker may be useful to indicate synovitis.

Initial work in the horse demonstrated that serum and synovial fluid levels of COMP were significantly lower in diseased joints.[23] Horses with osteoarthritis lesions of the third carpal bone had lower COMP levels in synovial fluid compared with normal joints.[24]

INDIVIDUAL SKELETAL BIOMARKERS OF BONE METABOLISM IN JOINT DISEASE

Anabolic and catabolic cascades exist in bone, but specific markers in normal and disease states are not clearly defined. This section deals only with bone markers thought to be significant in joint disease.

Anabolic Processes

Osteocalcin (OC) is a small non-collagenous protein associated with bone assembly and turnover and has been measured in serum and synovial fluid samples from people with osteoarthritis. Levels of OC correlated with bone scan findings and markers of cartilage metabolism.[22,25] However, because OC levels are higher in serum than synovial fluid, OC in synovial fluid may be derived from peripheral blood and may not reflect local joint disease.[25]

OC levels were measured in horses, and as in people, they appear to vary with age and with the administration of corticosteroids,[25,26] but the effect of gender remains unclear.[26] General anesthesia affects serum OC levels for 4 days.[27]

Bone-specific alkaline phosphatase is an isoform of alkaline phosphatase that is expressed at high levels on the cell surface of the bone-forming osteoblasts and plays an important role in bone formation. In a recent equine study a correlation was found between synovial fluid levels of bone-specific alkaline phosphatase, KS-5D4 epitope, and total GAG, and between all three biomarkers, and the amount of joint damage defined arthroscopically.[28] This supports a putative role for altered subchondral bone metabolism in equine osteoarthritis.

Catabolic Processes

Type I collagen C-telopeptides (CTX) may be useful markers of bone resorption. CTX levels in people with rheumatoid arthritis were positively correlated with indices of disease activity and joint destruction.[29,30] The marker was influenced by the administration of corticosteroids. CTX is present in equine serum, although its usefulness as a marker of pathological processes is unknown.[31]

Human bone sialoprotein is found only in adult bone, and levels are seven times higher at the interface of cartilage and bone compared with other locations in bone.[32] Serum levels are elevated significantly in people with clinical osteoarthritis

and those with bone scans consistent with osteoarthritis.[32,33] Detection of equine bone sialoprotein has not been reported, although development of an assay is currently under way. The hope is that this will be useful in identifying subchondral bone damage in horses with osteoarthritis.

FUTURE OF BIOMARKERS IN OSTEOARTHRITIS

Limited data are available regarding using biomarkers to diagnose and monitor equine joint disease. Factors influencing levels of biomarkers include liver and kidney clearance, circadian rhythms, intestinal peristalsis, exercise level, age, breed, diet, sex, drug administration, surgery, and general anesthesia. Methods of sample collection and storage also may be influential.

Although biomarkers may have a role in diagnosis and monitoring equine joint disease, a combination of markers likely will be required, especially because so many factors influence activity. Preliminary results from our ongoing study indicate that serum levels of OC, GAG, and a type I/II collagen degradation product, a biomarker of collagen cleavage, are significantly elevated in horses with an experimentally induced osteochondral fragment in the middle carpal joint. A prospective study is currently under way to investigate whether serum levels of bone and cartilage markers can predict important musculoskeletal injuries in Thoroughbreds in training.

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CHAPTER • 65

Models of Equine Joint Disease

Chris E. Kawcak

Animals have been used extensively as models of joint disease to represent clinical conditions in people. However, veterinary researchers have the luxury of using experimental animals from species that are clinically relevant. Unlike human research, one has no need to assume similarity in findings between species. Equine models of joint disease have been used for several decades to test the effects of drugs and various treatments on joints and to evaluate the pathogenesis of certain diseases. Joint disease can be assessed in horses with clinical disease; however, large numbers are needed for each treatment group to see significant differences in the face of great variation among individual horses. Owner compliance, differing treatment protocols between clinicians, and variability between horses in response to disease and treatment (such as conformation, limb use, and size) all contribute to this variation. Furthermore, clinical studies take a long time, not only to perform, but also to see the effects of treatment. Consequently, in vitro and in vivo models have been developed to give researchers better controlled studies that can be done in a relatively short period of time.

The model to be used should be designed to answer a question by using a testable hypothesis. Variability should be reduced as much as possible so that the question can be answered with little outside influence. However, as more and more variables are eliminated, the model becomes less representative of the clinical situation. For instance, the efficacy of oral joint supplements in reducing joint disease cannot rely solely on results from in vitro studies. The drugs must be tested in vivo to determine if and how they work. However, to determine the effects of a drug or chemical on articular cartilage matrix metabolism, a quick, relatively inexpensive in vitro test can be conducted. Therefore the type of model to be used depends on the question to be answered.

Two types of models are used to study equine joint disease. In vitro models can be used to study various treatments in cell lines and tissue cultures and involve using cells, cell lines, or tissues harvested from joints to test usually one specific pathological pathway or treatment scheme. In vivo systems can be used to test drugs and to determine the pathophysiological response to an insult. Unlike in vitro studies, in vivo studies involve the entire joint, allowing researchers to assess the whole organ to determine truly the clinical efficacy of a drug.

In this chapter the complexity of joint disease and the use of joint models are discussed. The difficulties in modeling joint disease, the rationale for selecting specific models, specific examples of models used for the study of the joint disease, and the current status and future use of equine models of joint disease are considered.

COMPLEXITY OF JOINT DISEASE

The joint can be considered an organ because it comprises several different types of tissues, which biomechanically and biochemically interact with each other. Joint disease can result from several factors. First, the disease has a direct biochemical effect on all tissues. For instance, with synovitis, inflammatory mediators can be released into the joint space and put the articular cartilage into a catabolic state.[1-4] Second, the pain produced by joint disease can result in a change in the character of the gait.[5,6] Consequently this change in joint loading alters biomechanical inputs on all tissues, resulting in a biochemical change in the response by tissues.[7,8] Third, disease of one tissue can result in a change in the mechanical input on another tissue. For instance, articular cartilage degeneration, which can result in increased stress to subchondral bone, can induce a sclerotic response.[9] As another example, subchondral bone sclerosis, which commonly occurs in racehorses, can lead to increased stress on overlying articular cartilage.[10] Therefore in the live animal all tissues are affected by one another.

Because several factors can influence joint disease, researchers have attempted to control these influences in experimental studies. However, not all of the factors can be controlled. For instance, several mechanical factors play a role in joint disease: specifically, mechanical input can vary with horse size, exercise intensity, conformation, neurological control, and lameness. Consequently, differences in the stress to joints can result in changes in biochemical pathways in those tissues. For instance, ponies are one third to one half the size of horses and have little naturally occurring joint disease. Therefore, they may not be the best equine models of joint disease for joint healing studies and exercise studies because of lower imposed stresses. However, they are still good models if imposed stresses do not play a role in the specific disease

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CHAPTER • 65

Models of Equine Joint Disease

Chris E. Kawcak

Animals have been used extensively as models of joint disease to represent clinical conditions in people. However, veterinary researchers have the luxury of using experimental animals from species that are clinically relevant. Unlike human research, one has no need to assume similarity in findings between species. Equine models of joint disease have been used for several decades to test the effects of drugs and various treatments on joints and to evaluate the pathogenesis of certain diseases. Joint disease can be assessed in horses with clinical disease; however, large numbers are needed for each treatment group to see significant differences in the face of great variation among individual horses. Owner compliance, differing treatment protocols between clinicians, and variability between horses in response to disease and treatment (such as conformation, limb use, and size) all contribute to this variation. Furthermore, clinical studies take a long time, not only to perform, but also to see the effects of treatment. Consequently, in vitro and in vivo models have been developed to give researchers better controlled studies that can be done in a relatively short period of time.

The model to be used should be designed to answer a question by using a testable hypothesis. Variability should be reduced as much as possible so that the question can be answered with little outside influence. However, as more and more variables are eliminated, the model becomes less representative of the clinical situation. For instance, the efficacy of oral joint supplements in reducing joint disease cannot rely solely on results from in vitro studies. The drugs must be tested in vivo to determine if and how they work. However, to determine the effects of a drug or chemical on articular cartilage matrix metabolism, a quick, relatively inexpensive in vitro test can be conducted. Therefore the type of model to be used depends on the question to be answered.

Two types of models are used to study equine joint disease. In vitro models can be used to study various treatments in cell lines and tissue cultures and involve using cells, cell lines, or tissues harvested from joints to test usually one specific pathological pathway or treatment scheme. In vivo systems can be used to test drugs and to determine the pathophysiological response to an insult. Unlike in vitro studies, in vivo studies involve the entire joint, allowing researchers to assess the whole organ to determine truly the clinical efficacy of a drug.

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being studied, such as induced synovitis models. Tissue material properties also have an influence on joint disease. For instance, weaker tissues undergo greater biomechanical changes than those that are stronger. Forces that control material properties of tissues include genetics, loading history, and age of the animal. The tissue remodeling status and the ability of animals to remodel articular cartilage, subchondral bone, and soft tissues can also influence joint disease, which is also affected by age, loading history, and genetics. The inflammatory response can also change the joint environment. Specifically, differences in the immune system at the time of disease can greatly influence the inflammatory response to the joint and influence the concentration of cytokines released into the joint.

Variables such as age, size, conformation, and neurological status can be controlled in experimental in vivo studies. However, loading history and the presence of subclinical disease are virtually impossible to control. With the advent of more sophisticated imaging equipment, such as computed tomography (CT), more information on loading history and subclinical disease can be obtained. For instance, subchondral bone density is indicative of loading history.[11] Therefore, researchers in the Equine Orthopaedic Research Laboratory (EORL) at Colorado State University are starting to perform pre-study CT examinations to determine subchondral bone density as an indicator of loading history. Another benefit of CT is that some subclinical diseases may be more easily detectable. Researchers in the EORL laboratory are also initiating a controlled exercise program on a high-speed treadmill before starting the study. The hope is that this can normalize the loading histories of experimental animals. Equally important as loading history and subclinical disease is the biomechanical and biochemical status of the articular cartilage, which at this time is difficult to assess non-invasively at the beginning of a project. With the advent of magnetic resonance imaging (MRI) and pressure probes, articular cartilage and bone matrix structure can be assessed more readily.

TYPES OF MODELS

In Vitro Models

Several types of models have been used to study joint disease. In vitro models have been used and are increasing in use for several reasons. Researchers in university settings are constantly driven to develop in vitro studies to reduce the use of live animals and to address the humane issues that surround live animal research. In vivo models at this time are essential for testing new drugs. However, animal care and use committees at universities critically evaluate in vivo research projects to be sure that they are necessary. These committees are under constant pressure to assure that research animal use falls within ethical guidelines. In vitro systems for studying joint disease can be performed on cells from the tissues of joints. Culture systems can be created for cells such a synoviocytes and chondrocytes in two-dimensional and three-dimensional matrices. Isolated cells also can be evaluated, within the individual matrices, or within artificial matrices. Isolated cells are used to evaluate cellular response to therapeutics agents. Specific outcomes are analyzed to determine cell metabolism and proliferation. Some cell cultures are allowed or stimulated to produce the matrix, and specific cell matrix interaction and matrix metabolism can be studied. Cells are often cultured within artificial matrices to evaluate cell-matrix interactions and cell proliferation within the matrix.

Tissues from living systems are also used for in vitro study of joint diseases. The advantages of such a system over isolated cells are that cells can be maintained within the natural matrix and the experiments are relatively easy to perform. However, tissues must equilibrate in the culture medium to a steady state. This metabolic state may not truly reflect the in vivo state, because no axial load exists and the tissue edges are unconfined. The change in stress patterns and bathing in artificial media then influence articular cartilage matrix metabolism, often leading to increased articular cartilage matrix degradation. Tissues are harvested from cadavers and specimens are placed within media, which is changed every 24 to 48 hours. These tissues then can be manipulated for study, and the media and tissue can be evaluated. Media collected during the study can be analyzed for release or degradation of matrix components and inflammatory mediators. Tissues can be assessed for changes in cell proliferation, matrix synthesis, and characteristics of matrix degradation.

Two types of tissue cultures are available. Single tissues can be studied to determine biochemical, molecular, mechanical, and histological changes that occur with certain influences.[12,13] However, co-culture systems also can be evaluated to determine interactions between tissues. Investigators in the EORL have studied synovium–articular cartilage and articular cartilage–subchondral bone co-culture systems. Exposure of articular cartilage matrix to subchondral bone caused a significant reduction in articular cartilage matrix metabolism.[14] Furthermore similar findings also were seen in the synovium–articular cartilage co-culture systems, in that exposure of articular cartilage matrix to subintimal tissues and vessels led to a significant reduction in articular cartilage matrix metabolism.[15] From these studies one can conclude that exposure of various depths of synovium and subchondral bone to articular cartilage can lead to release of mediators that can change articular cartilage metabolism. However, unlike the equilibration period for in vitro experiments, this change in metabolism is severe and long lasting.

In vitro experiments are relatively straightforward to perform and allow large numbers of repeats. Consequently the numbers are strong for statistical analysis. However, only one small portion of the disease process usually can be evaluated. Furthermore, the more tissues are removed from the natural joint environment, the greater the change in matrix metabolism and response to treatment.

In Vivo Studies

In vivo studies have been used for decades to study equine joint disease. The advantages of in vivo systems are that the cells and matrices are kept within the native environment and can be studied without the influence of harvest and culturing, and normal interaction between tissues can be maintained. The disadvantages are that live animals are sacrificed, and the costs can be prohibitive. In vivo studies are used for evaluating medications, articular cartilage healing techniques, and determining pathological responses to disease. An understanding of the clinical disease and an appreciation for strict experimental design are needed to produce useful in vivo models.

Various inciting mechanisms can be used to induce experimental joint disease. For instance, synovitis models are used to evaluate the sequence of events that occurs with synovitis and the influence of various drugs and medications. Change in gait, measurement of inflammatory mediators, assessment of articular cartilage matrix metabolism, and various clinical tests have been studied. Examples of these studies include injection of lipopolysaccharide,[1,16] interleukin-1,[17] sodium monoiodoacetate,[18,19] filipin,[2,20] polyvinyl alcohol foam particles,[21] carrageenan,[22] and amphotericin[6] into joints. Of these models, those that use the natural inflammatory mediators, such as lipolysaccharide and interleukin-1, seem to me to be the best for promoting the natural cycle of events in inflammation. The advantages of such studies are that they are quick, relatively well studied, and replicate a common clinical problem.

Instability models also have been studied in horses. The purposes of these studies have been to determine the sequence of events that occurs within tissues because of instability and to induce osteoarthritis.[15,23,24] Once osteoarthritis is induced, various treatments can be examined. An example of an instability model includes cutting the collateral and collateral sesamoidean ligaments in the metacarpophalangeal joints of horses.[24] Unlike the dog, complete surgical transection of the cranial cruciate ligament in horses has not resulted in progressive osteoarthritis.[25] Mild osteophyte formation occurred in these joints, but no progressive articular surface changes or lameness were appreciated. The results are unlike horses with naturally occurring cranial cruciate damage, in which some horses are lame and substantial articular cartilage damage may result. Unlike synovitis models, instability models are relatively long-term and represent an example of chronic, progressive disease.

Forced exercise can also lead to changes in joint environment. Examples include evaluation of osteochondral tissues in response to various levels of exercise. Significant changes were found in articular cartilage matrix biochemical and biomechanical properties in exercised horses compared with horses not exercised.[26] Strenuous exercise led to significant increases in calcified cartilage thickness, significant decreases in articular cartilage mechanical properties, and significant increases in fibronectin at the sites of degradation.[26-28] Treadmill exercise led to significant increase in clinical disease in the metacarpophalangeal joints of horses. This was detected grossly and using various imaging techniques.[29] Currently several studies are ongoing to evaluate the effects of exercise to determine the level of exercise most appropriate for protecting the tissues of the musculoskeletal system. For instance, researchers in the EORL are comparing horses with experimentally induced osteochondral disease to young normal horses that are exercised, in the hope of differentiating clinical and diagnostic results in horses with joint disease from those that are undergoing exercise-induced adaptation. Researchers also have shown a recent interest in the effects of exercise on foals, weanlings, and yearlings in the hope that early exercise will lead to stronger musculoskeletal tissues.

The osteochondral fragment model is a blend of in vivo models and involves inducing a clinically relevant disease through creation of an osteochondral fragment and imposed exercise. Researchers at the EORL have used this model extensively. Various medications have been studied, including betamethasone,[30] intravenous hyaluronan,[31] triamcinolone acetonide,[32] methylprednisolone acetate,[33] and gene therapy for joint disease.[34] The researchers are currently using the model to determine differences in diagnostic tests between adaptation to exercise and disease. The model is clinically relevant and induces progressive osteoarthritis, yet it is benign enough to induce grade 1 to grade 3 lameness.[35]

Models of disuse also have been shown to induce osteochondral damage. Disuse is a clinically relevant problem that induces articular cartilage and subchondral bone atrophy. Examples include a significant reduction in articular cartilage matrix metabolism in horses with a lower limb cast.[36] Furthermore, researchers[37,38] showed that application of a cast for 7 weeks, followed by treadmill exercise, led to a significant increase in lameness in the limb with a cast and significant decrease in bone formation.[37,38] Therefore disuse models are important, because casts are used clinically and can lead to substantial problems after removal. Often the race is between healing and the degradative changes caused by disuse.

Models of articular cartilage healing have been studied extensively because of the need to test various modes of treatment in vivo. These models also have been stimulated by the cutting edge research being done by several equine research laboratories around the world and the fact that the horse is becoming more accepted as a model of articular cartilage healing for people. If an implant or technique can stimulate and maintain articular cartilage healing in a horse, then the thought is the implant or technique should work in people. Several equine models are used to study joint healing. Besides the type of treatment that is tested, the models vary in the depth and size of articular cartilage defect formation. Partial-thickness and full-thickness articular cartilage, articular and calcified cartilage, and osteochondral defects have been evaluated in vivo and treated with various techniques.

The size and location of osteochondral lesions appear to have an effect on healing.[39] Small lesions in weight-bearing areas healed better than large lesions and lesions in non–weight-bearing areas. The physical characteristics of the lesions also affect healing. For instance, subchondral cystic lesions formed in horses with linear articular cartilage lesions but not in horses with elliptical lesions.[40] The subchondral defects did not fill in with bone. Trauma to the subchondral bone led to cyst formation.[41] The presence or absence of calcified cartilage also plays a role in articular cartilage healing. Calcified articular cartilage reduced defect filling compared with defects without calcified cartilage present.[42] However, the conclusion from this study and others was that calcified cartilage may provide support for improved healing. Consequently the influence of calcified cartilage on healing is currently being studied.[43]

Various articular cartilage resurfacing treatments have been tested in vivo. Periosteal grafts,[44,45] sternal cartilage grafts,[46,47] mosaicplasty,[48] subchondral micropicking,[42] and cell-based grafting have been evaluated.[45] Sternal cartilage grafts showed promise in the short term. However, significant degradation occurred after 4 months, with subchondral cyst formation.[46,47] Mosaicplasty also has shown promise; however, harvest sites are needed to obtain tissue for implantation.[48] Subchondral bone micropicking also has shown promise in improving healing of osteochondral defects and is simple to perform.[42] Cell-based grafting also has shown promise, but it requires special equipment and advanced training to perform.[49]

Models of infectious arthritis also have been studied (see Chapter 66). For instance, studies have aimed at identifying the ability of certain medications to potentiate infection when injected along with a sub-infective dose of bacteria. This work proved the clinical impression that polysulfated glycosaminoglycans increased the chances of infectious arthritis unless given with antibiotics.[50,51] Comparison of various treatment methods used to treat infectious arthritis also have been studied.[52,53]

Biomechanical Models

Computerized models currently are being developed to study joint disease not only in people, but also in horses and other animals. Specifically, these models are derived from the geometry of the joints, the forces imparted by limb loading and muscle force, and the material properties of tendons, ligaments, and articular cartilage. Consequently, joint disease can be imitated on the computer and the resulting change in joint forces determined. These models also can be used to determine changes in loading in clinical patients. Surgical procedures can be inserted into the program and resulting forces evaluated.

In vivo work will not be replaced in the near future, because it is the best means of evaluating tissue response to disease and treatment. However, newer in vitro systems and computer models are becoming more precise and better accepted by the research community and clinicians.

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CHAPTER • 66

Infectious Arthritis

Alicia L. Bertone

Classic clinical signs of infectious arthritis are heat and swelling and rapid development of non–weight-bearing lameness, often in less than 24 hours. The suspicion of joint infection increases if a predisposing risk factor is evident, such as prematurity, high sepsis score, or multisystemic disease in a foal[1] or preceding joint injection in an adult horse. Fracture and non-articular infection (cellulitis or foot abscess) need to be differentiated from infectious arthritis, because in these diseases, acute, severe lameness also develops.[2,3]

CAUSES

Musculoskeletal infection was reported to cause death in 5.2% of 2468 foals.[4] Yearly morbidity was 27.4% (677 foals), and morbidity attributed to musculoskeletal infection was 2.1%. Septicemia was the second most common cause of death, and hematogenous spread was the most common cause of infectious arthritis.[5] The risk of infection was highest in the first 30 days post partum and was lowest in practices that assessed the efficacy of transfer of passive immunity. Isolation of *Salmonella* species from synovial fluid and systemic disease were associated with an unfavorable prognosis for survival.[1]

In two retrospective studies of 153 mature horses[6,7] the most common causes of synovial infection were traumatic wounds (36.5% for joints and 55% for tendon sheaths), injections (34.1% and 22%), post-operative infection (19.8% for joints), and idiopathic causes (9.5% and 22%).[7,8] The Standardbred and the tarsocrural joint were over-represented in adult horses with joint infection, reflecting a greater number of joint injections in this breed and joint.

Of 424 bacterial isolates from 233 horses with joint, sheath, or bone infection, 91% were aerobic or facultative anaerobes. The most common organisms were Enterobacteriaceae (28.8%), followed by streptococci (13%) and staphylococci (11.8%).[9] In foals, Enterobacteriacae including *Escherichia coli* and *Staphylococcus* species[1,10] were more likely to be isolated. Staphylococci, specifically *Staphylococcus aureus*, is the most common organism isolated from infections occurring after surgery or injections. Foals or horses with infectious arthritis secondary to penetrating wounds are likely to have multiple bacterial infections. *Clostridium* species were the most common anaerobes isolated and were most common from wounds near the hoof. Fungal organisms were a rare cause of infectious arthritis but can be considered pathogens if identified in pure culture more than once.[11] Reactive arthritis in foals with septicemia from *Rhodococcus equi* can be confused with infectious arthritis, but lameness usually is not prominent, and synovial fluid nucleated cell counts are often within normal limits.[12]

EXAMINATION AND INITIAL MANAGEMENT

Potential infectious arthritis must be considered an emergency and is treated most effectively with early diagnosis. A systematic approach should include hematological examination, measurement of plasma fibrinogen, synovial fluid analysis, including cytological examination, Gram stain, and synovial fluid culture (up to 5 ml of synovial fluid in a broth culture bottle), and radiography.[3] In foals, particularly those with abnormal sepsis scores, blood culture should be performed

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CHAPTER • 66

Infectious Arthritis

Alicia L. Bertone

Classic clinical signs of infectious arthritis are heat and swelling and rapid development of non–weight-bearing lameness, often in less than 24 hours. The suspicion of joint infection increases if a predisposing risk factor is evident, such as prematurity, high sepsis score, or multisystemic disease in a foal[1] or preceding joint injection in an adult horse. Fracture and non-articular infection (cellulitis or foot abscess) need to be differentiated from infectious arthritis, because in these diseases, acute, severe lameness also develops.[2,3]

CAUSES

Musculoskeletal infection was reported to cause death in 5.2% of 2468 foals.[4] Yearly morbidity was 27.4% (677 foals), and morbidity attributed to musculoskeletal infection was 2.1%. Septicemia was the second most common cause of death, and hematogenous spread was the most common cause of infectious arthritis.[5] The risk of infection was highest in the first 30 days post partum and was lowest in practices that assessed the efficacy of transfer of passive immunity. Isolation of *Salmonella* species from synovial fluid and systemic disease were associated with an unfavorable prognosis for survival.[1]

In two retrospective studies of 153 mature horses[6,7] the most common causes of synovial infection were traumatic wounds (36.5% for joints and 55% for tendon sheaths), injections (34.1% and 22%), post-operative infection (19.8% for joints), and idiopathic causes (9.5% and 22%).[7,8] The Standardbred and the tarsocrural joint were over-represented in adult horses with joint infection, reflecting a greater number of joint injections in this breed and joint.

Of 424 bacterial isolates from 233 horses with joint, sheath, or bone infection, 91% were aerobic or facultative anaerobes. The most common organisms were Enterobacteriaceae (28.8%), followed by streptococci (13%) and staphylococci (11.8%).[9] In foals, Enterobacteriacae including *Escherichia coli* and *Staphylococcus* species[1,10] were more likely to be isolated. Staphylococci, specifically *Staphylococcus aureus*, is the most common organism isolated from infections occurring after surgery or injections. Foals or horses with infectious arthritis secondary to penetrating wounds are likely to have multiple bacterial infections. *Clostridium* species were the most common anaerobes isolated and were most common from wounds near the hoof. Fungal organisms were a rare cause of infectious arthritis but can be considered pathogens if identified in pure culture more than once.[11] Reactive arthritis in foals with septicemia from *Rhodococcus equi* can be confused with infectious arthritis, but lameness usually is not prominent, and synovial fluid nucleated cell counts are often within normal limits.[12]

EXAMINATION AND INITIAL MANAGEMENT

Potential infectious arthritis must be considered an emergency and is treated most effectively with early diagnosis. A systematic approach should include hematological examination, measurement of plasma fibrinogen, synovial fluid analysis, including cytological examination, Gram stain, and synovial fluid culture (up to 5 ml of synovial fluid in a broth culture bottle), and radiography.[3] In foals, particularly those with abnormal sepsis scores, blood culture should be performed

simultaneously. In adult horses, systemic blood examination is less rewarding than in foals, particularly in the early phases of clinical signs. In horses with experimental infectious arthritis, elevations in leukocyte count, total protein, and fibrinogen levels took several days to develop and were changed significantly from baseline values for individual horses, but they remained in the normal range for all horses.[13] Before performing arthrocentesis, surgical scrub materials, sterile gloves, needles, syringes, broth culture bottles, ethylenediamine tetraacetic acid (EDTA) or heparin tubes for cytological examinations, smear slides for Gram stain, and a dose of antimicrobial agents to instill directly into the joint after sampling should be available (see the following discussion).

Adult horses should be sedated. Foals can be placed in lateral recumbency with administration of an α_2-agonist and synthetic narcotic combination. Although data suggest that aseptic preparation of unclipped hair may be adequate and significantly decrease bacterial counts on the skin,[14] clipping the site for arthrocentesis is strongly recommended. In horses with peri-articular wounds, arthrocentesis should be performed well away from the wound to avoid the risk of joint contamination, if the joint is not contaminated already from the wound. To determine if a joint and wound communicate, the clinician should infuse 50 to 200 ml of balanced electrolyte solution into the joint after a synovial fluid sample has been obtained for culture and cytological examination. The clinician should watch closely for leakage of fluid from the wound. This is easier than using blue dye injections or contrast radiography. The joint should be drained and antibiotic instilled into the joint after samples have been obtained and any lavage or injection of fluid completed. Samples should be submitted immediately for evaluation.

If the joint fluid is grossly cloudy, turbid, or flocculent, broad-spectrum antibiotics initially should be given intravenously, and ingress and egress or through-and-through lavage of the joint should be performed until diagnosis of infectious arthritis can be confirmed. Antibiotic should be instilled into the joint after lavage is completed.

DIAGNOSIS

Gross evaluation of the synovial fluid can be informative. If newspaper print cannot be read through the fluid sample, it probably has a cell count of 30×10^9 nucleated cells/L. Fluid from infected joints is usually turbid, cloudy, and watery. Flocculence develops in chronically infected joints or joints that have been invaded with a needle or surgery. Blood contamination makes gross assessment of synovial fluid difficult, and determining if infection is present without clinicopathological analysis is often impossible. Serosanguineous fluid commonly is obtained from infected joints (Fig. 66-1). An estimate of the amount of blood (packed cell volume or red blood cell count) can be made to correct for the number of nucleated cells contaminating the sample (hemorrhage from arthrocentesis or leakage of blood from the infection):

$$\frac{\text{PCV (\%) of synovial fluid}}{\text{Actual or estimated PCV (\%) of blood}} = \frac{\text{X (WBC} \times 10^9\text{/L in synovial fluid)}}{\text{Actual or estimated WBCs} \times 10^9\text{/L blood}}$$

where PCV is packed cell volume and WBC is white blood cell.

Synovial fluid nucleated cell count, differential cell count, and total protein concentration are the most useful parameters to evaluate in diagnosing infectious arthritis. Synovial fluid nucleated cell counts in excess of 30×10^9/L cells, with >80% neutrophils, or total protein concentration in excess of 40 g/L are considered consistent with infection, particularly if these synovial fluid parameters correlate with clinical signs and predisposing circumstances.[3] The likelihood of isolating the causative organism is correlated positively to the nucleated cell count, that is, higher nucleated cell counts have greater isolation rates.[15] Approximately 25% of samples from horses suspected of having infectious arthritis demonstrate bacteria on Gram stain, confirming infection and offering the added benefit of assisting with identifying the organism and the initial selection of an antimicrobial agent.

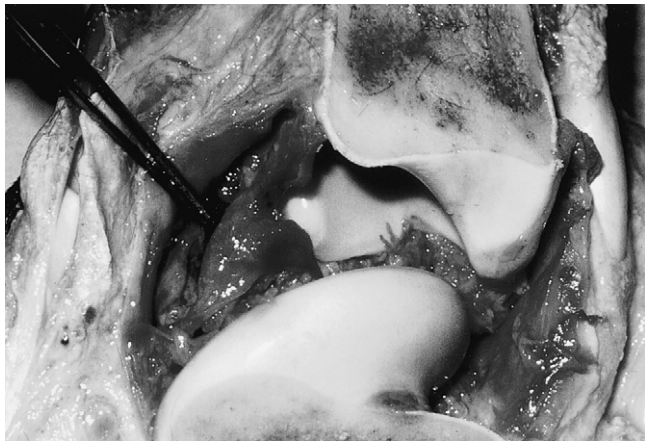

Fig. 66-1 At post-mortem examination, hemorrhagic synovitis associated with acute joint infection is seen in this tarsocrural joint.

For horses that do not meet obvious criteria for infectious arthritis, assessment of all parameters is necessary to conclude that the joint is infected. Importantly, joints can be infected with nucleated cell counts less than 30×10^9/L. Infection should be suspected if nucleated cell counts are between 10×10^9/L and 30×10^9/L and fluid is not serosanguineous, clinical signs of substantial lameness exist, fluid is flocculent (coagulating cells may falsely lower the measured cell count), a predisposing cause for infection is present (septicemia, surgery, or joint injection), and protein concentration exceeds more than 40 g/L. Previous intra-articular treatment with corticosteroids may delay onset of clinical signs and confuse interpretation of nucleated cell counts and total protein concentration in the acute phase.[16,17]

Synovial fluid protein levels continue to increase in chronically infected joints to more than 60 g/L in some horses.[13] Few other differential diagnoses, other than infection, produce significant elevations in total protein (>50 g/L). Isolation of a pure culture of a single organism, particularly a known pathogen, such as a coagulase-positive *Staphylococcus*, almost always indicates infection is present, even if the nucleated cell count is low.

In horses with chronic infectious arthritis refractory to aggressive treatment, nucleated cell counts can be low (5 to 10×10^9/L), but organisms, usually *Staphylococcus*, can still be isolated. In these horses the synovial fluid protein values are often high (>50 g/L) and other clinical signs of infectious arthritis (lameness, effusion, and heat) persist. In my experience these are horses that developed infectious arthritis after injection with a corticosteroid, were treated with systemic antibiotics and lavage, but not aggressive drainage, have a nidus of infected subchondral bone that keeps seeding the joint, or have a joint with severe cartilage erosion.

The distribution of nucleated cells in synovial fluid is an important aid to diagnosis. In horses with early infectious arthritis, almost always nucleated cells comprise more than 80% neutrophils and commonly more than 90% neutrophils.

The neutrophils usually appear healthy and not degenerate, although in overwhelming or aggressive infection, degeneration of neutrophils is seen. If synovial fluid has less than 75% neutrophils, infection is usually resolving.

Techniques that may be useful clinically in the future for diagnosis include polymerase chain reaction (PCR) analysis for detecting base pairs of bacterial or viral DNA in the synovial fluid[18,19] and determining enzyme and cytokine release that may be specific for infection.[20,21] Benefits of PCR include rapid (<24 hours), sensitive testing that can detect selected species of bacteria in the presence of antimicrobial drugs. PCR diagnostic techniques are sensitive because they amplify small quantities of bacterial DNA. Inherent in high sensitivity, however, is a possible high false-positive rate, because of skin contamination or contamination with bacteria at the time of arthrocentesis. Future clinical use and evaluation of this technique is expected.

Identification of enzyme/cytokine ratios or quantity may be specific for infection, because infection is the largest joint challenge that exists. Knowledge of the presence and interaction of mediators and inhibitors in joints is growing rapidly and is an active area of research. For further information on this aspect of joint infection, the reader is referred to other texts.[20,22]

It is important to determine whether bone or physeal involvement is concomitant, particularly in foals with refractory infectious arthritis. In most foals with bone involvement, radiolucent changes occur rapidly and are often detectable within 1 week after onset of clinical signs. In most bones radiographic changes may be apparent in 7 to 10 days, but if the small cuboidal bones of the carpus or tarsus are involved, the clinician may face an even greater diagnostic challenge. Infarction associated with infection may slow bone resorption, and evidence of radiolucency may lag for several days.

Nuclear scintigraphy may be useful to locate foci of infected bone, particularly in identifying involvement of subclinical sites of infection in polyarthritic foals, but the technique has practical restrictions. Normal bone scans or radiolabeled white cell studies can be performed. The foals become radioactive, and handling of the foal, blood samples, and synovial fluid samples becomes a small risk to attendants. Additionally, if the joints surrounding the small bones are infected, the resolution of the scan may be inadequate to identify specific bone involvement. Magnetic resonance imaging offers promise of improved identification of osteomyelitis[23,24] and often can be arranged for foals at facilities used for imaging people. Osteomyelitis is present in up to 59% of foals with infectious arthritis, but a favorable prognosis still can be achieved with treatment,[1] although the prognosis for athletic function is reduced when adjacent bone is involved or infectious arthritis is protracted.

ANTIMICROBIAL THERAPY

Systemic Therapy

Intravenous antimicrobial therapy should be initiated immediately, before bacterial culture results are available. Systemic administration of broad-spectrum antimicrobial drugs should be combined with the local administration of antimicrobial agents. Most common combinations include penicillin with an aminoglycoside or a third-generation cephalosporin such as ceftiofur sodium. In a retrospective study of equine musculoskeletal infections, gentamicin and amikacin were effective against 85% and 95% of equine isolates, respectively, indicating they are good choices for initial combination therapy.[9] Antimicrobial drugs given orally should be reserved for infection that is resolving, because gastrointestinal absorption is erratic, and blood and tissue levels are lower. Oral enrofloxacin is used to treat chronic bone and synovial infections in mature horses without reported incident, but enrofloxacin is not currently approved for use in horses. Enrofloxacin (Baytril 100) administered intravenously at 5 mg/kg once daily is effective and safe in mature (3 years and older) horses.[25] Enrofloxacin may cause lameness and cartilage lesions in foals and should not be used.[26] Enrofloxacin should not be administered to lactating mares, because the milk may concentrate the drug and subject the foal to chondrotoxic dosages.

Antimicrobial drugs should be given intravenously and in general at a dosing interval that maximizes peak serum levels and sustains trough levels that are at or above the MIC for the isolated organism. Serum antimicrobial peak and trough measurements should be periodically checked to maximize effectiveness and minimize toxicity (see the following discussion).

Aminoglycoside dosing three times a day was originally designed to achieve peak serum concentrations (5 to 10 μg/ml) and trough serum concentrations (0.5 to 2.0 μg/ml) to balance optimal clinical efficacy and to minimize toxicity.[27] However, once daily dosing may be more effective. The larger the serum aminoglycoside concentration to minimum inhibitory concentration (MIC) ratio, the greater the bactericidal effect. The aminoglycoside post-antibiotic effect (duration of sustained antimicrobial killing) is concentration dependent. With a single daily dose, trough levels could be lowered, thus lowering the risk of toxicity. Using in vivo models of infectious osteomyelitis, improved efficacy of gentamicin was demonstrated using once daily dosing (6.6 mg/kg body mass IV) compared with three times a day dosing (2.2 mg/kg body mass IV). In normal horses, gentamicin (administered at 6.6 mg/kg body mass once daily for 10 days) did not induce signs of nephrotoxicity and prolonged the post-antibiotic effect.[28] Therefore gentamicin and other aminoglycosides should be administered once daily in horses. However, the pharmacokinetics of aminoglycosides are altered in septic and premature hypoxic foals.[29] Serum drug concentrations should be measured to adjust dosing intervals. Sepsis score and creatinine concentration are inversely correlated to amikacin clearance in foals and could be useful indicators of altered drug disposition and delayed clearance.[30] The dosing interval may need to be lengthened beyond 24 hours in these foals to avoid toxicity (higher trough concentrations). In septic neonatal foals, administration of gentamicin at 3.3 mg/kg twice daily intravenously produced peak serum concentrations of more than 6 μg/ml and trough concentration less than 2 μg/ml in all foals without toxicity or development of new sites of infection.[31]

The drug must have excellent diffusion into the joint.[6] Most antimicrobial drugs penetrate the synovium in therapeutic concentrations when administered systemically at recommended dosages.[3] Concentrations of aminoglycosides actually may be greater in inflamed joints compared with normal joints.[32] If systemic blood trough levels drop below MIC for that antimicrobial, synovial fluid concentrations may also drop below MIC. Local antimicrobial administration (intra-articular), used to sustain high drug concentrations at the site of infection, may be most effective in both killing bacteria and penetrating organic debris.

Most positive synovial fluid cultures will be so within 24 hours after onset of infection, and a Gram stain can be immediately helpful. An additional 24 hours is usually needed to confirm the identity and susceptibility pattern of the organism. In my opinion, a broad-spectrum antimicrobial drug should be administered initially and continued at least until clinical signs begin to resolve substantially. Some joints may contain single or several organisms that were not isolated, particularly if infection was caused by a wound or by septicemia. Joints may be open, or repeatedly invaded during treatment, or a foal may be at risk of continued showers of bacteremia,

thereby increasing the risk of a shift in causative organism in the middle of treatment.

Local Therapy

For years local administration of antimicrobial agents was considered taboo, because solutions varied in pH and were believed to be injurious to tissues. The deleterious effects of local antibiotic administration were greatly overemphasized, and one of the most substantial advances in the management of horses with infectious arthritis and osteomyelitis has been the implementation of local antimicrobial therapy. High tissue concentration of antimicrobial agents causes rapid elimination of joint and bone infections.[33,34] Many innovative methods are being explored to provide high local antimicrobial drug levels. Administration of systemic antimicrobial agents remains an important adjunctive therapy, but it is recognized that tissue concentrations at the site of infection may be considerably lower than those achieved with local administration of drugs (at or below MIC for the organism) and therefore less effective.[35]

Direct Local Infusion of Antimicrobial Drugs

Intra-articular injection of antimicrobial drugs into the infected joint every 24 hours is currently the most common therapy for early infectious arthritis. Intra-carpal administration of 150 mg gentamicin maintained synovial fluid gentamicin concentrations well above the MIC for most equine pathogens (2 µg/ml) for 24 hours.[33,36] Most antimicrobial drugs, including penicillin, the cephalosporins, and aminoglycosides, are minimally irritating.

Appropriate intra-articular dosages of antimicrobial drugs are not scientifically identified, but anecdotally up to 500 mg gentamicin, 250 mg amikacin, 1×10^6 units of sodium penicillin, 500 mg cefazolin, and 500 mg ceftiofur sodium have been used without reported difficulties. Fluoroquinolones should not be used because at high concentrations the drugs are toxic to chondrocytes.[37]

Topical lavage of an infected joint or osteomyelitic bone is beneficial for the removal of infected organic debris, destructive enzymes, and neutrophils, but the inclusion of antimicrobial or antiseptic compounds in the lavage solution is still of questionable benefit. An increase in the local antimicrobial drug concentration is expected after lavage with antimicrobial drugs, but most of the drug leaves the joint with the lavage solution. Injection of antimicrobial drugs at the termination of lavage is probably more efficient. Use of antiseptics and potentiated antiseptics (EDTA and Tris buffer) in lavage may kill surface bacteria, but sustained killing is expected to be limited.[38] Even dilute antiseptic compounds such as 0.05% chlorhexidine can be irritating to equine joints.[39]

Antimicrobial-Impregnated Polymethylmethacrylate

One of the most practical methods for maintaining slow but effective release of antimicrobial agentss in bone and joint infections is the intra-articular insertion of impregnated polymethylmethacrylate (PMMA) beads.[40] In a study of 1085 open limb fractures in people, the post-operative infection rate was significantly reduced from 12% to 3.7% when aminoglycoside PMMA beads were inserted at surgery.[41] In horses, PMMA impregnated with aminoglycoside and cefazolin was used in horses with open fractures and with bone, implant, and joint infections, and survival rate was about 60%[42,43] (Fig. 66-2). In 12 horses with infectious arthritis treated with gentamicin-impregnated PMMA beads and lavage, including six with osteomyelitis, 92% survived.[44] Several antimicrobial drugs were shown to elute from PMMA in active concentrations, including aminoglycosides such as amikacin and gentamicin, and fluoroquinolones such as ciprofloxacin.[34,44,46] Use of fluoroquinolones in PMMA is not currently recommended in joints, because the concentrations expected to be released with local therapy may be toxic to equine chondrocytes.[37]

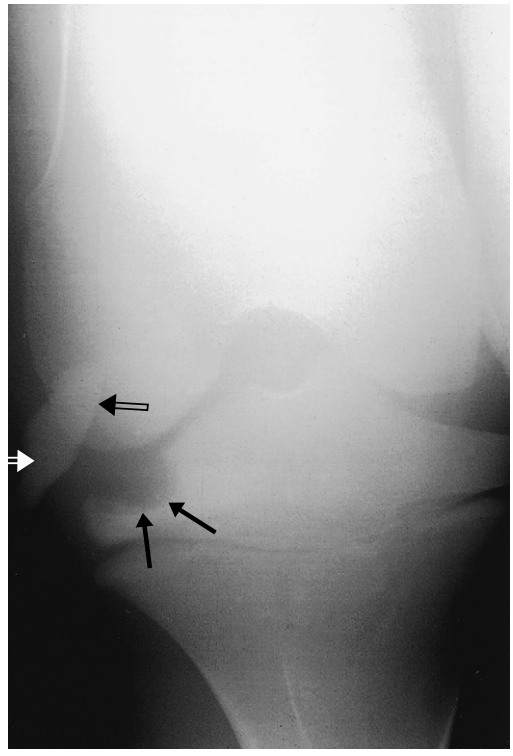

Fig. 66-2 Craniocaudal radiographic view of a foal's stifle joint demonstrating osteolysis of the medial tibial plateau *(solid black arrows)* that was debrided arthroscopically and a singular polymethylmethacrylate bead *(open arrows)* placed intra-articularly.

Preparation of implants (beads) can be performed in the operating room or beads can be gas sterilized and stored for future use. The antimicrobial compound (1 or 2 g of powder or liquid) is mixed thoroughly with 20 to 40 g of PMMA (medical grade) and shaped as desired. Beads can be placed on suture material to assist with retrieval from the joint. The beads can be placed into the joint through an arthrotomy, which is usually left open to drain. If infection recrudesces, existing beads can be exchanged with fresh ones or with those impregnated with different antimicrobial agents. Small beads (4 × 6 mm) can be placed through arthroscopic portals or cannulas, thus avoiding arthrotomy. Arthotomy, however, can also assist with joint drainage if left open to heal by contraction and epithelialization. In my experience, if infection is rapidly eliminated, the beads can be difficult if not impossible to retrieve. Beads frequently migrate to a joint pouch and become enveloped by synovium. If the arthrotomy heals quickly, the beads need to be removed surgically. Occasionally beads well removed from articular surfaces can be left in place permanently. Long-term sequelae appear minimal, but studies to date have not been performed. Complications with PMMA implants include the use of too many or too large beads, causing soft tissue trauma and pain, failure to exchange beads in unresolving infection and placement of beads under tendons and on articular cartilage. If osteomyelitis exists, lesions can be debrided and lavaged, and beads can be implanted directly into the bone bed. Beads placed directly into bone lesions are usually removed without difficulty.

Addition of antimicrobial compounds to PMMA alters the biomechanics of PMMA, particularly when using liquid forms. For purposes of treating joint infection, accuracy in drug elution and biomechanical properties of the PMMA are not critical. Use of the PMMA in orthopedic implants may require more precise preparation of implant material. The

elution rate of antimicrobial drugs varies with size and shape of the implant, the amount of antimicrobial agent impregnated, and the type and form of antimicrobial drug selected and depends on thorough and even mixing of the formulation.

Antimicrobial-Impregnated Biodegradable Drug Delivery Systems

Although PMMA offers advantages for local antibiotic delivery, its permanency is not ideal, particularly for certain tissue types. Many biodegradable compounds have been investigated as possible implants for antimicrobial drug delivery, most notably collagen,[47] DL–lactide-glycolide copolymers, polyanhydrides, polylactide, sebacic acid, tricalcium phosphate and calcium carbonate bone cement, and plaster of Paris.[48-53] Because of the ongoing degradation a greater amount and duration of antimicrobial drug release can be achieved. In a study using rabbits with infectious osteomyelitis, bacterial counts were significantly lower in those treated with gentamicin/polyanhydride implants than in those treated with gentamicin/PMMA implants.[50]

In horses, 50:50 DL–lactide-glycolide copolymers and poly(DL)lactide impregnated with gentamicin that eluted for 10 days eliminated infection of synovial explants in vitro without significant detrimental effects on synovial cell function (hyaluronan acid production), morphology, or viability.[48] In horses with experimentally induced *Staphylococcus* joint infection, intra-articular treatment with C44 fatty acid-sebacic acid (1:1) beads impregnated 20% with gentamicin as the sole treatment effectively eliminated joint infection in 33% of joints by day 3 and 66% of joints by day 13.[52] Lameness significantly improved. Gentamicin concentration peaked at 82 µg/ml at 24 hours after insertion of beads and remained higher than 10 µg/ml for 12 days (range of 5 to 41 times the MIC for the organism).[52] Currently these implants are being investigated for clinical use, but they are not yet commercially available. Plaster of Paris beads may offer a practical option for degradable implants, but biocompatibility studies should be performed.[54]

Regional Perfusion

Regional perfusion can be used to deliver high concentrations of antimicrobial agents to a selected region of the limb, usually using the venous system for drug administration. With horses under anesthesia, drugs also can be administered using an intra-osseous route. In either case a tourniquet is applied proximal to the infected bone or joint. Concentrations of gentamicin obtained in synovial fluid ranged from a peak of 589 µg/ml immediately after regional perfusion and declined to 4.8 µg/ml at 24 hours.[54] This technique was used to decrease white blood cell numbers in infected synovial fluid in experimentally induced equine arthritis and is most practical if applied when the horse is anesthetized for other treatment of infection, such as debridement and lavage, when intra-osseous infusion can be performed simultaneously.[55] Practically the technique is not commonly used for sustained high concentrations of antimicrobial agents, simply because daily general anesthetic procedures are costly and time consuming. Intravenous infusion can be performed in some standing, sedated horses. Limitations include the need for a tractable horse, minimal peri-articular swelling for ease of vein identification, and the horse must tolerate the pain of a tourniquet and infusion of antimicrobial drugs. Perineural analgesia can be performed (Editors). Intra-articular antimicrobial drug concentrations using intravenous regional infusion are not as high as those obtained with direct intra-articular injection.[36]

In contrast to joint concentrations, bone concentration of antimicrobial agents is expected to be higher after regional perfusion, because bone is also perfused. Therefore in foals with infectious arthritis and adjacent osteomyelitis, regional perfusion offers an advantage, because daily intra-articular administration of antimicrobial agents may not yield high concentrations of the drug in bone. Intra-articular administration relies on drug diffusion through cartilage or local blood supply to reach bone. Confirmation of bone antimicrobial concentrations after regional perfusion is warranted. Caution should be used when applying tourniquets in foals, because even short durations of application can induce clinical signs of distal limb ischemia. In foals with compromised bone blood flow from swelling and toxic effects of infection, this may be detrimental. I have seen two foals develop severe osteonecrosis after repeated regional perfusion procedures, a complication attributed to tourniquet application.

JOINT DRAINAGE AND DEBRIDEMENT

Joint lavage and drainage are vital for the effective management of infectious arthritis.[1,3,5,6,44] Ideally arthroscopic evaluation should be performed to remove foreign material and fibrin and to assess cartilage health. Removal of angry, infected synovium may reduce bacterial count and a source of infection. Healthy synovium should not be removed, because it may help combat the infection and may normalize joint health. In horses, maximal synovectomy was no better than open arthrotomy for management of infectious arthritis.[56] After synovectomy villous structures may not be regained for many months.[57-60] Repeated through-and-through lavage using needles placed on opposite sides of a joint can be effective in eliminating infection in some horses, but the lavage fluid may completely bypass some areas of a joint, allowing the infection to persist. Arthroscopy permits more thorough lavage of the entire joint, and together with a form of continued joint drainage, provides the most complete and rapid method to remove infective material and to estimate the extent of damage.[6,44]

Arthroscopic portals can be enlarged to function effectively as open arthrotomy incisions for continued drainage, or closed suction drainage can be initiated. The goal of either technique is continued joint decompression and drainage. I prefer open arthrotomy because it is simple and avoids the complications that can occur with drains, including drain breakage, ascending infection, joint irritation, and clogging. Arthrotomies usually heal without complication and with minimal scarring. With continued infection, arthrotomies stay open and may develop excessive granulation or fibrous tissue, but this complication may occur with other methods of chronic drainage as well. Open drainage in joints that cannot be bandaged, such as the stifle, can be performed by tying up the horses using cross ties or an overhead wire. Adhesive bandages applied using ether can be used to cover open drainage sites. I have not seen substantial permanent complications associated with arthrotomy and open drainage. Open arthrotomy can be used to insert PMMA beads for treatment of osteomyelitis or in horses with refractory infection.

Properly managed closed suction drainage is useful for large joints such as the tarsocrural and scapulohumeral joints, using a flat, fenestrated, latex drain. The drain should be tunneled under the skin to exit at a site removed from the joint. Negative pressure is applied using a large syringe, with a large guarded needle passed through the syringe to keep the plunger retracted.[60] The syringe is evacuated several times a day, and the drain is left in place until only small volumes of fluid are collected or the fluid appears relatively normal grossly and cytologically, and lameness improves. If the suction system fails, the drain is removed.

After initial debridement, lavage, and establishment of drainage, the joint can be flushed and local antimicrobial drug therapy applied daily, usually for 3 days. If clinical improvement is dramatic, further lavage is optional, but local antimicrobial injection should continue for 3 days. If clinical improvement continues, local antimicrobial therapy is discontinued. The joint is then rested for 24 hours and the synovial

fluid is reassessed. Ideally, follow-up synovial fluid contains fewer than 15×10^9 nucleated cells/L, but irritation from aggressive lavage and intra-articular antimicrobial compounds can result in higher cell counts.

Clinical improvement is assessed by evaluating rectal temperature, lameness, and heat over the joint surface. The synovial fluid nucleated cell counts should decline slowly over the next 7 days if infection remains under control. If nucleated cell counts rise again or if lameness recurs, lavage and local antimicrobial therapy can be re-instituted. Having to perform two series of lavage procedures (3 days each time) in foals is typical.

Articular Osteomyelitis

Fifty percent of foals with infectious arthritis also have osteomyelitis or bone necrosis.[1,6,7,10] Usually bone is infected, but bone infarction can also occur. These lesions are best treated using arthroscopic debridement and joint infection usually clears within days of removing infected bone. The importance of arthroscopic debridement cannot be overemphasized. In young foals with severe joint distention, lesions that would not normally be amenable to arthroscopic evaluation and debridement become accessible, such as those involving the tibial plateau (Fig. 66-2), caudal humeral head, or coxofemoral joint. In my experience those involving infection or necrosis of the tarsal bones and some aspects of the carpal bones are the most difficult to treat, because they are not directly accessible using arthroscopy. Insertion of PMMA beads close to the distal tarsal joints and prolonged systemic antimicrobial administration may be successful.

In adult horses with articular osteomyelitis it is critical to debride the infected bone as soon as the condition is recognized radiographically (Fig. 66-3). In chronic, progressive, infectious osteomyelitis in adult horses, arthrodesis or stimulated ankylosis with a bone graft may be the only way to eliminate joint pain.[61-63] Infection of implants does not always occur.[61] Bone infection can resolve with prolonged antimicrobial drug therapy, but joint immobilization is important to assist with elimination of infective organisms.

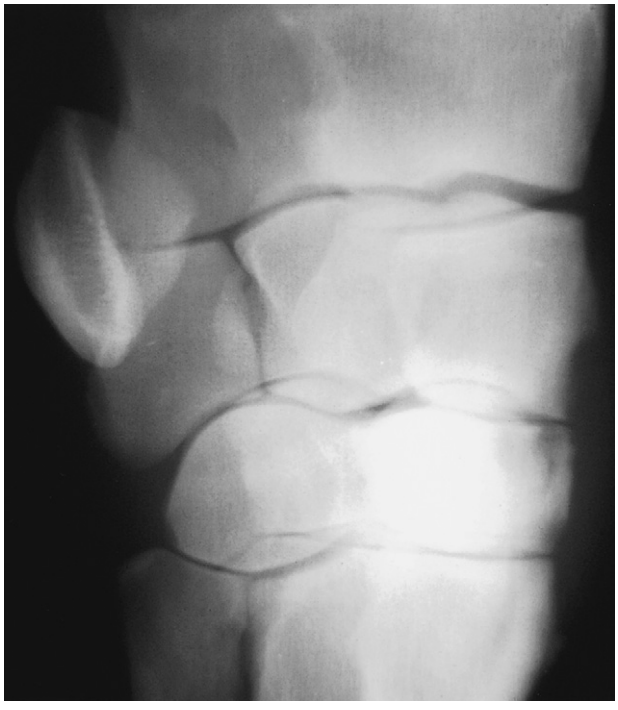

Fig. 66-3 Dorsolateral-palmaromedial radiographic view of a carpus of a Standardbred racehorse with infectious osteomyelitis and necrosis of the dorsomedial aspect of the third carpal bone. The infection occurred subsequent to an intra-articular injection.

PAIN MANAGEMENT

Non-Steroidal Anti-Inflammatory Drugs

In a rabbit staphylococcal model of infectious arthritis, use of NSAIDs with antimicrobial drugs significantly reduced joint swelling, prostaglandin E_2 release, and collagen joint destruction compared with the administration of antimicrobial drugs alone.[64] In mature horses phenylbutazone administration (4.4 to 8.8 mg/kg body mass daily) is commonly used to reduce pain and joint inflammation. Risk of laminitis in the supporting limbs of lame horses is an important consideration when treating infectious arthritis. Cyclooxygenase II inhibitors are currently being approved for use and seem promising at reducing the risk of toxic side effects, such as gastrointestinal ulcers and nephrotoxicity, seen with currently marketed NSAIDs. Cyclooxygenase II inhibitors reduce the production of inducible prostaglandin E_2 without suppressing the constitutive form of prostaglandin E_2 that protects epithelial mucosa homeostasis. Administration of NSAIDs in horses with infectious arthritis must be titrated to allow for the accurate assessment of joint pain as an indicator of clinical response to treatment. Most currently available NSAIDs marketed for use in horses, or used off label in horses, have been demonstrated to suppress equine synovial membrane inflammation and prostaglandin E_2 release in culture experiments.[65,66]

Dimethylsulfoxide (DMSO) solution (10% to 40%) has been used experimentally to treat synovitis and clinically to treat infectious arthritis in horses.[3] Lavage or intra-articular injection of up to 40% DMSO does not appear to have significant negative effects on articular cartilage. DMSO is a free radical scavenger, antiseptic, and analgesic and may inhibit chemotaxis of inflammatory cells. Practically, DMSO can be used in lavage fluid, particularly in the last liter of lavage. Because of the high solubility of DMSO, some residual drug probably is absorbed into the synovium during lavage.

Epidural Narcotics

Placement of indwelling epidural catheters and chronic infusion of α_2-agonists and narcotics such as morphine have been useful in alleviating hindlimb lameness and in horses with severe pain secondary to infectious arthritis (see Chapter 86).[67,68] In horses with chronic osteomyelitis or refractory infectious arthritis this technique may be useful in preventing recumbency or supporting limb laminitis.

TOPICAL TREATMENT, BANDAGING, AND ALTERNATIVE THERAPY

DMSO applied topically to horses with experimentally induced carpal synovitis significantly reduced synovial fluid nucleated cell counts.[14] DMSO was detectable in the synovial fluid of five of six horses and in the plasma of one of six horses, indicating its penetration of the tissues from topical application.[69] Topical application over infected joints may reduce synovitis.

Pressure bandages applied to reduce swelling also can reduce joint pain. Edema formation can be overwhelming in infected joints and produce stiffness, discomfort, elevated synovial fluid production (joint pressure), and poor joint clearance of infected material through the lymphatic system. Pressure bandages should be sterile, particularly when covering open arthrotomy incisions.

Once acute inflammation has resolved, passive flexion is indicated to enhance lymph flow, improve drainage, reduce edema, and enhance joint range of motion. Capsulitis associated

with infection can be a significant complication, and physiotherapy to restore joint function can be critical to improving long-term outcome and joint motion.

PROGNOSIS

Adult horses with infectious arthritis have a good prognosis for survival and for return to athletic function, provided that infection is recognized early. In most patients infection is eliminated.[2,6,7,44] Pre-existing osteoarthritis and articular cartilage damage are the most common reasons for failure to return to performance. Prompt recognition, aggressive drainage and lavage, and early treatment with local antimicrobial drugs have steadily improved prognosis to >80%[6] and, we hope, will achieve success rates close to 100%. Aggressive surgical debridement and use of implantable elution materials for chronic administration and multiple site delivery of antimicrobial agents have improved prognoses. The prognosis in foals is more guarded. Septicemia, osteomyelitis, and hypogammaglobulinemia result in a lower prognosis for life in foals, and infection is eliminated in only 50%.[7] Elimination of infection was achieved in 70%,[1,6] 50% survived,[6,10] and 30% reached racing performance.[1]

FUTURE TREATMENTS

Newer biodegradable polymers impregnated with antimicrobial drugs for direct joint insertion through an arthroscopic portal could greatly enhance the process of sustained drug delivery. Arthroscopic instrumentation can be used to insert the beads into the joints of standing horses.

Medical therapies for intra-articular drug therapy in the future may focus on prevention of *S. aureus* adhesion. A prominent feature of *S. aureus* virulence is the production of a bacterial surface marker that recognizes adhesive matrix molecules in collagen, precipitating bacterial adherence.[70,71] Monoclonal antibody, receptor antagonists, and vaccination challenges reduce the risk of obtaining infection with *S. aureus* in experimental animals.[70-72] These therapies may also be useful in the horse, since *S. aureus* is the most common cause of joint infection in adult horses.

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CHAPTER • 67

Non-Infectious Arthritis

Alicia L. Bertone

Non-infectious arthritis is characterized by pain, heat, swelling (effusion), erythema, and lameness. Joint structure and function, aspects of diagnosis and management of joint disease, and osteoarthritis are discussed in Chapters 63 and 64. This chapter focuses on normal and abnormal joint physiology and other specific non-infectious joint diseases.

JOINT PHYSIOLOGY

Fluid flow from the vascular space into the interstitium and joint space (third compartment space) and out into venules and lymphatics is tightly governed by Starling forces, which are a balance of arterial and venous pressures and colloid osmotic forces across the joint. The resultant fluid flow through the joint is modified by permeability of the synovium (osmotic reflection coefficient) and the vessel surface area available for fluid transport (filtration coefficient). Even in normal joints these forces are influenced by gravity (joint dependency), motion (exercise), and structure (joint compliance).[1-7]

Horses are unique in needing joint motion to maintain isogravimetric states of the joints (no fluid gain or loss), especially in peripheral joints.[1] In normal stationary equine limbs, lymphatic drainage from joints approximates zero until joint pressure exceeds 11 mm Hg for the fetlock joint (transitional microvascular pressure). In standing animals without counter forces, such as motion or external bandages to increase lymph flow forces, gravitational pressures increase arterial pressure to the joint and venous and lymphatic pressure from the joint. The result is tissue edema and joint effusion.

JOINT PATHOPHYSIOLOGY (SYNOVITIS)

The balance of these forces is altered in horses with synovitis because of the increased blood flow, altered synovial permeability, structural joint capsular changes, and loss of joint motion because of pain.[1,3,5,8,9] These factors profoundly affect joint physiology, contribute to clinical signs, and result in damage to the synovium and articular cartilage. Early signs of effusion that precede clinical detection of inflammation are related to changes in the hemodynamics of the joint, including increased blood flow and reduced joint motion.[8,9]

In chronic arthritis, capsular thickening, fibrosis, and altered synovial function likely influence fluid dynamics. Capsular fibrosis and joint enlargement produce decreased tissue compliance and an increase in intra-articular pressure occurs, even with slight increases in joint effusion. Articular cartilage is avascular and depends on synovial fluid for nutrition, so alteration in fluid flux affects nutritional exchange between the articular cartilage and synovium. Reduced nutrition exchange to articular cartilage exacerbates articular surface fibrillation and degenerative changes.[10,11]

JOINT PATHOLOGICAL CONDITIONS ASSOCIATED WITH ARTHRITIS

Effusion

Increased blood flow and capillary leakage occur early and contribute to increased fluid volume in the joint interstitium and synovial fluid, recognized as effusion. The condition is stimulated by vascular changes and neurotransmitter release, particularly β_2-adrenergic stimulation.[2,8,9] Because of low intra-articular pressures in normal equine joints (–5 mm Hg[6]; –1.25 mm Hg[12]), effusion precedes interstitial edema, until intra-articular pressure is greater than 11 mm Hg.[1] These physiological circumstances make detection of effusion one of the most sensitive indicators of early joint stress. Effusion, although common, is not normal and alters joint function. Congruent motion of joint surfaces depends on normal negative pressure and is important in decreasing shear force (side to side, sloppy movement).[7] Effusion is not painful as long as capsular tension is normal. Through the phenomenon of creep-relaxation, capsular tension is reduced in distended joints. Normal joints are relatively compliant and can accommodate fairly large changes in fluid volume with minimal increase in pressure, but elastance profiles of these joints may be permanently altered.[13] Joints with high structural congruence, such as the tarsocrural joint, are less affected biomechanically by joint effusion than are less congruent joints.[7]

High synovial fluid volume becomes more important during joint motion. Joint pressure profiles are profoundly altered by joint angle.[6,7,13] With effusion, as initial intra-articular pressure is increased, the capacity to accommodate rapid changes in pressure goes down, causing a rapid rise in intra-articular pressure and capsular wall tension at extreme joint

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angles, resulting in sharp pain during maximal joint excursion and simultaneous reduction in synovial perfusion.[14] These effects could be present even if synovitis is not recognized clinically. The influence of intermittent synovial ischemia is not elucidated fully, but it may be important in diseases of synovial proliferation.

Inflammation

Inflammation causes a humoral (vascular) and a cellular response. In early arthritis, cells are up-regulated to produce inflammatory mediators. Gene transcriptional profiles may be unique to certain forms of arthritis and potentially could be used in the future to recognize and prevent arthritis. Up-regulation of genes causes protein production, initiation of the inflammatory cascade, and further release of inflammatory mediators. Important mediators include cytokines interleukin-1, interleukin-6, and tumor necrosis factor.[2-4,15,16] Secondary mediators such as the eicosanoids (prostaglandin E_2 and leukotrienes),[2-4,15] free oxygen radicals,[17] substance P,[3] and nitric oxide[3,4] are also pro-arthritic and amplify pain.

Cellular influx causes effusion and subsequent clinical recognition of synovitis. The degree of synovitis and articular cartilage damage are directly proportional to synovial fluid nucleated cell count in infectious and non-infectious arthritis. Neutrophils are highly migratory and can move into joint fluid within 3 hours after a chemotactic stimulus, such as interleukin-1, paclitaxel, or endotoxin.[2,8,9,15,18] These cells are damaging to surrounding tissue because they have destructive enzymes and alter synovial fluid environment. In non-infectious arthritis, nucleated cell count is usually less than 30,000 nucleated cells per microliter,[15] but values greater than 100,000 nucleated cells per microliter occur in autoimmune arthritis,[19] endotoxin-induced arthritis,[16,20] and early in reactive arthritis.

Ischemia

Intra-articular pressures greater than 30 mm Hg cause significant reduction in the perfusion of the synovium and fibrous layer of the joint capsule.[14] Pressures of this magnitude occur in horses with palpable effusion of the fetlock joint and are present when synovitis is prominent.[12] Pressures greater than 60 mm Hg can reduce blood flow profoundly, and pressures approximating 100 mm Hg can cause capsule joint rupture (carpus and fetlock[21]). Clinically, joint rupture is uncommon but was hypothesized to contribute to dorsal fetlock capsular thickening.[12] I have seen this as a rare occurrence in the plantar pouch of the tarsocrural joint and the palmar pouch of the antebrachiocarpal joint. Intra-articular pressure can reach high levels during maximal flexion and extension, particularly in horses with resting pressures greater than 30 mm Hg.[6] Ischemia has been hypothesized to be a component of synovitis particularly in the proliferative form.[22] Low oxygen tension stimulates angiogenesis and granulation tissue formation, resulting in fibrosis. In chronically inflamed joints, clubbed and thickened synovial membrane often is seen during arthroscopic surgery, supporting the concept of synovial ischemia.

Synovial Fluid Lubrication: Hyaluronan

Synovial cells produce hyaluronan. A protective layer of high concentration hyaluronan remains close to the surface of the synovium and the remainder diffuses into the joint space, creating the unique viscosity of synovial fluid.[3,7] Adequate hyaluronan concentration is critical to provide lubrication of synovial soft tissues, particularly synovial villi. During inflammatory synovitis, lubrication of the soft tissues is reduced because of dilution of hyaluronan and a reduction in hyaluronan production. Swollen, edematous villi cause an elevated coefficient of friction. In horses with mild effusion, hyaluronan concentration can be normal because increased production by synovial cells matches elevation in synovial fluid volume.[23] When inflammation increases, synovial cell production decreases and degradation of hyaluronan increases. In horses with severe joint inflammation or hemarthrosis, hyaluronan concentration may be negligible.

Pain in Arthritis

Sensory and motor innervation help maintain joint stability, and in the absence of these protective reflexes, severe arthropathy may develop.[24] When activated, the peripheral nervous system can initiate the major features of acute inflammation, such as vasodilation and effusion, and lower the threshold for pain.[25] The C and A nerve fibers responsible for pain sensation in arthritis are activated by amines (such as serotonin) and neuropeptides (calcitonin gene-related peptide and substance P) that also act locally to exert pro-inflammatory effects on synovium. A role of substance P in joint pain is supported by the clinical effectiveness of the substance P depleting substance, capsaicin. Capsaicin initially activates C fibers, resulting in substance P release and pain, but subsequently desensitizes or causes degeneration of C fibers.[26]

The contribution of neuropeptides may be different in acute and chronic inflammatory arthritis. Edema formation in denervated limbs may indicate that loss of sensory innervation could play a role in acute arthritis.[27] Increased edema formation and decreased permeability to macromolecules were observed in denervated limbs subjected to interleukin-1 induction of synovitis.[2] The role of innervation in chronic arthritis is complex, because the neuropeptide substance P and calcitonin gene-related peptide were increased in sciatic nerve, dorsal root ganglia, and peri-articular tissues but were decreased in synovium.

The therapeutic implications are intriguing. Intramuscular gold or topically applied capsaicin could selectively destroy C fibers, thus lowering substance P levels, and these have been found to be clinically useful. Non-steroidal anti-inflammatory drugs (NSAIDs) decrease prostanoid production, and intra-articular corticosteroids inhibit the arachidonic acid cascade, thus having direct and indirect effects.[28] In addition, stimulation of primary afferent nociceptive fibers causes release of glutamate and substance P from central spinal pathways. This nociceptive input can be inhibited by stimulation of proprioceptive and tactile type I and II fibers. Stimulation of these fibers can be accomplished by high-frequency, low-intensity transcutaneous neural stimulation, frequently used in physiotherapy.

AUTOIMMMUNE-MEDIATED ARTHRITIS

Rheumatoid arthritis is a steroid-responsive arthritis, associated with high synovial nucleated cell counts, progressing to bone erosion and pannus formation. To establish a diagnosis of rheumatoid arthritis, an autoimmune component and production of rheumatoid factor must be documented. According to these criteria rheumatoid arthritis has not been reported in horses. In human systemic lupus erythematosus (SLE), systemic disease is also present and auto-antibodies are directed toward nuclear cellular material. An SLE-like disease was described in a young horse.[29] In horses, anti-collagen type II antibodies and immune complexes were identified in synovial fluid of horses with osteoarthritis and joint trauma. However, these immune complexes are much less common in horses with mild synovitis and have been found in sera. Relationship of cause and effect of these immunological findings is unclear, because immune complexes were found in many disease types. Although these auto-antibodies may be associated with equine diseases, it is unlikely that they initiate arthritis in horses.[30] They may develop after exposure to type II collagen,

after articular cartilage trauma or wear. Specific assays of synovial fluid for immunoglobulin M–rheumatoid factor (a feature of rheumatoid arthritis), antibodies to heat-shock protein, and anti-nuclear antibodies (ANA), a feature of SLE, revealed only modestly low levels of rheumatoid factor without correlation to disease and no ANA.[31]

IMMUNE-MEDIATED ARTHRITIS

The presence of synovitis and immunoglobulin G complex deposition in the synovium of foals has been reported.[32-34] This form of synovitis is called immune-mediated arthritis and may be associated with circulating immune complexes formed as a result of systemic disease.[32] Using specific monoclonal antibody techniques, immune-mediated arthritis was diagnosed in a 6-week-old pony foal infected with equine herpes virus-4.[33] Three horses were hyperimmunized with *Streptococcus* equine M protein vaccine and subsequently injected intra-articularly with purified streptococcal M protein. Severe suppurative synovitis developed, and synovial fluid nucleated cell counts were greater than 100,000 cells per microliter.[34] Eosinophils were prominent in the synovial fluid and synovial membrane in two horses.

A clinical syndrome of polysynovitis and vasculitis secondary to high circulating M protein (after streptococcal infection), or associated with *Rhodococcus equi* infections, is recognized and thought to be caused by immune-mediated arthritis. Seventeen (35%) of 48 foals with *R. equi* pneumonia infection had chronic active non-infectious arthritis. Pathogenesis involves immunocomplexes in the synovium. The hallmark of immune-mediated arthritis in foals is effusion in one or more joints but minimal lameness.[35] Synovitis often resolves in several weeks with or without treatment. Foals should be restricted to box rest, but no other specific treatment is necessary. Corticosteroids are contraindicated, because bacterial infection may be perpetuated.

REACTIVE SYNOVITIS

Reactive synovitis may occur after intra-articular injection of any product. Any intra-articular injection incites at least mild synovitis. The activated drug or a product in the solution may chemically induce reactive synovitis. Endotoxin contamination of multiple-dose vials or even single-dose products may cause reactive synovitis. In the case of a multiple-dose vial, a suspicion of endotoxin contamination should be high if more than one horse shows clinical signs within a short period. Horses are exquisitely sensitive to endotoxin, and concentrations above 0.125 ng per joint incite synovitis.[36] After intra-articular injection of methylprednisolone acetate, inflammatory cells surrounding vehicle crystals were identified in synovium 6 weeks later.[37] Reactive synovitis associated with methylprednisolone acetate may be most common in the distal interphalangeal joint. Although unusual, within a few hours after injection, horses can show severe lameness. Steroid arthropathy may be a form of reactive synovitis. The distal interphalangeal and tarsocrural joints appear most at risk to develop reactive arthritis after intra-articular injection of polysulfated glycosaminoglycans.[38]

Reactive synovitis must be distinguished from early infectious arthritis. Distinguishing features of reactive arthritis include early onset after injection (about 24 hours), synovial nucleated counts less than 30,000 cells per microliter, and resolution within 1 to 3 days. Lameness ranges from mild to severe, and in some horses distinguishing reactive arthritis from infectious arthritis may be difficult, and prompt management with intra-articular lavage, systemic and local antimicrobial drug administration, and anti-inflammatory therapy should be instituted. Culture and susceptibility testing should be performed if any suspicion exists that a bacterial infection is present, if synovitis does not resolve quickly, or in horses in which lameness persists.

Eosinophilic synovitis is rare and may represent an allergic reaction to an injected product, or to parasite migration, or could be truly idiopathic.[34,39] Joint lavage to assist in removing foreign material, and the administration of non-steroidal anti-inflammatory medication and anthelmintic treatment are indicated.

Foreign bodies rarely may be present within a joint or tendon sheath and incite chronic reactive synovitis. Broken needles, plant or seed awns or thorns, and debris from nearby wounds can cause reactive synovitis. Radiographic and ultrasonographic examinations can be helpful to identify the nature of the foreign material.[40] Cellulitis close to a joint may cause reactive synovitis that resolves with successful treatment of the primary infection.

TRAUMATIC SYNOVITIS

Primary traumatic synovitis is an early form of osteoarthritis. Horses at risk are usually in active sports training. Lameness usually is managed by intra-articular and systemic medication. Early medical intervention and appropriate joint rest and physiotherapy are critical to prevent loss of glycosaminoglycan from articular cartilage and permanent joint wear. Early loss of articular cartilage proteoglycan is reversible with medication and joint rest. If training is continued, some horses will develop proliferative synovitis, chip fractures, intra-articular ligament injury, and osteoarthritis. Intermittent hemarthrosis (see below) may be detected with primary traumatic synovitis, but often it indicates injury to subchondral bone, such as chip fracture or cartilage elevation.

PROLIFERATIVE (VILLONODULAR) SYNOVITIS

Chronic traumatic synovitis and continued exercise result in a painful thickening of the synovium, proliferative synovitis, particularly in areas of compression trauma.[41-45] The most common location is the dorsal fibrous pad (synovial pad) of the metacarpophalangeal joint, directly under the broad, flat common digital extensor tendon and joint capsule.[41] At maximal extension and flexion, pad compression results in intrasynovial hemorrhage, granulation tissue formation, fibrosis, and mineralization. Diagnosis and management are discussed in Chapter 37.

Chronic proliferative synovitis is a frequent finding in the carpal and tarsocrural joints during arthroscopic examination. Diffuse proliferative synovitis can be seen in horses that have had frequent intra-articular injections and have continued in exercise. Capsular fibrosis and loss of fine villous architecture occur. I do not recommend radical synovectomy, but removal of fibrotic tufts of capsule and synovium prone to pinching or demonstrating signs of internal hemorrhage and edema is warranted.

See Chapters 56 and 58 for a discussion of osteochondrosis.

IDIOPATHIC ARTHRITIS

Synovitis can occur without any known cause or associated trauma and can be truly idiopathic, although synovitis may be related to circulating toxins, including endotoxin, streptococcal cell wall, M protein, and viruses. In people, bacterial and viral deoxyribonucleic acid and bacterial peptidoglycans have been located in joints of patients with early rheumatoid and other non-infectious arthritides.[46,47] In horses synovitis can be associated with vasculitis, such as that seen with equine viral arteritis.

HEMARTHROSIS

Bleeding into a joint (hemarthrosis) causes joint capsule distention, severe pain, and lameness.[48] Draining the blood from the joint usually results in rapid relief of clinical signs. The cause of hemorrhage may be trauma to proliferative hemorrhagic synovium, an intra-articular fracture, or tearing of an intra-articular ligament. Hemorrhage may occur on a single occasion or may be recurrent. Diagnosis of hemorrhage is simple, by arthrocentesis, but identification of the primary cause can be more difficult. In the absence of radiological abnormalities, exploratory arthroscopy is warranted in a horse with recurrent episodic severe lameness associated with hemarthrosis. Hemorrhage associated with proliferative synovitis may be managed successfully by subtotal synovectomy.

LYME DISEASE

Although strictly speaking an infectious disease, Lyme disease is discussed in this section because it should be considered a differential diagnosis in tick-endemic areas in a horse with shifting limb lameness associated with synovitis in several joints.[49,50] However, many clinically normal horses have antibody titers to *Borrelia burdorferi*, and high titers are not synonymous with clinical disease. True Lyme disease is poorly documented in the horse, and definitive diagnosis would require identification of significantly raised titers in paired serum samples.

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CHAPTER • 68

Other Joint Conditions

Chris E. Kawcak and Gayle W. Trotter

Several types of joint diseases are rare and have been presented only in case reports or small retrospective studies. This makes generalizations about these diseases difficult and searching for information a tedious process.

DISEASES OF SOFT TISSUES OF THE JOINT

Soft tissue structures serve to support the joint, and disease can result in abnormal stresses to the articular cartilage and hence chronic progression of articular cartilage degeneration and osteoarthritis (see Chapter 63). Preoperative diagnosis of some soft tissue injuries is difficult, often leaving the surgeon with little to do other than evaluate the injury and debride fibrillated tissue during arthroscopic surgery. With the advent of newer diagnostic techniques such as magnetic resonance imaging (see Chapter 21) and increased use of ultrasonography (see Chapter 17), soft tissue injuries can be characterized better preoperatively.

Most soft tissues of joints, such as ligaments, menisci, and joint capsule, function to support joints. Joint ligaments maintain alignment of opposing and adjacent bones in joints. They form a connection between opposing bones (such as the tibia and femur in the stifle) and between adjacent bones in more complex joints (such as between the carpal bones in the carpus). They function to maintain alignment and allow for movement of the joint. The meniscal cartilages function as cushions between the tibia and femur of the femorotibial joints. Proper material characteristics are necessary for maintenance of the joint environment. Joint capsule is also responsible for maintaining joint support and provides a barrier for synovial fluid. Because the capsule surrounds the entire joint, appropriate elasticity is needed to maintain joint flexibility (see Chapter 67).

Ligament Injuries

Joint ligament injury can be an incidental finding during surgery or a devastating cause of lameness. For instance, medial palmar intercarpal ligament damage can be an incidental finding during arthroscopic surgery and has been seen incidentally in necropsy specimens (see Chapter 39).[1]

Injury to cruciate ligaments and menisci of the stifle usually cause significant pain (see Chapter 47), but cutting the cranial cruciate ligament in an attempt to create a model of osteoarthritis did not lead to osteoarthritis in horses. Therefore further destabilization of the joint beyond cruciate injury must usually occur, with possible damage to other soft tissue structures, including the joint capsule.

Collateral ligament injury caused by a bad step or laceration can result in subtle or severe lameness. We have seen low-grade lameness in a horse that had complete tearing of the medial collateral ligament of the metatarsophalangeal joint (Fig. 68-1). Laxity of the joint was detected by manipulation and stressed radiographs. The joint was immobilized in a cast for 6 weeks, followed by a splint for an additional 6 weeks. Reinjury occurred after splint removal and the joint was recast. After immobilization for 16 weeks the injury healed well enough for trail riding.

Ligament injuries usually heal slowly, and gradual return to function is needed to strengthen the tissues. The conse-

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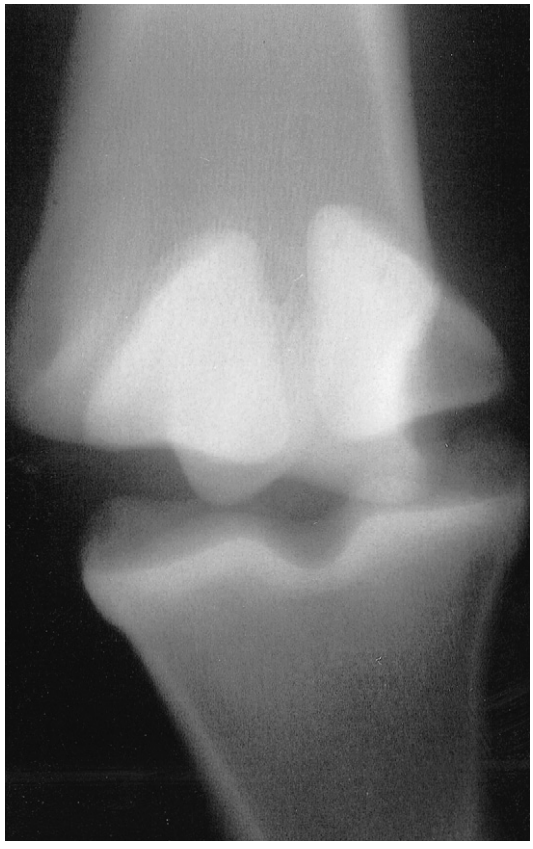

Fig. 68-1 A 3-year-old Paint gelding had acute swelling on the medial aspect of the metatarsophalangeal joint. A stressed dorsoplantar radiograph shows subluxation of the metatarsophalangeal joint caused by complete rupture of the medial collateral ligament.

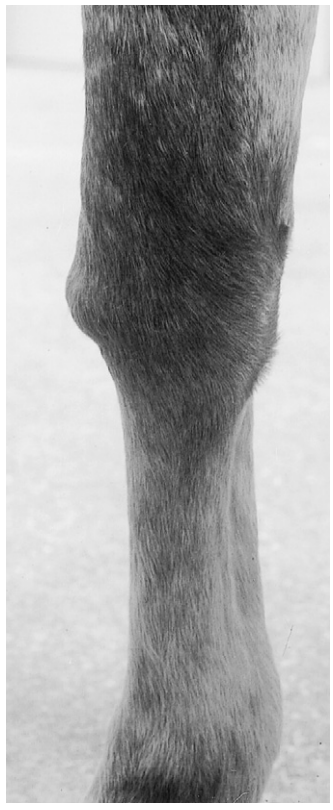

Fig. 68-2 A synovial hernia on the dorsum of the carpus.

quences of collateral ligament injury depend on the severity of damage to the ligament, contamination in the joint, and damage to other structures in the joint and may be difficult to predict at the time of injury. Long-term instability may place abnormal stresses on the joint and lead to progressive osteoarthritis. Cutting the lateral collateral and lateral collateral sesamoidean ligaments of the metacarpophalangeal joint resulted in lameness, increased joint circumference, decreased range of motion, and osteophyte formation in 8 weeks, resulting in osteoarthritis.[1]

Hygroma

A hygroma is an adventitious or acquired bursa on the dorsum of the carpus caused by trauma from falling, getting up and down, or hitting a fence; or by chronically pawing and hitting the dorsum of the carpus.[2-4] Non-painful, fluctuant, uniform soft tissue swelling occurs on the dorsal aspect of the carpus. Pressure does not induce swelling in any associated joints or tendon sheaths. Range of motion of the carpus may be reduced, but lameness is unusual. Injection of radiopaque contrast agent into the hygroma confirms its extra-articular position.

Spontaneous resolution of hygromas may occur, but treatment is often necessary. Drainage and injection of anti-inflammatory agents has been used with varying success; in many horses repeated injection is necessary.[3,4] We have seen spontaneous resolution after injection of the contrast agent. Injection of atropine (7 mg) may resolve the swelling. Owners should be warned that bandaging is an essential component of treatment and that long-term chronic thickening may occur.

Other treatments include incisional drainage; injection of irritants, such as iodine or Lugol's solution; and blistering,[3,4] but contrast radiography should be performed to ensure that the hygroma is an isolated structure. Although preoperative contrast radiographs may show no communication between a hygroma and a joint or tendon sheath, one might exist in the form of a one-way valve from the joint into the mass.[5] Drainage of the mass with a Penrose drain and bandaging has been used successfully for treatment of recurrent hygromas. Drains usually are removed once drainage stops, 2 to 7 days after placement.[6] Surgical excision can be performed in horses with chronic hygroma and is best accomplished if the fluid sac is left intact and dissected from the other tissues (see Chapter 39).[7] Soft tissue and skin closure are routine, and a splint can be used to prevent flexion for better healing. Prognosis for resolution of hygroma is often good, although some degree of thickening usually persists.

Synovial Hernia

A synovial hernia is a defect in a joint capsule or tendon sheath through which the synovial membrane can protrude. The condition rarely causes lameness but is a cosmetic blemish. A well-defined, round, soft tissue mass can be palpated over a joint, and with palpation fluid often can be moved between the hernia and the underlying joint or tendon sheath (Fig. 68-2). The hernia may disappear with joint flexion. Contrast agent injected into the hernial sac is detected in the underlying joint or tendon sheath, although a one-way valve may be present, limiting movement of contrast material. If the synovial hernia is of cosmetic concern, surgical excision can be performed, with a good prognosis for soundness provided no other joint diseases are present.[8]

Ganglion

A ganglion is defined as a fluid-filled structure that connects to a joint or tendon sheath through a one-way tract from the joint into the mass.[2] Unlike a synovial hernia, the mass lacks a synovial lining and often is filled with mucin. Ganglions are rare in

the horse, but common in people, and they have been reported around the stifle and the carpus.[2] A ganglion adjacent to the fetlock was associated with lameness that was alleviated by regional analgesia and after surgical excision of the mass.[9] However, communication with the tendon sheath and joint was not demonstrable by ultrasonography. Demonstrating connection between a ganglion and an adjacent joint by injection of radiographic contrast agent into the mass may or may not be possible.[6]

Synovial Fistula

Synovial fistulae are communications between two synovial structures, usually a joint and tendon sheath. They have occurred between the antebrachiocarpal joint and the common digital extensor tendon; the middle carpal joint and the extensor carpi radialis tendon sheath or the common digital extensor tendon sheath; the proximal interphalangeal joint and long digital extensor tendon sheath; and the extensor carpi radialis tendon sheath and a carpal hygroma.[5,10-12] Additional joint damage is often present in association with the fistula, causing lameness referable to the area.

Swelling in the joint and nearby tendon sheath occurs, and fluid is often movable between the structures (Fig. 68-3). Radiography may reveal additional joint or tendon sheath damage, and contrast agent injected into one of the structures is visible in the other.[10]

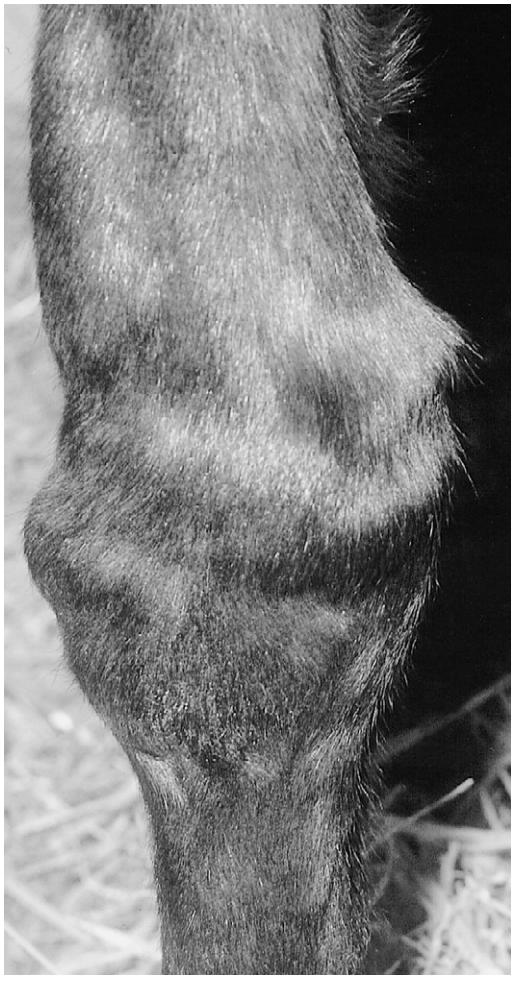

Fig. 68-3 Dorsal view of carpus with effusion of the antebrachiocarpal joint and common digital extensor tendon sheath. A synovial fistula was seen during arthroscopic surgery.

Occasionally a fistula can be seen during arthroscopic surgery, but closure of the fistula requires an arthrotomy. However, arthroscopic surgery for treatment of a primary problem, without repair of the fistula, has resulted in resolution of lameness, without resolution of the swelling. We do not close these fistulae unless a cosmetic effect is important, if the swelling itself is impeding performance, or if medical therapy fails to alleviate lameness.

NEOPLASIA

Joint-associated tumors in horses are rare and consequently behave unpredictably, and relying on treatment information from other species is difficult. Soft tissue tumors in horses are vascular, fibrous, or synovial in origin. Benign vascular masses such as hemangiomas have been seen in carpal and digital tendon sheaths of horses.[2] The tendon sheaths were distended with blood-stained fluid, but no associated lameness occurred, and surgical excision of the masses was curative.

Fibromas may occur as slow-growing masses near the tarsus, stifle, and distal radius. These masses are rarely erosive to associated bone. Complete surgical excision may be curative, but incomplete excision can result in recurrence.[11,13] A fibroma on the proximal lateral aspect of the tarsus was incompletely resected, and over a 4-month period the mass regrew to larger than its original size.[14]

Villonodular (proliferative) synovitis is a common traumatic injury on the proximal dorsal aspect of the metacarpophalangeal joints of racehorses and is not a tumor (see Chapters 36 and 67). Keratinization of a villonodular synovitis was associated with severe lameness.[15] It was suggested that this was a form of epidermal inclusion cyst, the result of inadvertent introduction of epidermal tissue into the joint after repeated arthrocentesis for previous infectious arthritis, resulting in a foreign body reaction. Surgical resection resulted in resolution of lameness. Pigmented villonodular synovitis has occurred in the metatarsophalangeal and femoropatellar joints resulting in chronic lameness in a mule.[16]

Synovial cell sarcomas have been identified in the antebrachium,[14] a digital sheath,[17] and proximal interphalangeal joint associated with soft tissue swelling and variable lameness. The masses had infiltrated the soft tissues and caused localized inflammatory bone loss because of pressure. Recurrence may occur after surgical excision.[18] Chondrosarcomas are rare, but they have been described in a metacarpophalangeal joint[19] and the carpal region,[20] associated with expansible radiolucent lesions.

A hemangiosarcoma occurred in the tarsal sheath, and surgical excision resulted in relief of lameness.[21] A secondary melanosarcoma in a shoulder joint caused severe lameness.[22]

OSTEOCHONDROMATOSIS

Synovial chondromatosis and osteochondromatosis describe pieces of uncalcified and calcified hyaline cartilage, respectively. Synovial chondromatosis is a disease in which hyaline cartilage can occur in the joint in pedunculated form, within the synovial membrane, or free within the joint. Osteochondromatosis results when endochondral ossification of the mass occurs, often making it difficult to differentiate from osteochondral fragmentation. Osteochondromatosis has occurred in the femorotibial joint.[2,23] Secondary osteochondromatosis also occurs within joints with osteoarthritis. The condition is often painful in people, and surgical removal is indicated. Osteochondromatosis has occurred incidentally in horses without lameness.

CALCINOSIS CIRCUMSCRIPTA

For a discussion of calcinosis circumscripta, see Chapter 47.

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CHAPTER • 69

Pathophysiology of Tendon Injury

Roger K.W. Smith

FUNCTIONS OF TENDONS AND LIGAMENTS

Tendons passively *transfer force* generated by muscle to bony attachments on the opposite side of a joint, or joints, to provide movement. In contrast the function of a ligament is to resist distraction of its two bony attachments (e.g., collateral ligaments and suspensory ligament [SL]). Although this function is true for most tendons and ligaments, the horse has evolved its digital flexor tendons and SL to exhibit additional functions. These tendons and ligaments, situated on the palmar aspect of the equine distal limb, receive large weight-bearing loads because of the hyperextended metacarpophalangeal and metatarsophalangeal joints. As a result the tendons and ligaments on the palmar aspect act to *support* the metacarpophalangeal and metatarsophalangeal joint during normal weight bearing.

In addition, the equine digital flexor tendons exhibit considerable elasticity that is used to *store energy* for energy-efficient locomotion.[1] In the case of the superficial digital flexor tendon (SDFT), its muscle is highly pennate (the muscle fibers are arranged at an oblique angle to the line of pull of the muscle, which maximizes power and minimizes contraction distance) and is unable to contract by more than a few millimeters. Therefore the action of the muscle, together with its accessory ligament, is largely passive to fix the origin of the SDFT in space. Although the muscle contracts only a short distance, its action, together with the tendon elasticity, also provides *shock absorption*. The gait of a horse at speed can be compared with a weight (the horse's body) bouncing up and down on elastic springs (the digital flexor tendons) in a similar fashion to a pogo stick.[2] This arrangement allows horses to reach and maintain high speeds while minimizing energy expenditure.

FUNCTIONAL CHARACTERISTICS

Biomechanical Properties

Stress-Strain Curves

Stress-strain curves for the tendon in which the force per unit area (stress) is plotted against the percentage of elongation (strain) can be used to provide values for the elastic modulus of tendon. Variation in the slopes between tendons and tendon sites occurs, but Fig. 69-1 shows a simplified stress-strain curve for tendon. The curve has four regions:

1. The toe region, where stretch to the tendon is nonlinear. This is associated with the elimination of the undulating pattern of the collagen fibrils (known as *crimp;* see the following discussion).
2. Linear deformation, which is this area of the curve from which the modulus of elasticity is determined and that characterizes the elastic stiffness of the tendon. The mechanism for this elongation is unclear, but it involves elongation of the collagenous network, with possible movement of collagen fibrils or fascicles relative to one another. A tendon is not homogeneous, and what parts of the tendon are responsible for this linear deformation is unclear.
3. Yield region, in which irreversible lengthening of the tendon occurs at these strains, possibly arising from covalent cross-link rupture and slippage of collagen fibrils.
4. Rupture, in which the stress falls quickly to zero as the collagen cross-links or fibrils sequentially rupture.

Biomechanical Parameters

A number of simple biomechanical parameters can be ascribed to tendons that are derived from its stress-strain characteristics: ultimate tensile stress (force per unit area at the point at which the tendon breaks), ultimate tensile strain (the percentage extension of the tendon at its breaking point), and the modulus of elasticity (E, a constant determined from the ratio of stress to strain for the linear part of the stress/strain curve). Some of the values of these parameters for the palmar supporting structures of the distal limb are shown in Table 69-1. A parameter not frequently calculated, but probably more relevant to the in vivo situation, is the force/stress at the yield point, after which irreversible damage is occurring.

Ultimate tensile force/stress. The SDFT receives in excess of 1 metric ton of weight (10 kN) at maximum weight bearing on a structure only about 1 cm^2 in cross-sectional area. The ultimate tensile stress (force at failure per area) for the SDFT (rupturing at the mid-metacarpal region) in the horse is therefore close to 100 MPa, which is at the upper limit of previously documented figures for other species (45 to 125 MPa).[3,4]

However, within any population of horses, large variation occurs in the ultimate tensile force, with up to a twofold dif-

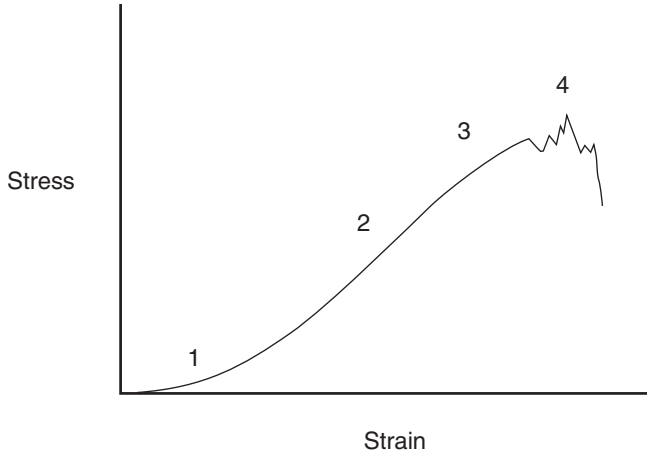

Fig. 69-1 Simplified stress-strain curve for tendon. *1*, Toe region; *2*, linear deformation; *3*, yield, *4*, rupture. (From Goodship AE, Birch HL, Wilson AM: The pathobiology and repair of tendon and ligament injury, *Vet Clin North Am Equine Pract* 10:323, 1994.)

ference between the weakest and strongest tendons.[5] This variation also exists for ultimate tensile stress,[6] indicating that the variation is not just due to differences in cross-sectional area. It is hypothesized that the horses with weaker tendons are more prone to tendon injury.

Modulus of elasticity/stiffness. The structural stiffness of a tendon is the force required to extend it by a unit length. This material property, the modulus of elasticity, is derived from the linear portion of the stress-strain curve for tendon, and for the SDFT it is about 1000 Mpa. It is normally correlated with ultimate tensile stress, so that the stronger the tendon, the stiffer it is.

Ultimate tendon strain. In vitro testing of equine flexor tendons indicates that they usually extend by 10% to 12% of the original length before they rupture, although values of up to 20% have been reported.[7] However, the ultimate tensile strain reflects only the final strain before rupture and includes that yield portion of the stress-strain curve that represents irreversible damage to the tendon tissue (Fig. 69-1). In addition the ultimate tensile strain is not constant along the length of the SDFT in vitro,[8] with the highest ultimate tensile strain in the metacarpal region (the region most frequently injured).

In vivo, the normal strains in the digital flexor tendons (in ponies) are about 2% to 4% at the walk and 4% to 6% at the trot.[9] At the gallop in the Thoroughbred, maximum strains in the metacarpal region of the SDFT can reach 16%.[10] Such strains are far greater than usually expected in tendons from most species and reflect the highly specialized nature of the equine digital flexor tendon. If these high strains are truly representative of the strains within the tendon, they indicate that equine tendon is operating at or close to its ultimate tensile strain. This suggests little tolerance in the system, which would explain the high incidence of injury in this structure. However, some caution in the interpretation of in vitro measurements is necessary, because studies have shown different results obtained between in vivo and in vitro tests.[9]

Hysteresis

Hysteresis refers to the energy loss between the loading and unloading cycles of tendon (Fig. 69-2), determined from the area between these two curves. Hysteresis is usually about 5% in equine tendons.[11] Some of this energy is responsible for the rise in temperature within the tendon core associated with repeated loading (as in an exercising horse), which has been suggested as a causative factor in equine superficial digital flexor tendonitis[12] (see the following discussion).

Classification of Tendons and The Relationship to Function

Recent research has demonstrated that tendons possess different properties depending on function. The tendons in the horse, like those in people, can be divided into two broad categories: those with the primary function of withstanding the weight of the horse (weight-bearing tendons) and those with the primary function of flexing, extending, or rotating joints (positional tendons). Weight-bearing tendons, such as the equine digital flexor tendons, are more elastic than positional tendons (e.g., the equine digital extensor tendons), which reflects the function of the digital flexor tendons as elastic energy stores. Positional tendons require stiffness for accurate positioning of the limb or digit. Human finger tendons are stiff for such a purpose, and although equine digital extensor tendons are not required for accurate placement of the digit, they nevertheless resemble this category of positional

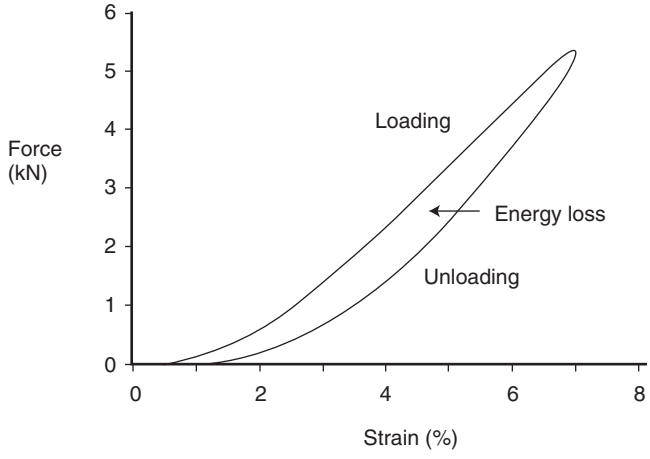

Fig. 69-2 Hysteresis loop for tendon. (From Goodship AE, Birch HL, Wilson AM: The pathobiology and repair of tendon and ligament injury, *Vet Clin North Am Equine Pract* 10:323, 1994.)

Table • **69-1**

In Vitro Biomechanical Parameters Quoted for the Palmar/Plantar Supporting Structures of the Equine Distal Limb

TENDON	ULTIMATE TENSILE FORCE (kN)	ULTIMATE TENSILE STRESS (MPa)	ULTIMATE TENSILE STRAIN (%)	E (MPa)	REFERENCE
SDFT	12.43	—	—	1096.5	63
	12.37	128.5	17.8	1188.9	67
	12.34	—	12.5	1189.0	8
	13.6 (range 9.5-20)				5
SDFT (HL)	—	—	12.3	1000-1282	11
DDFT	17.00	—	—	1585	63
	19.27	89	—	613	6
DDFT (HL)	—	—	10.0	738-1398	11
CDET	6.72	179	—	1523	6
ALDDFT	8.71		—	490	63
SL	17.15	—	10-12	1100	63
	17.43	78	—	510	6
SL (HL)	—	—	11.0	576-669	11

SDFT, Superficial digital flexor tendon; *HL,* hindlimb; *DDFT,* deep digital flexor tendon; *CDET,* common digital extensor tendon; *ALDDFT,* accessory ligament of the deep digital flexor tendon; *SL,* suspensory ligament.

tendons.[13] These differences in biomechanical properties are reflected in the anatomical features of the tendons.

ANATOMICAL STRUCTURE

Morphology of Tendons

Tendon is composed of a hierarchical structure of subunits. To the naked eye, in cross-section the tendon substance is divided into a number of fascicles, which are in turn composed of ever decreasingly sized subunits: fibers and then fibrils.

The fascicles are held together by the loose connective tissue, the endotenon, which is confluent with the outside of the tendon, the epitenon. The endotenon contains vascular and neural elements and separate cell populations, which may be a source of pluripotential cells. In regions where the tendons are not surrounded by a tendon sheath, a thick fibrous layer, the paratenon, further surrounds the tendon.

Crimp

In longitudinal section, under the light microscope, the collagen fibers in tendon have a wavy appearance known as crimp. This pattern is responsible in part for the elasticity of the tendon, and the toe region of the stress-strain curve when the mechanical behavior of the tendon is non-linear. A generalized reduction in the crimp angle occurs with aging, with a differentially greater reduction in the central fibers.[14,15] As the tendon stretches, the central fibers straighten first and therefore receive differentially greater load than the peripheral fibers, which may explain the site of pathological damage in those horses with centrally positioned core lesions. The reason for lesions situated peripherally at the tendon is less clear, unless these are also focal regions of the tendons that have developed atypically straightened fibers. Lesions involving the entire cross-section of the tendon represent a more generalized disruption of the tendon matrix.

Collagen Fibril Diameter

The collagen fibrils are composed of many triple helical collagen molecules arranged in a quarter stagger, which gives a characteristic banding pattern on electron microscopy. These collagen molecules are secreted through pores in the cell membrane as triple helical procollagen, which is subsequently assembled into fibrils extracellularly, by covalent intermolecular cross-linking (see the following discussion). Fusion of adjacent fibrils is responsible for the increasing size of collagen fibrils with age.[16,17] Foals at birth already have a bimodal or trimodal pattern of fibril diameters, and in the adult the fibrils can be grouped into two or three populations: small (40 nm), medium (120 nm), and large (>200 nm).[7]

Associated Structures

Blood Supply

Tendons obtain nutrients from two primary processes: perfusion and diffusion. Diffusion of nutrients from compartments other than blood occurs predominantly where the tendon is enclosed in a sheath, the synovial fluid playing an important role in tendon nutrition.

The principal blood supply in tendon arises from three sources: proximally, the musculotendonous junction; distally, the osseous insertion; and between these two, the tendon is supplied by intratendonous and extratendonous vessels. The extratendonous supply arises from the paratenon in extrasynovial tendon and from mesotenon attachments within synovial tendon sheaths (such as the vinculum between the fetlock annular ligament and the SDFT). The predominance of either source in the mid-tendon region depends on the species and the tendon. In the equine SDFT, two major parallel vessels run longitudinally in the lateral and medial borders of the mid-metacarpal tendon accompanied by an extensive anastomosing network of vessels.[18] These vessels anastomose with paratenon blood vessels, although removal of the paratenon blood supply in the horse failed to produce gross pathological damage. However, ligation of the intratendonous supply in the mid-metacarpal region produced ischemic pathological damage, demonstrating the importance of the intratendonous supply. The deep digital flexor tendon (DDFT) also has an anastomosing vascular network, except for its dorsal aspect as it passes over the metacarpophalangeal joint, where it has a more fibrocartilagenous phenotype to resist the compressive forces in this region.[19]

Tendon has been shown to have a good blood supply based on a number of techniques, usually involving clearance measurements of various radionuclides injected intratendonously (most commonly ^{133}Xe and ^{24}Na). The SDFT appears to have good blood supply similar to that of resting skeletal muscle, although findings have been inconsistent between studies and between animals on successive measurements. The large variation in the blood flow under different circumstances may indicate that external factors, as yet undefined, influence blood flow on a day-to-day basis.

Differences in blood flow between tendons are affected by age, exercise, and injury (Fig. 69-3).[20] The SDFT has a slightly higher blood flow than the DDFT, which reflects its good vascular anatomy (see the previous discussion). However, studies have shown similar functional blood flow throughout the metacarpal region of the SDFT although, histologically and microangiographically, the mid- and distal regions are less well vascularized.[21] However, not surprisingly, the DDFT in the metacarpophalangeal joint region has a significantly lower blood flow, associated with its fibrocartilagenous phenotype, with few blood vessels because of the high compressive forces in this region, which would limit any blood flow.

The blood flow appears to be considerably higher in foals than in adult horses, with a gradual decline in blood flow to the adult level by 3 years of age.

Exercise induces an increase in blood flow (about 200%), although this increase is delayed in animals not previously trained. The tendon blood supply therefore appears to exhibit a fitness memory.

Injury provokes a considerable increase in blood flow (>300%), which occurs in the clinically affected and the clinically unaffected limb, consistent with the bilateral nature of tendonitis in the horse, even though one limb is more severely affected than the other. Other measurements carried out in injured tendons have yielded variable results, which have been interpreted as representing the coexistence of fibrous tissue with low blood flow and hyperemic areas of acutely inflamed tendon.

Cellular Components

Although the biomechanical characteristics of tendon are determined by the composition and organization of the extracellular matrix, tenocytes are essential for the formation and maintenance of tendon tissue. At least three different populations of tenocytes are identifiable within normal equine tendon and ligament[7,22] (Fig. 69-4):

- Type I: Cells with thin, spindle-shaped nuclei
- Type II: Groups of cells with more rounded, thick, cigar-shaped nuclei
- Type III: Cartilage-like cells with round nuclei and visible nucleoli

The proportion of these cells varies between tendons and ligaments, with tendon site, and with age. Young tendon has considerably larger numbers of type II cells arranged between the collagen bundles. With aging, type I cells predominate, whereas in the areas subjected to compressive forces, type III cells can be identified.

The activity of these different cell types is unknown. The different cell types identifiable histologically may represent different cell lines or different states of extracellular matrix

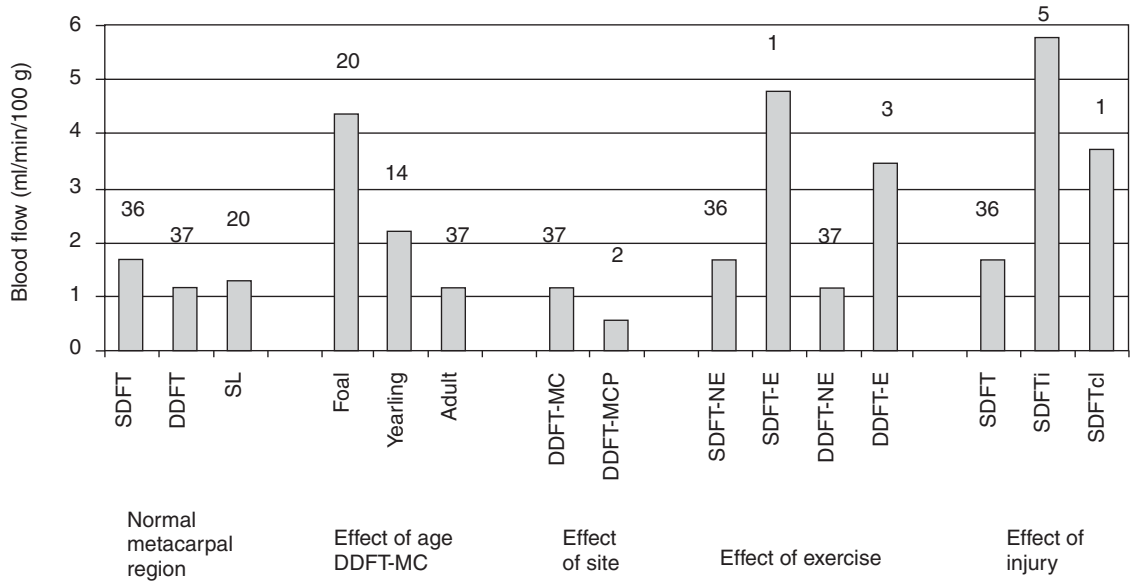

Fig. 69-3 Absolute blood flow in equine digital flexor tendons derived from ^{133}Xe clearance half-times. Numbers above the columns indicate the numbers of tendons evaluated. *DDFT,* Deep digital flexor tendon; *E,* exercised; *MC,* metacarpal region; *MCP,* metacarpophalangeal region; *NE,* not exercised; *SDFT,* superficial digital flexor tendon; *SDFTcl,* contralateral "normal" SDFT; *SDFTi,* superficial digital flexor tendonitis. (From Jones AJ: Normal and diseased equine digital flexor tendon: blood flow, biochemical and serological studies, PhD thesis, 1993, University of London.)

production. A reasonable assumption is to suppose that type II and III cells are metabolically more active and are responsible for maintaining the tendon extracellular matrix, although the metabolic activity of the type I cells cannot be discounted.

Ligament has a much higher cell population, with a predominance of type II cells arranged in columns. In the SDFT of the horse, which has greater numbers of cells during growth, total tendon cell numbers remain relatively constant after skeletal maturity. However, acellular areas develop, especially in the center of the SDFT in the metacarpal region, although the degree of acellularity is not particularly related to age.[23] More active, type II cells can be found surrounding the fibrils in these regions. Chondroid metaplasia can also be identified associated with these areas. Other areas, typified by the DDFT in the metacarpophalangeal region, also have acellular regions associated with a fibrocartilagenous phenotype (type III cells and cartilage-like matrix) as a result of concurrent compressive forces as the tendon wraps around the metacarpophalangeal joint. Other cells are associated with the tendon, namely the paratenon, epitenon, and endotenon fibroblasts and the synovial-like cells of the epitenon within the tendon sheaths. These cell populations may also play important roles in maintaining tendon tissue, especially because the endotenon harbors certain growth factors, such as transforming growth factor-β (TGF-β) (Fig. 69-5).[24]

The regulation of tenocyte metabolism still is not understood fully but probably relies on a combination of mechanical and cytokine stimuli. Tenocytes have been shown to sense and react rapidly to mechanical stimuli in vitro.[25] However, equine tenocytes in culture require the addition of a suitable growth factor to initiate a synthetic response to load.[26] Recently, the use of confocal microscopy has provided an insight into the relationship between tenocytes. Staining with a membrane dye has revealed extensive cytoplasmic extensions from tenocytes, which form a complex meshwork around the collagen bundles. Gap junctions have been shown to exist between these cytoplasmic extensions, which would provide an ideal arrangement for the coordinated biosynthetic reactions to mechanical stimuli.[27]

Of the multitude of growth factors having effects on connective tissues, TGF-β and insulin-like growth factor 1 have been investigated the most in equine tendon.[28,29] The synthesis and distribution of TGF-β isoforms in equine digital flexor tendon vary with age. The highest levels are observed in young equine digital flexor tendon, especially within the endotenon.[24] Levels decline after skeletal maturity, especially in the tendon fascicles themselves, and this may result in a relative lack of tenocyte synthetic activity after skeletal maturity. However, it is not yet clear which are the most fundamental growth factors in equine tendons and how the growth factor milieu acts to cause tendon matrix synthesis and repair.

Molecular Composition of Tendon Matrix

Tendons are composed predominantly of extracellular matrix, within which is a wide array of proteins, organized and interacting to produce the mechanical properties of tendon. The tendon extracellular matrix is composed predominantly of water (about 65% wet weight), collagen (about 30% wet weight), and non-collagenous glycoproteins (about 5% wet weight).

Collagen

About 80% of the dry weight of the tendon is collagen, of which the predominant collagen type is type I (>95%).[7] Type III collagen is present in the endotenon and appears to increase as the animal ages. Type II (the collagen of articular cartilage) is likely to occur at the same sites in the horse as described in other species; namely, tendon insertions and where tendons develop fibrocartilage-like tissue associated with a change in the direction of pull around bony prominences (e.g., at the metacarpophalangeal joint).

Collagen fibrils are strong, but the bonds formed between these fibrils and the higher order subunits are more likely to determine the strength of the tissue. The major covalent cross-link of type I collagen in tendon is between hydroxylysine and lysine residues.[30] Lysine and hydroxylysine are converted to the respective aldehydes by the action of the enzyme lysyl oxidase, which is inhibited by β-aminopropionitrile fumarate, a chemical that has been used in the treatment of equine

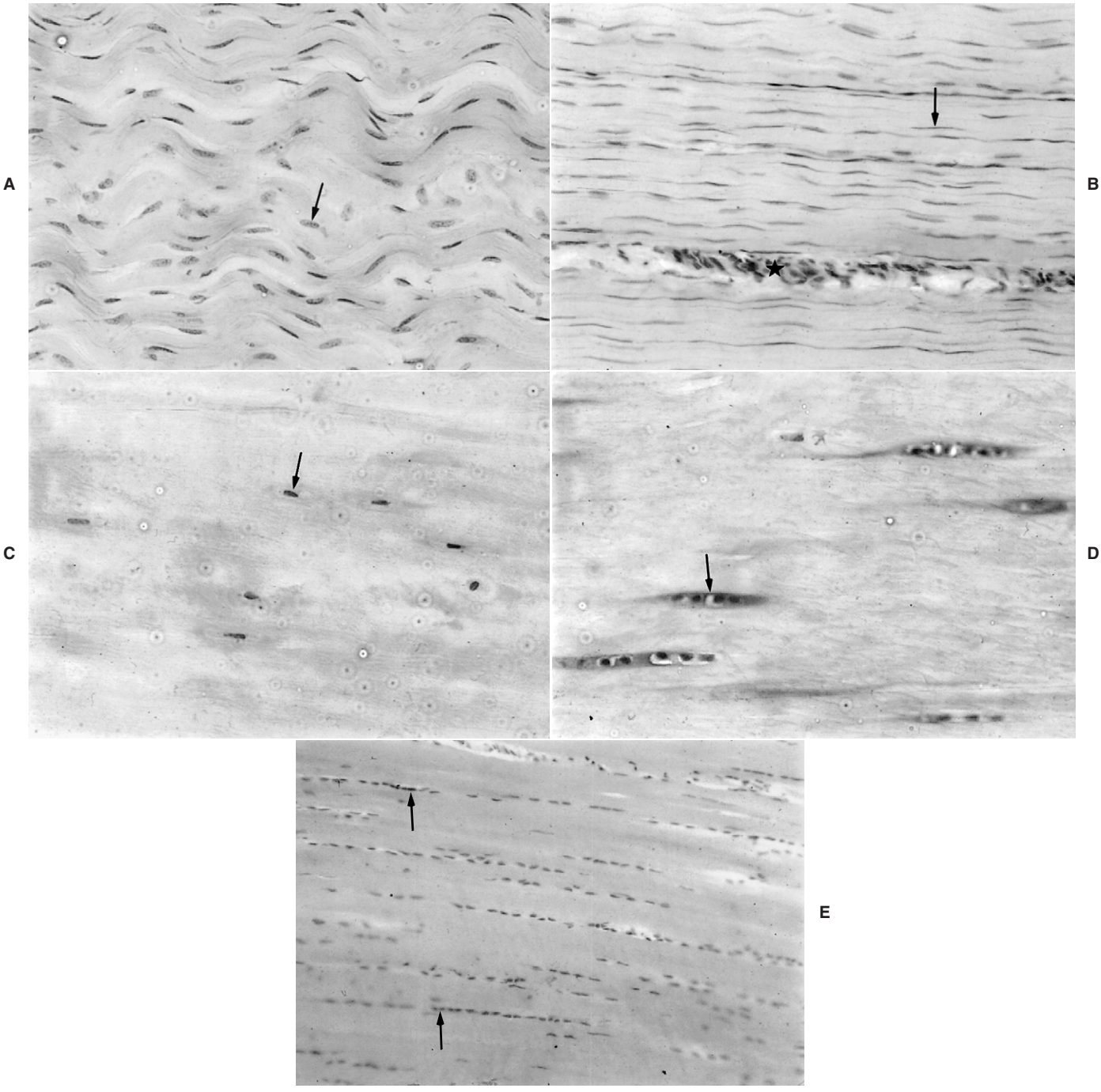

Fig. 69-4 Histological features of equine tendon and ligament. **A,** Foal superficial digital flexor tendon showing obvious crimp and predominantly type II cells (*arrow*). **B,** Young superficial digital flexor tendon showing reduction in crimp and increased number of type I cells (*arrow*). Note also the endotenon septa (*star*). **C,** Aged deep digital flexor tendon from the metacarpophalangeal region showing acellular regions and type III cells (*arrow*), resembling the chondrocyte phenotype associated with compressive loading in this region. **D,** Chondroid metaplasia (*arrow*) in an aged superficial digital flexor tendon. The acellular areas are visible between the regions of chondroid metaplasia. **E,** Suspensory ligament branch showing the lines of type II cells (*arrows*) characteristic of ligament.

tendon injuries. These lysine and hydroxylysine aldehydes then can form a number of different types of cross-links: reducible (e.g., dihydroxylysinonorleucine and hydroxylysinonorleucine) or non-reducible (hydroxylysylpyridinoline). The reducible cross-links become reduced with age so that at maturity their level is less than 10% of the level in the foal.[31]

Non-covalent cross-links (electrostatic in nature) are provided by the proteoglycans and other glycoproteins, especially the small proteoglycan decorin, which coats the collagen fibril. Although individually these cross-links are less strong than the covalent cross-links, the high number and involvement in the higher order organization of the collagen network

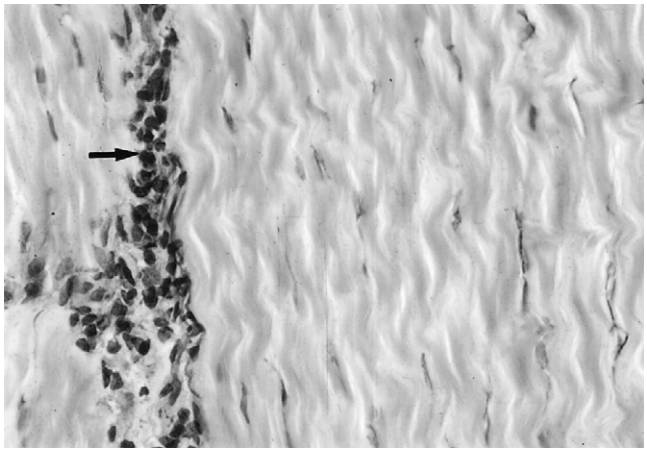

Fig. 69-5 Immunohistochemical staining for transforming growth factor-β3 in equine superficial digital flexor tendon. Note the concentration of stain (*arrow*) in the endotenon septa. (Courtesy Eddy Cauvin, Lyon, France.)

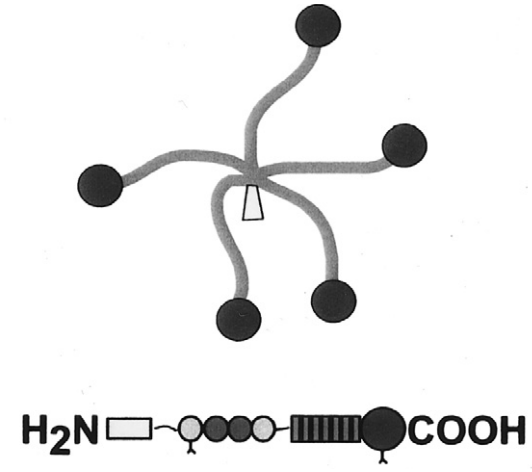

Fig. 69-6 Cartoon of the cartilage oligomeric matrix protein molecule. (Courtesy K. Rosenberg, Lund, Sweden.)

make them potentially major determinants of tendon mechanical properties.

Non-Collagenous Glycoproteins

Cartilage oligomeric matrix protein. The large cartilage oligomeric matrix protein (COMP) consists of five subunits, bound via disulfide bonds at their N-termini to form a five-armed protein, with a bouquet of arms with globular C-terminal domains that can interact with other matrix components[32,33] (Fig. 69-6). Although initially thought to be restricted in distribution to cartilage, COMP subsequently has been found largely in tissues whose function primarily is to resist load. Thus COMP is found in significant amounts in tendon, ligament, cartilage, intervertebral disk, and meniscus. In equine digital flexor tendons COMP shows large variation with site and age.[34] Levels are low at birth but accumulate rapidly associated with weight bearing. Levels peak in the metacarpal region of the SDFT (at about 3% dry weight of tendon) at skeletal maturity and subsequently decline (Fig. 69-7). Levels peak at a lower level in the metacarpophalangeal regions and in the DDFT but are maintained in the former.

The function of COMP has not yet been elucidated completely, but COMP is known to bind fibrillar collagens (I, II, and IX),[35,36] and a mutation in the human COMP gene is responsible for pseudoachondroplasia, characterized by lax tendons and ligaments, short stature, and early-onset osteoarthritis.[37,38] Although COMP may have a structural role, present research data suggest that it also may also act to bring collagen molecules together to form fibrils, and it may assist in the organization of the collagen network. This role may explain the decline in COMP levels after skeletal maturity in the metacarpal region, because the collagen matrix has been formed and limited remodeling occurs in the adult. Preliminary data on equine SDFT show a significant correlation between ultimate tensile stress and COMP levels at skeletal maturity.[39] Thus high levels of COMP during development potentially enable the formation of a high-quality tendon matrix.

Proteoglycans. Proteoglycans are a group of molecules that possess a protein core and a side chain of sugars (glycosaminoglycans, or GAGs). The sugar side chains are highly variable in type and length so that great diversity exists even within a given tissue. Work on a variety of soft tissues, especially articular cartilage, has demonstrated a large number of different proteoglycans that are vital to maintaining the structural integrity of the tissue by playing structural roles or by regulating the metabolism of the tissue.

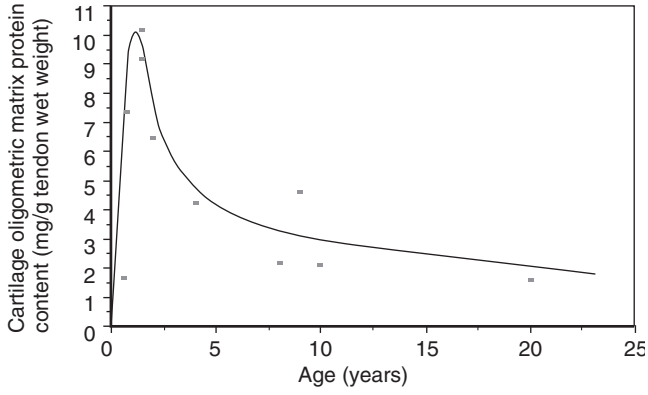

Fig. 69-7 The variation of cartilage oligomeric matrix protein levels with age in the metacarpal region of the superficial digital flexor tendon. (Modified from Smith RK, Zunino L, Webbon PM, et al: The distribution of cartilage oligomeric matrix protein (COMP) in tendon and its variation with tendon site, age and load, *Matrix Biol* 16:255, 1997.)

Proteoglycans are largely divided into two broad categories, the large and small proteoglycans. The large proteoglycans are exemplified by the major proteoglycan of cartilage, aggrecan, and the fibroblast-derived large proteoglycan, versican. These molecules possess a large number of GAG side chains and can form aggregates with hyaluronic acid. With the repulsion of the negatively charged GAG chains, this molecule has a bottle-brush shape and can trap large quantities of water. The swelling potential for this molecule, when restrained by the collagen network of cartilage, produces a structural matrix ideally suited to resisting compression. In tendon, areas subjected to compressive forces develop a matrix rich in these large proteoglycans, such as in the DDFT and SDFT in the metacarpophalangeal region.

The small proteoglycans such as decorin, biglycan, fibromodulin, and lumican usually have only one or two GAG side chains. Many of these proteoglycans have wide tissue distribution and have been shown to have structural and regulatory roles. A number of these proteoglycans bind to other members of the extracellular matrix. Thus decorin, the most common proteoglycan in tensional tendon (e.g., metacarpal region of the digital flexor tendons), has been shown to bind to fibrils of type I collagen.[40] Decorin is thought to be respon-

sible for regulating collagen fibril diameter and, together with the other small proteoglycans, may be responsible for providing electrostatic cross-links between fibrils, thus being also an important determinant of tendon strength. Targeted disruption of the genes for some of these small proteoglycans has confirmed a suggested role in maintaining tissue structural integrity. Deleting or knocking out the decorin gene results in variably sized collagen fibrils and poor mechanical strength in skin,[41] whereas targeted disruption of the fibromodulin gene causes altered collagen fibril morphology and reduced mechanical strength in tendon.[42]

Furthermore the small proteoglycans have been shown to bind various growth factors, especially TGF-β,[43] which may be important in regulating tendon homeostasis, adaptation, and response to injury.

Other non-collagenous glycoproteins. A number of other non-collagenous glycoproteins have been described in tendon, including elastin (not thought to be important for tendon elasticity in the horse),[31] fibronectin (which is upregulated after injury), thrombospondin 4, PRELP (*p*roline a*r*ginine-rich *e*nd *l*eucine-rich repeat *p*rotein), and tenascin-C. However, the functions of these, and others that have yet to be characterized, have not been determined fully.

Types of Tendon Injury

Tendons can suffer intrinsic (strain) or extrinsic (percutaneous) injury or displacement. The most common injury in the horse is the intrinsic injury of the SDFT in the metacarpal region. Recent epidemiological data have indicated an incidence of 43% in National Hunt horses in training.[44] Much of our understanding of tendon physiology (as described previously) and pathogenesis relates to this tendon. Insertional injuries, although common in the human athlete, are rarer in the horse and most commonly are seen associated with the SL rather than the SDFT.

Clinical superficial digital flexor tendonitis varies in severity from individual fibril or fiber slippage to individual fibril or fiber rupture and ultimately to complete rupture of tendon with progressive involvement of more groups of fibers and fascicles. Many horses with clinical tendonitis are believed to be preceded by subclinical degeneration of the tendon matrix. This is based on a number of observations. First, post-mortem examination of tendons of horses euthanized for reasons other than tendonitis revealed low-grade pathological damage ranging from acellular areas, chondroid metaplasia, and cyst formation.[23,45] Second, tendonitis is frequently a bilateral disease, although one limb is more severely affected than the other. Although bilateral changes sometimes can be difficult to identify clinically, ultrasonography invariably confirms some degree of bilateral involvement. Thirdly, recent research has identified a number of changes that occur within tendons associated with aging and exercise.

Mechanisms of Tendon Injury: Effect of Aging and Exercise

A number of controlled exercise studies in adult and young horses (Table 69-2) has provided considerable information on the effect of exercise on normal equine tendons. In none of these studies was there any indication of clinical tendonitis induced by the exercise protocols.

Regional differences in collagen fibril diameter were seen in long-term exercised older horses, but not in the short-term exercised, or younger horses.[46] Within the central region of the SDFT there was a higher proportion of smaller fibrils in comparison with the controls (Fig. 69-8). The higher proportion of small fibrils did not correlate with new collagen formation and thus appears to result from disassembly of the larger diameter fibrils, rather than the formation of new collagen, which would indicate an adaptive response. The reduction in crimp pattern seen with aging was accelerated by the exercise protocols in the adult.[47]

Changes in molecular composition also occurred in the long-term exercise studies, with a reduction in GAG content and an accelerated loss of COMP in the center of the tendon.[48,49] In contrast, molecular analysis of tendons recovered post mortem with central discoloration but no prior diagnosis of superficial digital flexor tendonitis demonstrated an increase in type III collagen and GAG.[50] Because these tendons appear to be enlarged significantly and have central hypoechoic lesions when examined by ultrasonography in vitro (Fig. 69-9), these molecular changes probably more reflect a reparative response rather than a degenerative change associated with aging and exercise.

In young growing animals, removal of load from tendon results in a lack of COMP accumulation in tendon, whereas removal of load after COMP has accumulated does not alter its levels in the tendon. Because recent data have shown an association between COMP levels and tendon strength at skeletal maturity,[39] too little exercise may inhibit the ability of the tendon to develop quality tendon matrix. However, exercise studies during skeletal development indicated that tendons are more easily damaged if the exercise level is too high.[51]

Table • **69-2**

Studies Aimed at Investigating the Effect of Exercise of Equine Digital Flexor Tendons

POPULATION	STUDY NAME	AGE OF ANIMAL AT ONSET OF EXERCISE	DURATION OF EXERCISE	NATURE OF EXERCISE	AGE AT ANALYSIS	REFERENCES FOR PROTOCOL
Thoroughbred	Bristol, long-term study	21 months	18 months	Treadmill	3 years 3 months	46, 49
Thoroughbred	Bristol, short-term study	19 months	4½ months	Treadmill	23½ months	49
Warmblood	Utrecht study	1 week	19 weeks (high intensity) followed by 6 months (low intensity)	Over ground	5 months (first group) 11 months (second group)	68

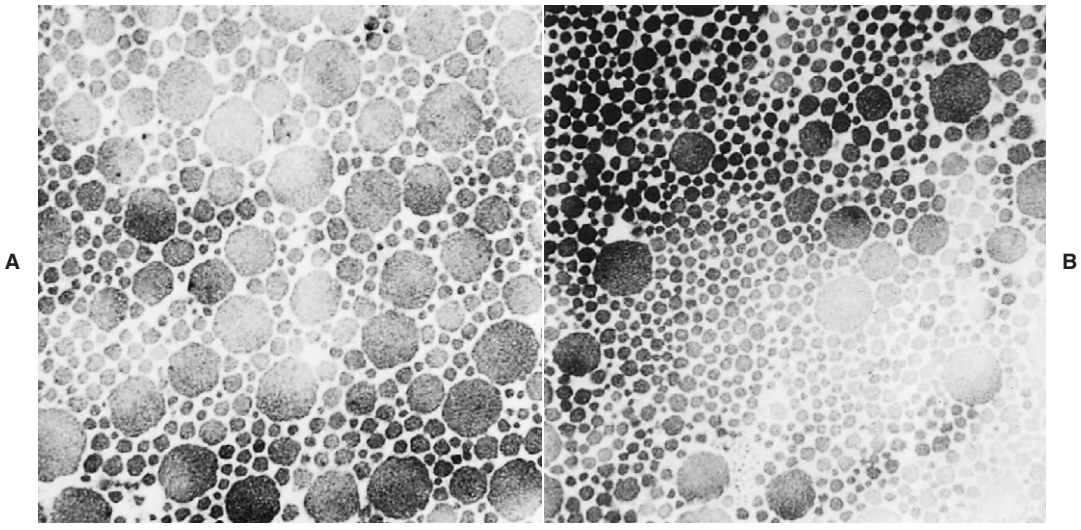

Fig. 69-8 Difference in collagen fibril populations in control and treadmill-exercised yearlings. **A,** Control. **B,** Treadmill exercised. Note the increased proportion of small-diameter fibrils compared with the non-exercised cohort (**A**). (From Patterson-Kane JC, Firth EC, Parry DAD, et al: Comparison of collagen fibril populations in the superficial digital flexor tendons of exercised and nonexercised thoroughbreds, *Equine Vet J* 29:121, 1997.)

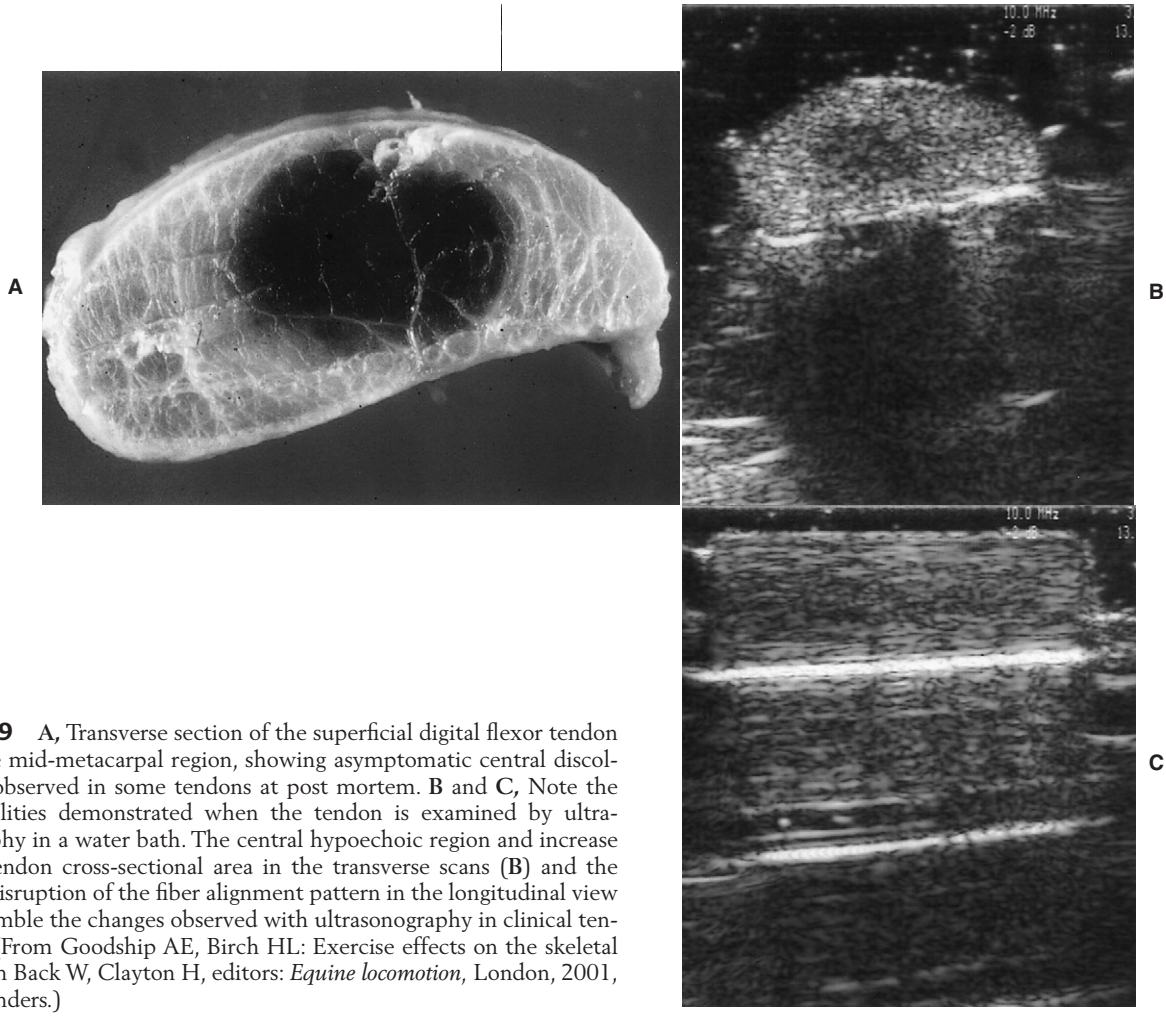

Fig. 69-9 **A,** Transverse section of the superficial digital flexor tendon from the mid-metacarpal region, showing asymptomatic central discoloration observed in some tendons at post mortem. **B** and **C,** Note the abnormalities demonstrated when the tendon is examined by ultrasonography in a water bath. The central hypoechoic region and increase in the tendon cross-sectional area in the transverse scans (**B**) and the central disruption of the fiber alignment pattern in the longitudinal view (**C**) resemble the changes observed with ultrasonography in clinical tendonitis. (From Goodship AE, Birch HL: Exercise effects on the skeletal tissues. In Back W, Clayton H, editors: *Equine locomotion*, London, 2001, WB Saunders.)

These controlled exercise studies have demonstrated that exercise accelerates a degenerative change that occurs inevitably with aging. Thus the research data would suggest that after skeletal maturity the tendon has limited ability to adapt. Instead, cumulative fatigue damage weakens the tendon matrix and allows the initiation of clinical tendonitis when loading overcomes the resistive strength of the tendon. Epidemiological studies have supported a strong association of age and exercise with the incidence of tendon injury in horses and people.[44,52]

Further confirmation of cellular activity during growth, but not after skeletal maturity, has been provided by studies of matrix turnover and gene expression. The turnover of collagen has been determined for experimental animals, but no definitive data are present for the horse. In experimental animals, collagen turnover is high in the neonate and growing animal, but it declined to low levels in the adult.[53] In bovine digital flexor tendons matrix gene expression, as determined by in situ hybridization, was easily detectable in young, growing animals, but no gene activity was present in the metacarpal region in the adult.[54] Interestingly, gene activity did persist in the metacarpophalangeal region, which may explain the relative resistance to injury of this region, because of its capability to remodel microdamage. In support of this hypothesis, COMP levels in this region of the equine SDFT and DDFT do not decline after skeletal maturity. The absence of gene activity in the metacarpal region may be from a lack of appropriate growth factors or cellular senescence. Certainly in these studies investigating TGF-β in equine tendons, young equine tendon had high levels, but amounts declined after skeletal maturity.[24]

In contrast, young growing tendon does appear to be sensitive to the effects of loading and exercise. The adaptive response of the growing animal may not be constant, and research data suggest that response may be pronounced most early in life and decline with growth. The level and amount of work necessary to induce this response is unknown and currently is being evaluated. However, by analogy with bone remodeling in response to load, high strain rates may be the most effective. An interesting note is that the natural exercise performed by a group of young foals at pasture includes a large amount of jumping activity at play, which is perfectly suited to these high strain rates on the digital flexor tendons.

Thus, as with other skeletal tissue development, such as cartilage, a window of opportunity apparently may be exploited to optimize conditioning of tendons for athletic performance (Fig. 69-10). The large variation seen in the mechanical properties within a population could be accounted for by variation in tendon development, caused by environmental factors, or genetic determinants.

From these observations a strategy for preventing tendonitis can be proposed. Maximizing the quality of tendon before skeletal maturity with the early introduction of controlled exercise may be possible, thereby reducing the incidence of tendon injury in subsequent racing and competition (Fig. 69-11).

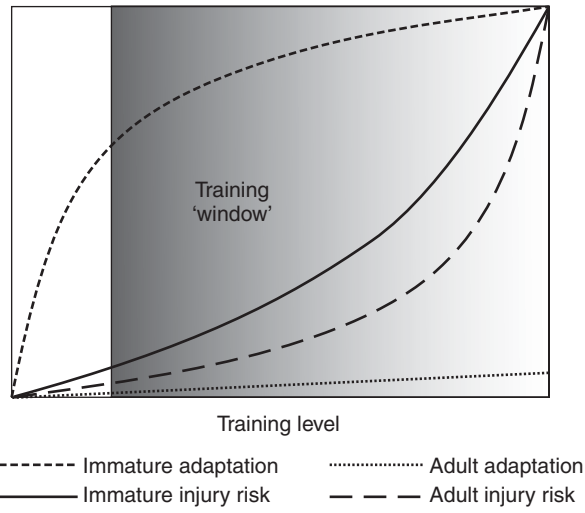

Fig. 69-10 Hypothesized schematic representation of the adaptive response and injury risk for growing (immature) and adult (>2 years of age) equine digital flexor tendons. (Modified from Goodship AE, Birch HL: Exercise effects on the skeletal tissues. In Back W, Clayton H, editors: *Equine locomotion*, London, 2001, WB Saunders.)

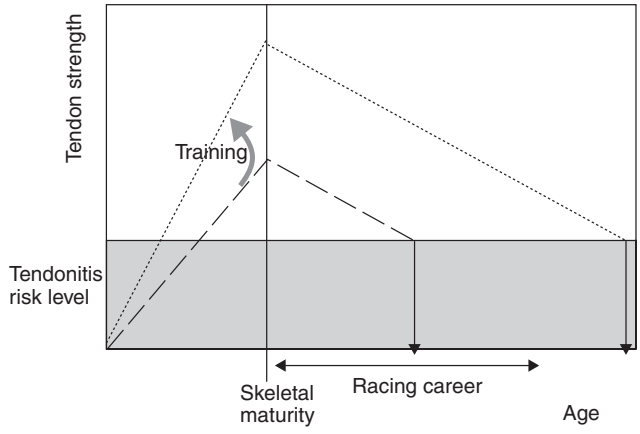

Fig. 69-11 Strategy for the prevention of tendonitis in the horse. The *dotted line* refers to a horse that develops strong tendons, whereas the *dashed line* represents a horse with poor-quality tendons at skeletal maturity (which appears to be at about 2 years of age). The latter suffers tendonitis during its racing/competitive career because of inevitable and cumulative fatigue damage to the tendon, whereas the former, although suffering the same degeneration, starts from a stronger point and therefore does not suffer tendonitis. The early introduction of exercise during development potentially improves tendon quality (*arrow*), thereby subsequently reducing the incidence of tendonitis.

Hypothesized Mechanisms of Tendon Degeneration

Mechanical Influences

Sudden over-extension of the metacarpophalangeal joint causes mechanical disruption of the tendon. Although this may be the mechanism of certain tendon injuries, such as deep digital flexor tendonitis, direct low-grade mechanical forces, such as experienced under maximal loading, could be responsible for the cumulative fatigue microdamage of the tendon matrix. Subsequent clinical tendonitis is initiated by similar, or sudden supra-maximal, loading after the accumulation of microdamage.

Physical Influences: Exercise-Induced Hyperthermia

Because of the hysteresis loop, when a tendon is loaded and unloaded, a loss of stored energy as heat results in temperature increases within the equine digital flexor tendons. Thermocouples have been placed inside the SDFT, and these have recorded temperatures of up to 45° C during periods of galloping.[12] Such temperatures are used to kill neoplastic cells therapeutically and so it was hypothesized that these temperatures would interfere with tenocyte metabolism and possibly destroy the cells. However, in vitro experiments have shown that tenocytes are much more resistant to these temperature increases in comparison with other fibroblast-like cells.[55] Interestingly, tenocytes tolerate such temperature increases even in utero, suggesting a genetically determined, rather than acquired, characteristic. Although

the cells remain viable with such temperature increases, hyperemia may still influence tendon matrix quality. A reduction in the normal synthetic activity of tenocytes or a direct denaturing effect on tendon extracellular matrix still may occur.

Vascular Theories

Blood flow through tendon is a complex issue and its relevance to clinical injury is still unsubstantiated. Under maximal loading, blood flow is limited or abolished within the tendon because of the compressive forces generated by the lengthening of the tendon, and this may give rise to relative hypoxia. Some areas of equine flexor tendon are relatively poorly perfused (e.g., the dorsal portion of the DDFT in the metacarpophalangeal joint region), and this level (but not just the dorsal surface) is a site predisposed to deep digital flexor tendonitis. However, this area also shows histological adaptation to the relatively ischemic environment, with fewer cells and increased amounts of the compression-resisting extracellular matrix components. Furthermore the tendon may receive some of its nutrition at this site by diffusion from the digital sheath synovial fluid. Similar alterations in the extracellular matrix composition are seen at the corresponding positions in the SDFT, and one could assume that the forces on this tendon would be similar to those of the DDFT at the same site. Therefore, a reduced blood flow would also be expected at this level, and yet clinically this region is invariably spared injury in all but the most severe tendonitis.

Equine tendon cells do rely at least in part on oxidative metabolism, although blocking aerobic metabolism does not prevent normal cell proliferation.[56] Based on studies in other species, tenocytes may be more resistant to hypoxia than other similar fibroblast-like cells.

Laser Doppler flowmetry has suggested that a change in blood supply is not the initiating cause in human Achilles tendonitis,[57] in contrast to the previously proposed hypoxic cause for tendonitis based on electron microscopic investigations of normal and diseased Achilles tendons.[58]

Another result of poor perfusion under loading is the generation of toxic free radicals when perfusion is restored. Such reperfusion injury also has been proposed as a causative factor for tendonitis through the destruction of tenocytes or tendon matrix by the free radicals, although this at present remains a speculative mechanism.

Proteolytic Enzymes

Various stimuli, including those mentioned previously, could result in the synthesis, release, or activation of proteolytic enzymes. Relatively little information is available on the constitutive or induced expression of proteases in tendon, although activity of procollagenase and aggrecanase has been described in human and bovine tendon explants in vitro.[59,60] An imbalance between the matrix synthesis and degradation of various extracellular matrix proteins is a possible mechanism whereby the tendon can be weakened and predisposed to clinical tendonitis.

Factors Affecting the Loading of the Superficial Digital Flexor Tendon and Initiation of Clinical Tendonitis

Peak SDFT forces are responsible for initiating clinical tendonitis. When the tendon has been weakened sufficiently by the preceding degenerative change, factors that increase the peak loading of the SDFT therefore also act to increase the risk of clinical tendonitis.

The SDFT is loaded preferentially at the early stage of the stride,[7,9,61] which represents the time of highest injury risk. External factors, such as the rider's weight or hard ground, increase these peak forces, although possibly only in certain tendons.[9] Recent data have suggested that landing from a jump increases the peak forces in the SDFT but not the SL.[62] The greater the height and number of fences jumped at Grand Prix–level show jumping would explain the higher incidence of injury in these horses to those competing at a lower level (see Chapters 70 and 116).

Foot conformation is also critical for affecting the loading of the SDFT (and SL). The lowering of the toe with respect to the heels, or the raising of the heels with respect to the toe, results in reduced loading of the DDFT and increased loading of the SL (and possibly the SDFT), often only detectable at the trot.[10,63,64] As a corollary to this, the long-toe, low-heel conformation characteristic of the Thoroughbred may actually protect the horse from superficial digital flexor tendonitis.[64]

The ground surface also influences the loading of the SDFT. Soft ground may predispose to increased strains in the SDFT by allowing the toe to sink. However, using sand has been shown largely to have little effect on the strains in the SDFT and DDFT.[9] The effect of ground surface on the incidence of tendonitis is probably more related to determining the speed of the horse. Speed is correlated with strains in the SDFT and also has been correlated with the incidence of SDFT tendonitis in racehorses (see Chapters 70, 107, 108, and 113). Thus ground surfaces that slow the horse tend to be protective of SDFT tendonitis, whereas the driest and hardest racecourses are associated with the highest incidence of tendonitis.

Many horses suffer tendonitis toward the end of a race or event when horses are fatigued. Fatigue will cause greater incoordination, which can result in increased peak loads on the SDFT, thereby increasing the risk of tendonitis.

Because tendon degeneration appears to be related to the number of loading cycles, the greater the exercise history and age, the more at risk the horse becomes. This certainly explains the strong association between age and tendonitis and may explain why older, sedentary horses still can develop tendonitis.

Because the subclinical phase of tendon degeneration affects both limbs similarly, clinical superficial digital flexor tendonitis is frequently bilateral. Changes are frequently observed with ultrasonography on both limbs, although one limb is usually more severely affected than the other.

Pathological Conditions and Phases of Tendon Healing

Tendonopathy can be divided into four phases. The subclinical phase of tendon degeneration, described previously, is difficult to identify clinically at present by palpation or ultrasonographic examination, because it causes minimal, if any, inflammatory reaction. In the future, serological assays, currently being developed to detect matrix proteins released from the tendon, may prove useful for detecting and monitoring this phase.

The clinically detectable pathological condition is divided into three phases: the acute inflammatory phase, the subacute reparative phase, and the chronic remodeling phase.

The acute inflammatory phase begins with the onset of the clinical injury and lasts usually only 1 to 2 weeks, although this is in part determined by the severity of the injury and the anti-inflammatory therapy initiated. This phase is characterized by substantial inflammation, with intratendonous hemorrhage, increased blood supply and edema, and the infiltration of leukocytes, initially neutrophils, but followed by macrophages and monocytes. The pronounced inflammation, if unchecked, results in the release of proteolytic enzymes, which, although directed at removing necrotic collagen, also digest relatively intact tendon collagen, which may cause the expansion of the lesion in the few days after the onset of the clinical tendonitis.

The sub-acute reparative phase begins within a few days of the injury, overlapping with the acute phase, and peaks after about 3 weeks.[65] This phase is characterized by a strong angiogenic response and the accumulation of fibroblasts within

the damaged tissue. These fibroblasts probably are derived from a number of sources including the resident tenocytes, endotenon and paratenon cells, and monocytes of vascular origin. These cells are then responsible for synthesizing scar tissue (tendon tissue is not regenerated), characterized by haphazardly arranged collagen, predominantly type III. The scar tissue formed is initially weaker than tendon tissue and hence healing tendon is predisposed to re-injury at the injury site. Such episodes of re-injury perpetuate the first two phases and increase the amount of damaged tendon and hence the severity of the injury.

The absence of a paratenon and an externally derived blood supply within a tendon sheath may explain the relative poor response in healing in these areas. The formation of adhesions within tendon sheaths, although responsible for limiting the movement of the tendons subsequently, provide a method of allowing angiogenesis and the infiltration of cells into damaged tendon tissue within a tendon sheath. Hence, although adhesions have deleterious effects on the function of tendons, they are a normal response to encourage tendon healing in this region.

During the chronic remodeling phase, which begins several months after the injury, the scar tissue slowly remodels over a number of months. This remodeling process is associated with a conversion of type III collagen to type I collagen, the major component of normal tendon. However, in spite of this conversion the tissue never becomes normal tendon tissue, although it is probably more functional. Controlled loading (exercise) during this phase may help promote this conversion and, even more importantly, align the collagen fibrils in the direction of force, which further improves the mechanical properties of the scar tissue. This aspect of the remodeling process is followed by assessing the fiber alignment score on ultrasound (see Chapter 70).

Re-injury is unfortunately common, even after healing is complete, in the same tendon, the contralateral tendon, or other supporting structures of the metacarpophalangeal joint. As the injured tendon remodels, it becomes stronger, so that fully healed tendon (15 to 18 months after injury) is frequently stronger than normal tendon.[66] However, remodeled tendon has poor elasticity, resulting in increased strain in adjacent, relatively undamaged regions of the tendon. Therefore if re-injury occurs in the same tendon, it frequently occurs at adjacent or remote sites to the original injury. Subsequent injury to the contralateral tendon is also effectively a re-injury because of the bilateral nature of the preceding degeneration and clinical tendonitis. Subsequent injury to the SL may be the consequence of some loss of support of the metacarpophalangeal joint, caused by significant lengthening of the SDFT, which can occur with severe superficial digital flexor tendonitis. The SL may also suffer cumulative microdamage, which would increase further its susceptibility to injury.

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CHAPTER • 70

Superficial Digital Flexor Tendonitis

The first two sections of this chapter consider the general clinical manifestations of tendonitis and then the specific surgical management of tendonitis in racehorses. The third section discusses some of the variable clinical presentations in other competition horses and factors influencing treatment and prognosis.

SUPERFICIAL DIGITAL FLEXOR TENDONITIS IN RACEHORSES

• Joan S. Jorgensen and Ronald L. Genovese

Superficial digital flexor tendon (SDFT) injuries significantly compromise athletic performance and may culminate in a career-ending injury. The incidence of SDFT injuries in Thoroughbred (TB) racehorses ranges from 7% to 43%,[1,2] and such horses are most at risk because of high racing speeds or high speeds associated with jumping (steeplechase racing, see Chapter 113).[3] Other performance horses, including upper-level event horses (see Chapter 118), have an increased risk of SDFT injury (see page 639). Horses used for dressage, high-level show jumpers (see Chapter 116), racing Arabians (see Chapter 112) and Quarter Horses (see Chapter 111) , polo ponies (see Chapter 120) , and fox hunters incur SDFT injuries less frequently.[3-5] SDFT injury from athletic use in racehorses commonly is seen because of repetitive speed cycles over distance and possibly genetic predisposition to SDFT injury.[6] We are aware of several TB racehorse mares and at least one TB stallion and one Standardbred (STB) stallion that are known to have progeny with an increased susceptibility to SDFT injury compared with the normal racehorse population. Additional factors that may predispose a horse to SDFT injury include conformation, working surfaces, shoeing, training methodology, and the relationship between the level of physical fitness and the current exercise.

SDFT injury also occurs spontaneously in sedentary or lightly used horses more than 15 years of age. These tendon injuries often are severe and generally involve the proximal metacarpal region and the carpus and extend to the musculotendonous junction in the antebrachium. Many of these injuries result in overt lameness, tendon thickening, and carpal sheath effusion. Sometimes, however, the only presenting sign is lameness, with little palpable thickening. These injuries occasionally can be difficult to diagnose, requiring local analgesia and ultrasonography.

SDFT injuries from athletic use occur in the forelimb far more frequently than in the hindlimb. In one American study of 143 TB racehorses, 58% of SDFT injuries occurred in the left forelimb and 42% in the right forelimb.[7] Bilateral injury is common and has been recognized more frequently since veterinarians have been examining both limbs routinely by ultrasonography.

Most injuries in the SDFT caused by athletic use occur in the mid-metacarpal region (zones 2B to 3B), but injuries also occur at the musculotendonous junction of the antebrachium, in the carpal canal and subcarpal region, and in the pastern (see Chapter 83). The plantar hock region is the most common site of SDFT injury in hindlimbs, especially in the STB racehorse (see Chapters 79 and 109). Occasionally this injury extends into the mid-metatarsal region. Infrequently, a subtle SDFT injury is associated with tenosynovitis of the digital flexor tendon sheath (DFTS) in hunters, jumpers, and dressage horses.

Racehorses with SDFT injuries in racehorses traditionally have been regarded as having a guarded to poor prognosis for return to racing, although the prognosis for other athletic disciplines is more optimistic. Before ultrasonography was used routinely, documentation of SDFT injury was limited. Diagnosis was based on gait evaluation and palpation of a swollen or thickened tendon. The injury was referred to as a bowed tendon, and morphological abnormality and severity of injury were little appreciated.

Recently, substantial progress has been made in understanding the nature of tendon injury and the mechanisms of healing. During repair, injured elastic tendon fibers are replaced with modified fibrous scar tissue, resulting in a tendon repair that is never totally normal. The quality of repair can vary greatly. Some tendon injuries repair and resolve with enough mature collagen so that they return to nearly normal size, with sufficient remodeling that results in approximately parallel alignment of the repair tissue. Other injuries develop a scar, with an overall increase in tendon size, poor or random fibrous tissue alignment, and peritendonous fibrosis.

Many of the proposed therapeutic approaches are directed at maximizing the chances for a more physiologically functioning tendon. Therapy requires a multifaceted approach

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Many of the proposed therapeutic approaches are directed at maximizing the chances for a more physiologically functioning tendon. Therapy requires a multifaceted approach

that reduces the acute inflammatory response and hemorrhage in the acute phase and improves fiber alignment during the long rehabilitation phase. The ultimate goal of any treatment and management programs is to maximize the chances for a tendon to repair with adequate strength and elasticity for a return to a similar level of performance with the lowest risk of re-injury.

It is important to recognize the variables that may affect the ultimate prognosis when managing SDFT injuries. Not all tendon injuries are the same, and case management depends on the specific injury, medical factors, and other non-medical factors.

CLINICAL SIGNS

Clinical signs of SDFT injury in racehorses vary considerably depending on the location of the primary injury, type of injury, severity, and the timing of the examination. Occasionally, clinical signs may be delayed by days or weeks. Furthermore, a lack of correlation may exist between the severity of the injury and the severity of tendonitis in any given individual, especially in the more common core tendon injuries experienced by TB racehorses. In contrast, STB racehorses more often experience lateral and medial border tendon injuries that result in significant swelling but less severely injured tendon fascicles. Identification of subtle, yet significant, re-injury by clinical evaluation may be difficult because of previous tendon thickening. Thus ultrasonographic imaging is essential to confirm clinical signs and to evaluate the extent of injury to the SDFT.

Lameness

In our experience the degree of lameness associated with a tendon lesion in the mid-metacarpal region is correlated with the severity of the injury. Slight to high-slight (category II to IV) injuries generally are not associated with any appreciable lameness, whereas mid-moderate to high-moderate (category V) injuries cause only transient lameness. Most severely injured tendons (category VI), or total rupture of the SDFT, result in at least transient lameness, which may be severe. In contrast, lesions in the carpal canal or proximal metacarpal region (zones 0 and lA) consistently are associated with lameness.

Swelling

For assessing tendon injuries, swelling is defined as subcutaneous or peritendonous fluid accumulation. Digital palpation reveals a soft or semi-firm, diffuse or focal fluid accumulation that may prohibit exact palpation of the SDFT. Subcutaneous swelling can be associated with tendon injury, especially in the acute stage of injury. Careful digital palpation of the limb held in a semi-flexed position may reveal slight crepitus in an acutely injured tendon. However, subcutaneous inflammation or hemorrhage is not associated invariably with tendon injury. Examples of focal edema or hemorrhage without significant SDFT injury include swelling associated with cording of the mid-metacarpal region secondary to a malpositioned bandage or subcutaneous swelling in the proximal or distal metacarpal region caused by malpositioned tendon boots or stable (stall) bandages. An example of diffuse swelling is pitting edema (see Chapter 14), occasionally caused by external blistering. Diffuse filling also may reflect a sub-solar abscess or cellulitis.

Thickening

Thickening, or enlargement specifically indicates SDFT swelling secondary to injury or a thickened end-stage repair from a previous injury. In this context, subcutaneous swelling is not appreciable, but a palpable enlargement of the SDFT occurs. In many slightly and diffusely injured tendons thickening may be difficult to appreciate and careful comparison to the normal contralateral limb may be required to identify SDFT enlargement. In more severely injured tendons examined in the subacute phase, enlargement generally can be felt. Two clinical situations in which assessing SDFT thickening is difficult are a focal SDFT injury in the sub-carpal region, where the tendon is enveloped by the retinaculum, especially in colder climates when limb hair is long, and instances of SDFT injury within the DFTS that also is associated with tenosynovitis. Tenosynovitis makes distinguishing between tendon thickening and sheath fibrosis difficult.

When palpating the SDFT, one should always determine if the medial and lateral borders of the SDFT can be separated clearly from the accessory ligament of the deep digital flexor tendon and the tendon itself. If both digital flexor tendons are slightly enlarged, detection of abnormality is more difficult; however, frequently the margins of both tendons are more rounded than normal. Assessing the flexibility of the SDFT also is useful, because abnormal stiffness usually reflects previous injury.

Heat

Increase in surface temperature often can be the earliest and most subtle clinical sign of SDFT injury or re-injury. Digital appreciation of an increase in skin temperature, or thermography, often can indicate tendon inflammation. Because extensive use of liniments and daily bandaging in racehorses also increases skin temperature, one must be careful when making this assessment.

Sensitivity to Direct Digital Palpation

A painful response to direct digital palpation is often a reliable clinical test for tendon injury. Examination is best performed by holding the limb in a semi-flexed position and palpating with the thumb and forefinger systematically from proximal to distal in the metacarpal region in an effort to elicit a painful response. When a sensitive area is palpated, the horse generally flinches. The examination has many caveats. If a sensitive response is elicited bilaterally, the horse may merely be hyperresponding to increased pressure and possibly has no injury. Not all horses with tendon injury have a painful response. Horses with blistering of the skin, adverse local drug reaction, infection, or cording are also hyper-responsive and more reactive than those with a tendon injury. In addition, extreme sensitivity to direct palpation coupled with focal or diffuse swelling may indicate a problem not related to the tendon.

Tendon Profile

Evaluation of the tendon profile with the limb in a full weight-bearing position can provide valuable information. In a normal limb the metacarpal region has a straight palmar profile. A normal SDFT should be superficial and parallel to the deep digital flexor tendon. It is important to examine the profile from all possible angles. With a slight injury, the tendon often has a normal profile when viewed from the lateral aspect and a convex or bowed profile from the medial aspect, or vice versa. In an acute total rupture, little swelling and thickening may be present if the leg is examined within 2 hours of the injury. However, with the limb in full weight-bearing position, one may note hyperextension of the metacarpophalangeal joint. In this case, digital palpation along the palmar aspect of the tendon reveals a 1- to 2-cm defect in the SDFT. Digital palpation with the limb in a semi-flexed position also reveals laxity and excessive mobility of the tendon.

Tenosynovitis of the Carpal Sheath or Digital Flexor Tendon Sheath

Tenosynovitis may be associated with a tendon injury or may be a clinical entity without tendon injury (see Chapters 75 and 76). Ultrasonographic evaluation is required to appreciate tendon injury in the presence of tenosynovitis.

Tendon Injury Limited to the Pastern

Injury to one or both of the SDFT branches of the pastern generally is associated with branch thickening and a painful response to direct digital pressure (see also Chapter 83). This is best appreciated with the limb held in a semi-flexed position and direct pressure placed on the branch with the clinician's thumb. Injury to the SDFT branch(s) may be associated with tenosynovitis.

MANAGEMENT OF THE ACUTE PHASE OF TENDON INJURY IN RACEHORSES

In most horses with sub-total SDFT injuries in the acute phase, anti-inflammatory and supportive management are instituted. A variety of treatment regimens are available. For the most part, systemic non-steroidal anti-inflammatory drugs (NSAIDs) such as phenylbutazone (4.4 mg/kg per day) for 7 to 10 days and a single dose of systemic corticosteroids such as dexamethasone (0.04 mg/kg) are included in the initial therapy. Perilesionally administered corticosteroids are considered contraindicated in tendon injuries, especially long-term usage, because they are thought to delay collagen formation. However, some clinicians use a single perilesional dose of triamcinolone (6 to 9 mg) or methylprednisolone acetate (40 mg) (dystrophic mineralization occasionally has been associated with methylprednisolone therapy) in horses with slight, peripheral tendon injuries in STB racehorses, especially when associated with a curb (see Chapter 79). Practitioners often administer a course of polysulfated glycosaminoglycans (PSGAGs; 1 vial per week for 4 weeks) in the acute stage.

Physical therapy is indicated, and we recommend icing for 1 to 2 hours once or twice a day, with application of a poultice, or simple support bandaging for subtotal injuries. Casting may be indicated for total ruptures. Exercise generally is restricted to stall (box) rest or limited hand walking. In horses with subtotal injuries, we prefer to keep the injured limb shod in a fully grooved bar shoe, with a straight hoof-pastern axis, so that the metacarpophalangeal joint position is normal in a standing position. We do not advocate raising or lowering the heel to an exaggerated position. We perform an ultrasonographic examination within the first few days and then again 3 to 4 weeks later. Sometimes tendon splitting is advised for small core lesions.

INJURY ASSESSMENT AND GOALS FOR ATHLETIC OUTCOME

Qualitative assessment combines the physical findings and a subjective ultrasonographic appraisal. This gives an accurate diagnosis, but we also strongly encourage the use of quantitative ultrasonographic evaluation. This includes data such as cross-sectional area (CSA) and echogenicity and fiber alignment scores (see Chapter 16).

Optimal healing of SDFT injuries depends on managing a number of variables including the personality of the horse, its age, sex, athletic use, conformation, injury episode number, maximal level of exercise attained, and severity of the injury. For instance, a 4-year-old TB gelding racehorse that has an upright conformation, has never raced, and suffers a severe (category VI) SDFT injury after one gallop has a poor prognosis with any treatment. If this horse sustained a small core lesion of the SDFT, its prognosis would be more guarded because of its conformation and the exercise level at which injury occurred compared with a similar injury in a well-conformed, seasoned racehorse.

Athletic outcome of a racehorse may be divided into three categories: successful, meaning completion of five or more races (I); partially successful, completion of one to four races (II); and failure, meaning re-injury occurred before the first race was completed (III). Successful horses can be further subdivided into those with re-injury or no re-injury. Partially successful can be further subdivided into those suffering re-injury or those injuring the contralateral SDFT or a forelimb suspensory ligament.

ULTRASONOGRAPHIC EVALUATION AND CATEGORIZATION OF INJURIES

If clinical evaluation indicates a possible SDFT injury, ultrasonography should be used to confirm the diagnosis and objectively assess the severity of injury. Sequential examinations provide a guide to controlled exercise management and are used to assess progress of repair and attempt to establish an optimal time to return to full work.

The acquisition and assessment of accurate ultrasonographic data requires high-quality images, and it is important to develop a rigid, standardized technique (see Chapter 16). The ultrasonographer must take primary responsibility for image interpretation. For a second person to give an opinion on images previously obtained by someone else is often difficult.

Quantitative ultrasonographic data includes CSA, percentage of CSA occupied by a lesion, grade of echogenicity of a lesion (type or echo score, TS), and assessment of fiber bundle alignment in longitudinal images (fiber alignment score, FAS). Each of these data points is assessed at every defined level of the limb (zone) and then summed to provide total scores. These scores then can be used to categorize an injury as minimal (category III), slight (category IV), moderate (category V), or severe (category VI) (see Chapter 16). The following comments apply to forelimb and hindlimb injuries, although reference is only made to the metacarpal region.

Initial Evaluation

Early examination of a suspected new injury of the SDFT may not reveal any anechoic or hypoechoic lesions. However, if the CSA of a single zone is more than 39% larger than the contralateral limb, or if the total of 6 of 7 zones is more than 14% larger than the contralateral limb, then tendonitis should be suspected (category II). If tendonitis cannot be substantiated by ultrasonography, despite soft tissue swelling, then conservative management is indicated. The horse should be restricted to walking exercise for at least 72 hours and then re-evaluated clinically and by ultrasonography. Symptomatic therapy includes systemic NSAIDs, daily icing, and mild leg liniments or sweats with limb bandaging. One must recognize that sometimes a lesion(s) cannot be appreciated by ultrasonography for at least several days or longer.

Within 7 days of injury a hypoechoic or anechoic lesion may represent tendon fascicle damage, hemorrhage, or inflammatory exudates and is most likely a combination of all three. Distinguishing the relative contributions of each or determining accurately the severity of injury, which may be underestimated or overestimated, is not possible. Initially, damaged collagen fibers may be grossly intact but non-functional, resulting in reflecting echoes. Ongoing enzymatic degradation and further injury caused by pressure necrosis may result in a lesion deteriorating over 3 to 4 weeks. In contrast, infrequently the hemorrhage and inflammatory exudates resolve over the following 3 to 4 weeks and result in a great improvement of the lesion, and the injury to fiber bundles may not be as serious as initially indicated.

Baseline Evaluation

If an initial examination is done within 1 to 7 days of a new injury or a re-injury, we strongly advise that a second ultra-

sonographic evaluation be performed 2 to 4 weeks (preferably 4 weeks) later to obtain baseline data about the severity of injury (see Chapter 16).

SUBACUTE PHASE TREATMENT AND LONG-TERM REHABILITATION

History of Treatment in Racehorses

A wide variety of short-term and long-term treatment programs for superficial digital flexor tendonitis have been used, sometimes implemented regardless of the severity of the injury or re-injury, and with or without prolonged rest or controlled exercise. No comprehensive reports comparing various treatments of similarly injured tendons are available, and therefore proposing specific recommendations that will give the best prognosis for return to racing is difficult.

A comprehensive retrospective study of TB and STB racehorses currently is being performed to compare the rate of return to racing between a variety of therapeutic regimens for minimal (category III), slight (category IV), moderate (category V), and severe (category VI) tendon injuries documented by ultrasonography. Therapies include pasture turnout, external blistering, internal blistering, intralesional therapies (excluding β-aminoproprionitrile fumarate [Bapten]), or a combination of these. In addition, the amount of layup time is being considered for each category of injury. If all therapeutic regimens are combined (Fig. 70-1), preliminary data indicate that few racehorses successfully return to racing without re-injury (athletic outcome I, completed five or more races), especially with severe injuries.[8] In addition, these data demonstrate that few racehorses are able to return to racing and not experience re-injury of the SDFT, sustain injury to the contralateral SDFT, or injure the suspensory apparatus (subcategories IB and IC). Ultimately, we hope that this research helps to determine optimum therapy for specific lesions and better equip the veterinarian to provide accurate prognostic information.

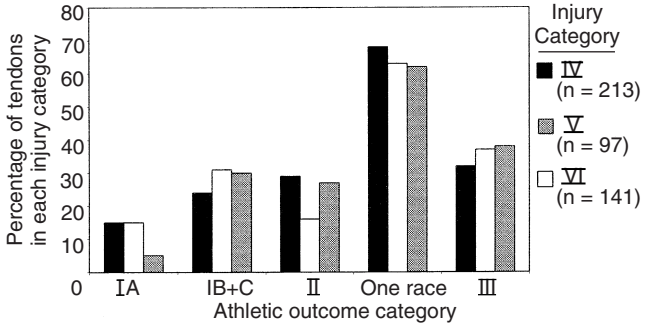

Fig. 70-1 A successful return to racing becomes increasingly difficult as the severity of tendon injury increases. Treatments for slight (category IV), moderate (category V), and severe (category VI) superficial digital flexor tendon injuries including pasture turnout, external or internal blistering, intralesional injections (excluding β-aminoproprionitrile fumarate), or a combination of these were assessed for their success in return to racing. Athletic outcome categories (AOCs) were separated into five groups. AOC IA refers to those horses that have successfully raced at least five times with no re-injury. AOC IB + C includes those that have raced at least five times but have re-injured the same tendon (group B) or the contralateral tendon (group C). AOC II includes those that have raced successfully one to four times and includes tendons that have been re-injured, whereas horses with tendon injuries in AOC III have never raced successfully. An additional group of all those that completed at least one race was included. Data are represented as the percentage of tendons in each injury category that resulted in the ultimate AOC.

Symptomatic Treatment with Continued Exercise

If the decision is made to treat a horse with an injured tendon symptomatically and continue exercise (racing), serial ultrasonographic examinations should be performed, because changes in size or echogenicity may be detected before clinical signs are obvious. If the ultrasonographic examination demonstrates progressive injury or instability and the horse is not athletically (economically) productive, stopping racing and considering long-term rehabilitation would be wise. Naturally, any racehorse racing with an injured tendon is risking more serious injury.

A retrospective study of 209 tendons from 207 TB racehorses was performed to determine if quantitative ultrasonographic assessment could aid in accurately defining a SDFT injury and provide evidence for determining a prognosis for racing in patients that underwent symptomatic treatment with continued exercise. Eighty-eight percent of horses that had no or minimal injury (categories I and III) started more than three races, whereas 12% failed. Thirty-five percent of horses with slight (IV), moderate (V), or severe (VI) injuries started more than three races, but 82% had recurrent injury.[7] Ultrasonography is therefore helpful to determine the prognosis for returning to racing. Symptomatic therapy while continuing to race is a viable therapeutic option for minimal tendon injuries.

Consider a 3-year-old TB gelding racehorse with a swollen left front SDFT after a race (Fig. 70-2). Quantitative ultrasonographic analysis revealed a total lesion area of 13%, TS of 7, and total FAS of 7, indicating a mildly injured (category IV) tendon. The horse was treated symptomatically with anti-inflammatory medication and continued to race. After 4½ months and 6 races the horse was racing well, having earned more than $34,000. After two additional races, the total lesional area increased and the horse's performance decreased. Long-term therapy with time off was instituted. STB racehorses are generally more successful than TBs in continued performance with a tendon injury.[8]

A retrospective study of pre-training (exercise level 5) ultrasonographic data from 106 injured racehorses provided four criteria that we use as a guideline for an optimum return to racing[9]:

1. At least a 60% decrease in category IV total lesional area, or <12% total hypoechogenic fiber bundles for all categories of severity of injury
2. At least a 10% to 15% decrease in total CSA from baseline for all categories of injury (a relatively greater decrease in more seriously injured tendons)
3. At least a 70% decrease in the total TS (ideally <4; the closer to zero the better).
4. At least a 75% decrease in the total FAS (ideally <4; the closer to zero the better) before advancing to exercise level 5.

After grouping the criteria, the horse should meet at least three of the four, with only minimal failure of the other criterion. Horses that met all criteria or failed only one had a 50% chance to be successful as defined by completing five races. If a horse failed more than one criterion, the chance of re-injury before racing one race was 85%. Horses that failed all criteria had a 100% failure rate.

Ultrasonographic evaluation is used to monitor tendon stability during training. For example, ultrasonographic examination of a right front SDFT 11½ months after the baseline scan and after 6 weeks of galloping indicated stable total CSA values but increased hypoechogenic tendon fascicles in zones 3B and 3C (Fig. 70-3). Clinically, increased heat and swelling in the distal metacarpal region were found, which indicated

Zone	Structure Size (mm^2)	Lesion Size (mm^2)	Fiber Alignment Score	Type/Echo Score	Clinical Findings
1A	87	—	—	—	Lameness: 0/5
1B	82	11 (14.25%)	—	1	Swelling: 2/5
2A	88	5 (6.18%)	—	1	Thickening: 1/5
2B	108	16 (14.84%)	1	1	Sensitivity: 2/5
3A	139	14 (10.47%)	3	2	Heat: 2/5
3B	**123**	**36 (29.19%)**	**3**	**2**	Tendon sheath involvement: 1/5
					Fetlock sinking: 0/5
Totals	627	82 (13%)	7	7	Fetlock flexion: Negligible

Bold type indicates maximal injury zone.

Fig. 70-2 Data derived from an initial computerized scan of a 3-year-old Thoroughbred racehorse actively racing with a category IV injury (<15% tendon lesion). The type (or echo) score and fiber alignment score are similar, indicating a new lesion. Zone 3B was determined to be the maximal injury zone with a 29% increase in size compared with the contralateral limb.

Zone	Structure Size (mm^2)	Lesion Size (mm^2)	Fiber Alignment Score	Type/Echo Score	Clinical Findings
1A	109	—	—	—	Lameness: 0/5
1B	95	—	—	—	Swelling: 1/5 (zone 3C)
2A	122	—	—	—	Thickening: 2/5
2B	157	11 (7.38%)	1	2	Sensitivity: 0/5
3A	136	14 (9.59%)	1	1	Heat: 1/5
3B	**178**	**28 (15.68%)***	**2**	**1**	Tendon sheath involvement: 0/5
3C	164	23 (13.90%)*	3	3	Fetlock sinking: 0/5
Totals	961	76 (8%)	7	7	Fetlock flexion: Negative or no abnormality detected

Bold type indicates maximal injury zone.
*Evidence of focal instability of echogenicity near the metacarpophalangeal joint.

Fig. 70-3 Data derived from a computerized scan of a right forelimb superficial digital flexor tendon injury 11½ months after the baseline scan and 6 weeks after galloping was resumed. Before galloping, the total cross-sectional area was 975 mm^2, which decreased to 961 mm^2. However, new hypoechoic lesions were documented in zones 3B and 3C, indicating an unstable healing process.

tendon instability at the current exercise level and a high risk of re-injury with continued training. The trainer was unwilling for economic reasons to pursue another long-term treatment program and decided on an intermediate program of 30 days of ponying (leading the horse from another horse) and swimming. Six weeks later significant re-injury of the SDFT occurred in the distal metacarpal region (Fig. 70-4). Using serial ultrasonographic monitoring is discussed in detail elsewhere (see Chapter 16).

Common Long-Term Treatment Programs

Controlled Exercise and Time Out of Training

A graded exercise program has been used based on severity of the injury and the ultrasonographic progress during rehabilitation.[10] Turnout in a large paddock was not permitted. The time out of training ranged from 9 to 12 months, depending on the initial severity of the injury and ultrasonographic progress. Twenty-eight TB racehorses were managed in this manner and 20 (71%) of the 28 returned to racing. Only two (25%) of eight horses treated with time off and large pasture exercise were successful. Currently no consensus exists on the treatment of choice for superficial digital flexor tendonitis in racehorses, but most treatment regimens are combined with a controlled exercise program and serial ultrasonographic assessment. Most horses with category II (tendonitis without lesions perceptible by ultrasonography) or III (total echogenicity score less than 3) lesions have a reasonably favorable prognosis for successful return to racing (defined as five or more races [athletic outcome I]) if they are confined to controlled exercise for sufficient time.

External/Internal Blistering, Pin Firing, and Time Off for Long-Term Rehabilitation

The most common therapeutic regimen in the United States for rehabilitating racehorse SDFT injuries includes some sort of counterirritation (see Chapter 89), plus controlled exercise

Zone	Structure Size (mm^2)	Lesion Size (mm^2)	Fiber Alignment Score	Type/Echo Score	Clinical Findings
1A	88	—	—	—	Lameness: 0/5
1B	116	—	—	—	Swelling: 0/5
2A	114	9 (7.71%)	1	1	Thickening: 2/5
2B	151	17 (11.27%)	1	1	Sensitivity: 0/5
3A	160	16 (10.40%)	1	1	Heat: 0/5
3B	**202**	**53 (26.37%)***	**3**	**3**	Tendon sheath involvement: 0/5
3C	191	27 (14.06%)*	3	3	Fetlock sinking: 0/5
Totals	1022	122 (12%)	9	9	Fetlock flexion: Negative or no abnormality detected

Bold type indicates maximal injury zone.
*Evidence of focal instability of echogenicity near the metacarpophalangeal joint.

Fig. 70-4 Data derived from a computerized scan of the same limb in Figure 70-3. After 16 days of continued exercise, significant re-injury is documented by an increase in total cross-sectional area to 1022 mm^2, worsening type score and fiber alignment score in zones 3B and 3C, and an increase in the total percentage hypoechogenic volume of the injury. These findings illustrate the importance of quantitative analysis in monitoring tendon healing and predicting responses to an aggressive treatment program.

or turnout into a large pasture. Many counterirritation options exist, including external blistering agents such as a variety of iodine-based liniments, internal peritendonous injection of 2% iodine in almond oil (internal blister), and pin firing. Several reasons exist for the persistent use of these treatment regimens, in spite of limited research suggesting that external blistering, and especially pin firing, have no beneficial effect on tendon injury repair.[11] No alternative therapeutic regimens result in a consistent return to racing without re-injury or injury to another soft tissue structure. Tradition is strong, and most newer surgical and medical treatment regimens are expensive, even though the prognosis remains guarded.

Counterirritation promotes angiogenesis and when used, exercise is restricted. In a retrospective study of 54 TB and STB racehorses treated with pin firing or external blistering and given more than 6 months out of training, 23 (43%) returned to race at least once.[12] Ten (19%) of these horses returned to racing without experiencing re-injury to the tendon. These data indicate that racehorses treated by counterirritation can return successfully to racing, and so counterirritation will continue to be used until alternative methods prove to have significant and consistent improved athletic outcomes. If counterirritation is the treatment option selected by the trainer or owner, athletic outcomes may be improved if treatment is combined with a controlled exercise program and serial ultrasonographic examinations. We recommend re-examination every 2 months to evaluate the quality of repair and to determine if the current exercise program is excessive for any given stage of healing.

Intralesional β-Aminoproprionitrile Fumarate and Controlled Exercise

β-Aminoproprionitrile fumarate is a toxic substance found in the seeds of the plant *Lathyrus odoratus* (sweet pea). If β-aminoproprionitrile fumarate is injected into an injured tendon 30 to 90 days after injury, it binds to the enzyme lysyl oxidase and inhibits the deamination of lysine. This temporarily blocks cross-linking between collagen fibers and improves the quality of repair, if combined with controlled exercise, to generate piezoelectric forces and encourage axial alignment of the repairing collagen fibers. In a naturally healing tendon the collagen fibers may be aligned randomly, whereas treatment with β-aminoproprionitrile fumarate may encourage parallel alignment of the fibers, resulting in a stronger, more physiological repair. Treatment with β-aminoproprionitrile fumarate combined with a controlled exercise program resulted in superior healing, with more type I collagen fibers and improved longitudinal fiber alignment compared with those treated without exercise.[13] β-Aminoproprionitrile fumarate does not increase tenocyte formation and only affects the orientation of the scar and does not hasten repair. Theoretically intralesionally administered β-aminoproprionitrile fumarate is indicated for horses with moderate to severe injuries, when a large volume of the tendon will be repaired by scarring.

β-Aminoproprionitrile fumarate is administered 30 to 90 days after injury and is used at a rate of 1 ml (0.7 mg) per 3% total lesional area, up to a maximum of 10 ml (7 mg). Pretreatment quantitative ultrasonographic evaluation is important for establishing the anatomical extent of the lesion to be treated and the dose required.

Several clinical trials with β-aminoproprionitrile fumarate have been performed. Genovese reported an improvement in quantitative ultrasonographic morphology.[14] In a study in the United Kingdom, six of seven TB flat racehorses failed to complete one race,[3] but a trial in the United States determined that 50% of treated horses returned to racing and completed at least five races.[15] Clinical trials performed in Ohio compared the results of β-aminoproprionitrile fumarate treatment (75 horses) with a placebo (10 horses). All horses followed a similar exercise program. In phase 1, horses were out of training for less than 6 months, but the rate of re-injury was high. In phase 2, convalescence was increased, based on the ultrasonographic grading of the severity of the injury. Horses with a category IV lesion had at least 6 months out of training; this was extended to 8 months for category V lesions, and 10 months for category VI injuries. In phase 2 the success rate increased (Fig. 70-5).

Thirty-seven percent of treated horses with moderate (category V) or severe (category VI) injuries returned to racing without recurrence of injury; 57% failed to race once. None of the placebo group returned to racing without recurrent injury

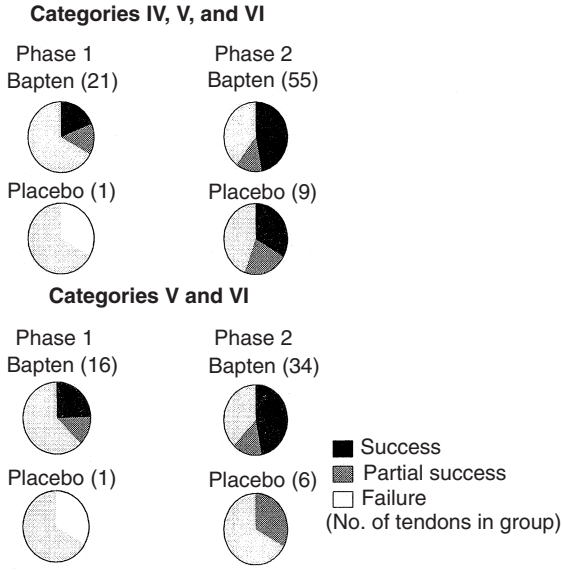

Fig. 70-5 Superficial digital flexor tendon injuries treated with Bapten were compared with placebo groups in two phases of the study. Phase 1 required less than 6 months of layup time, whereas phase 2 studies increased the convalescent time to at least 6 months for category IV injuries, 8 months for category V, and 10 months for category VI injuries. The percent of the total number of tendons was divided into three groups: success (raced at least five times after injury), partial success (raced one to four times), and failure (never raced). In phase 1, injuries including mild (category IV), moderate (category V), and severe (category VI) were grouped together to determine outcome; in phase 2 only moderate and severe categories were grouped.

and 71% failed to race once. Although slight (category IV) and moderate (category V) tendon lesions may appear healed clinically and by ultrasonography (with qualitative and quantitative assessment) 4 to 5 months after treatment, the strength of repair is inadequate to permit return to full training, and further time is essential for collagen maturation and remodeling.

Improvements in management have resulted in increased success with the use of β-aminoproprionitrile fumarate. Racehorses with career-ending category VI injuries also have been rehabilitated for low-level dressage or eventing, or as show hunters or show jumpers.

Intralesional or Perilesional Administration of Hyaluronan

Hyaluronan is a component of the ground substance of the tendon, directly influences collagen fibril formation and aggregation, and stimulates fibrillogenesis of type 1 collagen. Hyaluronan may decrease adhesion formation during tendon repair. In a clinical field trial, treatment of horses with acute anechoic tendon lesions with a single intralesional injection of high molecular weight hyaluronan (Hylartin-V) was compared with a saline placebo. Ultrasonography showed 60% of lesions resolved in the hyaluronan group compared with 24% resolved in the placebo-treated group, but athletic outcome results were not reported.[16] A separate long-term study comparing re-injury rate up to 2 years after treatment with intralesional high molecular weight hyaluronan combined with a controlled exercise program or controlled exercise alone found no significant difference in recurrence of injury. Neither study reported any adverse reaction to intralesionally administered high molecular weight hyaluronan.[16,17]

Corticosteroids

Repeated treatments with corticosteroids may impair tendon healing, but many veterinarians have anecdotally reported that low doses of corticosteroids combined with hyaluronan can be used successfully to manage category III/IV peripheral SDFT lesions in STB racehorses. This practice seldom is used in TB racehorses because of the concern of acute breakdown, an uncommon occurrence in the STB racehorse. A single perilesional injection of 6 to 9 mg triamcinolone with 10 to 20 mg hyaluronan is suggested. Methylprednisolone is not recommended, because it may be associated with development of dystrophic mineralization. It is important to note that continued exercise during this treatment may lead to more serious injury. Therefore close clinical and ultrasonographic monitoring, modification of training schedules (downscaled), and spaced, selected races for optimal results are recommended.

Intralesional and Systemic Administration of Polysulfated Glycosaminoglycans

PSGAGs are reported to inhibit macrophage activation and collagenase and metalloproteinase activity and therefore may be useful in the acute stage of tendon injury or re-injury.[2] The suggestion has been made that PSGAG may stimulate tenocyte repair. However, in a long-term clinical study, no significant difference was found in the recurrent injury rate of tendons treated with systemic or intralesionally administered PSGAGs compared with controlled exercise alone.[17] However, because tendon injuries are potentially career threatening, one author (R.L.G.) often recommends weekly systemic administration of PSGAGs for 4 to 6 weeks for horses with acute injuries of less than 8 weeks' duration. This is combined with other long-term rehabilitation procedures.

Physical Therapies

Physical therapeutic approaches for long-term rehabilitation of a tendon injury, such as therapeutic ultrasound, low-frequency laser, extracorporeal shock wave therapy, cryotherapy, and electromagnetic field therapy frequently are used by owners, but no clinical studies document their therapeutic value over conservative, controlled exercise management.

Other Proposed Long-Term Treatments

Use of insulin-like growth factor-1 and transforming growth factor β-1 are two intralesional hormonal treatments proposed to stimulate intrinsic healing of tendon injuries, with the overall goal to stimulate tenocyte replication and production of type 1 collagen. Clinical trials in racehorse tendon injuries have not been reported. Research in these areas is promising and is greatly improving the biochemical understanding of tendon injury and the biomechanics of repair.

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SURGICAL MANAGEMENT OF SUPERFICIAL DIGITAL FLEXOR TENDONITIS

• Mike W. Ross

TRANSECTION OF THE ACCESSORY LIGAMENT OF THE SUPERFICIAL DIGITAL FLEXOR TENDON

Since Bramlage first described transection of the accessory ligament of the superficial digital flexor tendon (ALSDFT), also known as superior or proximal check desmotomy, as a novel surgical treatment for tendonitis of the SDFT, there has been controversy regarding efficacy of the procedure.[1-6] Early, optimistic results were reported in TB racehorses and included 32 of 36 horses (89%) that returned to racing and 25 horses that competed at a level equal to or above the pre-injury level.[3] The results of that study were criticized because criteria for success, defined as completing two races and starting a third, were lenient, and horses developing contralateral limb lameness, including tendonitis, were excluded. Recently, results from a larger group of TB racehorses, using a more strict definition of success, revealed that 97 of 137 horses raced (71%), 70 horses (51%) made more than five starts after surgery, but average earnings decreased in 58% of horses. In that study the mean time from surgery to first start was 353 days.[4] The proposal was that the ALSDFT heals after transection but in an elongated fashion, causing an increased length of the bone-ligament-tendon-bone (radius-ALSDFT-SDFT-proximal and middle phalanges) construct.[5] In a smaller, separate study evaluating the long-term effects of transection of the ALSDFT and other treatments, 53% of flat racehorses, 58% of steeplechasers, and 73% of hurdlers competed in five or more races after surgery.[6] Clearly, with more strict definition of success, earlier results have been downgraded, but do appear superior to results from unmonitored conservative therapy alone. For instance, one estimate claims that only 20% of TB racehorses will compete three or more races after injury.[7] In a separate study of TB horses treated without surgery, 52% returned to racing, but 48% had recurrent tendonitis.[8] More recently, 20 of 28 (71%) TB racehorses were managed successfully with careful rehabilitation (controlled exercise, no turnout), serial ultrasonographic examinations, and a minimum of 8 to 9 months of rest.[9] Clearly, controlled exercise and careful clinical and ultrasonographic examinations are important components of any rehabilitation program. In contrast to other reported results a recent study found no difference in prognosis in TB racehorses treated with transection of the ALSDFT compared with conservative management and in fact found horses that underwent surgery to be at risk to develop suspensory desmitis.[10]

In the STB racehorse, results after transection of the ALSDFT are clearly superior to those achieved in the TB racehorse and published results in horses receiving only conservative management. In our study, 35 of 38 horses raced after surgery (92%), and 33 horses (87%) started more than five races, but tendonitis recurred in six horses.[11] Using a strict definition of success, 71% of horses started five or more times after surgery without recurrence of tendonitis, median earnings per start decreased significantly, and mean time from surgery to first start was 237 days. Suspensory desmitis developed in five horses, all of which had bilateral transection of the ALSDFT.[11] In a similar study, 82% of STB horses raced after transection of the ALSDFT, and 69% competed in five or more starts. In that study, horses that raced before injury had a better prognosis.[5] Published results of conservative management in STB racehorses are scant, but in one retrospective study, 31 (75%) of 41 STB racehorses completed two races and started a third, but tendonitis recurred in 43% of these horses.[12]

In summary, transection of the ALSDFT is clearly beneficial in the STB racehorse. Results of transection of the ALSDFT in other types of sport horses have not been published, but I suspect hunters, jumpers, event, and dressage horses have a prognosis somewhere between that published for the TB and STB racehorse. Unpublished results in the Three Day Event horse suggest that transection of the ALSDFT may be of limited value, although reasons for this are not known.[13] In a study of 33 horses in which 22 were non-racehorses, tendon splitting under ultrasound guidance combined in some horses with transection of the ALSDFT, resulted in 68% return to previous level of competition.[14] In the TB racehorse, results of transection of the ALSDFT may be superior to those achieved with intratendonous injections and uncontrolled rehabilitation, but are not as favorable as conservative management with controlled exercise.

Surgical Procedure

Currently, I prefer conventional surgery using a transthecal approach through the flexor carpi radialis tendon sheath.[3] Compared with the original description of transection of the ALSDFT, this modified approach allows the surgeon to close the incision in three layers, providing a much more secure closure. Transection of the ALSDFT is performed in a caudal location where the ligament is well defined. The surgical procedure is performed using a medial approach, with the horse in lateral recumbency (repositioned if bilateral transection of the ALSDFT is performed) without tourniquet application. The initial incision is made directly over or just cranial to the cephalic vein. The vein is carefully dissected free from underlying antebrachial fascia and retracted caudally. The cranial approach to the vein is less vascular than is the caudal approach, and in most horses a vein penetrating the antebrachial fascia is clamped and ligated. It is important to sever the ALSDFT completely, because incomplete division does not allow immediate transfer of load to the muscle and intuitively would promote faster healing of the structure after

surgery and reduce the possibility the ligament would heal in elongated fashion. To sever the ligament completely, carefully dissecting the proximal fibers from the nutrient artery and vein often is necessary, and in some horses these structures are cut inadvertently, or division is necessary. Division of the vessels causes no known clinical problem, but the vessel ends should be ligated to prevent edema and seroma formation.

Often, the proximal aspect of the carpal canal is penetrated, because the ALSDFT is attached to this structure distally. In horses undergoing transection of the ALSDFT an occasional complication is inadvertent transection of the nutrient artery. Although ligation of the vessel ends has no adverse clinical consequence, swelling in the SDFT seems to abate quickly after surgery, suggesting that hemorrhage may be an important factor early after injury. Complications with the surgical procedure are unusual, but the clinician should be aware of what normal healing of the surgical site entails. Nearly all sites develop diffuse swelling within 1 to 3 days after surgery, and in some horses hematoma or seroma formation occurs. The tendon region is vascular, and swelling is common and expected. If necessary, fluid can be evacuated, but this is seldom necessary. Firm, fibrous tissue forms at the surgical site by 4 to 6 weeks after surgery and may persist for several months. However, in most horses it is difficult to see residual swelling by 6 to 8 months after surgery, and cosmetic appearance is acceptable, if not normal, thereafter.

Transection of the ALSDFT can be accomplished using an endoscopic approach through the carpal sheath. A lateral approach is preferred, but the surgical procedure could be done with the horse in dorsal recumbency if bilateral transection of the ALSDFT was necessary.[15] The ALSDFT can be seen through the sheath and severed, but problems with hemorrhage from transection of the nutrient vessel can be encountered, and whether the entire ligament has been transected is sometimes unclear. Some surgeons prefer the endoscopic approach to avoid swelling that occurs with conventional surgery, but I feel this concern is overrated. Although this approach may be considered more elegant than conventional surgery, I prefer the conventional approach described previously.

How Transection Works

Originally, transection of the ALSDFT was thought to reduce tendon strain, thereby reducing the risk of recurrence of tendonitis. If a gap remained in the ALSDFT after transection, one could reasonably assume load was transferred to the superficial digital flexor muscle. In this case the muscle rather than the inelastic scar within the tendon could stretch, thereby protecting the healed portion of tendon. Experimental evidence in cadaver specimens suggests that after transection of the ALSDFT, load is transferred immediately to the muscle, but tendon strain increases because of a decrease in the metacarpophalangeal joint angle (hyperextension).[16] It has been proposed that the ALSDFT likely heals after transection, but in an elongated fashion, allowing increased length of the bone-ligament-tendon-bone axis, rather than replacing this load bearing axis with muscle. This in theory would increase elastic limit of the damaged tendon and negate the intrinsic loss of elasticity found in healed but scarred tendon.[5] I was initially skeptical of the mechanical effects of transection of the ALSDFT, but immediately after surgery horses may exhibit back-at-the-knee conformation (calf knee). Circumstantially the fact that suspensory desmitis occurs in horses after transection of the ALSDFT implicates a shift in distribution of load from the SDFT to the suspensory ligament.[10,11] Although suspensory desmitis is a serious soft tissue injury and possibility of suspensory injury has altered recommendations for transection of the ALSDFT, I do not consider the risk of suspensory desmitis after surgery a reason to abandon the procedure in certain sport horses.

When Transection Should Be Performed

I used to perform transection of the ALSDFT with the assumption that the procedure was useful in preventing recurrence of tendonitis, and no attempt was made to perform surgery immediately after discovery of tendonitis. However, my current recommendation is that the procedure be done as early as possible after injury. Obvious, visible reduction in tendon size occurs in the first 5 to 10 days after surgery, and although I cannot substantiate this claim, the procedure may be important in limiting or reducing inflammation and thus improve the quality of early healing. Immediate reduction of load on the damaged tendon may explain decreased swelling, but possibly transection of the ALSDFT may function to decompress the injured tendon or may alter blood flow. Reduction in tendon swelling is most marked in horses undergoing transection of the ALSDFT and annular desmotomy.

Patient Selection for Transection

Racehorses with mild, diffuse tendonitis or those with core lesions involving less than or equal to 10% of the CSA of the tendon likely will heal with conservative management and generally are not considered surgical candidates, unless injury is recurrent. Racehorses with recurrent diffuse tendonitis, severe diffuse tendonitis, or core lesions involving 10% to 15% or more of the CSA of the tendon are surgical candidates. In other sport horses, transection of the ALSDFT is recommended in the affected limb in upper-level performance horses using similar guidelines as racehorses, but in lower-level horses conservative management of more severe injuries is often successful.

Bilateral Transection

Bramlage's early results using transection of the ALSDFT in TB racehorses were complicated by the development of superficial digital flexor tendonitis in the contralateral limb.[2,3] In any racehorse, bilateral tendonitis is not uncommon. Ultrasonographic evaluation of young racehorses with obvious tendonitis in one limb often reveals mild tendonitis in the contralateral limb, and I suggest that both limbs be examined carefully before surgery. These facts led me to consider performing transection of the ALSDFT in racehorses bilaterally, even when the procedure was done prophylactically. However, horse's age, existence of concomitant suspensory desmitis in the contralateral limb, gait (in STB racehorses), type of sport horse, and cause of tendonitis play a role in decision making. Bilateral transection of the ALSDFT is recommended in horses with bilateral tendonitis, young 2- or 3-year-old TB or STB racehorses with unilateral tendonitis in which the contralateral limb is operated on prophylactically, or in horses with subtle ultrasonographic evidence of contralateral tendonitis. Unilateral transection of the ALSDFT (surgical procedure performed in affected limb only) is recommended in show horses; in young racehorses, particularly STB racehorses with suspensory desmitis in the contralateral limb; or in horses with tendonitis caused by direct trauma (tendonitis is unlikely to occur in the contralateral limb), such as a bandage bow. In older STB racehorses I often recommend contralateral transection of the ALSDFT in pacers, but not in trotters, because the prevalence of superficial digital flexor tendonitis is much lower in trotters.

Aftercare

Horses are given 2 weeks of absolute stall rest, followed by 6 weeks of stall rest with an increasing hand walking program, beginning with 10 to 15 minutes twice daily. After 8 weeks, an additional hand walking program, walking in the jog cart (STB racehorses), or swimming physiotherapy is recommended for an additional 8 weeks. Turnout exercise is not permitted. Horses then are placed back into early training by trotting and light galloping (TB) or by walking and light jogging (STB).

Serial ultrasonographic examinations should be performed each time an incremental increase in exercise is planned or when clinical evidence of mild inflammation exists. Time to first start varies considerably but in TB and STB racehorses is between 11 and 12 months and 8 and 9 months, respectively.

Large, full-limb support bandages are considered important in limiting motion and swelling at the surgical site(s) and are maintained and changed as needed for a minimum of 14 days. Once bandages are removed, swelling inevitably occurs at the site(s) of transection of the ALSDFT. An inner, lighter bandage is covered by a heavy cotton, full-limb dressing. Concomitant systemic use of PSGAGs or hyaluronan is controversial, and clear benefits have not been demonstrated, but the use of these medications makes sense to me theoretically. Eight weekly injections of PSGAGs are recommended. Dramatic clinical improvement in tendon size occurs when transection of the ALSDFT is combined with other surgical procedures, or in horses in which peritendonous injection of corticosteroids or a combination of corticosteroids and other anti-inflammatory products is used adjunctively with surgery. Phenylbutazone (4.4 mg/kg, intravenously or PO, twice daily for 10 to 14 days) administration appears useful in reducing swelling and improving comfort after surgery.

ANNULAR DESMOTOMY

Recently in equine literature much attention has been given to transection of the ALSDFT and tendon splitting, but little to no mention has been made of annular desmotomy. This procedure remains a most useful surgical treatment, usually combined with transection of the ALSDFT, to manage distal tendonitis of the SDFT. Two distinct clinical situations occur in which annular desmotomy is recommended. The most successful is adjunct use of annular desmotomy in horses with superficial digital flexor tendonitis. The palmar annular ligament (PAL) is a passenger in the disease process, which results in annular constriction of the SDFT. As the SDFT enlarges, the PAL impedes gliding function of the tendon. Further tendon enlargement and inflammation may cause thickening of the PAL, but the primary disease process involves the SDFT, not the PAL or DFTS. The PAL often becomes thickened and adheres to underlying DFTS and SDFT. Once the PAL impedes SDFT function, lameness, continued swelling, and inflammation occur. Annular desmotomy usually is combined with transection of the ALSDFT but can be a career-saving procedure when done alone. Annular desmotomy provides immediate decompression and improved gliding function of the SDFT, which are important, time-honored surgical principles to follow. The decision to transect the PAL is based on clinical observations. Typically a notch appears along the palmar aspect of the limb when viewed from the side, at the location of the PAL. Even if impingement is not severe, I recommend annular desmotomy. Viewed with ultrasonography the PAL may be normal to moderately thickened and adhesions may exist, but actual evidence of PAL desmitis is lacking. The SDFT is abnormal. After annular desmotomy a reduction in tendon size is seen within 5 to 10 days, and clinically initial swelling appears to redistribute proximal to the PAL and distal to the level of the surgical site. The PAL likely reforms in an elongated fashion after surgery but well after tendon size has decreased. Adhesion formation between the PAL, DFTS, and SDFT may occur after surgery, and inadvertent damage to the edge of the SDFT is possible, but the benefits appear to outweigh the risks of the procedure.

The second and less successful clinical situation in which annular desmotomy is used is in horses with chronic tenosynovitis without tendonitis of the SDFT, in horses with tendonitis of the deep digital flexor tendon, or in those with rarely seen primary palmar annular desmitis (see Chapter 75).

I prefer a minimally invasive conventional surgical approach. A 1.5-cm stab incision is made medially (when used with transection of the ALSDFT) just proximal to the PAL through skin, subcutaneous tissues, and DFTS, but care should be taken to avoid incision of the medial (lateral) aspect of the SDFT. A curved bistoury (Sontec Instruments, Englewood, CO) or one blade of a straight Mayo scissors is inserted deep to the PAL, superficial to the SDFT. The PAL is incised in its entirety, and care must be taken to transect the distal aspect completely. Reversing the bistoury or scissors and transecting the fibers of the PAL and thickened DFTS proximal to the incision may be necessary. The small incision then is closed using subcutaneous and skin sutures. Occasionally a larger incision is necessary in horses with severe and chronic tendonitis, because adhesions preclude accurate insertion of the bistoury. Care after surgery is the same as described for transection of the ALSDFT when the procedures are done simultaneously, but if annular desmotomy is performed alone, an accelerated exercise program often is advised. Long stall rest periods after annular desmotomy may promote earlier healing of the severed PAL and adhesion formation. After an initial 2-week period of stall rest, hand walking is given for 2 weeks and the tendon is re-evaluated. Acceleration of the exercise program at this point may be recommended depending on factors such as horse's value, class of racing, time of season, and owner's and trainer's proposed schedule.

Other surgical approaches, including an extended open approach or an open approach without entering the DFTS, have been described, but I do not recommend them. The approach described previously can be done with horses in the standing position or while they are under general anesthesia. Endoscopic examination of the DFTS and transection of the PAL have been described elegantly (see Chapters 24 and 75). This is a novel visually pleasing surgical procedure, but in my hospital it is expensive and time consuming to perform.[17] Complications with any approach include incisional dehiscence, delayed healing, infection, and damage to the ipsilateral edge of the SDFT. Horses with severe tendonitis in which the SDFT is severely enlarged and those with extensive adhesions and thickening of the annular desmotomy and DFTS are at most risk for complications.

TENOSCOPY OF THE DIGITAL FLEXOR TENDON SHEATH AND CARPAL SHEATH

See Chapter 24 for a discussion of tenoscopy of the digital flexor tendon sheath and carpal sheaths.

PROXIMAL METACARPAL FASCIOTOMY/CARPAL RETINACULAR RELEASE

For horses with proximally located superficial digital flexor tendonitis an effective method to decompress the damaged, enlarged SDFT and improve gliding function is to transect the proximal metacarpal fascia and carpal retinaculum. Clinical diagnosis in horses with only proximal superficial digital flexor tendonitis can be challenging because swelling and pain can be easily missed during palpation. Lameness is often inappropriately severe based on the minimal clinical signs present, horses often stand with an over-at-the-knee conformation (bucked knee), a positive response to carpal flexion is manifested, and horses may have mild carpal tenosynovitis. Horses with severe mid to distal superficial digital flexor tendonitis may have proximal involvement, and fasciotomy/carpal retinacular release appears beneficial. Old horses with inexplicably severe superficial digital flexor tendonitis often have extension of tendonitis to the region of the carpal canal and may have moderate to severe carpal tenosynovitis. Carpal retinacular release is

described for use in horses with carpal tenosynovitis, but it can be useful in horses with superficial digital flexor tendonitis in which transection of the metacarpal fascia is done concomitantly. This procedure most often is performed in combination with transection of the ALSDFT but can be combined with annular desmotomy also. Prognosis in horses with superficial digital flexor tendonitis in which transection of the ALSDFT and fasciotomy/carpal retinacular release are performed is guarded to good. Prognosis in old horses requiring transection of the ALSDFT, fasciotomy/carpal retinacular release, and in some annular desmotomy is better than expected, because swelling and lameness are often severe in these horses. Substantial reduction in tendon size and lameness score after surgery can be expected, and some horses have returned to light riding or field hunting.

Fasciotomy/carpal retinacular release is done using conventional surgical techniques and usually via a medial approach, because the procedure often is combined with transection of the ALSDFT. A 2- to 3-cm incision is made through skin and subcutaneous tissues just dorsal to the cephalic vein at the level of the proximal aspect of the second metacarpal bone. Because underlying tendonitis is present, bleeding can be excessive. A 1-cm incision is made in the dense, underlying metacarpal fascia, and a straight Mayo scissors is used to extend the fasciotomy proximally, through carpal retinaculum to the level of the distal aspect of the accessory carpal bone and distally to the mid-metacarpal region. No distinction is perceivable, between fascia and retinaculum because the tissues blend together. Subcutaneous tissues and skin are closed routinely. Bandaging and care after surgery are the same as given for transection of the ALSDFT, because the procedure usually is performed in combination.

TENDON SPLITTING

Interest in tendon splitting as an adjunct method to manage superficial digital flexor tendonitis has been renewed. Originally the procedure was developed to promote vascularization of the tendon in horses with chronic tendonitis, and early results showed promise.[18] Experimental studies questioned the value of tendon splitting, and the conclusion was that splitting induced excessive granulation tissue and slow healing of areas of tendon necrosis.[18,20] Clinical use of the technique then fell out of favor, but renewed interest was sparked by reports of combined use of splitting with transection of the ALSDFT in a clinical study and improved healing and revascularization of acute collagenase-induced tendon injuries in an experimental study.[21,22] The collagenase model produces severe tendonitis with extensive necrosis, and I question the value of this model in mimicking the naturally occurring disease. Various authors have reported clinical experiences with tendon splitting used with other procedures, but clinical studies using splitting alone are lacking.[4,5,11,14] Tendon splitting currently is used in horses with core lesions early in the disease process rather than in horses with chronic tendonitis, the group for which the procedure was designed originally. Tendon splitting, done to decompress areas of hemorrhage, or to provide vascular access channels early after injury, makes theoretical sense for horses with anechoic lesions, but it is of questionable value if done once granulation tissue has formed or mature collagen fibers exist. The rationale given for use in early lesions is to decompress the area of hemorrhage and to provide vascular access channels to improve vascularization by vessel ingrowth. Soon after injury areas of hemorrhage become granulation tissue in which the value of decompression would be minimal. The surgical procedure would have to be performed before granulation tissue develops (3 to 5 days, but certainly before 2 weeks) and should be reserved for horses with anechoic lesions. Damage caused to peritendonous tissues and surrounding intact tendon fibers must be considered and may outweigh any benefit. However, the procedure has been done apparently successfully or at least without outward harmful effects.

In clinical practice I use the procedure when requested by referring veterinarians or in TB racehorses with anechoic core lesions. The percutaneous technique is preferred, and using a double-edged tenotome or a No. 11 scalpel blade is recommended. The procedure seldom is used in the STB racehorse, and in our report detailing results of transection of the ALSDFT in this racing breed, tendon splitting was done in only one horse.[11] In STB racehorses managed with transection of the ALSDFT, I recommend annular desmotomy, but not tendon splitting. Currently I recommend that the procedure be done in TB racehorses or other non-STB sport horses with transection of the ALSDFT. Needle decompression of anechoic lesions early after injury may be of value to provide early decompression, but creation of vascular access channels using this method appears implausible.

COMBINED SURGICAL PROCEDURES

Because superficial digital flexor tendonitis is career limiting in many sport horses, early and aggressive surgical management should be considered. Potentially, injection of growth factors, liquid bone marrow, or other substances used with surgery and strict rehabilitation may offer the best hope for a successful outcome. Combined surgical management using transection of the ALSDFT and annular desmotomy; transection of the ALSDFT, annular desmotomy, and fasciotomy/carpal retinacular release; or in some horses various combinations with tendon splitting has been successful even in horses with severe tendonitis. In my experience, using transection of the ALSDFT without annular desmotomy in horses with distal superficial digital flexor tendonitis often results in failure, as does using transection of the ALSDFT without fasciotomy/carpal retinacular release in those with lesions involving the proximal aspect of the tendon.

The ideal management program for horses with superficial digital flexor tendonitis has yet to be discovered but should rely on the principles of tendon healing, including minimizing peritendonous scar tissue formation, minimizing the effect of hemorrhage and subsequent granulation tissue formation and disorganized fiber alignment, maximizing gliding function within and external to the SDFT, reduction of load and protection of inelastic scar tissue by increasing the length of the bone-ligament-tendon-bone construct, and an exercise regimen that allows healing and maturation of collagen fibers without deleterious effects of uncontrolled exercise. Surgical management preserves many of these time-honored principles and has proved useful in many types of sport horses.

Treatment of tendon injuries in racehorses is a challenge to clinicians and researchers. Return to racing is associated with a high rate of recurrent injury. However, much progress has been made in the past 2 decades in diagnostic identification and classification of injury, and the biochemical and biomechanical aspects of tendon injury. Treatment and management programs by necessity depend heavily on economics and trainer decisions relative to continued exercise or long-term rehabilitation. Of vital importance is accurate injury severity categorization, controlled exercise in long-term rehabilitation programs, and ultrasonographic monitoring at all levels of treatment. Improved athletic outcome results gradually evolve as continued basic science research and clinical investigations of past and proposed therapeutic regimens unfold.

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SUPERFICIAL DIGITAL FLEXOR TENDONITIS IN EVENT HORSES, SHOW JUMPERS, AND DRESSAGE HORSES

• Sue J. Dyson

Superficial digital flexor (SDF) tendonitis is a potentially career-limiting injury in event horses and Grand Prix–level show jumpers. Although the incidence of SDF tendonitis is much lower in dressage horses, it is also an important injury. The clinical manifestations vary considerably.

SUPERFICIAL DIGITAL FLEXOR TENDONITIS IN EVENT HORSES

Clinical Signs

Although all event horses are at risk of injury of the SDFT, the incidence of injury is highest in horses competing at advanced or international level, especially those competing in Three Day Events. This is probably due to the combination of galloping long distances and jumping on very variable terrain and footing. After training or competing, riders commonly apply a proprietary clay to the forelimbs and bandage the limbs at least overnight. This practice may result in early warning signs of impending tendon damage being missed. Because lesions frequently occur bilaterally, subtle changes in limb temperature may be missed except by the most astute and vigilant riders or grooms.

The clinical signs associated with tendon damage vary markedly. A horse may pull up lame after the cross country phase of an event and rapidly develop peritendonous soft tissue swelling and pain on palpation of the tendon. In some horses the degree of pain is severe and the horse may be distressed and reluctant to bear weight on the limb. If lesions occur bilaterally, the horse may be reluctant to move and its behaviour may mimic the appearance of a horse that is tying up. These horses may require both analgesia and sedation to relieve the distress. Clinicians should be aware that a horse may appear very lame, but it may not be possible to elicit pain by palpation of the tendon, although the tendon may feel abnormally soft.

In contrast a horse may complete the cross country phase and appear sound, with lameness developing several hours later. Although soft tissue swelling may develop, in some horses there may be absolutely no clues as to the cause of lameness (i.e., no heat, pain, or swelling). This may persist for many days, and then suddenly enlargement, subtle or obvious, of the SDFT may be seen despite resolution of lameness.

In other horses, lameness is never present. The horse initially may exhibit poor performance and clinical examination may reveal that the SDF tendons are slightly enlarged. Alternatively, the horse may have localized heat and pain on pressure applied to the SDFT, with no history of lameness. Some horses have obvious clinical signs of SDF tendonitis after the first training gallop, cross country schooling, first event, or following a period of reduced work for some months after a Three Day Event. It seems highly likely that these horses sustained damage to the SDF tendon(s) at the Three Day Event without associated detectable clinical signs.

Clinical signs and the severity of the tendon injury are not necessarily correlated. The clinical signs of SDF tendonitis can easily be masked. I have examined a number of horses at events when the horses have finished lame and had clinical signs compatible with SDF tendonitis. The horses have been treated by application of a modified Robert Jones bandage and systemic NSAIDs. Ultrasonographic examination after 5 to 7 days has been recommended. The horse has then been examined by the owner's regular veterinary surgeon, who has been unable to detect any palpable abnormality after removal

of the bandage and therefore elected not to perform an ultrasonographic examination. Return to work has been recommended by the second veterinary surgeon with catastrophic consequences. Some horses that have completed the speed and endurance phase of a Three Day Event and sustained a tendon injury can be managed to pass the final horse inspection and complete the show jumping, without marked deterioration of the tendon injury.

Diagnostic Ultrasonography

In my opinion, whenever unexplained lameness occurs in an event horse, the SDF tendons should be examined ultrasonographically. I also recommend routine ultrasonographic examination 10 to 14 days after the horse completes a Three Day Event. Ultrasonographic examination should be mandatory for any horse with a history of slight heat or filling in the metacarpal region, unless there is an obvious cause (Fig. 70-6). However, it is equally important to recognize the constraints of ultrasonographic examinations, due both to the skill of the veterinarian and limitations of the resolution of ultrasonographic images. If a SDF tendon appears normal ultrasonographically, but the clinical signs are suspicious of injury, the horse should be treated as if it had a tendon injury, with repeat examination after a further 7 to 14 days.

In horses with early subtle tendonitis the only abnormality detectable may be slight, localized enlargement in CSA of the tendon, therefore area measurements and comparisons with the contralateral limb can be extremely valuable. If the injury is bilateral, subtle enlargement in both SDF tendons is easily missed. It is important to examine the relative sizes of the superficial and deep digital flexor (DDF) tendons and to be aware of the way in which a normal SDF tendon changes shape from proximally to distally in the metacarpal region (see Chapter 16).

It is also important to look carefully at the echogenicity of the SDFT and compare it with the DDFT and more proximal and distal sites within the SDFT. Subtle lesions often result in a slight diffuse reduction in echogenicity of part or all of the CSA of the tendon, but only in a localized region. Such lesions are easily missed, especially if the gain controls of the ultrasound machine are set too high or if the limb is examined without fine clipping of the hair coat. Detection of these early injuries is very important, because a horse can make a relatively rapid complete recovery at this stage, whereas continued work may result in a much more severe, career-limiting injury. In these early injuries, longitudinal images of the tendon often appear normal. Both forelimbs should be examined routinely.

Because lesions may be very localized, it is important to carefully and systematically examine the tendon from proximally to distally. Although gross core lesions of the SDFT are relatively easily identified without fine clipping of the hair coat, image resolution is inferior. Application of mineral oil helps to improve resolution, but this can destroy the ultrasound standoff pad. Subtle lesions will be missed unless the hair is clipped, although shaving is unnecessary. Owners may be reluctant to allow the limbs to be clipped, suggesting that this may jeopardize the horse's evaluation at an inspection at a forthcoming Three Day Event if the limbs actually appear normal and the horse is unable to compete. Owners must be persuaded that it is essential to clip the limbs and that only a narrow strip is necessary; the clinician should suggest that if the horse is fit to compete, the entire horse is then clipped.

Treatment

Management of SDF tendonitis in the event horse is difficult. Individual horses' capacities to recover from tendon injuries vary considerably. In some horses a marked improvement is

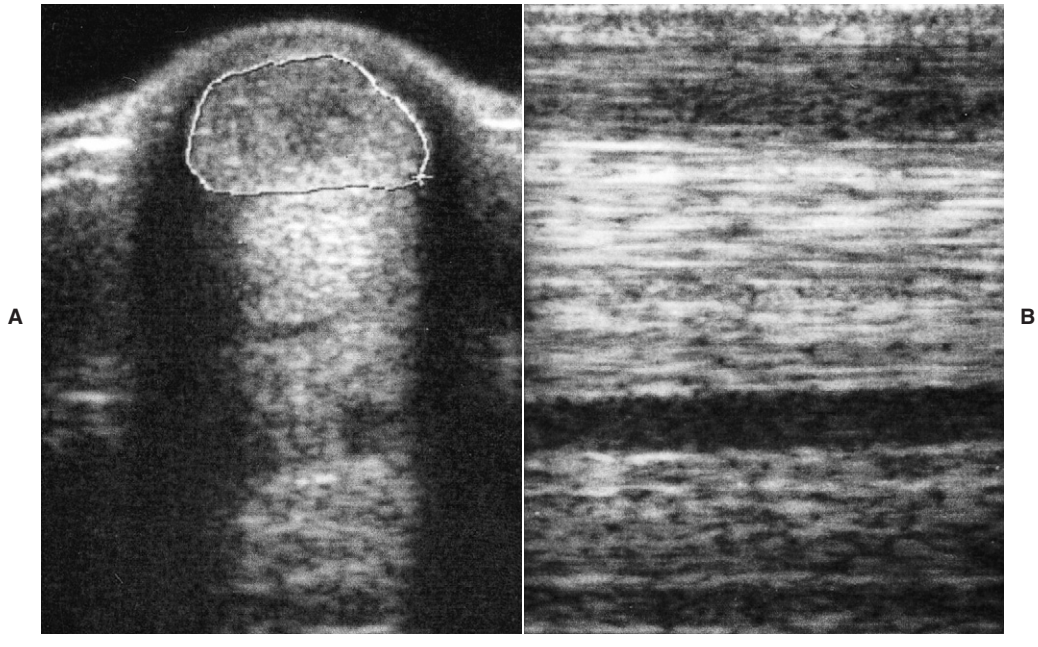

Fig. 70-6 **A,** Transverse ultrasonographic image of the palmar aspect of the right forelimb of a 9-year-old advanced event horse; the scan was obtained at 16 cm distal to the accessory carpal bone. Medial is to the left. The horse initially had slight heat in the metacarpal region but no lameness. The superficial digital flexor tendon (SDFT) is enlarged (cross-sectional area 1.2 cm^2), and there is a diffuse reduction in echogenicity with an acentric, more hypoechogenic region. The lesion extended from 12 to 24 cm distal to the accessory carpal bone. **B,** Longitudinal ultrasonographic image of zone 2B; proximal is to the left. There is diffuse reduction in echogenicity of the SDFT with loss of long linear echoes.

seen in the clinical and ultrasonographic appearance of the tendon within 3 months of first injury, whereas little progress is seen in other horses with a similar injury. In almost all horses a convalescent period of 1 year after injury is required. In some horses, normal echogenicity is never restored and a fairly obvious central hypoechoic area persists ultrasonographically. These horses may be able to withstand one day events, but frequently sustain re-injury at a Three Day Event. Lesions in the distal one fourth of the metacarpal region (zones 3B and 3C) are particularly at risk to re-injury. Even in those horses in which relatively normal echogenicity is restored, it is relatively unusual for them to be able to complete more than one or two Three Day Events without re-injury.

Most published studies of the treatment of SDF tendonitis relate to the racing TB and STB. It is generally accepted that the rate of re-injury is high, especially for horses competing in Three Day Events. Although a horse may be managed successfully to complete one Three Day Event after injury, the risks of subsequent injury are very high.

Of 23 event horses treated with a controlled exercise program alone, 57% were able to return to full athletic function without recurrent injury in a 2-year period after resumption of full work,[1] compared with 56% of 25 horses that received intralesional hyaluronan and the same controlled exercise program and 58% of 31 horses that received intralesional PSGAGs administered intralesionally, systemically, or by both routes. Almost all horses with a unilateral injury re-injured the same tendon.

The results of intralesional treatment with β-aminopropionitrile fumarate appear superficially to be slightly worse; in a 2-year follow-up period after return to full work, only 12 (52%) of 23 horses competed without re-injury.[2] Five (22%) horses re-injured the same SDFT. However, an additional 6 horses competed successfully and then injured either the SDFT of the contralateral limb (5) or the ALDDFT in the ipsilateral limb (1). Eight of the 12 successfully treated horses have completed three or more Three Day Events at championship level without recurrent injury. A further 6 (of 6) horses have been competing successfully for 12 to 18 months after treatment. Overall, in my experience, this treatment seems to offer the best long-term prognosis for event horses with moderate to severe injuries of the SDFT. After treatment, horses are walked for 30 to 60 minutes daily for 4 months and are then re-examined ultrasonographically. Successfully treated horses had a remarkably good strength of fiber pattern in longitudinal ultrasonographic images 4 months after treatment. This feature appears to be a good prognostic indicator. Work intensity is slowly and progressively increased thereafter, with horses generally reaching full work by 12 months after treatment.

Desmotomy of the accessory ligament of the SDFT has been less successful in the management of SDF tendon lesions in event horses than in the racehorse, and the disappointing results achieved in the author's clinic have led to this technique being abandoned. It has also fallen from favor in the United States.[3,4] Tendon splitting has been used for anechoic central core lesions, combined with a controlled exercise program with successful results, but the number of treated horses does not compare with the studies described previously.[2]

Regardless of the method of management, serial ultrasonographic examinations during the convalescent period seem to be the most accurate predictors of the final outcome. If the echogenicity and fiber pattern have improved markedly by 4 months after injury, then the longer-term prognosis is much better than in those horses where improvement is only slight. The strength of the fiber pattern when full work is resumed is a good predictor of whether the horse will sustain re-injury.[1] It does appear that some horses have an innately better ability to repair tendon lesions than others.

Pastern Lesions

Injury to the medial or the lateral branch of the SDFT in the pastern region occurs most commonly in forelimbs but also occasionally in hindlimbs (see Chapter 83). Such injury can occur as an isolated acute lesion or as a sequel to previous tendonitis in the metacarpal region. There is usually acute-onset, moderate lameness. In some horses, soft tissue swelling is immediately apparent on the palmar aspect of the pastern, but in others, obvious swelling may take several days to develop. In these horses, perineural analgesia may be required in the acute stage to localize the source of pain. Lameness is alleviated by perineural analgesia of the palmar nerves at the level of the proximal sesamoid bones. Diagnosis is confirmed ultrasonographically. The affected branch is usually enlarged with poor demarcation of its margins and diffuse or focal areas of reduced echogenicity. Horses with acute lesions with no preceding tendonitis have a fair prognosis for complete recovery after adequate rest for at least 6 months. However, horses with lesions secondary to tendonitis in the metacarpal region have a more guarded prognosis.

SUPERFICIAL DIGITAL FLEXOR TENDONITIS IN SHOW JUMPERS

Injury to the SDFT is comparatively unusual in show jumping horses, except those competing at international level, or horses approximately 15 years of age or older. Injury to the SDFT is most commonly unilateral. It may occur as a primary lesion or as a sequel to previous desmitis of the ALDDFT. Injuries occur most frequently during competition and may result in an acute, severe lameness with the horse abruptly stopping during a round. Alternatively, the horse may finish lame. Clinical signs of heat, swelling, and pain may or may not be obvious at the time. In some horses, SDF tendonitis is insidious in onset, with development of swelling and localized heat without recognition of any gait abnormality (Fig. 70-7, *A*). Occasionally traumatic injuries occur to the SDFT within the carpal sheath after the horses has sustained a fall (see Chapter 76).

Injuries restricted to the proximal one third of the metacarpal region are common, and there appears to be a tendency for the development of peritendonous adhesions and recurrent lameness. Horses with these lesions have been difficult to manage successfully, as are those with lesions that extend proximally into the carpal canal. However, horses with lesions restricted to the metacarpal region, without peritendonous adhesions, can be rehabilitated and returned to competition with judicious, symptomatic management relatively quickly compared with event horses with a comparable injury. Some horses can withstand return to full work, despite persistence of quite obvious ultrasonographic abnormalities of the SDFT. However, the tendon must be monitored carefully, because some horses continue to sustain recurrent low-grade injuries, which result in the tendon becoming progressively larger and wrapping around the DDFT and becoming contiguous with the ALDDFT. Severe injuries predispose to the development of secondary desmitis of the ALDDFT (see Chapter 72).

Desmitis of the ALDDFT may occur as a primary lesion. Severe lesions predispose to the development of secondary SDF tendonitis. Horses with these injuries are difficult to manage successfully. Marginal tears of the SDFT within the DFTS occur occasionally, presenting as an acute-onset lameness associated with distension of the DFTS (Fig. 70-7, *B*).

Fourteen (93%) of 15 show jumpers with unilateral SDF tendonitis that were treated conservatively after a controlled exercise program were able to return to full athletic function for at least 2 years without recurrent injury, compared with 4 (80%) of 5 horses treated with intralesional hyaluronan and

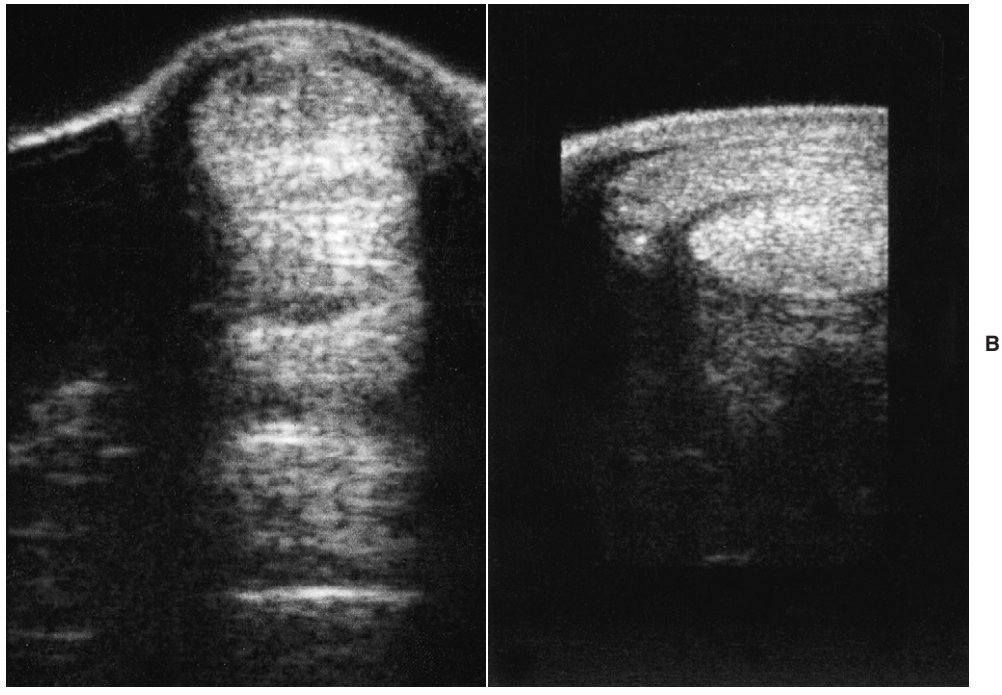

Fig. 70-7 **A,** Transverse ultrasonographic image of the palmar aspect of the left forelimb of a 13-year-old Grand Prix show jumper; the image was obtained at 11 cm distal to the accessory carpal bone. The horse initially had localized heat and swelling but no lameness. A focal hypoechoic lesion is seen on the palmar margin of the superficial digital flexor tendon (SDFT). The lesion extended less than 1 cm proximodistally. The horse's condition was managed symptomatically and the horse continued to compete successfully for 6 months with no change in the lesion; the horse then developed acute lameness with exacerbation of the injury. **B,** Transverse ultrasonographic image of the palmar aspect of the distal metacarpal region of a 14-year-old Grand Prix show jumper with acute-onset, severe lameness associated with distention of the digital flexor tendon sheath. Medial is to the left. The medial aspect of the superficial digital flexor tendon is enlarged, its margin is irregular, and there are focal anechoic lesions.

3 (75%) of 4 horses treated with PSGAG.[1] Five horses with severe lesions that had not responded adequately to conservative management were subsequently treated with β-aminoproprionitrile fumarate, and all have withstood return to international competition without recurrent injury.[2]

Older horses (>15 years of age) with SDT tendonitis generally carry a more guarded prognosis. Some of these injuries appear to be progressive degenerative lesions, which deteriorate despite rest.

SUPERFICIAL DIGITAL FLEXOR TENDONITIS IN DRESSAGE HORSES

SDF tendonitis is not a common injury in dressage horses but it does occur occasionally (Fig. 70-8). Most lesions occur either in the proximal one third of the metacarpal region or in the distal metatarsal and pastern regions of hindlimbs. Mild lesions in the distal two thirds of the metacarpal region are less common but are easily managed. The horse is restricted to stall rest and controlled exercise and usually resumes work after resolution of localized heat, pain, and peritendonous swelling. Lesions in the proximal one third of the metacarpal region are much more difficult to manage, and clinical signs resolve very slowly regardless of the method of management. Hindlimb lesions have been restricted to the DFTS and are sometimes bilateral. They often are associated with adhesion formation within the DFTS and tend to result in chronic lameness.

Some marginal tears of the SDFT are only recognized during exploratory tenoscopy of a distended DFTS. Concurrent tears of the manica flexuria have also been identified during tenoscopic evaluation. The results of debridement of marginal tears of the SDFT within the DFTS in dressage horses have been rather disappointing. Although lameness has often improved, minor career-limiting gait abnormalities have persisted.[2]

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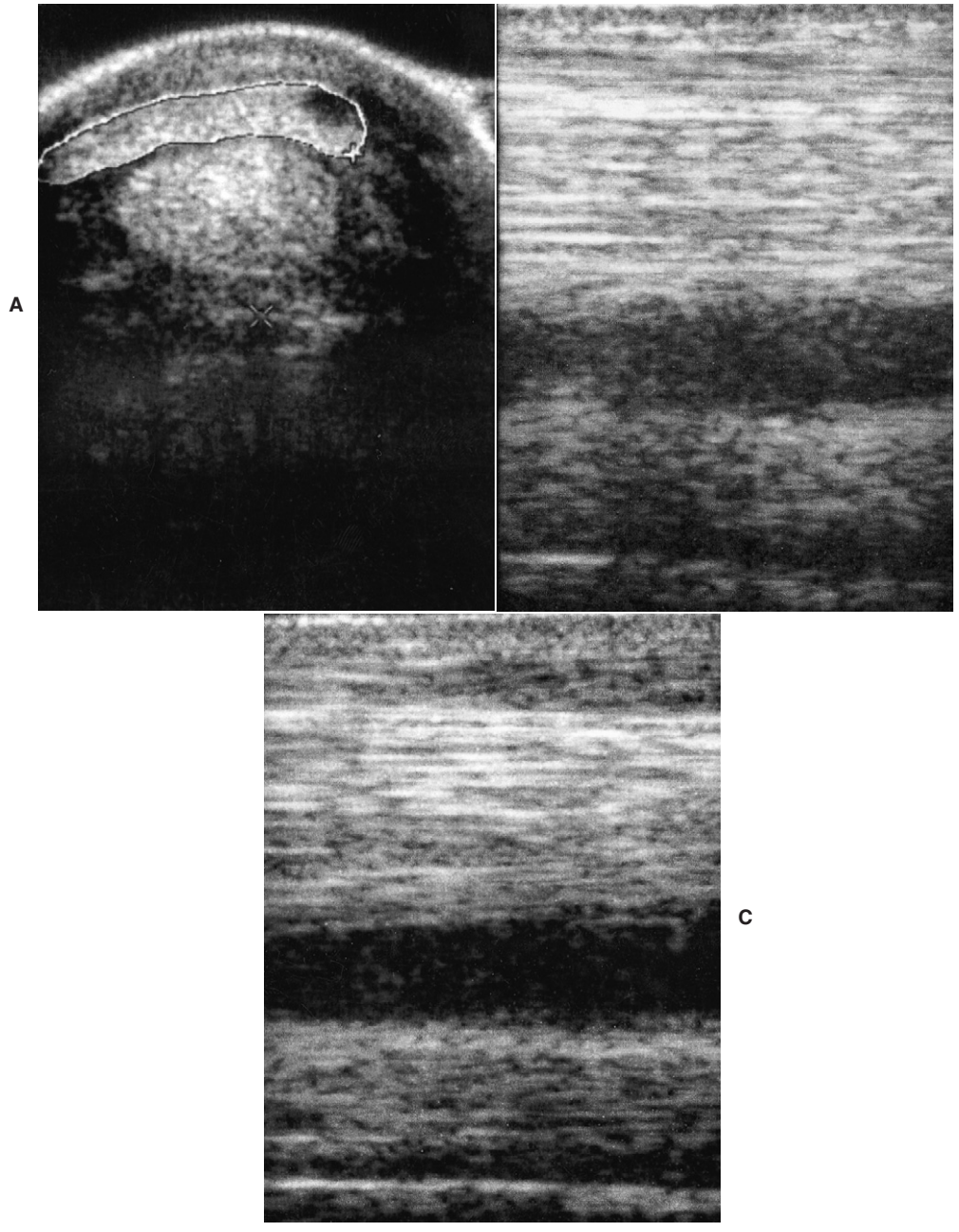

Fig. 70-8 **A,** Transverse ultrasonographic image of the palmar aspect of the left forelimb of a 12-year-old Grand Prix dressage horse. The image was obtained at 22 cm distal to the accessory carpal bone; medial is to the left. The horse had exceptionally extravagant forelimb paces and acute-onset, moderate lameness. There is a focal hypoechoic lesion on the lateral aspect of the superficial digital flexor tendon (SDFT). The lesion extended from 16 to 26 cm distal to the accessory carpal bone. At this level the tendon is slightly enlarged (cross-sectional area 1.2 cm^2), but at all other levels it was of normal size. **B,** Longtitudinal ultrasonographic image of zone 3A obtained from the palmar midline; proximal is to the left. The SDFT appears normal. **C,** Longitudinal ultrasonographic image of zone 3A obtained from the palmarolateral aspect of the metacarpal region shows reduced echogenicity of the SDFT.

CHAPTER • 71

The Deep Digital Flexor Tendon

Sue J. Dyson

ANATOMY

In the forelimb the deep digital flexor tendon (DDFT) has three heads: the humeral head, the largest, and the smaller radial and ulnar heads.[1] The tendon of the humeral head develops 8 to 10 cm proximal to the antebrachiocarpal joint, but muscular tissue persists until the level of the antebrachiocarpal joint, where the ulnar and radial heads join. The merged tendon is triangular in cross-section within the carpal canal but becomes more rounded in the metacarpal region. The accessory ligament of the DDFT (ALDDFT) merges with the DDFT in the middle third of the metacarpal region.

In the hindlimb the DDFT is formed by a large lateral digital flexor tendon and a smaller medial digital flexor tendon. The lateral digital flexor tendon incorporates the caudal tibialis tendon and passes over the sustentaculum tali within the tarsal sheath. The medial digital flexor tendon passes over the proximal tubercle of the talus, on the medial aspect of the talus, in its own synovial sheath. These two tendons fuse in the proximal metatarsal region. In the proximal metatarsal region the DDFT is a large oval-shaped structure that becomes smaller farther distally. The ALDDFT in the hindlimb varies in size and is generally comparatively smaller than in the forelimb.

At the fetlock region the DDFT becomes wider, elliptical, and fibrocartilaginous and is enclosed within the digital flexor tendon sheath (DFTS). In the pastern region the tendon becomes bilobed. At the level of the proximal part of the middle phalanx the dorsal part of the tendon becomes a fibrocartilaginous pad. Distally the DDFT is molded to the palmar/plantar aspect of the navicular bone. The DDFT is broad and has a terminal fan-like expansion containing cartilage and inserts on the facies flexoria of the distal phalanx, delineated dorsally by the semilunar line and the adjacent surface of the ungular cartilage.

The DDFT has a high modulus of elasticity (1585 MPa) and a considerable strength to rupture (approximately 1700 daN).[1] The ALDDFT has a low modulus of elasticity (490 MPa) and a moderate strength to rupture (approximately 490 daN). The DDFT limits carpal and fetlock extension under high loads. In the fetlock region the DDFT is under tension and compression. It is therefore fibrocartilaginous in this region and in the pastern, where the tendon is under pressure from the tuberositas flexoria, a transverse prominence on the proximal palmar aspect of the middle phalanx. In the digit the DDFT facilitates flexion of the proximal interphalangeal joint during weight bearing and stabilizes the distal interphalangeal joint.

The position of the DDFT and the navicular bone varies considerably during the stance phase. In the full weight-bearing position the DDFT is only in close contact with the distal border of the navicular bone, but during propulsion it comes into full contact with the palmar aspect of the bone. The tendon is stretched maximally as active contraction of the muscle bellies and the elasticity in the tendon result in elevation of the fetlock and extension of the distal interphalangeal joint. During the swing phase of the stride the DDFT relaxes. The DFTS facilitates displacement of the flexor tendons during flexion and extension.

DEEP DIGITAL FLEXOR TENDONITIS ASSOCIATED WITH RECURRENT DESMITIS OF THE ACCESSORY LIGAMENT OF THE DEEP DIGITAL FLEXOR TENDON

Injuries of the DDFT in the carpal or metacarpal region, proximal to the DFTS, are rare except in association with chronic desmitis of the ALDDFT (see Chapter 72). Recurrent desmitis may be accompanied by pathological lesions of the DDFT.[2] Because of the close proximity of the DDFT and its accessory ligament, it is difficult by palpation to assess each structure accurately, especially with chronic enlargement of the ALDDFT, which may wrap around the borders of the DDFT. Ultrasonographic examination may reveal slight enlargement of the DDFT. The dorsal border may be less well defined, and diffuse hypoechogenic regions may occur within the DDFT, extending a variable distance proximodistally. These injuries usually result in recurrent lameness.

It has also been noted that in association with substantial enlargement of the superficial digital flexor tendon (SDFT) because of chronic tendonitis, the DDFT becomes smaller in cross-sectional area.[3] With chronic enlargement of the ALDDFT, the DDFT may also reduce in size.

I have not examined a horse with primary deep digital flexor tendonitis in the proximal metacarpal region, but a single case was recorded by Genovese and Rantanen[4] in an 8-year-old Quarter Horse used for English pleasure riding. Occasionally traumatic injuries of the DDFT have been seen within the carpal sheath.

DEEP DIGITAL FLEXOR TENDONITIS IN THE CARPAL SHEATH SECONDARY TO SOLITARY OSTEOCHONDROMA

Lesions of the DDFT within the carpal sheath are an unusual cause of lameness except secondary to irritation by a solitary osteochondroma. An osteochondroma is an exostosis continuous with the cortex of the bone and is covered by cartilage. The osteochondroma develops immediately proximal to the distal radial physis, often medial to the midline. Lameness is sudden in onset and usually is accentuated by carpal flexion. Invariably distention of the carpal sheath occurs. An osteochondroma is readily identifiable radiographically. Ultrasonographic examination from the medial aspect of the distal antebrachium also reveals the abnormal bone contour, an abnormal amount of fluid within the carpal sheath, and an irregular dorsal contour of the DDFT. Treatment is by surgical removal of the osteochondroma and debridement of any torn fibers of the DDFT. The prognosis for return to athletic function is excellent.

DEEP DIGITAL FLEXOR TENDONITIS WITHIN THE DIGITAL FLEXOR TENDON SHEATH IN THE FETLOCK REGION

Some enlargement of the DFTS is common in hindlimbs, often unassociated with lameness, but occurs less frequently in forelimbs. Sudden-onset lameness associated with distention of a DFTS in a forelimb or a hindlimb may be caused by a variety of different lesions, but deep digital flexor tendonitis always should be considered[5,6] (see Chapter 75). It is rare to identify lesions of the DDFT within a DFTS that is not distended. Some horses develop deep digital flexor tendonitis *after* long-term chronic enlargement of the DFTS.

Lameness associated with DDFT lesions within the DFTS occurs more frequently in hindlimbs than in forelimbs and in horses from a variety of disciplines. The condition usually occurs unilaterally, although it has been seen bilaterally in the hindlimbs of several Warmblood dressage horses.[3]

Lameness varies from mild to moderately severe. Distention and thickening of the DFTS may make accurate palpation of the DDFT difficult. In some horses pain can be elicited by palpation of the margins of the tendon or by firm pressure being applied to its palmar (plantar) aspect. The tendon should be assessed throughout its length, proximal and distal to the fetlock. In the acute stage there may be localized heat. Passive flexion of the lower limb may induce pain. If forelimb lameness is only mild in straight lines, it may be exaggerated on the lunge on a soft surface, especially in medium and extended trot. Distal limb flexion often accentuates the lameness.

Intrathecal analgesia of the DFTS usually results in significant improvement but rarely alleviates lameness. Better improvement is seen after low palmar/low plantar perineural analgesia. In horses in which the metacarpophalangeal (metatarsophalangeal) joint capsule also is distended, performing intra-articular analgesia may be necessary to be sure that distention is not contributing to pain.

Definitive diagnosis requires ultrasonographic examination. Four types of lesions involving the DDFT have been identified: enlargement and change in shape of the tendon, focal hypoechoic lesions within the tendon or on its border, mineralization within the DDFT, and marginal tears.[5-7] The first three are readily diagnosed using diagnostic ultrasonography, but the marginal tears are much more difficult to identify. Surgical exploration may be required for definitive diagnosis.[8] Acute-onset focal hypoechoic areas generally are not seen with pre-existing adhesion formation, although any of the other lesions may be.

The normal DDFT changes in its shape and cross-sectional area from proximally to distally, but it is usually bilaterally symmetrical. The normal DDFT is uniform in its echogenicity, and its margins are clearly defined. Hypoechoic artifacts are induced readily in the distal fetlock and pastern regions if the ultrasound transducer is not perpendicular to the tendon, and in these regions evaluating the SDFT and DDFT simultaneously is difficult. Echogenic synovial plicae (mesotendon) extend medially and laterally from the DDFT to the DFTS wall in the proximal recess of the DFTS (Fig. 71-1, *A*). These are seen much more obviously when the tendon sheath is distended and should not be mistaken as marginal tears or adhesions. With chronic tenosynovitis, these plicae may become thickened. Distal to the fetlock is an echogenic palmar (plantar) synovial fold that should not be confused with an adhesion (Fig. 71-1, *B*). The ergot on the palmar (plantar) distal aspect of the fetlock prohibits ultrasonographic evaluation at this level and lesions of the DDFT may be missed.

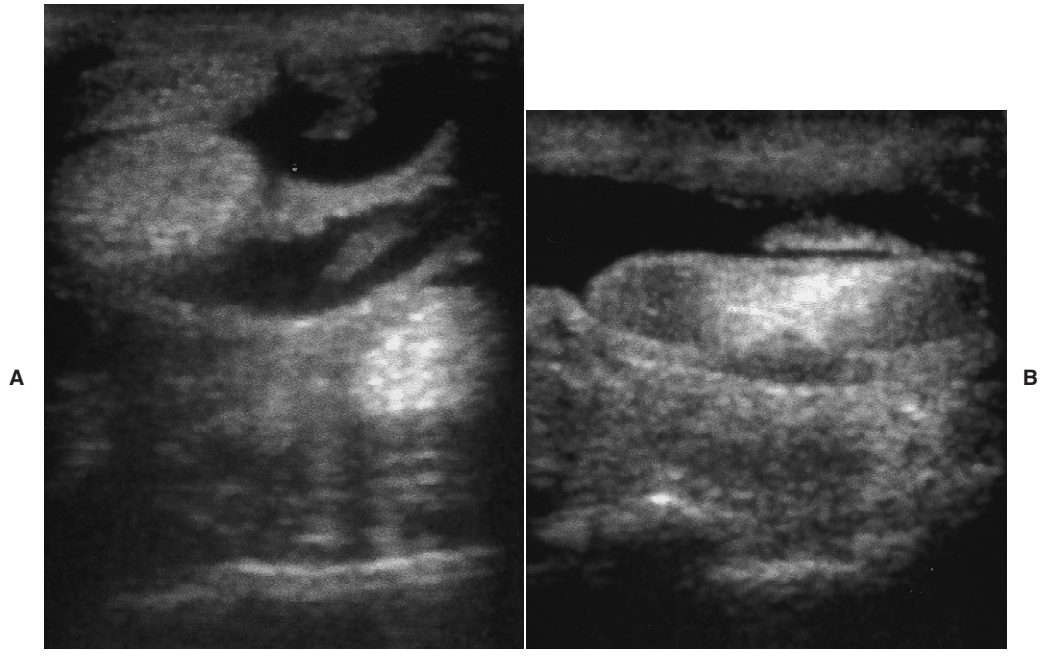

Fig. 71-1 Transverse ultrasonographic images of the digital flexor tendon sheath in (**A**) the distal metacarpal region and (**B**) the pastern. Medial is to the left. **A,** An abnormal amount of fluid is within the digital flexor tendon sheath, but the deep digital flexor tendon appears normal. Note the prominent synovial plica extending from the lateral aspect of the deep digital flexor tendon. This plica also is thickened slightly, and other echogenic material is within the anechogenic synovial fluid, but the horse had no associated lameness. **B,** Note the abnormal amount of fluid within the digital flexor tendon sheath, and a synovial plica is on the palmar aspect of the deep digital flexor tendon. These are normal anatomical structures that are visible most readily when the digital flexor tendon sheath is distended.

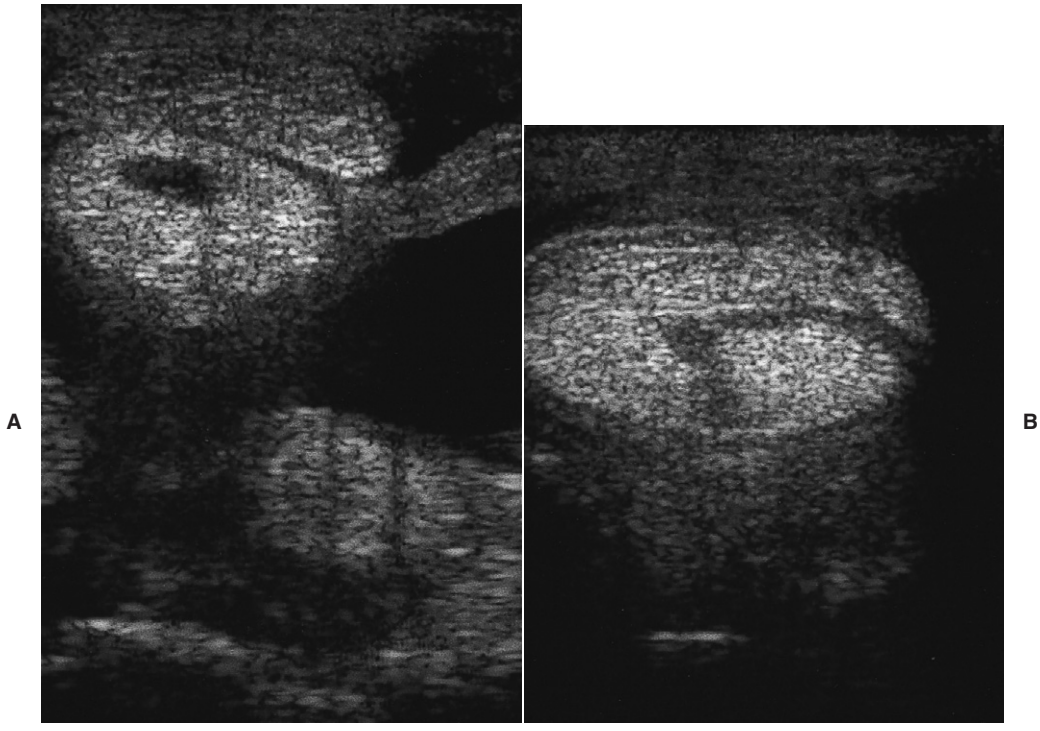

Fig. 71-2 Transverse ultrasonographic images of the distal metatarsal region of a horse with acute-onset right hindlimb lameness associated with distention of the digital flexor tendon sheath. Medial is to the left. An abnormal amount of fluid is within the digital flexor tendon sheath. **A,** There is a well-defined anechoic lesion within the deep digital flexor tendon, close to the plantar border. Note also the rather poorly defined dorsal border. **B,** Slightly farther distally the lesion can be seen to involve the dorsal and plantar borders of the tendon.

Enlargement or Change in Shape of the Deep Digital Flexor Tendon

In horses with low-grade injuries the only detectable ultrasonographic abnormality is a change in shape or size of the DDFT. The echogenicity and fiber pattern may appear normal; therefore these injuries are easily overlooked. The DDFT may look rounder in cross-section, rather than elliptical. Careful comparison with the contralateral limb at the same distance distal to the accessory carpal bone is important for accurate diagnosis. Comparison of the relative sizes of the SDFT and DDFT can also be helpful. Cross-sectional area measurements of the SDFT are prone to error at this level, and therefore measurements of the dorsopalmar thickness of the SDFT and DDFT are more accurate. If the horse fails to respond satisfactorily to conservative management, then consideration should be given to the presence of a marginal tear that may not be detectable with ultrasonography.[3]

Constriction of the DFTS and its contents by an enlarged palmar annular ligament may result in secondary compression of the DDFT.

Focal Hypoechogenic Lesions

The cross-sectional area of the DDFT may be enlarged. Focal hypoechoic defects vary in size and position within the cross-section of the tendon and in length. Some are small, occupying less than one tenth of the cross-sectional area of the tendon and extending less than 1 cm proximodistally, whereas others are considerably more extensive (Fig. 71-2).[5] Generally, larger lesions are associated with more severe lameness. These lesions generally occur immediately proximal to, or at the level of, the fetlock joint. An abnormal amount of synovial fluid is found within the DFTS, and sometimes adhesions are identified.

In some horses, inspection of the medial or lateral border of the DDFT reveals loss of definition of the margin of the tendon and an area of reduced echogenicity, indicating major fiber disruption. These lesions are easy to diagnose. The marginal tears described subsequently (see page 647) are more difficult to detect.

Small lesions have resolved with rest and horses have returned to full athletic function. However, large lesions tend to persist, and ultrasonographic examination may reveal no change, even if the horse is rested for more than 1 year. Large lesions have been associated with recurrent lameness.[5] Intrathecal medication with hyaluronan, triamcinolone, or methylprednisolone acetate has resulted in only temporary relief. Desmotomy of the palmar (plantar) annular ligament has resulted in only temporary remission of clinical signs. The prognosis for return to full athletic function is guarded.

Fibrosis and Mineralization Within the Deep Digital Flexor Tendon

Some horses develop widespread hyperechogenic foci within the DDFT in the fetlock region. Some of these create shadowing artifacts and therefore represent mineralization (Fig. 71-3). The foci generally but not invariably reflect a chronic injury.[7] If extensive, they make evaluation of the remaining tendon architecture difficult because of accoustic shadowing. The cross-sectional area of the tendon usually is enlarged. Ill-defined hypoechogenic regions often extend proximally or distally from hyperechogenic foci or are remote from them. Fibrous adhesions may extend from the DDFT to the DFTS wall.

The cause of these hyperechogenic lesions is unknown, although some affected horses have a history of previous injections of corticosteroids into the DFTS. Most of the affected horses are middle-aged Warmblood-type horses used

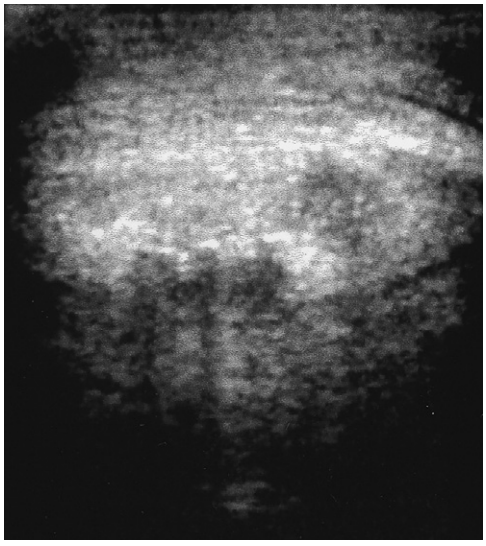

Fig. 71-3 Transverse ultrasonographic image of the distal metatarsal region of an 8-year-old event horse with chronic right hindlimb lameness associated with distention of the digital flexor tendon sheath. Medial is to the left. The deep digital flexor tendon is enlarged. Many hyperechogenic foci are within the deep digital flexor tendon, resulting in shadowing artifacts that make evaluating accurately the structure of the more dorsal aspects of the tendon impossible.

for show jumping or dressage. These lesions tend to result in persistent long-term lameness.

Marginal Tears of the Deep Digital Flexor Tendon

Marginal tears of the DDFT have been identified on the medial, lateral, and dorsal borders.[3,8] Lesions involving the lateral margin have been identified most commonly, varying in length (Fig. 71-4). Frequently the proximal extent of the tear is at the level of the manica flexoria. Some lesions extend as far distally as the proximal digital annular ligament. In some horses the lesion is a frontal plane split in the margin of the tendon; in other horses a sagittal tear of fibers occurs. Frontal plane splits in the DDFT may not be detectable by ultrasonography, although often a detectable enlargement of the cross-sectional area of the DDFT exists. The affected margin may be slightly less well defined compared with normal. Occasionally a torn echogenic strand is seen partially detached from the DDFT. Care must be taken not to confuse the synovial plicae on the medial and lateral borders of the tendon with a marginal tear.

Diagnosis is based on surgical exploration of the DFTS, which is indicated if a horse has pain associated with the DFTS along with abnormalities of the DDFT detected by ultrasonography, or no detectable changes, but a failure to respond to conservative management (see Chapter 75). Tenoscopic evaluation via a portal on the proximopalmar aspect of the pastern enables the most comprehensive view of the flexor tendons.[9] However, evaluating properly the DDFT underneath the manica flexoria may not be possible, unless the manica flexoria is transected. Treatment can be by debridement of the lesion, which can be done tenoscopically, or by debridement and suturing. The latter may be achieved most effectively by transection of the palmar (plantar) annular ligament (PAL) and opening the DFTS, followed by primary closure of the PAL. Four horses treated by tensocopic debridement became sound.[8] Five horses treated by debridement and suturing became sound, but five had persistent lameness.

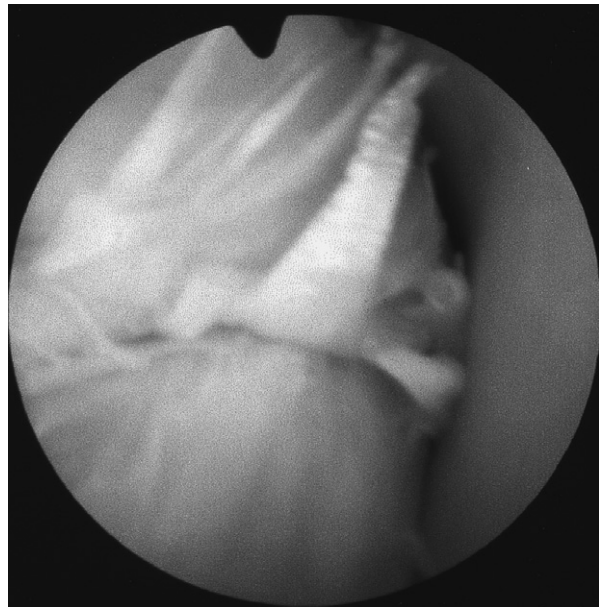

Fig. 71-4 The lateral margin of the deep digital flexor tendon (DDFT) within the digital flexor tendon sheath in the distal metacarpal region, viewed tenoscopically. The deep digital flexor tendon has a marginal tear. Ultrasonographic examination revealed slight enlargement of the DDFT, but otherwise the tendon appeared structurally normal. (Courtesy I.M. Wright, Newmarket, England.)

However, some horses develop thickening of the PAL and or the proximal digital annular ligament post-operatively, together with subcutaneous fibrosis, which results in persistent lameness. Five of six horses used for dressage, show jumping, or horse trials treated by open surgery to repair defects in the margin of the DDFT had persistent lameness associated with this type of reaction.[10] Therefore tenoscopic debridement is the preferred method of management.

DEEP DIGITAL FLEXOR INJURIES IN THE PASTERN REGION

The DDFT within the pastern remains within the DFTS, over which lie the thin proximal and distal digital annular ligaments. The tendon is therefore relatively close to the skin surface and is therefore vulnerable to the effects of direct trauma and puncture wounds.

In the pastern region the DDFT is a bilobed structure, each lobe being similar in size and shape at any level. Care must be taken when evaluating the tendon by ultrasonography because off-incidence artifacts are created readily. These may be seen as central, round hypoechoic areas, which should not be confused as lesions.

Deep Digital Flexor Tendonitis

Deep digital flexor tendonitis in the pastern region is not a common cause of lameness and occurs less often than sprain of the oblique distal sesamoidean ligaments or strain of one of the branches of the SDFT (see Chapter 83). Tendonitis of the DDFT is seen most often in skeletally mature horses and occurs more often in forelimbs than in hindlimbs. Deep digital flexor tendonitis occurs in a variety of sports horses, but the incidence in racehorses is lower than in other performance horses.

Deep digital flexor tendonitis in the pastern usually results in acute-onset, unilateral, moderate to severe lameness that is

persistent. Less commonly lameness is only detectable after maximal exertion and has resolved with rest but has progressively deteriorated over 1 to 2 years.[3] Slight, firm soft tissue swelling may occur on the palmar midline of the pastern region. Often slight distention of the DFTS occurs in the pastern region, but an obvious windgall may not be apparent. Firm palpation on, or just to one side of, the palmar midline of the pastern region may cause pain. Lameness is often worse on a soft surface compared with a hard surface and generally is improved substantially by perineural analgesia of the palmar nerves at the level of the proximal sesamoid bones.

Diagnosis is based on ultrasonographic evaluation. Careful comparison of the size and shape of the contralateral DDFT is useful. The ease with which the tendon can be evaluated distally in the pastern depends on the conformation of the pastern and foot. Evaluation is more difficult in horses with a narrow heel and a deep cleft between each bulb. A lesion may involve just one lobe of the tendon or both and is usually characterized by enlargement and alteration in shape of the tendon, with or without hypoechoic regions within the tendon. The medial and lateral margins of the tendon should be carefully evaluated. In chronic injuries focal hyperechoic foci consistent with dystrophic mineralization may be seen. Occasionally a complex of injuries is identified involving not only the DDFT but also a distal sesamoidean ligament. All structures should be examined carefully and systematically. Lesions restricted to the DDFT, which have been recognized early, usually respond reasonably to prolonged rest (6 to 12 months), with progressive resolution of hypoechoic defects as assessed by ultrasonography. Some enlargement of the tendon may persist long term. Horses with more chronic injuries or complex injuries respond less favorably, with a high incidence of recurrent lameness.

Injury Caused by Blunt Trauma in the Pastern Region

Direct blunt trauma caused by, for example, a severe overreach can result in severe inflammation of the subcutaneous tissue, the digital annular ligaments, and sometimes also the DDFT. If the proximal or distal digital annular ligament becomes thickened and fibrosed, this can create undue pressure on the DDFT and chronic pain, without any structural abnormality of the DDFT. In the acute stage, generalized swelling occurs in the pastern region, and palpation elicits pain. Ultrasonographic examination is required to determine which structures have been damaged. This may need to be repeated as fibrotic reactions develop in the ensuing weeks. The prognosis is generally favorable unless the digital annular ligaments become substantially thickened or the palmar aspect of the DDFT is torn.

An overreach sometimes results in laceration of the skin and the palmar aspect of the DDFT. These lacerations may not coincide because of the mobile nature of the skin, and damage to the DDFT may be overlooked, unless the tendon is examined by ultrasonography. One or both branches of the SDFT may also be damaged, so all structures should be evaluated carefully and systematically. Such lesions often heal with extensive fibrotic reactions and adhesion formation to the DFTS, which may result in chronic lameness.

Puncture Wounds of the Deep Digital Flexor Tendon

Puncture wounds in the palmar (plantar) aspect of the pastern region, caused by sharp objects such as a flint, can cause sudden-onset, moderate to severe lameness and rapid development of effusion within the DFTS. Ultrasonographic examination can be used to determine whether the DDFT was penetrated. An anechoic defect may be seen in its palmar border. With prompt treatment by vigorous lavage of the DFTS combined with systemic broad-spectrum antimicrobial therapy, the outcome may be favorable. Ideally the DFTS and its contents should be inspected using an arthroscope to determine if any foreign material was embedded within the DDFT that might seriously compromise the outcome.

Rupture of the Deep Digital Flexor Tendon

Rupture of the DDFT in the pastern region or within the hoof capsule is usually a sequel to a previous neurectomy of the palmar digital nerves. Neurectomy is usually performed because of suspected navicular disease or to relieve chronic foot pain of unknown cause. Pre-existing lesions of the DDFT are likely to predispose to tendon rupture. I have never examined a horse with spontaneous rupture of the DDFT that did not have a history of neurectomy. Lameness may be insidious and progressive or sudden in onset, associated with extensive swelling in the pastern region. Thickening results from enlargement of the DDFT, peritendonous fibrosis, and in horses with long-term injuries, secondary superficial digital flexor tendonitis. Complete rupture results in the toe of the limb flipping up when the limb is bearing weight (Fig. 71-5). These clinical signs are pathognomonic. In such horses the proximal end of the DDFT may retract proximally, resulting in the SDFT becoming opposed to the fibrocartilaginous scutum on the palmar aspect of the pastern. Radiographically a variable degree of subluxation of the distal interphalangeal joint exists, depending on the degree of integrity of the DDFT. The prognosis is hopeless for athletic function. Extensive peritendonous fibrosis ultimately develops, providing adequate support to preserve life for breeding, assuming that the contralateral limb can withstand the strain in the interim.

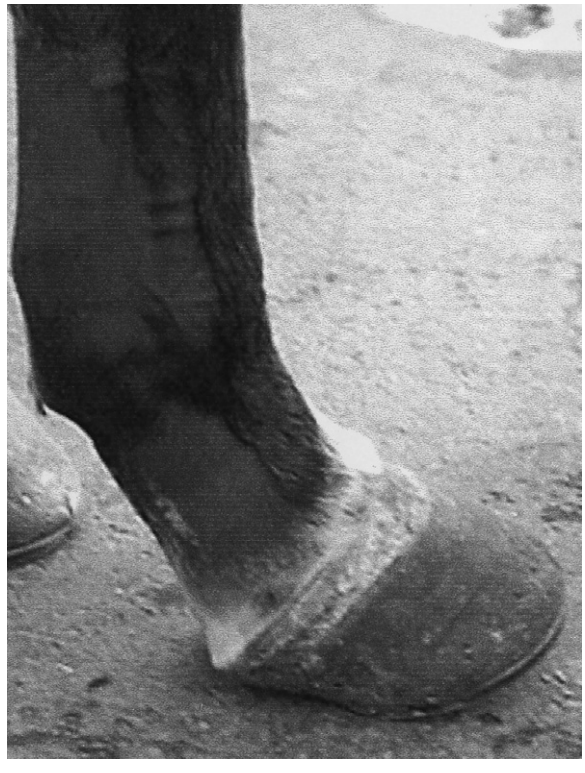

Fig. 71-5 The distal part of the limb of a horse with rupture of the deep digital flexor tendon. The toe is flipped upward associated with subluxation of the distal interphalangeal joint. There is considerable thickening in the pastern region caused by peritendonous fibrosis.

LESIONS OF THE DEEP DIGITAL FLEXOR TENDON WITHIN THE HOOF CAPSULE

Lesions of the deep digital flexor tendon within the hoof capsule are discussed in Chapter 33.

LESIONS OF THE DEEP DIGITAL FLEXOR TENDON IN THE HOCK AND PROXIMAL METATARSAL REGIONS

Primary deep digital flexor tendonitis in the hock or proximal metatarsal region is an unusual cause of hindlimb lameness (see Chapter 77). Lesions in the proximal metatarsal region have been identified in young Thoroughbreds in race training, with lameness associated with mild swelling in the proximal plantar metatarsal region. Lameness was improved transiently by intra-articular analgesia of the tarsometatarsal joint in two horses, presumably because of local diffusion of local anesthetic solution.[11] Subtarsal analgesia of the plantar and plantar metatarsal nerves alleviated lameness in one horse. Ultrasonographic examination revealed enlargement of the DDFT in the proximal metatarsal region, poor definition of the margins, and diffuse hypoechoic areas within the tendon. Rest for 3 months has resulted in resolution of lameness, and the prognosis is favorable for return to racing.

Primary lesions of the DDFT in the hock region have been identified only in skeletally mature horses and are comparatively rare. Lameness is sudden in onset and moderate in degree. Mild distention of tarsal sheath may occur, but localizing signs may be absent. Lameness is improved by perineural analgesia of the tibial and fibular nerves, but it is unaffected by intra-articular analgesia of the hock joints. Diagnosis relies on ultrasonographic examination. The DDFT may be examined from the plantaromedial aspect of the hock within the tarsal sheath. The DDFT is a large oval-shaped structure with well-defined margins. The dorsolateral aspect of the tendon may appear slightly less echogenic than the remainder of the tendon, and care should be taken not to misinterpret this as a lesion. Careful comparison with the contralateral limb is essential. Abnormalities include enlargement of the tendon, poor demarcation of the borders, and hypoechoic areas (Fig. 71-6). Lesions may also be identified by tenoscopic examination of the tarsal sheath (see Chapters 24 and 77).

Lesions of the DDFT also have been identified in association with bony lesions of the sustentaculum tali of the fibular tarsal bone (calcaneus) and ectopic mineralization within the tarsal sheath.[12] These horses may have fibrillation of the DDFT and adhesion formation. These lesions generally are seen with obvious distention of the tarsal sheath and long-term lameness and probably occur secondary to primary pathological conditions of the bone. The sustentaculum tali should be evaluated radiographically using plantarolateral-dorsomedial oblique and flexed dorsoplantar views. The prognosis for athletic performance is poor with conservative or surgical management.

Dorsomedial luxation of the DDFT secondary to congenital malformation of the sustentaculum tali has been recognized in young Saddlebred horses and occasionally in Thoroughbreds.[3,13] Clinically the plantar aspect of the tarsal region appears broader than normal, with tarsus valgus. Lameness may not be obvious. Diagnosis is based on radiographic examination: the sustentaculum tali appears flattened in a dorsoplantar (flexed) view.[14] This may result in a mechanical lameness. Surgical treatment has been attempted, but the horse did not race.[15]

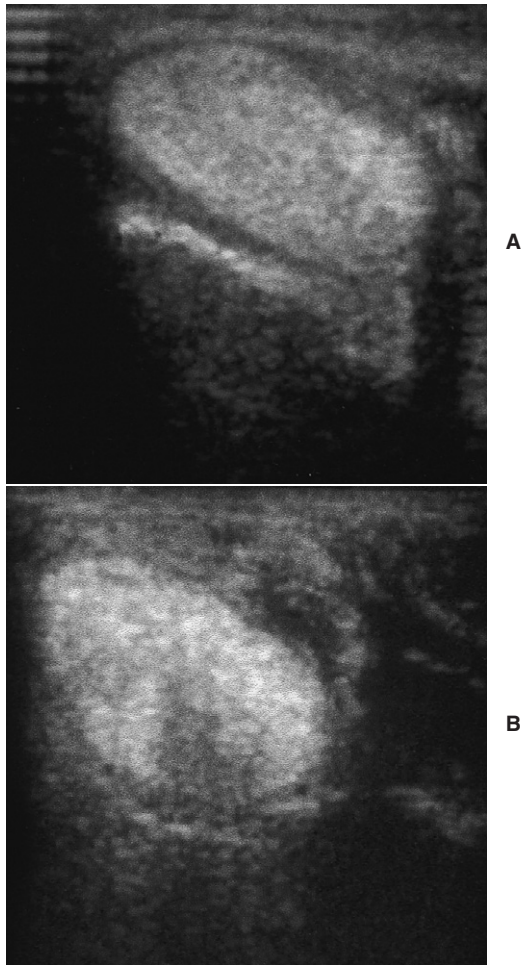

Fig. 71-6 **A,** Transverse ultrasonographic image of the plantaromedial aspect of the distal hock region of normal horse. Medial is to the left. **B,** Transverse ultrasonographic image of the plantaromedial aspect of the distal hock of a 7-year-old event horse with moderate lameness alleviated by tibial and fibular nerve blocks. No abnormalities were detected radiographically or scintigraphically. Medial is left. The deep digital flexor tendon has a large hypoechoic lesion dorsolaterally.

INFECTION OF THE DEEP DIGITAL FLEXOR TENDON

Infection of the deep digital flexor tendon is discussed in Chapter 38 (see page 373).

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CHAPTER • 72

Desmitis of the Accessory Ligament of the Deep Digital Flexor Tendon

Sue J. Dyson

ANATOMY

In the forelimbs the accessory ligament of the deep digital flexor tendon (ALDDFT) is a substantial structure, similar in size to the deep digital flexor tendon (DDFT). The ALDDFT continues the common palmar ligament of the carpus.[1] Proximally the ALDDFT is broad and rectangular in cross-section; farther distally it becomes narrower and thicker and blends with the DDFT in the middle one third of the metacarpal region. Fibrous bundles run from the medial and lateral borders of the DDFT to the superficial digital flexor tendon (SDFT) in the proximal half of the metacarpal region, and these predispose the horse to develop adhesions between the ALDDFT in horses with severe superficial digital flexor tendonitis or severe desmitis of the ALDDFT. The ALDDFT forms the dorsal wall of the carpal canal, within which is the carpal synovial sheath, which is interspersed between the DDFT and its accessory ligament.

In the hindlimbs the size of the ALDDFT varies extremely, but it is generally smaller than in the forelimb and rarely more than half the thickness of the DDFT. The ligament is usually symmetrical in the left and right hindlimbs of an individual horse.

The ALDDFT has a low modulus of elasticity and a moderate strength to rupture, whereas the DDFT has a high modulus of elasticity and more than three times the strength to rupture. In the forelimb the ALDDFT is loaded during the late stance phase, during extension of the digital joints,[1] or when landing over a fence.[2] The ALDDFT prevents over-stretching of the DDFT by passively carrying the load during maximal extension of the distal interphalangeal and metacarpophalangeal joints. The ALDDFT also functions to facilitate carpal extension when the limb is loaded.[1] During flexion of the limb the ALDDFT is relaxed completely, and active muscle contraction results in the DDFT sliding proximally within the carpal sheath. The role of the ALDDFT in the hindlimb is less clear.

Desmitis of the ALDDFT usually occurs in forelimbs[3-6] but *occasionally* is recognized as a cause of hindlimb lameness.[3-7] Desmitis may occur alone[3,6] as an acute injury or develop secondarily to previous severe tendonitis of the SDFT. In the latter case the SDFT is enlarged substantially and wraps around the medial and lateral margins of the DDFT. Adhesions develop between adjacent structures. In horses with chronic, severe desmitis of the ALDDFT, additional injury may occur to the adjacent DDFT.[6,8] A flexural deformity of the metacarpophalangeal or metatarsophalangeal joint may develop after severe injury to the ALDDFT.[3,9] Occasionally a flexural deformity of the metatarsophalangeal joint develops in one or both hindlimbs without detectable lesions in the ALDDFT.[10] The cause of this condition is unknown, but the flexural deformity may be relieved by desmotomy of the ALDDFT, provided that contracture of peri-articular soft tissues has not already occurred.

PATHOGENESIS

Degenerative aging changes take place in the ALDDFT, and these may be a significant predisposing factor in the development of desmitis. The amount of fibrillar collagen and number of large collagen fibers in the ALDDFT decrease with increasing age.[11] In a study of mechanical properties of the ALDDFT in relationship to age, failure forces of the ALDDFT of older horses were significantly lower than those of the ALDDFT in younger horses.[12] Fibrillar ruptures developed in the ALDDFT of old horses at forces and strains that were approximately half

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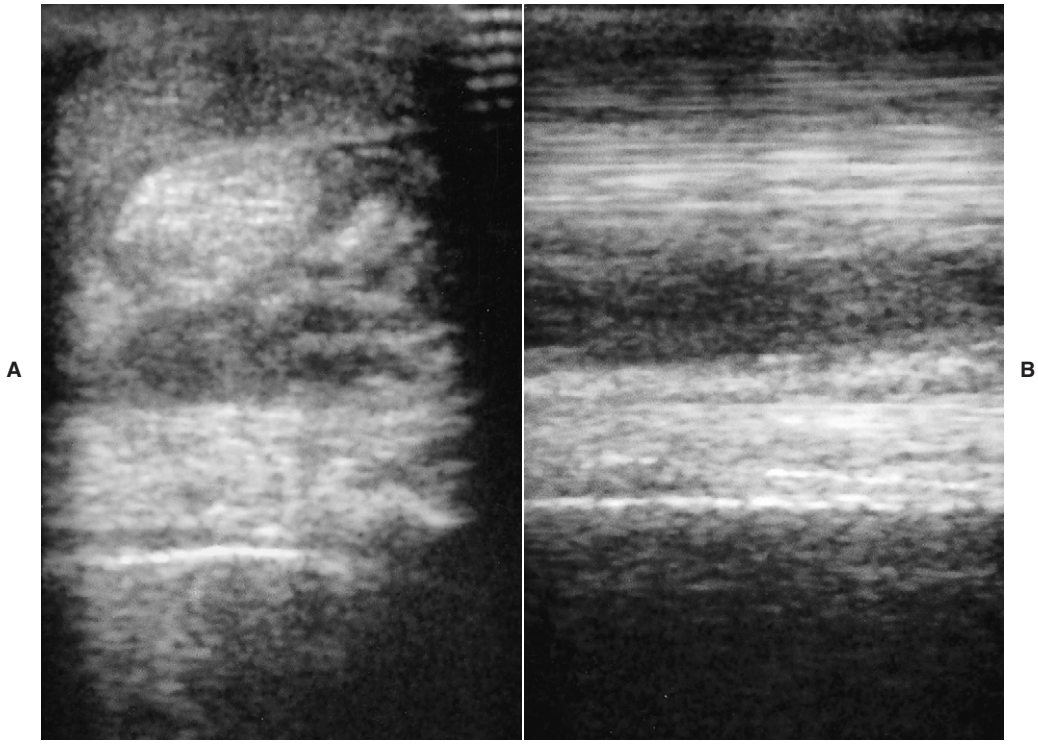

Fig. 72-1 **A,** Transverse ultrasonographic image of the palmar metacarpal soft tissue structures, 11 cm distal to the accessory carpal bone, of an 8-year-old Grand Prix show jumper. Medial is to the left. The accessory ligament of the deep digital flexor tendon is considerably enlarged and diffusely hypoechoic. **B,** Longitudinal ultrasonographic image of the palmar metacarpal sort tissues in zone 2A of the same horse as in **A.** Proximal is to the left. There is a diffuse reduction in echogenicity of the accessory ligament of the deep digital flexor tendon and poor fiber pattern.

those of the forces inducing total failure in young horses. It has been suggested that fibrillar rupture occurs at relatively low strains in older horses and that repetitive microtrauma may lead to clinical desmitis.[11]

The incidence of desmitis of the ALDDFT is rather different to other tendonous and ligamentous injuries. Injuries occur more often in horses older than 8 years of age.[3-5] The incidence in Thoroughbreds is comparatively low, and therefore this is an unusual injury in event horses or racehorses, except in older steeplechasers. Desmitis is a relatively common injury in ponies (see Chapter 127) and crossbred horses, including pleasure horses.[3,4] The incidence in Warmblood horses is also high.[5] Desmitis occurs in older show jumpers (see Chapter 116), especially Grand Prix level horses,[10] and also older dressage horses[10,13] and also sometimes occurs in young, extravagantly moving dressage horses (see Chapter 117). Desmitis is generally a unilateral forelimb injury (Fig. 72-1), although occasionally it occurs bilaterally, and is comparatively unusual in hindlimbs.

In hindlimbs, desmitis of the ALDDFT has been seen most frequently in cob-type breeds[10] and Quarter Horses or Quarter Horse crosses,[14] even in some horses or ponies of a comparatively young age, and does not appear to have been induced traumatically in all horses. In some of these horses the condition has been bilateral, developing simultaneously or sequentially in each hindlimb. Desmitis frequently has been associated with a tendency to stand with the fetlock of the affected limb partially flexed (Fig. 72-2), resulting in the development of a flexural deformity. The condition also has been recognized together with swelling on the plantar aspect of the pastern, associated with concurrent desmitis of the straight or oblique distal sesamoidean ligaments.[10]

HISTORY AND CLINICAL SIGNS

There is usually an acute-onset moderate to severe lameness during exercise.[3-5] A show jumper may pull up lame after jumping a large fence or after a water jump.[10] Swelling develops rapidly in the proximal one third of the metacarpal region, dorsal to the SDFT. Determining by palpation whether the DDFT or accessory ligament is enlarged can be difficult, but injuries of the DDFT in this area are unusual. Some horses develop desmitis secondary to previous superficial digital flexor tendonitis, and in these horses separating the margins of the SDFT from the ALDDFT often is difficult.

Clinical signs include swelling, heat, pain on palpation, and lameness. The horse may stand with slight elevation of the heel and flexion of the metacarpophalangeal joint. Some degree of swelling often persists, even after long-term convalescence. In horses with re-injury, new swelling may be only slight and careful palpation is required to identify a focus of pain.

Some horses with desmitis in the hindlimb may have similar clinical signs. However, some do not have an acute-onset lameness but show insidious enlargement and contracture of the ALDDFT, resulting in inability of the horse to place the heel of the affected limb(s) on the ground.

ULTRASONOGRAPHY

Diagnosis is confirmed by ultrasonography (see Fig. 72-1). The transducer should be focused initially at the depth of the ALDDFT, which should be examined in transverse and longitudinal planes. Comparative images of the contralateral limb

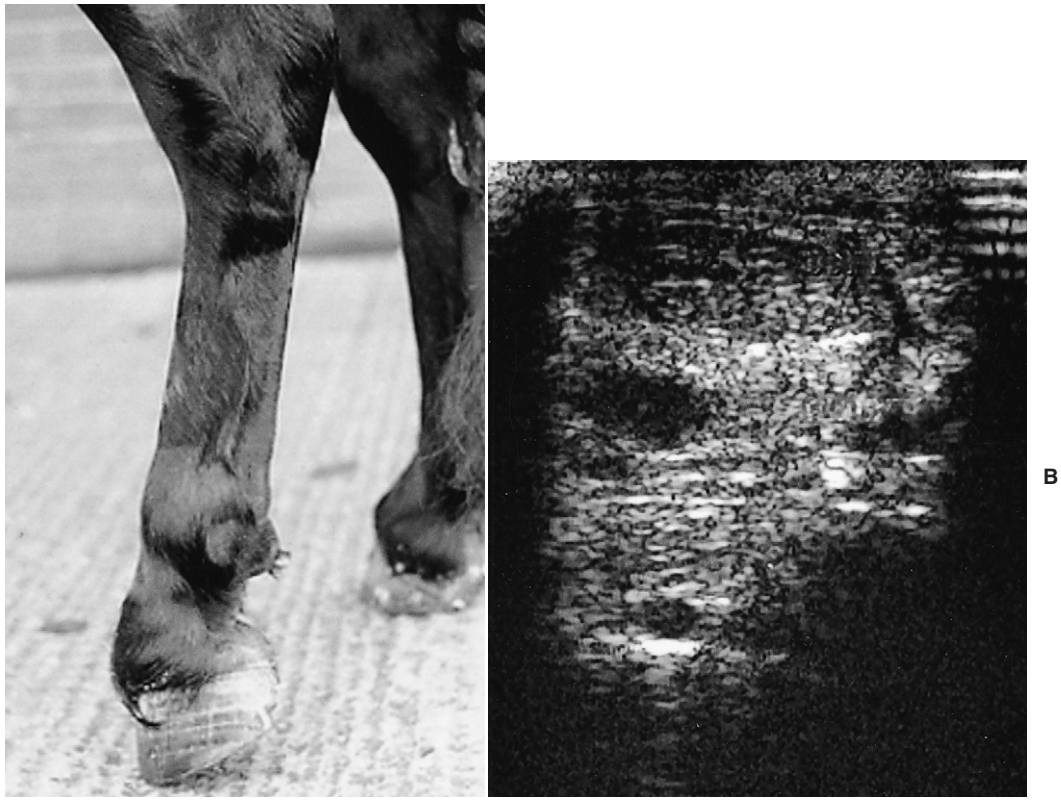

Fig. 72-2 **A,** A 6-year-old Fell pony with a flexural deformity of the left hind fetlock associated with chronic desmitis of the accessory ligament of the deep digital flexor tendon. The pony was unable to place the heel of the left hind foot on the ground. Even when the pony was heavily sedated, extending the left hind fetlock was not possible. Firm enlargement on the plantar aspect of the pastern associated with desmitis of the straight distal sesamoidean ligament was also apparent. Similar abnormalities had developed in the right hindlimb approximately 12 months previously. **B,** Transverse ultrasonographic image of the plantar metatarsal soft tissues approximately 10 cm distal to the tarsometatarsal joint. Medial is to the left. The accessory ligament of the deep digital flexor tendon is enlarged considerably, and there is a large anechoic lesion medially.

also should be obtained. If the transducer has a built-in standoff this may create artifacts at the level of the ALDDFT.

The ALDDFT is normally the most echogenic of the palmar metacarpal (plantar metatarsal) soft tissue structures, and its borders are well defined. In some ponies the ALDDFT is less echogenic than the DDFT and suspensory ligament.

With injury, the ALDDFT invariably is enlarged and tends to expand around the borders of the DDFT, especially laterally. With severe injuries, it may be necessary to move the transducer medially and laterally to evaluate properly the margins of the ligament. Often definition of the borders of the ligament is lost, with a diffuse reduction in echogenicity of the ligament, sometimes with anechoic areas. Occasionally a large proportion of the ligament is anechogenic. Central core lesions are comparatively rare. The dorsal border of the DDFT should be inspected carefully for evidence of concurrent injury, especially in horses with recurrent injury. In horses in which injury has been sustained secondary to previous superficial digital flexor tendonitis or in which desmitis of the ALDDFT is recurrent, the SDFT also should be evaluated for evidence of simultaneous recurrent injury or recent injury. The transducer should be focused on the SDFT, using a standoff. Adhesions between the ALDDFT and adjacent structures may be best identified in longitudinal images obtained with the limb not bearing weight. With passive flexion and extension of the fetlock it should be possible to see independent movement of the SDFT, DDFT, and ALDDFT, provided that no significant adhesion formation has occurred.

In horses with chronic desmitis, when substantial enlargement of the ALDDFT occurs, with or without enlargement of the SDFT, the cross-sectional area of the DDFT often is reduced significantly.[10]

In those horses that initially had an enlarged ALDDFT and inability to load the heel of the affected limb, the ALDDFT usually is enlarged massively, with poorly demarcated borders, and is diffusely hypoechogenic. Other structures in the metacarpal or metatarsal regions and pastern should be examined, because lesions have been identified simultaneously in the SDFT[14] and the distal sesamoidean ligaments.[10]

TREATMENT

In horses with acute, first-time primary injuries of the ALDDFT conservative treatment is usually satisfactory, but the prognosis is more guarded for longer-term injuries when the horse has continued to exercise. The horse should be restricted to box rest and controlled walking exercise for a minimum of 3 months and then should be re-evaluated ultrasonographically. Non-steroidal anti-inflammatory drugs should be given if the horse will not load the limb normally at rest to avoid development of a secondary flexural deformity of the metacarpophalangeal joint. Any foot imbalance should be corrected.

Improvement in echogenicity generally is seen more quickly than in comparable injuries of the SDFT. Box rest and

controlled walking exercise should continue until the ligament is of similar echogenicity to the DDFT in transverse and longitudinal images. Progress should be monitored monthly after the first re-evaluation 3 months after injury.

Horses that are allowed uncontrolled turnout in the initial 3-month period tend to have persistent clinical signs, and lesions persist ultrasonographically.

Treatment with β-aminoproprionitrile fumarate could be considered, especially in horses suffering a recurrent injury; however, no published reports of its efficacy in treating this condition are available, although successful results have been achieved.[15]

Differences in the reported success of conservative treatment have been significant.[3-5] A number of factors, including the chronicity of the injury when therapy was first instituted and the type of horse, probably account for this disparity in success. Dyson[3] reported complete functional recovery in 76% of 27 horses and ponies, whereas McDiarmid[4] reported only 43% success, and Van den Belt, Becker, and Dik[5] reported only 18%. Most of the horses in the series of Van den Belt, Becker, and Dik were large Warmbloods, whereas Dyson's series[3] included a large number of ponies. In my experience early recognition and aggressive treatment are key factors to a successful outcome.

In horses with chronic, recurrent desmitis, in horses with evidence of adhesions between the ALDDFT and the SDFT, or in those that tend to stand with the fetlock flexed, surgical treatment by desmotomy of the ALDDFT may be indicated.[3,6] The treated ligament heals by scar tissue that is inferior in strength to normal ligament, but the ligament will be longer, and this may reduce the strain on it[16] and thus reduce the risks of re-injury. Experimental removal of a full-thickness piece of the ALDDFT of 1 cm length resulted in long-term repair by scar tissue, in which orientation of the collagen was random. The healed ligaments were 1 cm longer than control ligaments and enlarged in cross-sectional area. The functional characteristics, force, and elongation at failure reached 80% of control values.[17]

The number of published long-term follow-up results for treatment of chronic desmitis of the ALDDFT by desmotomy is limited. In my experience desmotomy has been successful in horses with chronic desmitis of the ALDDFT alone, but in those with concurrent superficial digital flexor tendonitis, or a flexural deformity of the metacarpophalangeal joint that cannot be corrected passively, the results have been disappointing. Horses with concurrent superficial digital flexor tendonitis have been relieved of evidence of resting pain, but restoring these horses to full athletic function has not been possible.

The prognosis depends on the chronicity of the injury and evidence of any concurrent injury to the DDFT or SDFT. Horses with acute injuries have a better prognosis than those with chronic injuries. Horses with recurrent injuries have a more guarded prognosis, especially if lesions are identified in the DDFT. Horses with lesions of the ALDDFT that have developed secondary to superficial digital flexor tendonitis have the most guarded prognosis, especially if any evidence of flexural deformity exists.

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CHAPTER • 73

The Suspensory Apparatus

Sue J. Dyson and Ronald L. Genovese

ANATOMY AND PATHOPHYSIOLOGY

The suspensory ligament (SL) is correctly called the third interosseous muscle, but it is referred to as the SL throughout this text. The anatomy in the forelimbs and hindlimbs is similar and separate mention of the hindlimb is only made when substantial differences exist.

The SL can be divided into three separate regions that are subject to injury: the proximal part, the body, and the branches. For clinical purposes, in the forelimb the proximal part extends from 4 to 12 cm distal to the accessory carpal bone, and in the hindlimb, from 2 to 10 cm distal to the tarsometatarsal joint. The distal sesamoidean ligaments are considered individually.

In the forelimb the SL originates from two heads that rapidly fuse. In the hindlimb this division is less obvious. The SL contains a variable amount of muscular tissue (2% to 11%), which tends to be bilaterally symmetrical.[1] In the forelimb the SL originates from the palmar carpal ligament and the proximal aspect of the third metacarpal bone (McIII), whereas in the hindlimb it originates principally from the proximoplantar aspect of the third metatarsal bone (MtIII). The SL in the forelimb is approximately rectangular in cross-section, but it is more rounded in the hindlimb.

The body of the SL descends between the second (McII, MtII) and fourth (McIV, MtIV) metacarpal (metatarsal) bones and divides into two branches at a variable site in the mid-metacarpal (metatarsal) region. The level of division is usually bilaterally symmetrical. Each branch inserts on the abaxial surface of the corresponding proximal sesamoid bone (PSB). Each branch detaches a thin extensor branch dorsodistally that courses obliquely across the pastern to join the dorsal digital extensor tendon just above the proximal interphalangeal joint. Each extensor branch also blends with the corresponding collateral sesamoidean ligament.

The distal sesamoidean ligaments are the functional continuation of the SL in the digit and comprise the straight sesamoidean ligament, oblique sesamoidean ligaments, cruciate sesamoidean ligaments, and short sesamoidean ligaments. All attach proximally to the base of the PSBs and the proximal scutum. The straight sesamoidean ligament is a flat trapezoid-shaped band that inserts via the scutum medium onto the proximal aspect of the middle phalanx. The oblique sesamoidean ligaments are triangular-shaped structures that converge to insert on the palmar aspect of the proximal phalanx. The cruciate sesamoidean ligaments form the palmar wall of the distal palmar synovial recess of the metacarpophalangeal joint. They consist of two thin layers of tissue that cross each other and insert on the proximal palmar tuberosities of the proximal phalanx. The short sesamoidean ligaments insert on the proximal palmar aspect of the proximal phalanx and are difficult to separate from the dorsal aspect of the oblique sesamoidean ligaments.

The palmar ligament of the fetlock, also referred to as the intersesamoidean ligament, is a thick collagen structure that completely covers the palmar and axial surfaces of the PSBs and is strongly attached to them. Together with the PSBs, the palmar ligament forms the proximal scutum. The concave palmar surface of the proximal scutum provides a smooth surface over which the digital flexor tendons glide.

In the forelimb the SL is innervated by the palmar metacarpal nerves, derived from the lateral palmar nerve, which receives fibers from the ulnar and median nerves.[2] The hindlimb SL is innervated by the plantar metatarsal nerves, branches from the deep branch of the lateral plantar nerve, which is derived from the tibial nerve. The proximal SL is closely related to the palmar outpouching of the carpometacarpal joint in the forelimb[3] and the plantar outpouching of the tarsometatarsal joint in the hindlimb.[4]

The principal function of the SL is to prevent excessive extension of the fetlock joint.[5] During weight bearing the relative tension in the SL and flexor tendons regulates the stresses applied to different aspects of the McIII bone. When a limb is fully load bearing, the distal part of the SL branches is apposed closely to the abaxial aspects of the metacarpal condyles and then moves to the palmar aspect as the fetlock drops. During hyperextension, the PSBs move distally and dorsally, so the branches of the SL act as articular surfaces to balance the position of the McIII bone. If the limb is loaded asymmetrically, so that torque is on the fetlock, the SL branches contribute to joint stability on the side opposite compression of the joint.

Some evidence exists that training increases the strength of the SL; the mean absolute load to failure in a single load-to-failure compression test was significantly higher in horses that had been in racehorse training compared with those that had been confined to box or paddock rest.[6] In the trained group failure was most likely to be by fracture of a PSB, whereas in the untrained group the SL failed. However, when six 2-year-old Thoroughbred (TB) fillies underwent an 18-month controlled exercise program including galloping and were compared with six fillies that were restricted to walking exercise, no differences in the collagen fibril mass-average diameter in the body of the SL were found.[7] Mass-average diameter is correlated with ligament strength.

PROXIMAL SUSPENSORY DESMITIS IN THE FORELIMB

Proximal suspensory desmitis (PSD) is a common injury in the forelimbs[8-12] of athletic horses and may occur unilaterally or bilaterally. Some confusion has occurred about what constitutes PSD, and many clinicians have used this term for lameness that is worse with the affected limb on the outside of a circle and that is alleviated by analgesia of the proximal palmar metacarpal region but in which radiographic and ultrasonographic findings have been negative. In our experience this case scenario is relatively unusual. Ultrasonographic abnormalities of the proximal SL are usually detectable, and the absence of detectable structural abnormality should alert the veterinarian to search for an alternative diagnosis.

PSD results in sudden onset lameness, which can be remarkably transient, resolving within 24 hours unless the horse is worked hard. In horses with more chronic desmitis, lameness may be persistent. Lameness varies from mild to moderate and is rarely severe, unless the lesion is extensive. Lameness in Standardbred (STB) racehorses may be apparent only at high speeds. Bilateral PSD may result in loss of action rather than overt lameness, which occurs more commonly in flat racehorses, probably because of failure to recognize earlier, subtle unilateral lameness. Lameness is usually worse on soft ground, especially with the affected limb on the outside of a circle, and when subtle may be more easily felt by a rider than seen by an observer. Lameness may not be apparent at the working trot but may be detectable at the medium or extended trot. Recognition of these features in the history may be important, because acute lameness often resolves rapidly, and working the horse hard to reproduce lameness, with the inherent risk of worsening the injury, may be undesirable. Lameness is often transiently accentuated by distal limb flexion.

In the acute phase slight edema in the proximal metacarpal region, localized heat, and distention of the medial palmar vein may occur, but these features may be transient or absent. Pressure applied to the SL against the palmar aspect of the McIII bone or forced extension and protraction of the limb may elicit pain.

The feet should be evaluated carefully, because frequently foot imbalance is a predisposing factor. Back-at-the-knee and tied-in below the knee conformation also may be predisposing factors.

PSD is a common compensatory injury; therefore the whole horse should be evaluated to ensure that other causes of lameness are not missed.

Diagnostic Analgesic Techniques

If PSD is suspected, perineural analgesia of the lateral palmar nerve[3] (2 ml mepivacaine) or the medial and lateral palmar metacarpal nerves (2 ml per site) is indicated (see Chapter 10). This should result in substantial improvement in. or alleviation of, lameness within 10 minutes, assuming PSD is the only cause of lameness. However, neither technique is necessarily specific. Blockade of the lateral palmar nerve also has the potential to alleviate pain associated with a lateral source of pain in the more distal limb (e.g., a splint). The risks of influencing middle carpal joint pain are less than using the subcarpal approach, but local anesthetic solution may diffuse and improve lameness associated with the middle carpal joint[13] or with the carpal canal. Perineural analgesia of the palmar metacarpal nerves may alleviate pain associated with the middle carpal or carpometacarpal joints because of local diffusion or inadvertent deposition of local anesthetic solution into the distopalmar outpouchings of the carpometacarpal joint capsule. A false-negative result may be achieved because of inadvertent injection into the carpal sheath or failure of the local anesthetic solution to diffuse proximally to the most proximal extent of a lesion. Although the SL receives innervation from fibers from the median and ulnar nerves, perineural analgesia of the ulnar nerve usually resolves or substantially improves lameness associated with PSD.

Intra-articular analgesia of the middle carpal joint may result in partial improvement or complete alleviation of pain associated with the proximal suspensory ligament in some horses (15 [65%] of 25 horses).[13] Using a dorsal approach to the middle carpal joint rather than a palmarolateral approach should theoretically reduce the risks of diffusion of local anesthetic solution to the proximal SL and palmar metacarpal nerves; however, in practice the difference seems minor. Comparison of the relative responses to middle carpal joint analgesia (6 ml mepivacaine; assessed 10 minutes after injection) and perineural analgesia of the lateral palmar nerve or the palmar metacarpal nerves is potentially useful but can be highly misleading. Generally a horse with lameness caused by PSD responds better to perineural analgesia than intra-articular analgesia, but this is not universal. Similarly primary middle carpal joint pain usually is improved best by intra-articular analgesia, but this is not always the case. Middle carpal joint pain and PSD may occur concurrently, especially in STB racehorses. The clinician should evaluate the response to these diagnostic analgesic techniques in the light of the following:

- The use of the horse and thus the likelihood of the site of injury, and other clinical signs
- Other clinical signs: for example, distention of the middle carpal joint capsule and pain on passive manipulation of the carpus
- The degree and character of the lameness

Perineural analgesia of the palmar nerves at the level of the base of the proximal sesamoid bones often results in lameness because of PSD appearing worse. Perineural analgesia of the palmar nerves (at mid-metacarpal level) and palmar metacarpal nerves (distal to the button of McII and McIV) (four-point or low palmar block) often results in some improvement in pain associated with PSD, presumably because of proximal diffusion of local anesthetic solution via lymphatic vessels or along fascial planes.

More than one source of pain may be contributing to lameness. PSD and concurrent foot pain occur commonly. Hindlimb lameness also may be concurrent, especially in the contralateral hindlimb, so it is important to assess and to re-evaluate the whole horse.

Differential Diagnosis

PSD should be differentiated from middle carpal joint pain, being aware that especially in young TB racehorses and STB racehorses lesions may occur in both locations simultaneously. Osteoarthritis of the carpometacarpal joint occasionally occurs (see page 368). Pain associated with palmar cortical fatigue fractures or stress reactions of McIII[11,14-16] responds similarly to diagnostic analgesic techniques; however, lameness tends to be more severe and worse on firm ground and often deteriorates the farther the horse trots (see page 364). Avulsion fractures of McIII at the origin of the SL (see page 367) occur less frequently and tend to be associated with more persistent and severe lameness.[1,17] Pain associated with the carpal sheath or carpal retinaculum also should be considered (see Chapter 76). Perineural analgesia of the deep branch of the lateral palmar nerve or the palmar metacarpal nerves alone should not alleviate pain associated with the deep digital flexor tendon (DDFT) or its accessory ligament (ALDDFT), the superficial digital flexor tendon (SDFT), or the fetlock region, without simultaneous blockade of the palmar nerves.

Diagnostic Ultrasonography

Diagnostic ultrasonography is essential for accurate diagnosis of PSD. The limb should be evaluated in transverse and longitudinal planes, and careful comparison should be made with the contralateral limb. High-quality images are required, because lesions can be subtle and easily missed if the gain controls are too high or if the transducer is not focused on the SL. Artifacts are readily created if the transducer is not in complete contact with the limb. The contours of the proximal palmar aspect of the metacarpal region can make obtaining longitudinal images difficult, especially because the proximal palmar aspect of the McIII bone slopes backward. Therefore creating hypoechoic artifacts at the enthesis of the SL on the McIII bone is easy. Cross-sectional area measurements may be extremely valuable, especially in horses with acute PSD, because enlargement of the ligament may be the only detectable ultrasonographic abnormality. Bear in mind that muscular tissue appears less echogenic than does ligamentous

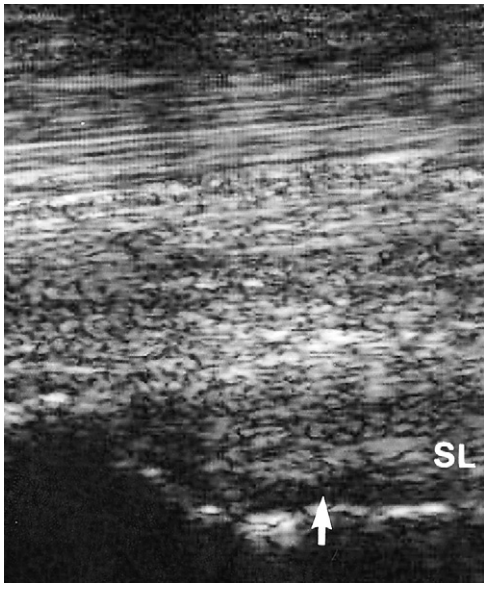

Fig. 73-1 Longitudinal (proximal is to the left) images of the proximal metacarpal region. The thin echogenic band from the suspensory ligament (*SL*) passes proximally from the enthesis of the suspensory ligament on the third metacarpal bone to blend with the palmar fascia. Dorsal to the suspensory ligament is anechoic fluid within the palmar recess of the carpometacarpal joint capsule (*arrow*).

tissue and that proximally the SL originates in two halves. Previous injuries to the SL may not resolve fully to restore normal, uniform echogenicity. Be aware that poor diagnostic analgesic technique may result in aspiration of air, which creates artifacts. A thin band of the SL passes proximally from the enthesis on the McIII bone to blend with the palmar carpal fascia. The anechoic space seen dorsal to the SL at this level is fluid in the palmar recess of the carpometacarpal joint and should not be confused as a lesion (Fig. 73-1).

Abnormalities associated with PSD include the following[18] (Figs. 73-2 and 73-3):

- Enlargement of the cross-sectional area. This may result in reduction of space between the SL and the palmar cortex of the McIII bone or reduced space between the SL and ALDDFT.
- Poor demarcation of the margins of the SL, especially the dorsal margin
- Focal or diffuse areas of reduced echogenicity. These may extend less than 1 cm proximodistally and occupy from less than 10% to up to the entire cross-sectional area of the ligament.
- Focal anechoic core lesions
- Reduced strength of fiber pattern
- Focal mineralization (rare in acute injuries)
- Mild increased echogenicity of the entire SL in long-term, chronic injuries

In a horse with bilateral PSD an obvious lesion may be detectable in the lamer limb, but abnormalities may be much more subtle and occasionally not apparent in the less lame limb. In a 3-year-old TB that sustains PSD at 2 years of age, mild lameness may be recurrent, and discerning any structural abnormality other than enlargement of the SL may not be possible.

The degree of ultrasonographic abnormality (cross-sectional area involved and proximodistal extent of the lesion) usually reflects the severity of the lameness. In horses with acute PSD the ultrasonographic abnormalities may be subtle, although if lameness is unilateral, slight enlargement of cross-sectional area may be detectable. Care should be taken to compare measurements in the contralateral limb at the same distance distal to the accessory carpal bone. Ultrasonographic abnormalities may deteriorate over the next 10 to 14 days, and re-evaluation may be useful to confirm the diagnosis.

In horses with an avulsion fracture of the McIII bone at the origin of the SL, the fracture fragment is usually readily identifiable and generally is associated with only a focal lesion in the SL itself, usually restricted to the dorsal aspect (see page 367).

Radiography

Usually no detectable radiographic abnormalities of the McIII bone occur in horses with acute PSD. With chronic PSD, increased opacity of the proximal aspect of the McIII bone may be seen in dorsopalmar views. This sclerosis should be differentiated from that associated with a palmar cortical fatigue fracture,[19] which is invariably medial. In a lateromedial projection subcortical sclerosis in the proximal palmar aspect of the McIII bone may be apparent. These secondary bony changes in a forelimb are associated with a more guarded prognosis.

Nuclear Scintigraphy

Reports in the literature about the usefulness of nuclear scintigraphy for diagnosing PSD are confusing because of failure to correlate scintigraphic findings with ultrasonographic and radiographic findings and because avulsion fractures of the McIII bone are not considered separately.[20,21] Nuclear scintigraphy is generally unnecessary for diagnosing PSD, provided that good-quality ultrasonographic images are obtained, but may give additional information about associated bone turnover at the insertion of the SL. Pool- and bone-phase images may be negative. Abnormal radiopharmaceutical uptake in the pool phase may actually reflect early bone uptake. Increased uptake of ^{99m}Tc–methylene diphosphonate was identified in the proximal palmar metacarpal region in 55% of pool-phase images and 44% of bone-phase images in 25 horses with ultrasonographic evidence of PSD.[22] Therefore negative scintigraphic images do not preclude the presence of PSD. Radiopharmaceutical uptake associated with either an avulsion fracture of the McIII bone at the insertion of the SL or a stress fracture is likely to be more intense. Abnormal radiopharmaceutical uptake in the bone phase, seen in the absence of ultrasonographic and radiographic abnormalities, is more likely to reflect a primary pathological condition of bone.

Treatment

Most horses with acute forelimb PSD respond well to box rest and controlled walking exercise for 3 months. Attention to correct foot balance is important. A premature resumption of work usually results in recurrent injury. Approximately 90% of horses resume full athletic function without recurrent injury.[1] Horses with chronic PSD may require more prolonged rehabilitation, and in a small proportion lameness is persistent. Some TB racehorses with chronic lesions have been able to be maintained in training with judicious use of phenylbutazone, without significant deterioration of the lesion. No fatalities associated with PSD occurred in 630 TB racehorses examined post mortem because of musculoskeletal injuries.[23] Some STB racehorses with acute lesions have been managed by slightly reducing the training schedule, local injections of corticosteroids and hyaluronan, symptomatic anti-inflammatory therapy (local icing, liniments such as dimethylsulfoxide [DMSO] and corticosteroid paints, and phenylbutazone as necessary), and shortening the toes and increasing hoof angle. Although a few treated horses were able to race successfully, prognosis after rest was better.[24]

Extracorporeal shock wave treatment (three treatments at 2-week intervals) has been successful in some horses with

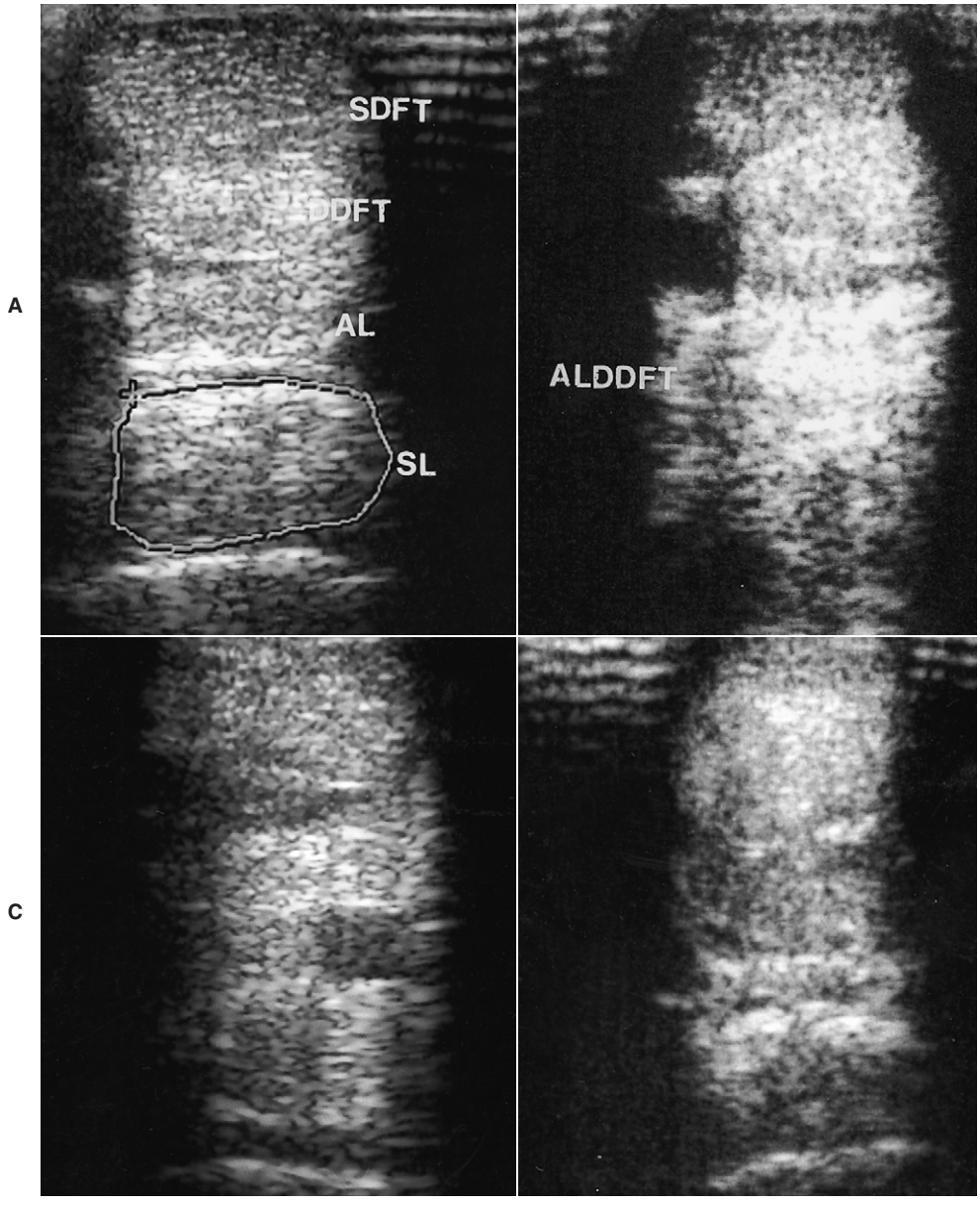

Fig. 73-2 **A,** Transverse ultrasonographic image of the metacarpal region of 3-year-old Thoroughbred, with mild forelimb lameness, at 9 cm distal to the accessory carpal bone. The suspensory ligament (*SL*) shows a slight overall reduction in echogenicity compatible with proximal suspensory desmitis. The ligament is enlarged (cross-sectional area 1.39 cm^2 compared with 1.25 cm^2 in the contralateral limb). The lesion extended less than 1 cm proximodistally. **B,** Transverse ultrasonographic image of the metacarpal region at 8 cm distal to the accessory carpal bone of the right forelimb of 7-year-old medium-level dressage horse. The entire cross-sectional area of the suspensory ligament is reduced in echogenicity. The lesion extended 1.5 cm proximodistally. Note also the hyperechoic appearance of the accessory ligament of the deep digital flexor tendon (*ALDDFT*). **C,** Transverse ultrasonographic image of the proximal metacarpal region of 6-year-old event horse with acute onset right forelimb lameness. There is a hypoechoic lesion in the medial aspect of the suspensory ligament that extends less than 1 cm proximodistally. Medial is left. **D,** Transverse ultrasonographic image of the right forelimb of 7-year-old dressage horse with bilateral forelimb lameness. The palmar aspect of the suspensory ligament is slightly increased in echogenicity, but the dorsal aspect is diffusely hypoechoic.

chronic lesions that had failed to respond to conservative management.[25] Ten (50%) of 20 horses with chronic forelimb PSD with lameness of greater than 3 weeks' duration were in full work 6 months after treatment.[26] Local infiltration with 4 to 6 ml of 2% iodine in almond oil also has been used successfully.[8]

In some horses the lesions disappear completely ultrasonographically. In others echogenicity may increase, but uniform echogenicity is never restored. Rest should be continued until the ultrasonographic appearance remains stable.

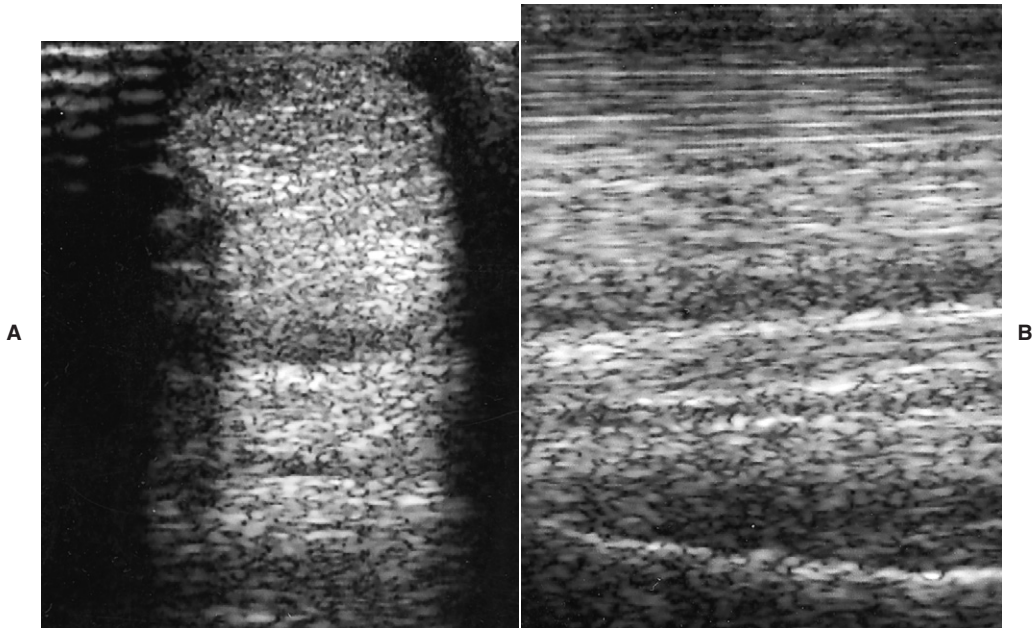

Fig. 73-3 **A,** Transverse ultrasonographic image of the proximal metacarpal region at 8 cm distal to the accessory carpal bone of the left forelimb of a Grand Prix show jumper. The horse had developed acute severe lameness immediately after completing a jumping round 2 weeks previously. The dorsal half of the suspensory ligament is diffusely hypoechoic. **B,** Longitudinal ultrasonographic image of the proximal metacarpal region of the same horse. Proximal is to the left. The dorsal aspect of the suspensory ligament is diffusely hypoechoic.

PROXIMAL SUSPENSORY DESMITIS IN THE HINDLIMB

PSD in the hindlimb results in either an insidious or a sudden onset lameness that may be mild or severe. Some horses show poor performance rather than a recognized lameness. In contrast to the forelimb, lameness may persist and remain severe, despite restriction to box rest. Such persistence probably is caused by a compartment-like syndrome and pressure on the adjacent plantar metatarsal nerves.[18] In view of the chronicity of some lesions when first identified and the finding of secondary radiological changes in sound horses, some lesions likely exist subclinically or are associated with a low-grade lameness that goes unrecognized. The incidence of bilateral lesions is higher than in forelimbs.

PSD in the hindlimb occurs in horses in all athletic disciplines and of all ages and is a particular problem in dressage horses working at advanced level. Horses with straight hock conformation or hyperextension of the metatarsophalangeal joint appear predisposed to injury (Fig. 73-4). Such conformational abnormalities were identified in 9 (21%) of 42 horses with hindlimb PSD but in only 4 (8%) of 50 horses examined consecutively with hindlimb lameness unrelated to the suspensory apparatus.[27] Hyperextension of the metatarsophalangeal joint may develop as a sequel to PSD. A long-toe and low-heel conformation also may be a predisposing factor, especially if associated with abnormal orientation of the distal phalanx, with the plantar aspect lower than the toe.[22]

PSD in the hindlimb in a STB racehorse is common and usually results in an abnormal gait at high speeds, which may or may not be apparent at the trot. Unilateral left hindlimb lameness may present as the horse drifting to the right shaft and being on the right line.

Horses with acute hindlimb PSD may have localized heat and swelling and pain on pressure applied to the SL, but frequently no localizing clinical features are apparent.

Lameness is often characterized by a reduced height of arc of foot flight, with or without intermittent catching of the toe. The cranial phase of the stride may be shortened. Lameness may be accentuated by proximal or distal limb flexion. Bilateral lesions may result in poor hindlimb action rather than obvious hindlimb lameness. Lameness may be more obvious on a circle on the lunge, but unlike forelimb PSD the lameness is not necessarily worse with the lamer limb on the outside. Like many horses with hindlimb lameness, lameness is often more obvious when the horse is ridden, especially when the rider sits on the diagonal of the lame or lamer limb.

Diagnostic Analgesic Techniques

Perineural analgesia of the plantar nerves (mid-metatarsal level) and plantar metatarsal nerves may result in slight improvement in lameness because of proximal diffusion of the local anesthetic solution. Lameness usually is improved substantially by perineural analgesia of the medial and lateral plantar metatarsal nerves (2 ml of mepivacaine 2% per site) or the deep branch of the lateral plantar nerve distal to the tarsus, but lameness may not be alleviated fully. Improvement is usually seen within 10 minutes of injection. Depositing the local anesthetic solution as proximal as ideal may be difficult because of the shape of the MtIV bone. This may result in only partial improvement in lameness. False-negative results also may be obtained because of inadvertent injection into the tarsal sheath or the tarsometatarsal joint capsule. Subtarsal analgesia can influence tarsometatarsal joint pain, and occasionally (2 [8%] of 24 horses[28]) intra-articular analgesia of the tarsometatarsal joint alleviates pain associated with PSD. Perineural analgesia of the tibial nerve alone alleviates pain associated with PSD without significantly influencing tarsal pain. However, the tibial nerve is large; therefore 20 minutes may pass before analgesia is achieved effectively. Occasionally PSD occurs together with pain associated with osteoarthritis of the tarsometatarsal joint.

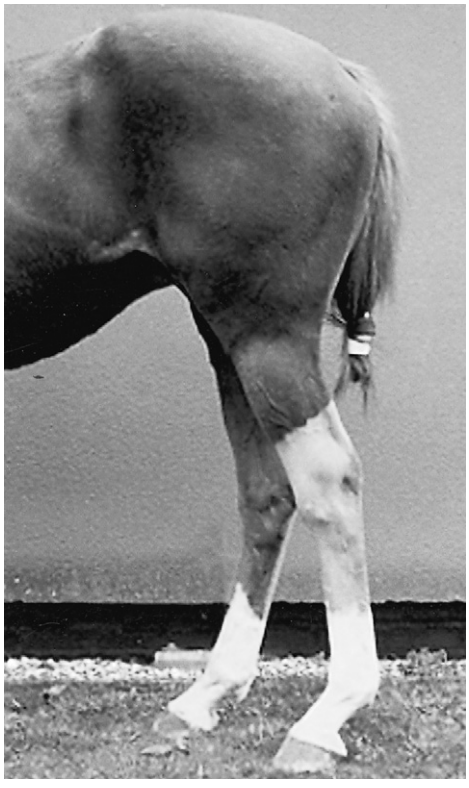

Fig. 73-4 The hindlimbs of 7-year-old show jumper with proximal suspensory desmitis of the left hindlimb. Note the relatively straight hock conformation and the sloping pasterns associated with hyperextension of the hind fetlocks. The horse also had a false thoroughpin on the left hindlimb that did not contribute to lameness.

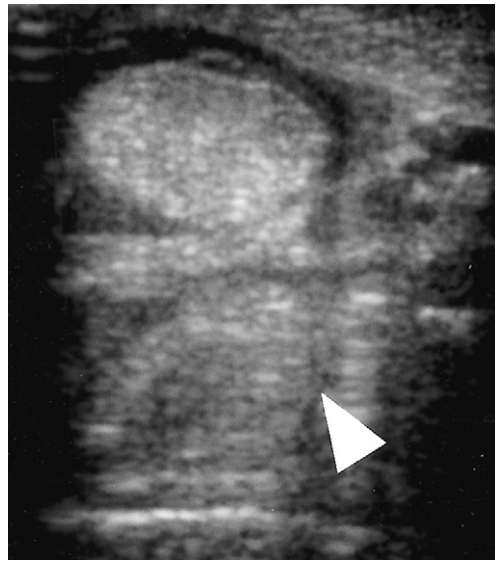

Fig. 73-5 Transverse ultrasonographic image of the proximal metatarsal region of a horse with proximal suspensory desmitis. Medial is to the left. The broad anechoic band through the suspensory ligament (*arrowhead*) is an artifact caused by the overlying plantar vessels. There is a hypoechoic region in the medial aspect of the suspensory ligament.

Diagnostic Ultrasonography

High-quality ultrasonographic images are essential for accurate diagnosis. Large vessels plantarolateral to the SL may result in broad linear anechoic artifacts within the SL (Fig. 73-5). In large Warmblood horses in particular the SL is situated deeply, and the ultrasound transducer must be focused accordingly. In transverse and longitudinal images the most proximal part of the SL in a normal horse may appear slightly less echogenic than the DDFT (Fig. 73-6). Detection of subtle abnormalities requires careful comparison with the contralateral limb and measurement of cross-sectional area. The SL should be imaged routinely in transverse and longitudinal planes.

In hindlimb PSD focal anechoic areas are relatively unusual, except in the STB racehorse. More commonly the SL is enlarged, with poor demarcation of its borders and a diffuse reduction in echogenicity of part or all of the cross-sectional area of the ligament (Figs. 73-5, 73-7, and 73-8). Ectopic mineralization occurs more often in hindlimbs compared with forelimbs. An irregular contour of the plantar aspect of the MtIII bone may reflect enthesophyte formation. In some horses, especially those with abnormal conformation, the lesions may progress despite box rest.

Radiography

Diagnosis should never be based on radiography alone, because some sound horse have some sclerosis of the proximal aspect of the MtIII bone. In horses with chronic active PSD this tends to be more extensive. In the dorsoplantar view opacity of the proximal aspect of the MtIII bone is increased, often more obvious laterally. In a lateromedial projection subcortical sclerosis and alteration of the trabecular pattern of the proximoplantar aspect of the MtIII bone may be caused by endosteal new bone, extending up to 4 cm proximodistally (Fig. 73-9). The plantar cortex may itself be thickened, and in addition enthesophyte formation may be visible on the plantar aspect. However, in some acute horses with injury no radiological abnormality is detectable.

Nuclear Scintigraphy

Nuclear scintigraphy is not a sensitive means of detecting PSD in hindlimbs. Pool-phase images were positive in only 25% of 20 horses with ultrasonographic evidence of PSD.[22] In bone-phase images increased radiopharmaceutical uptake in the proximoplantar aspect of the MtIII bone was found in 42% (Fig. 73-10). Increased radiopharmaceutical uptake in the proximoplantar aspect of the MtIII bone, with no detectable ultrasonographic abnormality of the SL and no radiographic change associated with enthesopathy, should be associated with a primary pathological condition of bone.

Differential Diagnosis

PSD should be differentiated from pain associated with the tarsometatarsal joint, an avulsion fracture of the MtIII bone at the origin of the SL, and primary stress reactions in the MtIII bone.

Treatment

The prognosis for horses with PSD in the hindlimb generally has been poor. Only 6 (14%) of 42 horses seen in a referral practice were able to resume full work without detectable lameness for at least 1 year, all of which had been lame for less than 5 weeks.[27] All these horses showed substantial improvement in clinical signs within 3 months of the onset of lameness. Two additional horses resumed full work but suffered lameness in another limb. Seven horses improved greatly and were able to work, despite persistent mild lameness. Twenty-four horses (57%) had persistent or recurrent lameness. Results from a first opinion practice also were disappointing, with only 10 (58%) of 17 horses resuming work.[1]

Horses with acute (less than 4 to 6 weeks' duration) hindlimb PSD respond reasonably well to local infiltration with

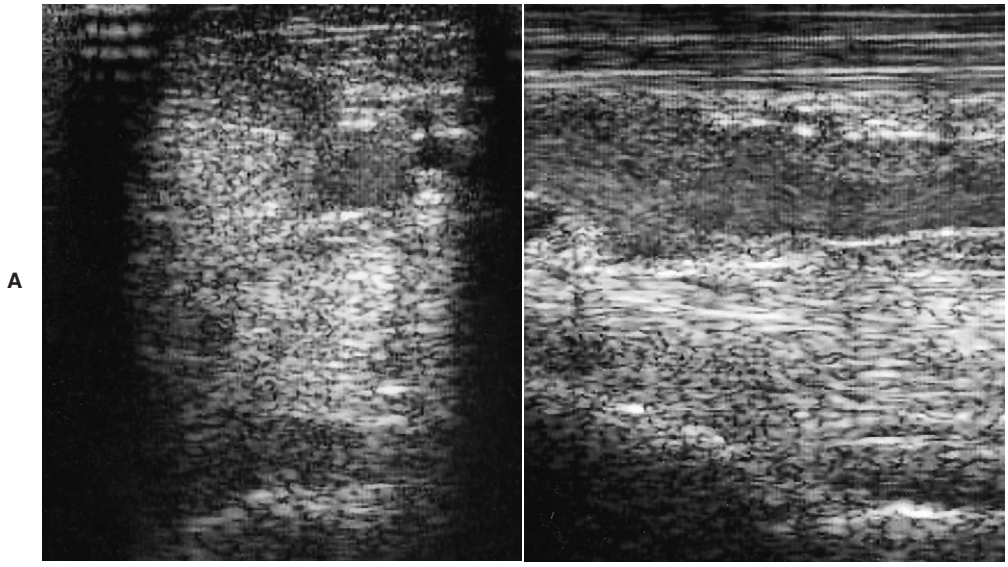

Fig. 73-6 **A,** Transverse ultrasonographic image of the proximal metatarsal region of a normal horse, 4 cm distal to the tarsometatarsal joint. Medial is to the left. The suspensory ligament is similar in echogenicity to the deep digital flexor tendon. **B,** Longitudinal ultrasonographic image of the proximal metatarsal region of a normal horse. Proximal is to the left.

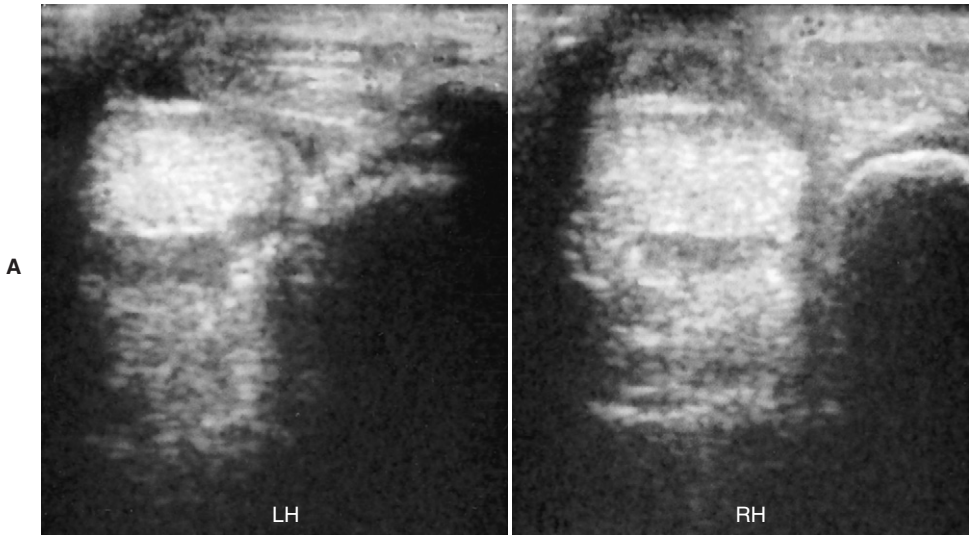

Fig. 73-7 Transverse ultrasonographic images of left (*LH*) and right (*RH*) hindlimbs, at 4 cm distal to the tarsometatarsal joint, of an 11-year-old show jumper with left hindlimb lameness caused by proximal suspensory desmitis. The damaged suspensory ligament is diffusely hypoechoic and enlarged.

corticosteroids, aimed at reducing inflammation and therefore swelling, and thus minimizing the risk of developing a compartment syndrome (see the following discussion). Foot imbalance is corrected and egg bar shoes are used to reduce extension of the fetlock. Horses with chronic PSD have a guarded prognosis regardless of the treatment. Lameness often tends to persist unchanged, even after prolonged box rest, which is unusual for a primary soft tissue lesion. In some horses lesions are progressive. Local infiltration with corticosteroids, glycosaminoglycan polysulfate, hyaluronan, or homeopathic drugs such as actovegin and traumil has given disappointing results.

In some horses an initial improvement in lameness is seen after box rest and controlled walking exercise for 2 to 3 months, and then no further improvement is seen. Increasing the exercise despite the lameness sometimes results in further improvement. Some horses have worked satisfactorily while being treated with phenylbutazone, without apparent deterioration of clinical signs.

Extracorporeal shock wave therapy appears to be helpful in some horses.[27] Eighteen of 30 horses with PSD in forelimbs or hindlimbs were in work 6 months after treatment. Eighteen (40%) of 45 horses with unilateral or bilateral hindlimb PSD were in full work 6 months after treatment[26]; however, a small proportion of these horses have experienced recurrent lameness subsequently. Injection of 2% iodine in almond oil resulted in 12 (54%) of 22 horses returning to work.[29]

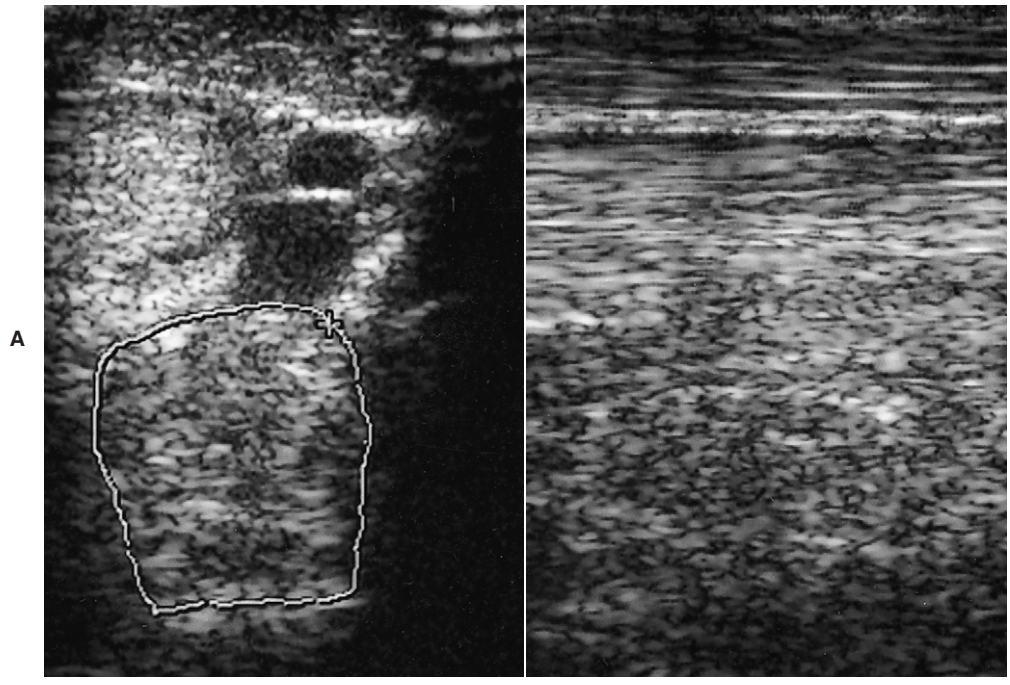

Fig. 73-8 **A,** Transverse ultrasonographic image of the left hindlimb at 3 cm distal to the tarsometatarsal joint of 12-year-old pleasure riding horse that had a lack of hindlimb impulsion. The horse had bilateral proximal suspensory desmitis. The suspensory ligament is enlarged (cross-sectional area 2.4 cm^2) and diffusely hypoechoic. **B,** Longitudinal ultrasonographic image of the proximal metatarsal region of the left hindlimb of the same horse. The suspensory ligament has poor fiber pattern.

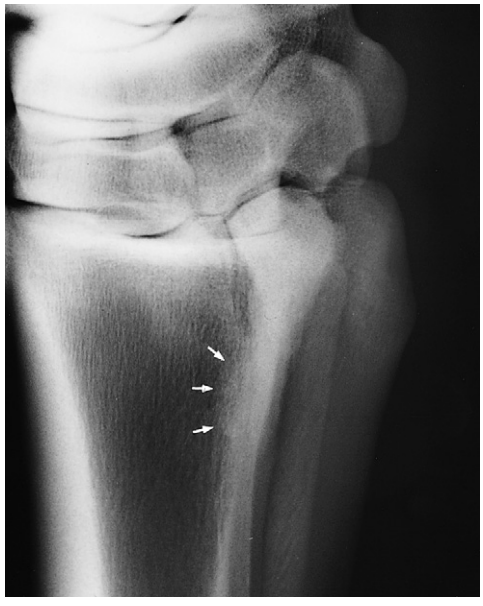

Fig. 73-9 Lateromedial radiographic view of the right hindlimb of 7-year-old dressage horse with chronic proximal suspensory desmitis. The proximoplantar aspect of the third metatarsal bone (*arrows*) has sub-cortical sclerosis.

Fig. 73-10 Lateral scintigraphic image of the left hindlimb of 9-year-old Warmblood show jumper with proximal suspensory desmitis. There is marked focal increased radiopharmaceutical uptake in the proximoplantar aspect of the third metatarsal bone.

Tibial neurectomy performed in eight horses enabled six to return to full athletic function (show jumping and horse trials) for at least 2 years after surgery, with no post-operative complications.[22] Neurectomy of the deep branch of the lateral plantar nerve has been combined with incising the thin fascial covering of the plantar aspect of the SL and was successful in five of six horses.[29] A further six of eight horses treated by one author (S.J.D.) have returned to full athletic function after neurectomy of the deep branch of the lateral planter nerve, but two horses experienced deterioration because of progressive lesions in the SL.[22] Both of these horses had pre-existing excessively straight hock conformation and hyperextension of the fetlock of the lame limb. Fasciotomy of the deep plantar metatarsal fascia has been successful in some horses.[30] Injection of about 30 ml of bone marrow also is claimed to be successful, especially if combined with fasciotomy, allowing 85% of horses to return to the former level of function[31] (see Chapter 74).

Gross Pathological and Histopathological Findings

Post-mortem examinations have been performed on both hindlimbs of eight horses, six with unilateral lameness and two with bilateral lameness.[17,22,28] Abnormalities of the SLs were confined to the lame limbs. The SLs were grossly enlarged, with thickening of surrounding fascia and periligamentous tissues, especially on the plantar aspect. Histological changes in the SL included hypercellularity and acellular areas; hemosiderin deposition; fibrosis; hyalinization of collagen; an increased number of fibrous septae, some with blood vessels; neovascularization; and chondroid metaplasia. Although chondroid metaplasia was seen at the ligament bone interface in lame and sound limbs, intraligamentous chondroid metaplasia was only seem in the lame limbs.

Evidence of compression of adjacent peripheral nerves was found in the lame limb of five horses. Abnormalities of the plantar metatarsal nerves included thickening of the perineurium, perineural fibrosis, reduction or absence of nerve fibers, and Renaut bodies.

These changes support the theory that PSD in the hindlimb results is a compartment syndrome.

AVULSION FRACTURES OF THE THIRD METACARPAL OR METATARSAL BONE AT THE ORIGIN OF THE SUSPENSORY LIGAMENT

Avulsion fractures of the third metacarpal or metatarsal bone at the region of the SL are discussed on page 367.

SUSPENSORY DESMITIS: BODY LESIONS

Desmitis of the body of the SL is principally an injury of horses that race, STBs and TBs (flat racehorses and jumpers). In TBs in Europe the incidence is much higher in horses that race over fences (National Hunt Racing and point to pointing; see Chapter 113) compared with flat racehorses. Although PSD and desmitis of the medial and or lateral branches of the SL are relatively common injuries in event horses, show jumpers, dressage horses, and endurance horses, body lesions are recognized less frequently, except as associated with an exostosis of the McII or MtIV (splint) bones or as a sequel to a branch injury. Injuries occur in forelimbs and hindlimbs, and in STBs several limbs may be affected concurrently, whereas in TBs lesions generally are restricted to the forelimb.[23,33] There is frequently a poor correlation between the extent of the lesions and the degree of lameness. Performance, however, can be compromised despite no evidence of overt lameness. In STB racehorses lameness may be observed or tolerated while athletic function continues. This continued exercise may result in progressive injury or injury to other tendonous or ligamentous structures.

Soreness on palpation of the SL is not synonymous with structural damage. Event horses frequently have sore SLs for several days after fast work or competition, but rarely does this appear to be associated with a significant problem. Soreness does not appear to be a warning sign of impending desmitis.

Clinical Signs

The clinical signs associated with suspensory body desmitis vary in presence and degree and include the following:

- Localized heat
- Periligamentous edema. Severe periligamentous soft tissue swelling may make accurate palpation of the SL difficult.
- Rounding of the margins of the SL
- Enlargement of the body of the SL
- Pain on palpation of the margins of the SL
- Abnormal stiffness of the SL
- Pain on palpation of the distal third of the McII or MtIV bones, if there is a concurrent fracture
- Lameness. The absence of lameness does not preclude significant desmitis.

Diagnosis

Diagnosis is based on clinical signs and ultrasonographic assessment. Diagnostic analgesic techniques rarely are required unless another contributory cause of lameness or recurrence of previous desmitis is suspected. In a horse with previous desmitis a previously enlarged SL may have no detectable change in size, shape, or reaction to palpation. Perineural analgesia of the palmar metacarpal nerves proximal to the site of the suspect lesion should remove associated lameness.

The body of the SL of a normal horse is not always uniform in echogenicity because of variable amounts of muscle tissue in the ligament and variations in the level of bifurcation of the SL between horses. This can make detection of subtle lesions difficult. The maximum injury zone is frequently at the region of the bifurcation of the SL, which may appear hypoechoic in normal horses (Fig. 73-11). Careful comparison with the contralateral limb may help if clinical signs are unilateral. Ultrasonographic abnormalities associated with active desmitis include the following:

- Enlargement of the body of the ligament in transverse and median planes
- Loss of definition of one or more of the margins of the ligament
- Focal hypoechoic areas, peripheral or central, extending a variable distance proximodistally
- A diffuse reduction in echogenicity of some or all of the cross-sectional area of the ligament
- In horses with chronic desmitis, focal hyperechoic areas

Enlargement may be the only detectable abnormality. Careful comparison with the contralateral limb can be helpful. Reference can be made to studies of sizes in normal horses of similar breed, but caution is urged because notable differences do exist between American and European TBs. The extent of ultrasonographic abnormality is not always correlated with the severity of clinical signs. A reasonable correlation exists with the severity of the ultrasonographic grade of the injury and the prognosis; however, a diffuse reduction in echogenicity of the cross-sectional area has a similarly poor prognosis to a large anechoic lesion. Lesions may extend into the branches, which should also be examined by ultrasonography.

Many lesions persist long term, although focal lesions in STBs may fill in. In TBs a readily detectable defect often persists, despite some increase in echogenicity and slight reduction in size. This can limit the value of serial ultrasonographic

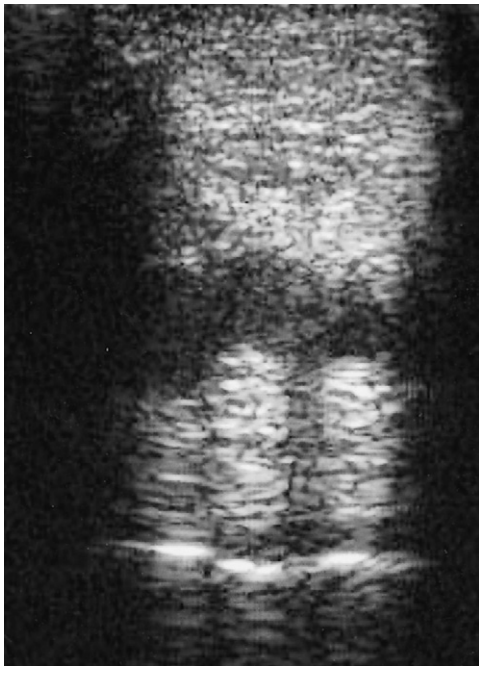

Fig. 73-11 Transverse ultrasonographic image of a normal forelimb suspensory ligament at the level of the beginning of the bifurcation, 18 cm distal to the accessory carpal bone. The central, less echogenic region should not be confused with a lesion.

evaluations to determine when a horse might be able to withstand work. Determining objectively to what extent healing has taken place and the strength of the repair tissue often is not possible. This may explain the high rate of recurrence of body lesions in STBs and TBs. Racing careers of STBs can usually be extended, whereas managing TBs is much more difficult, because lameness in TBs rarely stabilizes.[23] Horses with slight lesions may be responsive to symptomatic treatment, and a horse may train and race well; however, the lesion is likely to be progressive. The risk of catastrophic breakdown of the suspensory apparatus must always be considered in a TB racehorse. In STB racehorses rest for about 3 months may confer no extra advantage to symptomatic treatment and continued training[24] with respect to racing performance. Lesions are usually progressive, and periodic ultrasonographic monitoring is recommended.

There is a high rate of re-injury to the body of the SL of the same or contralateral limb or to a branch of the same or contralateral limb subsequent to desmitis of the body of the SL.[32]

Radiographic examination of the McII and McIV bones is indicated if clinical examination suggests abnormal modeling or a fracture, because surgical amputation may be indicated[23] (see page 371).

Management

Treatment is aimed at reducing inflammation by the administration of systemic NSAIDs, local or systemic corticosteroids, hydrotherapy, and controlled exercise. Cryotherapy is used by some clinicians to provide pain relief, but a real risk of progressive deterioration of the lesion exists (see Chapter 90).

Progress is monitored clinically and by serial ultrasonographic examinations. Ideally there should be progressive reduction in cross-sectional area, improvement in echogenicity and fiber alignment, no restrictive periligamentous fibrosis, and reasonable stability in all of these feature as exercise is increased. In STBs hand walking exercise is advocated for the first 4 weeks.[23] Provided that some improvement in the ultrasonographic appearance of the ligament occurs and the horse is sound when jogged in hand, the horse is harnessed and walked in the jog cart for another 4 weeks. Flat shoes are preferred. The horse is re-assessed by ultrasonography after 8 weeks, and if further filling in of the defect occurs, short slow jogging is reintroduced and gradually increased. A third ultrasonographic examination is performed after 8 weeks of jogging, and provided that the lesion is uniformly echogenic to the remainder of the SL and has a linear arrangement of fibers, normal jogging and speed work are resumed. Although this method is successful in some STBs, in others a longer period of convalescence is required.

Continued medical therapy includes daily hydrotherapy; for example, whirlpool boots, stable and exercise bandages, massage with emollient oils, leg sweats, and leg tighteners with or without DMSO. The aim is to stimulate circulation, reduce inflammation, and provide pain relief. The work program should be adapted so that days of light work are interspersed with hard work. Swimming is a useful method of maintaining cardiovascular fitness but should not be used alone, because the musculoskeletal system requires regular stimulus to avoid other injuries.

Intralesional treatment with β-aminoproprionitrile fumarate has been used in STB and TB racehorses with moderate to severe injuries of the SL body.[24] Eleven of 18 horses successfully returned to racing, although some dropped in class.

Surgical splitting of the body of the SL usually is not advocated in STBs or TBs.[23] However, occasionally, large chronic lesions with associated McII or McIV bone fractures are split, combined with surgical removal of the fracture piece in STB and, less commonly, TB racehorses. Pin firing combined with rest may be of benefit in some horses with chronic desmitis[23] (see Chapter 89).

SUSPENSORY DESMITIS ASSOCIATED WITH AN EXOSTOSIS ON THE SECOND OR FOURTH METACARPAL OR METATARSAL BONE (SPLINT)

Suspensory desmitis associated with exostosis of the second or fourth metacarpal (metatarsal) bone is discussed on page 370 (Fig. 73-12).

SUSPENSORY DESMITIS ASSOCIATED WITH FRACTURE OF THE DISTAL THIRD OF THE SECOND OR FOURTH METACARPAL OR METATARSAL BONE

Suspensory desmitis associated fracture of the distal third of the second or fourth metacarpal or metatarsal bone is discussed on page 371.

SUSPENSORY DESMITIS: BRANCH LESIONS

Desmitis of the medial and or lateral branches of the SL in forelimbs and hindlimbs is a relatively common injury in all types of sports horses. Usually only a single branch is affected in a single limb, although both branches may be affected, especially in hindlimbs. Foot imbalance often is recognized in affected horses and may be a predisposing factor. Some horses, particularly event horses, have acute onset distention of the metacarpophalangeal joint capsule concurrent with SL branch desmitis. Detection of radiographic abnormalities (modeling or fracture of the distal aspect of the McII or McIV bone or a PSB) in some horses at the time of recognition of an acute, first time injury, suggests a sub-clinical pre-existing problem.

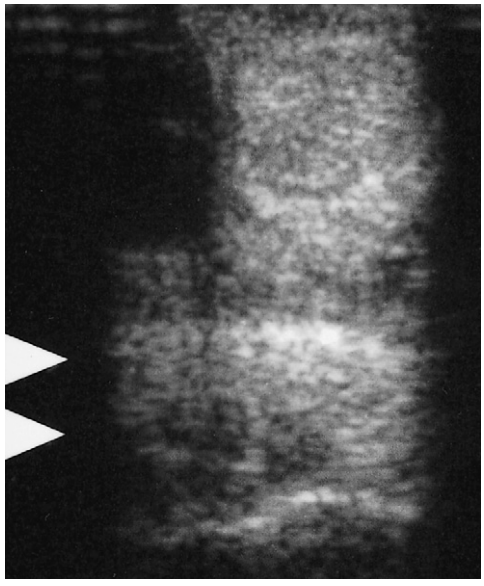

Fig. 73-12 Transverse ultrasonographic image of the mid-metacarpal region of 7-year-old show jumper with intermittent left forelimb lameness associated with localized enlargement of the axial aspect of the second metacarpal bone (a splint). Slight pain could be elicited by pressure. Separating the axial border of the bone from the suspensory ligament by palpation was not possible. Lameness was alleviated by local infiltration of local anesthetic solution. Echogenic material (*arrowheads*) extends from the medial aspect of the suspensory ligament, which was slightly hypoechoic dorsomedially. Surgical exploration revealed a loose spicule of bone adjacent to the splint and a granulomatous reaction between it and the suspensory ligament. A focal defect was found in the medial border of the suspensory ligament.

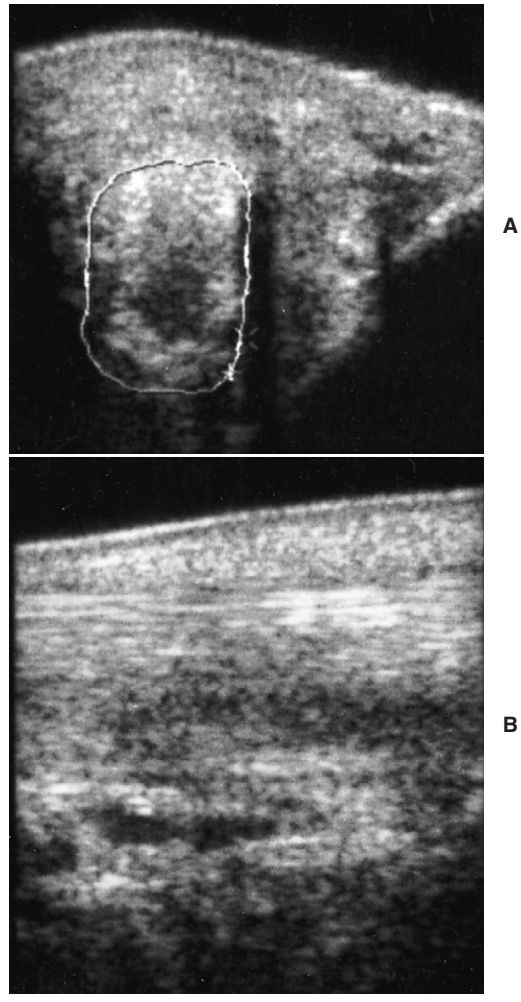

Fig. 73-13 **A,** Transverse ultrasonographic image of the medial branch of the suspensory ligament of the right forelimb of 14-year-old Grand Prix dressage horse. The suspensory ligament branch is enlarged (cross-sectional area 2.02 cm^2) and has a large, almost anechoic lesion. The echogenic material subcutaneously also contributed to the soft tissue swelling. **B,** Longitudinal ultrasonographic image of the medial branch of the suspensory ligament of the right forelimb of the same horse. Proximal is to the left. There is a marked loss of fiber pattern within the suspensory ligament. Note also the subcutaneous echogenic material.

Clinical Signs

The clinical signs depend on the degree of damage and chronicity of the lesion(s) and include localized heat and swelling. Swelling often is caused by enlargement of the branch, together with periligamentous edema or fibrosis. Associated distention of the metacarpophalangeal joint capsule may occur. In hindlimbs sometimes considerable distention of the digital flexor tendon sheath (DFTS) occurs, making assessing the SL branches by palpation difficult. Pain usually is elicited by direct pressure applied to the injured branch or by passive flexion of the fetlock. Lameness varies, may be absent, usually is proportional to the degree of damage, and is related inversely to the duration of injury. A notable exception is in the hindlimbs of older dressage horses, in which occasionally both branches sustain damage, and strong adhesions develop between them. These horses experience persistent, severe lameness. Occasionally younger horses from any discipline develop acute-onset, severe, and persistent hindlimb lameness associated with progressive stretching of a single branch and the development of periligamentous fibrosis. There is associated hyperextension of the fetlock.

Diagnosis

Diagnosis is based on clinical signs and ultrasonographic examination. Diagnostic analgesic techniques usually are required only if more than one lesion is suspected as the cause of lameness. In horses with acute branch injury, with concurrent distention of the metacarpophalangeal joint capsule, we suggest that the horse be re-evaluated after 2 to 3 weeks. If joint capsule distention and pain on manipulation persist, then the joint should be blocked intra-articularly.

The entire SL should be examined by ultrasonography, because lesions may extend beyond areas that are palpably abnormal. Ultrasonographic abnormalities include the following (Figs. 73-13 to 73-15):

- Abnormalities of the body of the SL (see page 662)
- Enlargement of the branch
- Change in shape of the branch
- Loss of definition of one or more margins of the branch
- Well-defined or poorly defined hypoechoic areas, central or peripheral
- A diffuse reduction of echogenicity involving some or all of the cross-sectional area of the branch
- Echogenic material subcutaneously
- Echogenic material between the medial and lateral branches
- Hyperechoic foci or larger masses within the branch
- An irregular contour or fracture of the ipsilateral PSB
- An abnormal amount of fluid within the DFTS

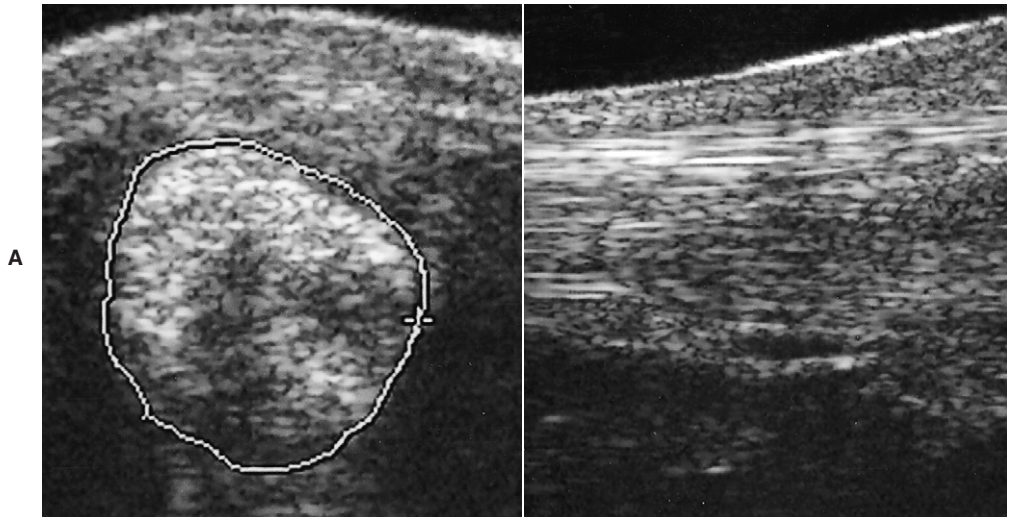

Fig. 73-14 **A,** Transverse ultrasonographic image of the lateral branch of the suspensory ligament of the left hindlimb of 7-year-old intermediate-level event horse with lameness of 2 weeks' duration. The suspensory branch is considerably enlarged (cross-sectional area 2.0 cm^2). Its margins are poorly defined and there is marked loss of echogenicity. **B,** Longitudinal ultrasonographic image of the lateral branch of the suspensory ligament of the same horse. Proximal is to the left. The ligament is hypoechoic, with loss of fiber pattern.

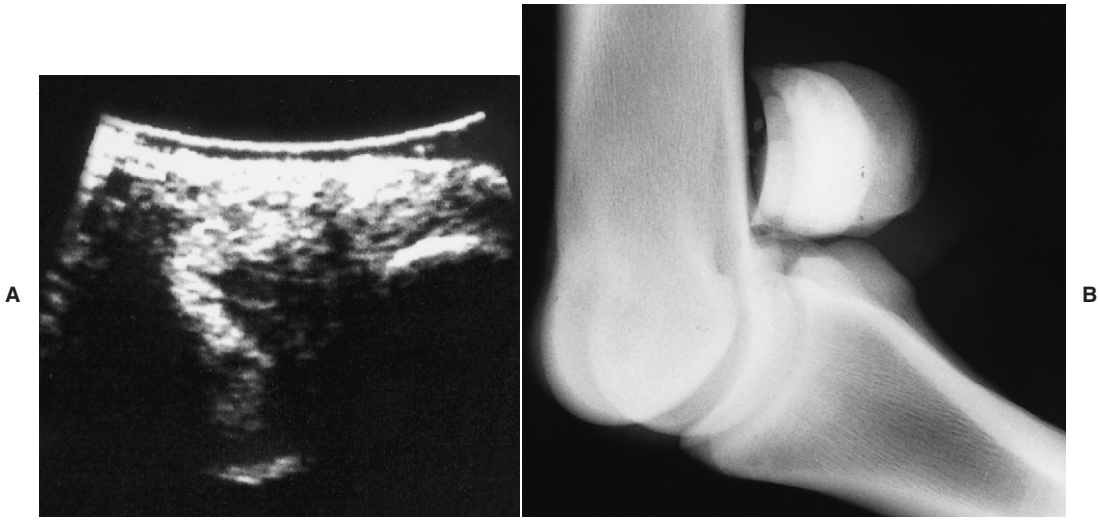

Fig. 73-15 **A,** Transverse ultrasonographic image of the lateral branch of the suspensory ligament, close to its insertion on the lateral proximal sesamoid bone, of the right hindlimb of 9-year-old advanced event horse with acute onset lameness. The ligament has a large, almost anechoic defect. **B,** Flexed, slightly oblique lateromedial radiographic view of the right hind fetlock of the same horse. The lateral proximal sesamoid bone is positioned slightly more dorsally. Several small osseous opacities are seen dorsal to it. These were removed arthroscopically, and torn fibers at the suspensory ligament branch insertion were debrided.

The branches should be examined in transverse and longitudinal planes. Lesions restricted to the insertion are sometimes only detectable in longitudinal images. Sometimes horses show slight localized swelling and heat in the region of a SL branch, with a subtle alteration in gait, in which no ultrasonographic abnormality of the SL can be identified. The swelling appears to be principally periligamentous. Some of these horses can be maintained safely in work, whereas in others clinical signs deteriorate with the development of a branch lesion.

Radiographic examination of the ipsilateral splint bone and PSB should be performed. Abnormalities may include the following:

- Dystrophic mineralization in the SL
- Distortion in shape of the ipsilateral splint bone
- Fracture of the distal aspect of the ipsilateral splint bone
- Fracture(s) of the ipsilateral PSB (see Fig. 73-15)
- Radiating lucent lines within the ipsilateral PSB
- Modeling of the palmar aspect of the PSB

Fractures influence the treatment and prognosis. However, radiographic evidence of sesamoiditis is not well correlated with the outcome, although primary sesamoiditis unassociated with SL desmitis can cause recurrent lameness (see Chapter 37).

Management

Treatment depends on the occupation of the horse, the breed, and the severity of the clinical signs and ultrasonographic abnormalities. Horses with acute central core lesions may be treated by splitting, with horses returning to work within 9 months. Horses with more peripheral lesions or those poorly demarcated are not suitable for splitting, but their injuries can be managed conservatively by appropriate trimming and shoeing, box rest, and a controlled increasing exercise program. Intra-lesional injection of β-aminoproprionitrile fumarate (five 7-mg injections on alternate days) combined with a controlled increasing work program over 6 to 9 months has been successful.[22]

Dressage horses and show jumpers with minor lesions and only subtle ultrasonographic abnormalities have been managed successfully by appropriate trimming and shoeing with egg bar shoes, combined with modification of the training program for 6 to 8 weeks. Some horses can be managed successfully and maintained in work by aggressive local therapy (e.g., use of whirlpool boots, laser or ultrasound therapy, leg sweats, and cold therapy). Continued work in an event horse usually results in significant progression of the lesion. In a TB racehorse, continued training in the face of SL branch injury runs the risk of an acute breakdown injury of the suspensory apparatus. In STB racehorses symptomatic treatment is common, with NSAIDs as required, daily icing, topical application of a DMSO-cortisone liniment, and modification of the training schedule to light jogging or swimming between races. Lesions may remain stable or progress slowly and usually ultimately compromise performance significantly. Management is more difficult in horses that race at a high level, so training cannot be reduced enough, or the speed of the race results in overload of the compromised structure. Horses seem to do less well on a slower track surface, with a deep cushion, compared with a fast track surface.

Ultrasonographic lesions are often slow to resolve and some persist long term (longer than 18 months). Some horses are able to resume full work after a convalescent period of at least 9 months, despite the persistence of a lesion, although the incidence of recurrent injury is high. If a lesion persists as viewed by ultrasonography, knowing when to recommend resumption of work is difficult. Generally the horse should have been rested at least 6 months and no appreciable change should be found in the ultrasonographic appearance of the SL branch between two examinations 3 months apart. With increased exercise the ligament should remain stable in size and echogenicity.

Horses with hindlimb suspensory branch lesions with periligamentous fibrosis and shortening of a SL branch or echogenic material extending between the two branches have a poor prognosis. This echogenic material represents firm fibrous adhesions between the branches.

PROXIMAL SESAMOID BONE FRACTURE ASSOCIATED WITH A BRANCH INJURY

Sesamoid fractures associated with SL branch injury are discussed on page 354.

AVULSION FRACTURE OF THE PROXIMAL SESAMOID BONE AT THE INSERTION OF THE PALMAR ANNULAR LIGAMENT

Avulsion injuries of the palmar annular ligament (PAL) at its insertion on a PSB are not common[22,34] and must be differentiated carefully from abaxial proximal sesamoid bone fractures (see Chapter 37, page 354). Injuries occur in forelimbs and hindlimbs. Horses with acute injury show lameness associated with diffuse soft tissue swelling palmar to a proximal sesamoid bone.[22] Swelling does not involve the ipsilateral SL branch unless that branch has been damaged concurrently. There may also be mild distention of the fetlock joint capsule and the digital flexor tendon sheath. It may be possible to elicit pain by firm pressure applied to the abaxial aspect of the sesamoid bone. In horses with more chronic injury no localizing clinical signs may be apparent. Radiographic examination may reveal a punched-out lesion of the PSB (Fig. 73-16) or a small detached fragment of bone (Fig. 73-17), the origin of which cannot be determined. Ultrasonographic examination helps to determine the origin of the fragment. The PAL should be examined from the palmar aspect of the limb and then followed around to its insertion. An osseous echogenic body may be seen attached to the PAL close to the PSB. Treatment by surgical removal of the fragment or conservative management has resulted in return to full athletic function.[22,34]

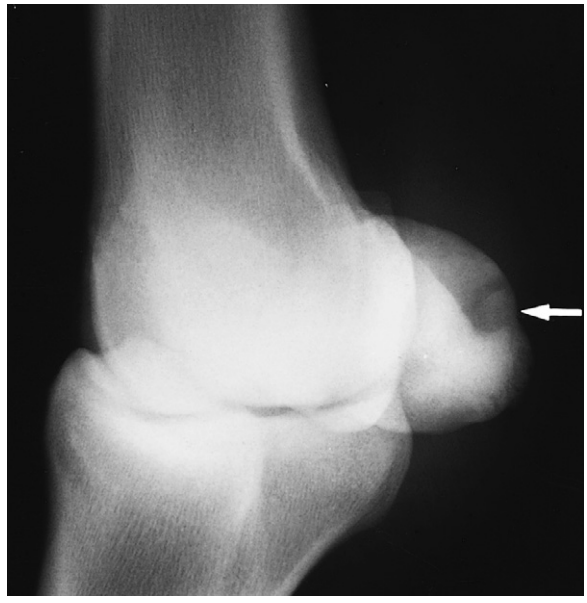

Fig. 73-16 Dorsolateral-palmaromedial oblique radiographic view of the right front fetlock of 7-year-old advanced event horse. A punched-out opacity is visible on the palmar aspect of the bone (*arrow*). This is an avulsion injury at the insertion of the palmar annular ligament. Note also the new bone on the palmar distal aspect of the bone.

DAMAGE OF THE PALMAR (PLANTAR) LIGAMENT OF THE FETLOCK (INTERSESAMOIDEAN LIGAMENT)

Focal Tears in the Body of the Intersesamoidean Ligament

Focal tears in the body of the intersesamoidean ligament are an unusual cause of lameness and can only be diagnosed definitively by arthroscopic evaluation of the palmar (plantar) pouch of the metacarpophalangeal (metatarsophalangeal) joint.[22] Lameness is usually acute in onset and moderate to severe. Lameness improves with rest but often persists. There may be effusion in the metacarpophalangeal joint. Lameness is improved by intra-articular analgesia. Usually no identifiable radiographic abnormalities occur. Small focal tears are extremely difficult to identify using ultrasonography. Nuclear

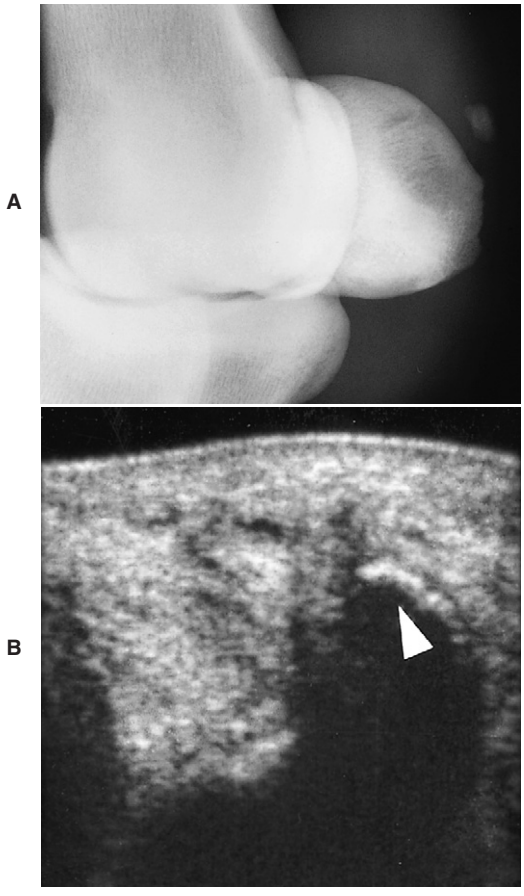

Fig. 73-17 **A,** Dorsomedial-palmarolateral oblique radiographic view of the right front fetlock of 9-year-old Grand Prix show jumper with acute-onset lameness associated with soft tissue swelling on the medial aspect of the fetlock, palmar to the medial branch of the suspensory ligament. There was also slight edematous swelling around the distal aspect of the medial branch of the suspensory ligament. There is an osseous fragment palmar to the proximal sesamoid bones, but its origin could not be determined radiographically. **B,** Transverse ultrasonographic image of the palmaromedial aspect of the fetlock. A thick hyperechoic line represents the osseous fragment (*arrow*) that was an avulsion of the insertion of the palmar annular ligament.

scintigraphic examination of a small number of horses with small focal tears not involving the insertion of the ligament has revealed slight generalized increased radiopharmaceutical uptake in the fetlock region but not localized to the PSBs. Horses with focal ligament tears have a poor response to surgical debridement and prolonged rest, with or without intra-articular medication, and the prognosis for return to athletic function is guarded.

Degeneration or Partial Rupture of the Intersesamoidean Ligament

Degenerative lesions and partial rupture of the intersesamoidean ligament have been seen only in older, mature athletic horses and are relatively rare.[35] The condition has been recognized in forelimbs and hindlimbs and may occur as a single injury or with other soft tissue or bony lesions.

Lameness is acute in onset and moderate to severe. Mild distention of the metacarpophalangeal (metatarsophalangeal) joint capsule or the DFTS may occur. Lameness may be improved by intra-articular analgesia, but the response to perineural analgesia of the palmar and palmar metacarpal nerves is often better.

Diagnosis is based on ultrasonography, and a number of abnormalities have been identified:

- An increase in the space between the PSBs
- An increased thickness of the palmar ligament
- Reduced echogenicity of the palmar ligament
- Focal hyperechoic regions in the palmar ligament
- Dorsal displacement of the flexor tendons
- Thinning of the palmar ligament and reduction in the space between the ligament and the dorsal aspect of the DDFT
- Irregularity of the facies flexoria of a PSB (This may correlate with radiolucent areas in the axial aspect of the bone.)

Careful examination of the flexor tendons, the PAL, and the distal sesamoidean ligaments should be performed to identify any concurrent injuries.

Radiographic examination occasionally may reveal osteolytic lesions on the axial aspect of the PSBs (see the following discussion).

The prognosis for return to athletic function is extremely guarded.[22]

Insertional Injury of the Intersesamoidean Ligament

Aseptic necrosis of the axial aspect of the PSBs has been described as a cause of lameness in forelimbs and hindlimbs.[35,36] The condition has been characterized by the development of radiolucent zones in the axial aspect of one or both PSBs (Fig. 73-18), lesions which have also been seen with infection (see the following discussion). Histological examination of two horses indicated that these lesions reflect trauma of the insertion of the intersesamoidean ligament.[22] Insertional lesions of the palmar ligament of the fetlock also may occur without radiographic change and have been identified arthroscopically (Fig. 73-19).

In contrast to horses with associated infection, usually no substantial soft tissue swelling occurs in the fetlock region, although distention of the metacarpophalangeal (metatarsophalangeal) joint capsule, the DFTS, or both may occur. In some horses eliciting pain by palpation may be difficult. Lameness may vary from moderate to severe and is often worse with the horse walking in a circle compared with walking in a straight line.

Regional analgesia by a four-point block usually results in much better improvement in lameness than intra-articular analgesia. In the acute stage radiographic examination may be negative. Ultrasonographic evaluation may be unrewarding, but in some horses an irregular outline of one of the PSBs may be seen, with reduced echogenicity of the intersesamoidean ligament (see Fig. 73-18).

Nuclear scintigraphic examination usually reveals increased radiopharmaceutical uptake in one or both PSBs in contrast to the pattern of uptake that has been seen with focal lesions in the body of the intersesamoidean ligament (see the previous discussion). Increased radiopharmaceutical uptake localized to a PSB also may be caused by an incomplete fracture or subchondral trauma. Sequential radiographic examination may reveal the development of lucent zones on the axial aspect of the sesamoid bones.

These lesions may be associated with long-term lameness, although surgical debridement has resulted in return to former athletic function in some horses.[36,37] Arthroscopic evaluation usually reveals a defect in the palmar ligament, through which a probe can be inserted into the bony defect. Defects usually are more pronounced in one PSB, but they can involve both PSBs. Damaged palmar ligament and bone are debrided. Occasionally, to approach lesions not involving the articular surface, it may be necessary to create a small apical fracture to gain access to the bony defect.

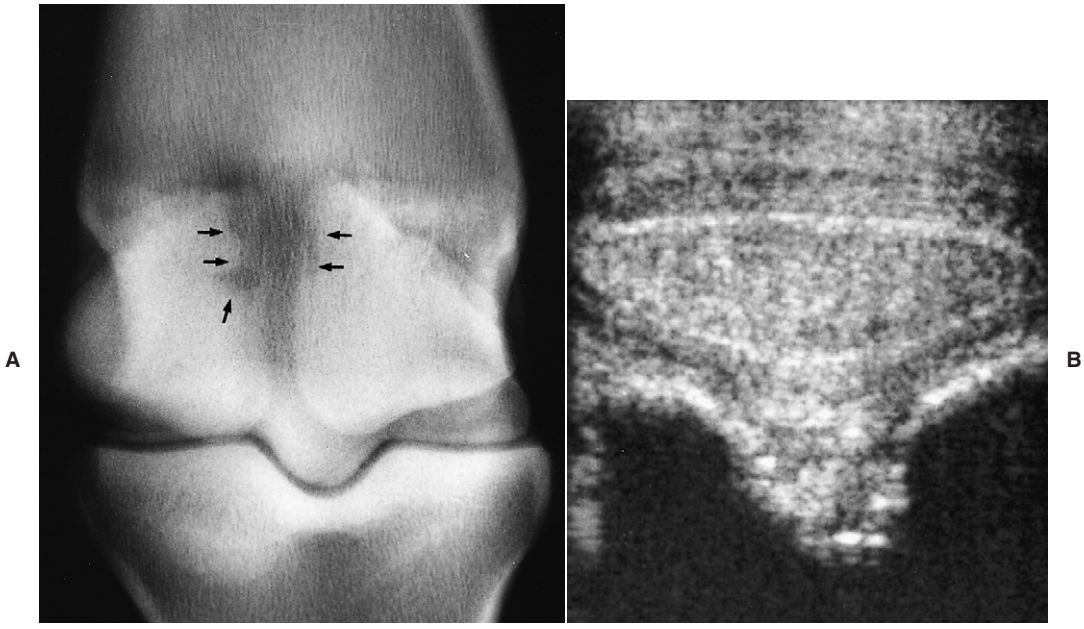

Fig. 73-18 A, Dorsoplantar radiographic image of the right hind fetlock of 6-year-old Thoroughbred with severe lameness associated with moderate distention of the metatarsophalangeal joint capsule and mild distention of the digital flexor tendon sheath. Medial is to the left. The axial borders of the proximal sesamoid bones are poorly defined proximally, with lucent areas in the bone (*arrows*). Histological examination confirmed traumatic insertional injuries of the palmar ligament of the fetlock. **B,** Transverse ultrasonographic image of the plantar aspect of the fetlock of the same horse. Medial is to the left. Note the irregularity of the outline of the proximal sesamoid bones and the rather heterogeneous echogenicity of the intersesamoidean ligament.

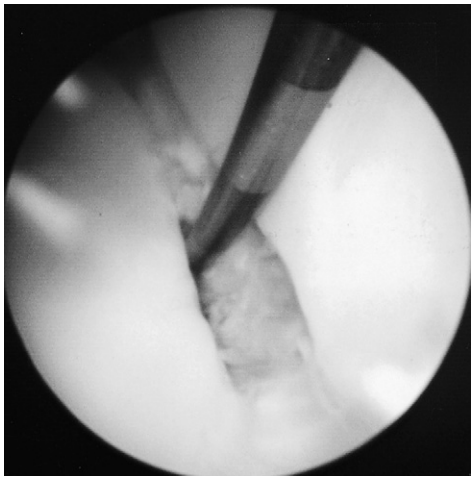

Fig. 73-19 Arthroscopic picture of focal tear in the palmar ligament of the fetlock at its insertion on the medial proximal sesamoid bone. (Courtesy I.M. Wright, Newmarket, Suffolk, England.)

INFECTION OF THE AXIAL ASPECT OF THE PROXIMAL SESAMOID BONES

Aseptic necrosis[36,37] and osteomyelitis[38] of the PSBs have been described, but in one author's experience (S.J.D.) aseptic necrosis occurs more commonly. Aseptic necrosis is probably an inappropriate term because the Editors believe that lesions develop secondary to trauma of the insertion of the intersesamoidean ligament (see the previous discussion). Infection is not necessarily associated with a known route of infection, but several horses with infection have been seen after puncture wounds in the pastern region or cellulitis[22] (see Fig. 73-19).

Infection of the PSBs may occur in forelimbs and hindlimbs and is characterized by radiolucent zones involving the axial margin of the medial or lateral PSB, or both. Pain may be elicited by pressure over the PSBs. Fetlock flexion usually is resented. Distention of the fetlock joint capsule and the DFTS and surrounding soft tissue swelling are variable features. Synoviocentesis may reveal evidence of infection in adjacent structures. In some horses a penetrating wound can be identified in the palmar aspect of the fetlock or pastern. Lameness varies from moderate to severe.

High-resolution dorsal 15° to 20° proximal-palmaro (plantaro) distal oblique radiographic views, with adequate penetration, are essential for diagnosis. If the radiographs are underexposed, lesions will be missed. Ultrasonography is useful for confirming the diagnosis and for identifying other concurrent lesions (Fig. 73-20), such as extensive tenosynovitis of the DFTS, deposition of echogenic material around the affected PSB, reduced echogenicity of the intersesamoidean ligament, and lesions of the flexor tendons or adhesions between them.[1]

The prognosis with conservative or surgical management is extremely guarded.

STRAIGHT DISTAL SESAMOIDEAN DESMITIS

Desmitis of the straight distal sesamoidean ligament is an unusual cause of lameness that usually occurs in a forelimb, but it has been recognized in hindlimbs.[39] One of us (S.J.D.) has recognized it most commonly in event horses. The condition may occur alone or together with injury to an oblique distal sesamoidean ligament. Lameness is sudden in onset, usually with no detectable swelling in the acute phase. Some

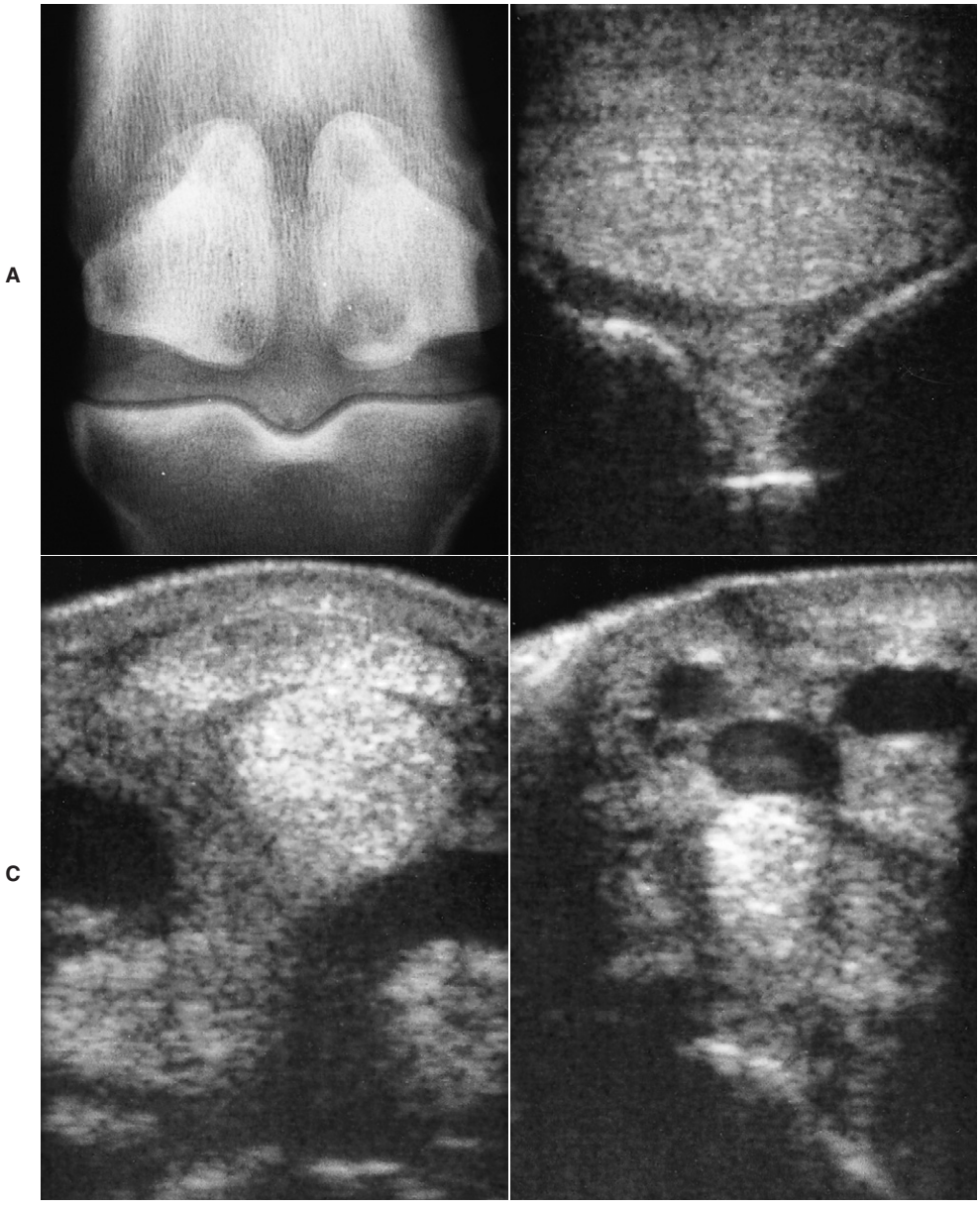

Fig. 73-20 **A,** Dorsopalmar radiographic view of the right metacarpophalangeal joint of 8-year-old Thoroughbred with massive swelling of the metacarpal region, restricted flexibility of the fetlock and distention of the digital flexor tendon sheath. There are large radiolucent zones involving the distal axial borders of the proximal sesamoid bones. **B,** Transverse ultrasonographic image of the distal aspect of the metacarpal region of the same horse. Note the asymmetrical appearance of the proximal sesamoid bones and the difference in distance between them and the dorsal border of the deep digital flexor tendon. **C,** Transverse ultrasonographic image of the distal metacarpal region of the same horse. Note the abnormal amount of fluid within the digital flexor tendon sheath and the echogenic material around the deep digital flexor tendon. **D,** Palmar oblique ultrasonographic view. Note the subcutaneous echogenic material and the distended vessels.

swelling may develop subsequently, but this can be difficult to detect if the lesion is far proximal. Concurrent effusion may occur in the DFTS, and palpation on the palmar midline of the pastern region may elicit pain, but not invariably so. Lameness may be severe acutely and then rapidly improving but persisting. Lameness is alleviated by palmar nerve blocks at the level of the base of the PSBs. Diagnosis is based on ultrasonography and abnormalities include the following (Fig. 73-21):

- Enlargement of the straight sesamoidean ligament and therefore reduction in the space between it and the DDFT
- Focal or diffuse reduction in echogenicity
- Increased fluid within the DFTS in some horses

Care should be taken not to misinterpret the normal hypoechoic area in the most distal part of the ligament as a lesion. This represents the cartilaginous scutum at the insertion.

Lesions may occur localized to the insertion, either unilaterally or bilaterally, and alone or in conjunction with other causes of palmar foot pain, such as navicular disease. There is usually no palpable abnormality. Lameness is removed by perineural analgesia of the palmar nerves at the base of the proximal sesamoid bones. However, improvement may be seen after

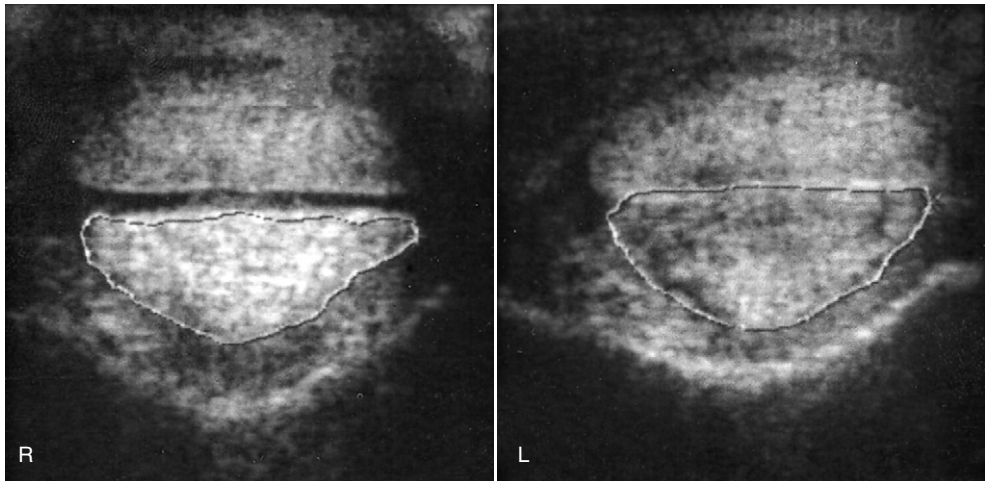

Fig. 73-21 Transverse ultrasonographic images of the left (on the left) and right proximal pastern regions of an advanced event horse with acute onset, severe right forelimb lameness after completing a cross-country course. The lameness improved greatly within 24 hours. Flexion of the right front fetlock elicited pain, but no swelling or pain on direct palpation could be detected. Lameness was alleviated by perineural analgesia of the palmar nerves at the level of the fetlock joint. The right straight sesamoidean ligament is enlarged (cross-sectional area 1.46 cm^2) compared with the left (1.35 cm^2), resulting in loss of space between it and the deep digital flexor tendon. The ligament is also hypoechoic.

palmar digital nerve blocks if these are performed in the midpastern region or because of proximal diffusion from a more distal injection site. Although some lesions can be identified ultrasonographically, the shape of the pastern influences the ease with which these structures can be imaged accurately. Recent experience with magnetic resonance imaging (MRI) suggests that these injuries may occur more frequently than previously recognized. MRI seems more sensitive for the detection of insertional injuries compared with ultrasonography.

Occasionally concurrent lesions of the straight and oblique sesamoidean ligaments occur and have a more guarded prognosis.

Treatment has consisted of box rest, corrective trimming and shoeing, and controlled walking exercise, with periodic reevaluation with ultrasonography. Usually progressive resolution of lameness occurs over 6 to 8 weeks, with gradual improvement in the ultrasonographic appearance of the ligament over 6 months. However, the prognosis for return to full athletic function at a high level of competition is guarded, because the incidence of recurrent injury is high.

Loss of tension (relaxation) of the straight sesamoidean ligament may occur if the SL is ruptured, resulting in instability or luxation of the proximal interphalangeal joint (palmar/plantar subluxation). Ultrasonographic abnormalities include the following:

- Apparent enlargement of the ligament cross-sectional area
- Loss of fiber alignment
- Focal hypoechoic areas caused by lack of tension

OBLIQUE (MIDDLE) DISTAL SESAMOIDEAN DESMITIS

Desmitis of one or both oblique sesamoidean ligament occurs in forelimbs and hindlimbs, but it is a more common source of forelimb lameness. Lameness is usually acute in onset and moderate to severe. In the acute stage often no localizing clinical signs are apparent, but in the following 7 to 10 days swelling may develop in the pastern region, and pain may be elicited by firm palpation. However, lesions may occur without detectable palpable abnormalities, alone or with another cause of lameness. Lameness, which is often worst on a soft surface, is alleviated by palmar (abaxial sesamoid) nerve blocks. One should note that tendonitis of the medial or lateral branch of the SDFT is a more common injury in the pastern, has a similar clinical presentation, and is more likely to be associated with detectable soft tissue swelling.

One also must recognize that enthesophyte formation on the palmar aspect of the proximal phalanx at the site of insertion of the oblique sesamoidean ligaments is a common incidental radiographic abnormality frequently unassociated with clinical signs and often not associated with any detectable ultrasonographic structural abnormality of the ligament. Well-circumscribed, smoothly marginated osseous bodies, possibly old avulsion fractures from the base of a PSB, are common radiographic and ultrasonographic observations in mature horses in which the oblique sesamoidean ligament appears to be structurally normal (see Fig. 115-7).

Ultrasonographic abnormalities of the oblique sesamoidean ligament are often identified concurrently with some other cause of lameness; for example, osteoarthritis of the proximal interphalangeal joint or desmitis of a branch of the SL.

Ultrasonographic abnormalities include one or more of the following (Figs. 73-22 and 73-23):

- Enlargement
- Diffuse reduction of echogenicity
- Focal hypoechoic areas
- Poor demarcation of the margins of the ligament
- Reduction in space between the ligament and the SDFT
- Entheseophyte formation on the palmar (plantar) aspect of the proximal phalanx or the base of the ipsilateral PSB
- An avulsion fracture of the base of the ipsilateral PSB or at the insertion on the palmar aspect of the proximal phalanx

Each ligament should be evaluated carefully in transverse and longitudinal planes. Occasionally lesions occur together with injuries to other soft tissues of the pastern, so all structures should be evaluated systematically.

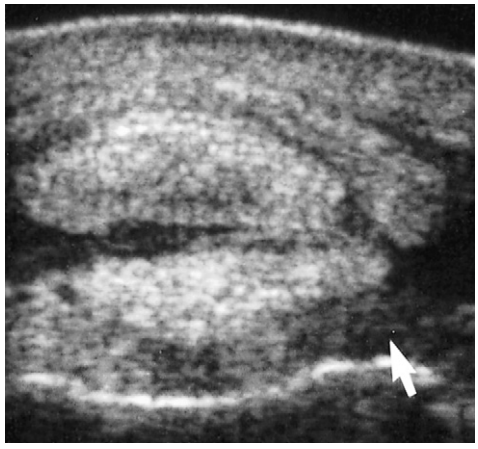

Fig. 73-22 Transverse ultrasonographic image of 7-year-old Thoroughbred with left forelimb lameness of 10 days' duration. Medial is to the left. The lateral oblique sesamoidean ligament is diffusely hypoechoic (*arrow*).

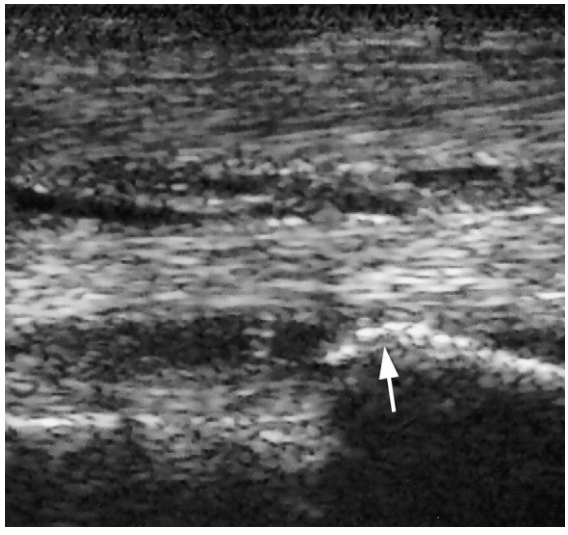

Fig. 73-23 Longitudinal ultrasonographic image of the right front pastern of 7-year-old pleasure riding horse with moderate lameness. Proximal is to the left. A large enthesophyte (*arrow*) is visible on the palmar aspect of the proximal phalanx. The region of insertion of the oblique sesamoidean ligament is reduced markedly in echogenicity. The horse also had osteoarthritis of the proximal interphalangeal joint.

Horses with a primary injury of the distal sesamoidean ligaments have a high incidence of recurrent injury if returned to full athletic function after conservative management. However, horses with primary lameness of another cause may be seen with an enlarged oblique sesamoidean ligament of normal echogenicity, apparently unassociated with clinical signs.

PROGRESSIVE ATRAUMATIC BREAKDOWN OF THE HINDLIMB SUSPENSORY LIGAMENTS

Progressive degenerative changes in hindlimb SLs have been seen in a small number of horses that originally showed PSD and had straight hock conformation. These horses developed hyperextension of the hind fetlock joints (Fig. 73-24). The condition has been characterized by a diffuse decrease in echogenicity of the proximal suspensory ligaments, which

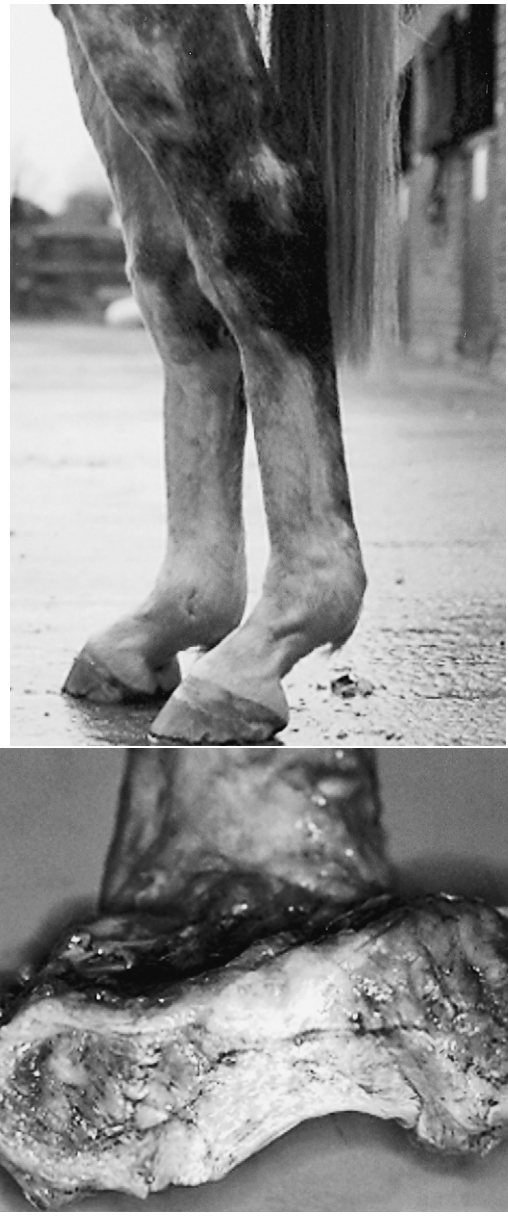

Fig. 73-24 **A,** The hindlimbs of 14-year-old Thoroughbred gelding with hyperextension of the hind fetlocks and soft tissue enlargement around the distal aspect of the branches of the suspensory ligaments. The superficial digital flexor tendon of the right hindlimb was subluxated laterally. **B,** The distal aspect of the suspensory ligament branches of the same horse. Considerable fibrous tissue surrounds the branches. The ligaments were discolored substantially, with no normal-appearing ligamentous tissue.

becomes progressively more extensive distally. A similar clinical condition has been recognized in older horses, especially broodmares, with progressive hyperextension (dorsiflexion) of the hind fetlock joints, which may result in abrasion of the plantar aspect of the fetlocks. Progressive hyperextension of the hind fetlock joints also has been seen in middle-aged performance horses with apparent lengthening of branches of the SL and periligamentous fibrous reaction. Some breeds, including the Peruvian paso and Andalusian horses, seem particularly at risk. The condition is generally bilateral and is associated with a deterioration in hindlimb gait rather than an overt lameness. Secondary lateral luxation of the SDFT from the point of hock occurs occasionally, probably associated with the straight angle of the hock.

The cause of these conditions is not known. They are generally progressive. Flat shoes with plantar extensions help to lift the fetlock and may prevent secondary trauma, but the prognosis for proper athletic function is guarded.

TRAUMATIC DISRUPTION OF THE SUSPENSORY APPARATUS

Traumatic disruption of the suspensory apparatus is discussed on page 359.

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CHAPTER • 74

Clinical Use of Stem Cells and Marrow Components to Stimulate Suspensory Ligament Regeneration

Doug Herthel

As a result of 22 years of diagnosing and treating suspensory ligament (SL) injuries and experiencing few advances in returning horses to full soundness, a novel, minimally invasive method of obtaining bone marrow aspirates from an anesthetized patient's sternum and injecting it into damaged ligament was initiated 6 years ago at Alamo Pintado Equine Medical Center. As a result of using fresh autologous stem cells and bone marrow components intralesionally, we were able to accelerate and improve the ultrasonographic quality of ligament healing and return horses to soundness sooner and with much less incidence of recurrence than previously experienced. The technique is a useful alternative and adjunct to other non-invasive and invasive therapies and is safe, economical, and effective. The procedure has been used in more than 500 horses with ligament or tendon damage at the medical center and in hundreds of horses with suspensory desmitis at many private and university equine hospitals in the United States and in Europe.

SCIENTIFIC RATIONALE FOR USING BONE MARROW

Autologous bone marrow contains many of the elements required for ligament regeneration, such as growth and differentiation factors, cell transplants, pluripotential cells (mesenchymal stem cells), and fibrin matrices. Stem cells are primitive undifferentiated cells that exist in bone marrow. These cells have the capacity to reproduce themselves and to form the entirety of bone marrow. In people, bone marrow transplants are used to achieve permanent marrow engraftment. Bone marrow aspirate contains about 2 stem cells per 100,000 cells. Stem cells have potential for limitless replication and differentiation into multiple cell lineages in response to the local environment (e.g., local cytokines and growth factors). One hundred stem cells have the ability completely to repopulate the entire immune system of a laboratory mouse, and 1×10^6 stem cells are enough to repopulate a person's immune system.

Bone marrow aspirates also contain numerous growth factor peptides, macrophages, neutrophils, platelets, fibrinonectin, fibrinogen, and endothelial cells, all of which have potential to facilitate ligament regeneration. Certain growth factors such as platelet-derived growth factor are chemoattractant agents for fibroblasts and can cause migration of these cells to the recipient site. Growth factors can induce mitogenesis of existing cells and up-regulation of extracellular matrix and collagen production. Similar results were seen in dog wound healing studies using transforming growth factor β (TGF-β). Platelet-derived growth factors have the additional ability to stimulate angiogenesis, inducing mitosis and formation of new functioning capillary blood vessels. In our unpublished studies new capillary formation has been prominent after bone marrow injection. Bone marrow contains about 0.06 ng of platelet-derived growth factor per 1 million platelets, or about 1,200 molecules of platelet-derived growth factor per individual platelet. Bone marrow aspirate contains about 6×10^9 platelets per 30 ml. Experimental studies using as little as 2 ng of platelet-derived growth factor applied to laboratory animal wound models increased fibroblast migration, collagen synthesis, and wound strength by 70%. Percutaneous injection of bone marrow for ligament healing was first reported in 1987; platelet-derived growth factor and TGF-β in aspirates enhanced wound healing in rats by stimulating collagen synthesis.[1] Seeding cultured stem cells into a tendon gap resulted in significantly improved repair biomechanics.[2] With the stimulation and signaling of cytokines and growth factors we hypothesized that equine stem cells may have the ability to morph into collagen-producing fibroblasts that may be deficient in damaged equine SLs and that fibroblasts already present in the damaged SL may be stimulated to produce collagen.

SUSPENSORY DESMITIS

Suspensory desmitis is a common musculoskeletal injury in sport horses and is discussed in detail in Chapter 73. Most injuries are mild and heal uneventfully, but some horses develop severe, acute suspensory desmitis or chronic suspensory desmitis that causes protracted clinical signs and horses are prone to re-injury. Many different therapeutic approaches, including ligament splitting, hyaluronan and iodine injections, blistering, pin firing, acupuncture, cold laser, and rest and rehabilitation have been used to promote ligament healing, but success rate is poorly documented.

TECHNIQUE

All horses are placed under general anesthesia and positioned in dorsal recumbency. Aseptic preparation of the affected suspensory ligament and the sternum is performed. A 60-ml syringe attached to a Jamshidi 10-gauge bone marrow needle is used to aspirate 20 to 30 ml of liquid bone marrow from the sternebrae (12 to 15 cm cranial to the xyphoid). Bone marrow is transferred immediately to 6-ml syringes using a sterile three-way stopcock, and lesions are injected through preplaced 18-gauge needles using ultrasound guidance. A total volume of 20 to 30 ml is used for each suspensory lesion, but additional bone marrow can be harvested from other sites in the sternum. No antibiotics or anticoagulants are added to bone marrow to prevent injury or to depress cells and proteins. Multiple lesions are treated with bone marrow aspirated from different locations to prevent dilution of the marrow with blood.

In horses with proximal suspensory desmitis in which dramatic clinical and ultrasonographic evidence of ligament

enlargement existed, concurrent forelimb or hindlimb fasciotomy was performed. Using a medial approach the fascia superficial to the proximal SL was transected to allow direct open injection of bone marrow. In most horses the original 2-cm incision was extended in both proximal and distal directions using a straight Mayo scissors.

RESULTS OF INTRALESIONAL BONE MARROW INJECTIONS

Between October 1995 and December 1998, 100 horses were treated for suspensory desmitis using bone marrow injection. Fifteen horses had concurrent fasciotomy. Horses satisfied one of the following criteria: clinical and ultrasonographic evidence of a severe, acute suspensory desmitis or moderate to severe lameness from chronic suspensory desmitis. Ultrasonographic changes included circumscribed peripheral or central hypoechoic lesions, significant increase in cross-sectional area, loss of ligament margins, and associated third metatarsal or metacarpal bone avulsion fractures, or combinations of these. Some horses were included that lacked substantial evidence of suspensory desmitis, but they had lameness abolished by diagnostic analgesia in the proximal palmar metatarsal or metacarpal regions and a substantial increase in radiopharmaceutical uptake in scintigraphic images.

Nine breeds of horses were represented, including in descending order, Warmbloods, Thoroughbreds, Quarter Horses, Andalusians, Peruvian pasos, Arabians, Standardbreds, Saddlebreds, and Morgans. Horses ranged from 2 to 24 years of age. Eighty-one horses had forelimb suspensory desmitis, and 19 horses had hindlimb suspensory desmitis. The most common sporting activities were dressage, jumping, and racing. Follow-up clinical and ultrasonographic examinations were performed at 60, 90, 120, and 180 days. Two horses had second injections.

After 6 months, 84 horses were sound and had returned to full work. Eight horses improved and went back to work but had mild but manageable lameness. Five horses remained too lame to return to normal work. Three horses died from unrelated causes and could not be followed past 6 months.

In one horse a hematoma developed at the injection site, likely from inadvertent puncture of a nearby artery. In two horses mild lameness was seen for 2 days after surgery, but in all others lameness did not increase at any evaluation period.

DISCUSSION

Although this study lacked controls, results were impressive. Theoretical benefits of bone marrow injection in returning damaged SL to near its previous biochemical, morphological, and biomechanical properties could be expected. Bone marrow injection, compared with natural healing, may improve blood supply and establish cell numbers to produce high-quality collagen and matrix. The clinical results of these 100 horses are promising, and many have competed in upper levels of sporting events. Bone marrow injection can be used for horses with tendonitis of the superficial or deep digital flexor tendons, desmitis of the accessory ligament of the deep digital flexor tendon, and desmitis of the straight distal sesamoidean and sacroiliac ligaments. Based on my clinical and ultrasonographic findings this procedure has proved to be a dramatic advance over conventional therapies and normal healing of these structures.

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CHAPTER • 75

Diseases of the Digital Synovial Sheath, Palmar Annular Ligament, and Digital Annular Ligaments

Michael C. Schramme and Roger K.W. Smith

Synovial effusion of the digital synovial sheath is common in all types of working horses. Frequently, effusion is idiopathic in origin and affects the digital sheath of both hindlimbs without causing lameness. Occasionally synovial effusion is seen in a single limb along with lameness. Before the advent of ultrasonography and tenoscopy, injury to the soft tissue structures of the digital synovial sheath often remained unrecognized. The cause of chronic synovial effusion and lameness remained elusive, and a diagnosis of idiopathic tenosynovitis was made readily. Because modern diagnostic techniques have become commonplace in equine lameness practice, specific injuries of the structures of the digital sheath have been identified.

ANATOMICAL CONSIDERATIONS

The detailed anatomy of the digital flexor tendon sheath (DFTS) and its contents has been well described[1] and is similar in forelimbs and hindlimbs. Reference is made to palmar and metacarpal region throughout, but the terms *plantar region* and *metatarsal region* strictly apply to the hindlimb. The DFTS is a thin-walled structure that encompasses the superficial digital flexor tendon (SDFT) and the deep digital flexor tendon (DDFT) from the level of the distal third of the metacarpal region to the T ligament, just proximal to the navicular bursa and the palmar pouch of the distal interphalangeal joint. The DFTS is mesenchymal in cell origin

enlargement existed, concurrent forelimb or hindlimb fasciotomy was performed. Using a medial approach the fascia superficial to the proximal SL was transected to allow direct open injection of bone marrow. In most horses the original 2-cm incision was extended in both proximal and distal directions using a straight Mayo scissors.

RESULTS OF INTRALESIONAL BONE MARROW INJECTIONS

Between October 1995 and December 1998, 100 horses were treated for suspensory desmitis using bone marrow injection. Fifteen horses had concurrent fasciotomy. Horses satisfied one of the following criteria: clinical and ultrasonographic evidence of a severe, acute suspensory desmitis or moderate to severe lameness from chronic suspensory desmitis. Ultrasonographic changes included circumscribed peripheral or central hypoechoic lesions, significant increase in cross-sectional area, loss of ligament margins, and associated third metatarsal or metacarpal bone avulsion fractures, or combinations of these. Some horses were included that lacked substantial evidence of suspensory desmitis, but they had lameness abolished by diagnostic analgesia in the proximal palmar metatarsal or metacarpal regions and a substantial increase in radiopharmaceutical uptake in scintigraphic images.

Nine breeds of horses were represented, including in descending order, Warmbloods, Thoroughbreds, Quarter Horses, Andalusians, Peruvian pasos, Arabians, Standardbreds, Saddlebreds, and Morgans. Horses ranged from 2 to 24 years of age. Eighty-one horses had forelimb suspensory desmitis, and 19 horses had hindlimb suspensory desmitis. The most common sporting activities were dressage, jumping, and racing. Follow-up clinical and ultrasonographic examinations were performed at 60, 90, 120, and 180 days. Two horses had second injections.

After 6 months, 84 horses were sound and had returned to full work. Eight horses improved and went back to work but had mild but manageable lameness. Five horses remained too lame to return to normal work. Three horses died from unrelated causes and could not be followed past 6 months.

In one horse a hematoma developed at the injection site, likely from inadvertent puncture of a nearby artery. In two horses mild lameness was seen for 2 days after surgery, but in all others lameness did not increase at any evaluation period.

DISCUSSION

Although this study lacked controls, results were impressive. Theoretical benefits of bone marrow injection in returning damaged SL to near its previous biochemical, morphological, and biomechanical properties could be expected. Bone marrow injection, compared with natural healing, may improve blood supply and establish cell numbers to produce high-quality collagen and matrix. The clinical results of these 100 horses are promising, and many have competed in upper levels of sporting events. Bone marrow injection can be used for horses with tendonitis of the superficial or deep digital flexor tendons, desmitis of the accessory ligament of the deep digital flexor tendon, and desmitis of the straight distal sesamoidean and sacroiliac ligaments. Based on my clinical and ultrasonographic findings this procedure has proved to be a dramatic advance over conventional therapies and normal healing of these structures.

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CHAPTER • 75

Diseases of the Digital Synovial Sheath, Palmar Annular Ligament, and Digital Annular Ligaments

Michael C. Schramme and Roger K.W. Smith

Synovial effusion of the digital synovial sheath is common in all types of working horses. Frequently, effusion is idiopathic in origin and affects the digital sheath of both hindlimbs without causing lameness. Occasionally synovial effusion is seen in a single limb along with lameness. Before the advent of ultrasonography and tenoscopy, injury to the soft tissue structures of the digital synovial sheath often remained unrecognized. The cause of chronic synovial effusion and lameness remained elusive, and a diagnosis of idiopathic tenosynovitis was made readily. Because modern diagnostic techniques have become commonplace in equine lameness practice, specific injuries of the structures of the digital sheath have been identified.

ANATOMICAL CONSIDERATIONS

The detailed anatomy of the digital flexor tendon sheath (DFTS) and its contents has been well described[1] and is similar in forelimbs and hindlimbs. Reference is made to palmar and metacarpal region throughout, but the terms *plantar region* and *metatarsal region* strictly apply to the hindlimb. The DFTS is a thin-walled structure that encompasses the superficial digital flexor tendon (SDFT) and the deep digital flexor tendon (DDFT) from the level of the distal third of the metacarpal region to the T ligament, just proximal to the navicular bursa and the palmar pouch of the distal interphalangeal joint. The DFTS is mesenchymal in cell origin

and is composed of two layers, an outer fibrous layer and an inner synovial layer. The fibrous layer provides strength and vascularity to the sheath. The synovial layer provides a smooth, frictionless surface and produces constituents of the synovial fluid. The palmar wall of the sheath incorporates three annular ligaments: the palmar annular ligament (PAL) and the proximal and the distal digital annular ligaments. These annular ligaments bind the flexor tendons to the palmar aspect of the digit, are effectively thickenings of the fibrous layer of the sheath wall with a transverse fiber orientation, and measure 2 mm or less in thickness.

The PAL or annular ligament of the fetlock joint inserts on the palmar border of the proximal sesamoid bones (PSBs), is continuous with the palmar (intersesamoidean) ligament of the fetlock joint, and thus converts the proximal scutum into an inelastic canal. The strong, transversely arranged fibers of the PAL bind down the flexor tendons into the proximal scutum. Distal to the PAL and immediately under the skin, the deep fascia forms a second, quadrilateral ligament, the proximal digital annular ligament. This ligament is a fibrous sheet that covers and adheres to the palmar surface of the SDFT and attaches laterally and medially by two bands to the proximal phalanx: one to the proximopalmar tuberosity and one that joins the insertion of the distal branch of the SDFT to the distal part of the proximal phalanx. This arrangement firmly binds the SDFT and DDFT, enveloped by the DFTS, in the palmar pastern region. The distal digital annular ligament adheres to the palmar surface of the distal part of the DDFT and binds down the terminal part of this tendon. The ligament is a crescent-shaped fibrous sheet attached by a strong band on either side of the middle of the proximal phalanx, covering the distal branches of the SDFT.

The dorsal wall of the DFTS is formed by the proximal scutum, middle scutum, and distal sesamoidean ligaments. The proximal scutum and the middle scutum are strong fibrocartilaginous pads that allow sliding of the flexor tendons along the palmar aspect of the fetlock and pastern regions, respectively. The proximal scutum is composed of the two PSBs and the thick intersesamoidean ligament. The latter is a thick sagittal structure made of transversely aligned collagen fibers. The intersesamoidean ligament covers and is attached strongly to the whole palmar and axial aspect of the PSBs and creates a solid union between these bones. The concave palmar face of the proximal scutum allows sliding of the flexor tendons in the palmar fetlock region. The proximal scutum extends proximally to the apex of each PSB between the two distal branches of the suspensory ligament (SL). Distally the proximal scutum gives insertion to the distal sesamoidean ligaments, which represent the functional continuation of the SL and consist of the straight, oblique, cruciate, and short distal sesamoidean ligaments. The straight distal sesamoidean ligament inserts distally on the middle scutum, together with the distal branches of the SDFT and the palmar ligaments of the proximal interphalangeal joint. The middle scutum is a thick, fibrocartilaginous structure attached to the proximopalmar aspect of the middle phalanx. The middle scutum contacts the palmar aspect of the distal condyles of the proximal phalanx dorsally and the DDFT palmarly.

Within the DFTS, the SDFT and DDFT are intimately related. A fibrous ring (the manica flexoria) emanates from the lateral and medial borders of the SDFT and encircles the DDFT completely, from the proximal limit of the DFTS to the proximal aspect of the PSBs. The synovial lining of the DFTS adheres to the palmar surface of the SDFT in the sagittal midline proximal to the PAL, along the dorsal surface of the PAL and along the dorsal surface of the proximal digital annular ligament. The synovial lining of the DFTS also adheres to the palmar surface of the DDFT between the proximal digital annular ligament and the distal digital annular ligament, and along the dorsal surface of the distal digital annular ligament. The sagittal adhesion-like mesotenon between the SDFT and PAL is referred to as the vinculum of the SDFT. The DDFT also has a mesotenon that attaches to its palmar surface at the level of the proximal interphalangeal joint (see Fig. 71-1, *B*). These mesotenon attachments contain vascular branches that contribute to the arterial supply of the intra-synovial part of the tendon.

The DFTS facilitates displacement of the flexor tendons during flexion and extension of the fetlock and interphalangeal joints. During metacarpophalangeal (metatarsophalangeal) joint movements, the two flexor tendons displace together, but during interphalangeal joint movements, displacement of the DDFT is greater than that of the SDFT.

DIAGNOSTIC TECHNIQUES

Diagnostic techniques that localize disease to the DFTS include synoviocentesis and synovial fluid analysis and intrathecal or perineural injection of local anesthetic solutions. Synoviocentesis of the DFTS can be performed in one of the several recesses of the sheath. Access to the proximal pouch is possible when the sheath is distended with synovial fluid but difficult when it is not. Synoviocentesis can be achieved by introducing a 2.5-cm needle along the dorsal aspect of the DDFT, between the DDFT and the lateral branch of the SL, a few centimeters proximal to the lateral PSB. Easier access can be gained via the distal palmar pouch of the sheath, which extends between the two distal branches of the SDFT and between the two digital annular ligaments, along the palmar surface of the DDFT. One should remember that this pouch is divided sagittally by the mesotenon of the DDFT in its distal part. The needle therefore should be aimed to access this pouch between the lateral or medial border of the DDFT and the ipsilateral distal branch of the SDFT to minimize the risk of inadvertent needle penetration of the DDFT. The DFTS also can be accessed through its proximal or distal collateral recesses. The proximal collateral recess is situated in the triangular space palmaromedially or palmarolaterally, between the base of a PSB, the proximal insertion of the proximal digital annular ligament, and the dorsal border of the DDFT. The space can be entered 1 cm distal to the base of a PSB and 1 cm palmar or plantar to the neurovascular bundle. The distal collateral recess is located on the lateral (or medial) aspect of the pastern, between the flexor tendons and the distal sesamoidean ligament and between the proximal and distal insertions of the proximal digital annular ligament. A cadaver study has shown that synoviocentesis of the DFTS is most consistently successful when performed at the level of the proximal lateral recess on a non–weight-bearing limb.[2] Ten milliliters of local anesthetic solution is injected for adequate desensitization of the DFTS (see Chapter 10).

Characteristics of synovial fluid of the DFTS do not vary from those of the distal limb synovial joints. Normal synovial fluid is clear yellow and has a nucleated cell count of 770 cells/μl or less and a total protein concentration of 1.0 g/dl or less.[3]

IMAGING OF THE DIGITAL SYNOVIAL SHEATH

Diagnostic ultrasonography is by far the most commonly used technique for evaluating the DFTS. The DFTS is first encountered at level 3A and continues distally to level P1C and beyond (see Chapter 16).[4] The PAL can be demonstrated in normal horses as a thin (1 to 2 mm) echogenic band immediately adjacent to the palmar surface of the SDFT at level 3C. The proximal digital annular ligament and distal digital annular ligament usually cannot be recognized in the palmar

midline, unless they are abnormally thickened. The vinculum of the SDFT at the level of the PSB is easily identifiable by ultrasonography, but the distal mesotenon of the DDFT in the phalangeal region is only occasionally visible, usually when distention of the DFTS provides negative contrast (see Fig. 71-1, *B*). A normal synovial reflection or mesotenon joins the lateral and medial borders of the DDFT in the proximal recess of the DFTS, which should not be mistaken for an adhesion (see Fig. 71-1, *A*). The thickness of the DFTS can be assessed at levels 3A and 3B, where the capsule is identifiable as an echogenic band dorsal to the DDFT and the manica flexoria.

Before the widespread use of ultrasonography for examining the soft tissues of the DFTS, negative contrast radiography was described for the assessment of tenosynovitis and annular desmitis.[5] To perform this technique, a tourniquet is applied distal to the carpus in the standing or anesthetized horse, 50 to 100 ml of air is injected into the DFTS, and another 200 to 300 ml of air is injected subcutaneously. Sometimes extra air is injected between the SDFT and DDFT at mid-metacarpal level. One post-inflation lateromedial radiograph taken at half the milliampere second (mAs) value of standard skeletal exposure for this region is normally sufficient to make an accurate diagnosis. Although this technique effectively demonstrates the thickness of the PAL, its use has become superseded by the widespread availability of diagnostic ultrasound.

Survey radiography of the DFTS is performed to demonstrate evidence of intrathecal air or gas caused by a penetrating wound, the presence of metaplastic mineralization of injured soft tissue structures, or concurrent pathological conditions of the bone. Positive contrast radiography may provide the most conclusive evidence of wound communication with the synovial space. Although fistulography and filling of the intra-thecal space with sterile iodine-based contrast medium is diagnostic of communication between the wound and the DFTS, we prefer to access the DFTS by placing a needle at a site remote from the wound. This minimizes the risk of forcing bacteria or foreign material present in the deeper layers of the wound into the synovial space. Using a remote site further avoids the risk of inadvertently introducing bacteria while passing the needle through an area of cellulitis into the synovial cavity. Infiltration of 10 to 20 ml of sterile contrast medium followed by manipulation of the digit should result in flow of contrast medium from the wound and can be demonstrated radiographically (Fig. 75-1).

Tenoscopy is the ultimate imaging modality for evaluating the internal structures of the DFTS.[6] The endooscope is introduced routinely in the proximal collateral recess of the sheath, 1 cm distal to the base of PSB and 1 cm palmar or plantar to the neurovascular bundle, but access to other synovial recesses is also possible (see Chapter 24). This approach allows for a complete examination of the DFTS and its contents, except for the palmar surface of the SDFT, unless significant thickening of any of the annular ligaments or extensive subcutaneous fibrosis has occurred. The approach also facilitates therapeutic maneuvers within the DFTS, such as PAL desmotomy, adhesiotomy, synovial mass removal, and debridement of fibrillated or torn areas of the flexor tendons, manica flexoria, or intersesamoidean ligament.

The superior soft tissue contrast of magnetic resonance imaging is likely to improve diagnostic imaging of the DFTS in the near future.

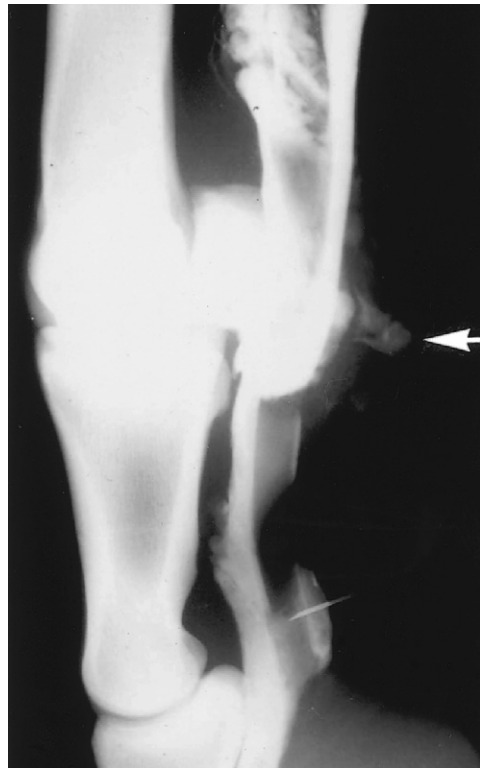

Fig. 75-1 Lateromedial radiographic view of the fetlock region. A positive-contrast tenogram of the digital synovial sheath, followed by manipulation of the digit, resulted in flow of radiopaque contrast medium from the wound (*arrow*).

DISEASES OF THE DIGITAL SYNOVIAL SHEATH

Non-Infectious Tenosynovitis

Etiopathogenesis

Acute non-infectious tenosynovitis is a traumatic synovitis/capsulitis of the sheath lining. As for joints, a traumatic synovitis/capsulitis can be caused by accumulative low-grade trauma associated with normal exercise, acute trauma associated with direct impact force (e.g., overreach), or an abnormal force outside the normal range of movement of the fetlock region (e.g., hyperextension). Synovitis/capsulitis can be complicated by damage to the internal or supporting structures of the DFTS, such as disruption of the visceral or parietal synovial layers, tearing of the vincula, tearing of the fibrous capsule with herniation, central or marginal damage to the flexor tendons, tearing of the manica flexoria, and spraining of the PAL or proximal digital annular ligament. Each of these complicating conditions is likely to result in continuous irritation of the sheath and cause chronic tenosynovitis. Chronic tenosynovitis may be associated with villonodular thickening of the sheath lining, especially in the proximal recess; adhesion formation between the visceral and parietal synovial lining; and fibrosis with reduced elasticity of the DFTS capsule. When these conditions are present, a self-perpetuating cycle occurs of improvement with rest, followed by repeat tearing of fibrous tissue (in adhesions and the fibrosed sheath capsule) with exercise. This results in increased inflammation and lameness, further fibrosis, and eventually thickening of the annular ligament and stenosis of the fetlock canal (see the following discussion of annular ligament syndrome). *Complex tenosynovitis* has been defined as tenosynovitis with thickening of the PAL, synovial distention, and adhesions or synovial masses, or both.[7] Windgalls, especially those occurring in the hindlimbs, are another form of low-grade chronic tenosynovitis. Although lameness is not usually a feature of windgalls, the synovial effusion is still likely to reflect the presence of low-grade chronic synovitis of the DFTS, caused by the continuous stress of use-induced overloading.

Diagnosis

Acute tenosynovitis is characterized by a sudden onset of mild to severe lameness, accompanied by DFTS distention that can

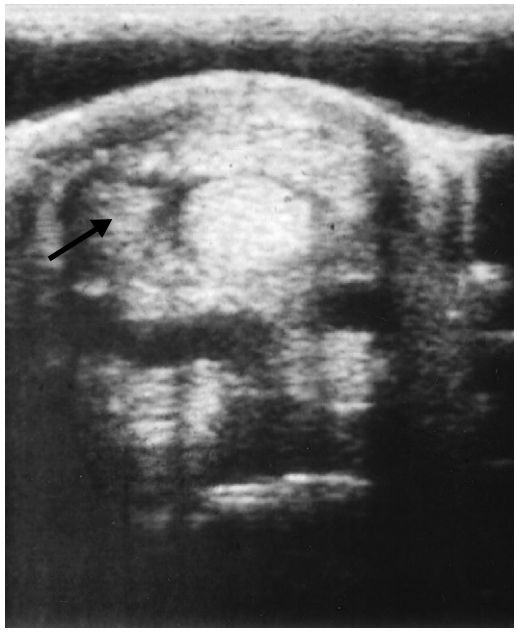

Fig. 75-2 Stage 3 tenosynovitis of the digital synovial sheath with adhesions and synovial mass formation (*arrow*).

be palpated in the proximolateral and proximomedial pouches of the sheath and in the palmarodistal recess between both branches of the SDFT. The palmar or plantar region of the fetlock has increased skin temperature, and forced flexion of the fetlock is painful and exacerbates lameness. Chronic tenosynovitis produces similar signs, except for the signs of acute inflammation, although repeat injury may cause these signs to be superimposed on an established tenosynovitis.

Ultrasonographic examination is essential for identifying adhesions and complicating injuries of the flexor tendons (see Figs. 70-7, *B* and 70-8, *A*), the intersesamoidean ligament, or annular ligaments and to document the staging of the condition.[8] DFTS effusion may accompany injury to the intrathecal part of the SDFT associated with a classic bowed tendon (see Chapter 70). However, intrathecal tendon injuries occur more commonly as focal core lesions in the DDFT (see Chapter 71) or as longitudinal tears of the SDFT, DDFT, or manica flexoria.

Tenosynovitis has been described in three ultrasonographic stages of progression.[8] Symmetrical distention of the DFTS, without evidence of synovial proliferation represents stage 1, or the effusive stage of synovitis. More pronounced, often asymmetrical distention of the proximal pouch, which feels firm on palpation and is accompanied by synovial proliferation, is stage 2. In stage 3 synovitis, extensive synovial proliferation occurs with adhesion formation and synovial masses in the sheath. Unless obviously abnormal synovial masses occur within the sheath, the presence of adhesions easily can be overestimated with ultrasonography (Fig. 75-2).

Treatment

Treatment of horses with acute tenosynovitis consists of rest with bandage immobilization, cold therapy, and systemic anti-inflammatory medication. The latter can be commenced with a single systemic dose of a short-acting corticosteroid (dexamethasone phosphate 0.06 mg/kg IV, or betamethasone phosphate 0.04 mg/kg IV), followed by a 5-day systemic course of flunixin meglumine (1.1 mg/kg sid) or phenylbutazone (4.4 mg/kg sid). After the initial 7 to 14 days, in-hand walking can be resumed for a period of 2 weeks, before returning the horse to riding. If the clinical signs have not resolved after 7 to 14 days, intrathecal injection of hyaluronan and a corticosteroid (40 to 80 mg methylprednisolone or 10 mg triamcinolone acetonide) can be considered. However, an intrathecal injection of a corticosteroid may enable a horse with a complicating flexor tendon injury to return to soundness temporarily, thereby potentially allowing it to exacerbate the underlying cause for continued irritation of the digital sheath. The clinician therefore should ensure with the aid of ultrasonographic evaluation that no injuries to the flexor tendons in the DFTS exist before intrathecal corticosteroids are administered. However, one should remember that many marginal tears of the SDFT or the DDFT within the DFTS may not be identifiable with ultrasonography, especially if they are located at the level of the ultrasonographic blind spot beneath the ergot. Measuring the cross-sectional area of each flexor tendon (see Chapter 16) and comparing the same measurements in the contralateral limb may help veterinarians recognize subtle flexor tendon injuries, without obvious changes in echogenicity.

Treatment of horses with chronic tenosynovitis usually involves the aspiration of excess synovial fluid, followed by intrathecal injection of hyaluronan and corticosteroids. In horses that are unresponsive to this treatment, or in horses with complex tenosynovitis (those characterized by thickening and fibrosis of the PAL, adhesions, synovial masses, or focal and accessible tendon lesions), tenoscopic exploration of the DFTS and intrathecal soft tissue debridement are indicated.

Prognosis

The prognosis for horses with acute, uncomplicated noninfectious tenosynovitis is favorable if treatment starts immediately. The prognosis for those with complex tenosynovitis with synovial masses and adhesions is favorable after tenoscopic debridement and PAL desmotomy. In one report, 18 of 25 horses returned to athletic soundness.[7] Tendon injuries have an unfavorable effect on the prognosis for return to athletic soundness. Only 11 of 20 horses with longitudinal tears of the flexor tendons within the DFTS[9] and 7 of 24 horses with DDFT tendonitis within the digital sheath[10] made a full recovery (see Chapters 70 and 71).

Infectious Tenosynovitis

Infectious tenosynovitis is a critical condition in the horse because of the severity of lameness, the difficulty in eliminating infection from the DFTS, and the high risk of long-term sequelae such as adhesions and fibrosis, which contribute to permanent lameness even if infection is eliminated.

Etiopathogenesis

The most common cause of DFTS infection is a penetrating wound. Occasionally, small puncture wounds that readily seal over may not be recognized as the cause of acute, severe lameness associated with DFTS infection. Penetrating injuries to the DDFT in the pastern region are frequently undiagnosed at the outset.[11] Most penetrations in this region lead to infectious tenosynovitis and rarely infectious tendonitis of the DDFT. However, occasionally a traumatic penetration may enter the DDFT through its distal mesotenon and form a localized abscess that remains separate from the intrathecal space, thereby preventing infectious tenosynovitis. Infection may also occur after intrathecal injection or surgery of the PAL. Rarely hematogenous spread of infection to the digital sheath may result from bacteremia.

Diagnosis

Heat and effusion of the DFTS and severe lameness are classic signs of DFTS infection. Typically horses are reluctant to put the heel of the affected limb to the ground. Exception to this may be seen in horses with an open wound to the DFTS that allows free drainage of infected synovial fluid. The reduction in intrathecal pressure and bacterial numbers provided by continuous flow of synovial fluid may reduce inflammation and lameness considerably. With a closed wound, however, the affected sheath is grossly distended. Generalized and painful

lower limb edema caused by cellulitis associated with the entry wound may hinder specific palpation that would aid in identifying DFTS distention. Horses with penetrating injuries to the DDFT in the pastern region are characterized by moderate to severe lameness, localized swelling in the pastern, and a pronounced pain response to focal pressure on the palmar surface of the DDFT in the pastern region.

Radiography and ultrasonography are useful to recognize complicating factors such as osteomyelitis, concurrent tendon injury, foreign bodies, and infectious tendonitis. Contrast radiography may help confirm a penetrating tract. In the absence of a penetrating tract, confirmation of the diagnosis relies on synovial fluid analysis. This should be performed as early as possible when DFTS infection is suspected. A total nucleated cell count greater than or equal to 30,000/μl, with more than 90% neutrophils, and a total protein concentration greater than or equal to 4.0 g/dl is considered pathognomonic for infection. Attempts should be made to identify bacteria by Gram stain and culture. Using broth culture bottles enhances the likelihood of obtaining a positive culture. Bacteria cultured from wounds or draining tracts are not representative of intrathecal bacterial populations. Positive cultures have been reported from 67% to 81% of infectious tendon sheaths.[12] Of the positive cultures in one study, 46% were mixed cultures. *Streptococcus* and *Klebsiella* species were the most common isolates from adult horses with infectious tenosynovitis.[13] In another study, iatrogenic synovial infection was more likely to result in a pure culture of *Staphylococcus aureus*, where as Enterobacteriaceae were most frequently cultured from synovial sepsis caused by a penetrating wound.[14]

Treatment

As for joint infections (see Chapter 66), the principles of antimicrobial therapy, synovial debridement, and drainage apply to infections of the DFTS. Treatment of infectious tenosynovitis must consist of aggressive intrasynovial and systemic broad-spectrum antimicrobial therapy with lavage of the sheath. Regional intravenous perfusion of antibiotics,[15] slow-release antibiotic-depot systems in collagen or polymethylmetacrylate,[16,17] and antibiotic infusion pumps (MILA Joint Infusion System; MILA International, Inc., Florence, KY)[18] all have been used to maximize the intra-thecal concentration of antibiotics over a protracted period of time.

Although primary closure of an open DFTS shortly after injury usually is indicated, closure may be contraindicated in some horses with extensive contamination, because closure may trap bacteria in the sheath. In such horses, cleaning the wound thoroughly and maintaining it under sterile wraps to allow for healing by second intention to occur may be preferable, or consideration may be given to delayed closure when the wound is sufficiently clean and lameness has resolved.[12]

The ideal surgical treatment for DFTS infection consists of tenoscopic debridement of fibrin, adhesions, and synovial masses along with simultaneous lavage. Transection of the PAL often relieves pain that results from excessive fluid accumulation and fibrosis and thickening of the sheath wall. This is best performed through a tenoscopic approach to minimize the risk of wound dehiscence that accompanies a transcutaneous approach. After tenoscopy, portals usually are closed or can be left open for continued drainage. Indwelling fenestrated drains may be placed at surgery for continued through-and-through lavage. Fenestrated polyvinylchloride or silicone drains are preferable to Penrose drains, because they allow for lavage and drainage. The drain is covered with a sterile dressing under a bulky pressure bandage, and lavage is continued twice daily for 3 to 5 days. Because adhesion formation and fibrosis are the most common causes for surviving horses' failure to return to the intended use, specific recommendations have been made to reduce the incidence of these crippling complications. After removal of the drain and healing of the skin portals, hyaluronan is injected into the sheath at 14-day intervals to reduce adhesion formation. These injections are best started between 7 and 14 days after injury during the time of fibrosis and adhesion formation. Early passive motion and hand-walking exercise have been advocated to prevent restriction of range of motion.[12,13]

Elevation of the heel often causes improved weight bearing and ambulation during this period. Cast immobilization of the distal limb is contraindicated during treatment for DFTS infection if the flexor tendons are intact. Although immobilization limits recurrent inflammation associated with continuous movement of the damaged tissues, it limits drainage and promotes the restrictive nature of any scar tissue and adhesions. However, immobilization effects more rapid soft tissue healing and is therefore indicated to decrease wound motion once infection has been resolved. A commercial or homemade splint is adequate for immobilization.

Prognosis

Horses with wounds of the DFTS have a good prognosis for return to soundness if the wound is diagnosed and treated before infectious tenosynovitis develops. If infection occurs, the prognosis is guarded. Although the prognosis for elimination of infection and survival is good in most studies (as high as 100% in one study[14]), another study reported a 45% failure of return to soundness and intended use for horses with infectious DFTS tenosynovitis. Even if infection can be resolved successfully, digital sheath fibrosis, adhesions, and occasionally tendon rupture or osteomyelitis of the PSB may cause permanent lameness. Most authors believe that the chance of resolution is related directly to the duration of infection.[12]

ANNULAR LIGAMENT SYNDROME

Etiopathogenesis

The PAL counteracts a tendency of the PSBs to move in a dorsal and abaxial direction during weight bearing. The distal branches and the extensor branches of the suspensory ligament effect strong traction in a dorsal direction at the respective insertion sites on the abaxial surface of the PSBs during extension of the fetlock joint. This traction is balanced by the intersesamoidean ligament and the PAL.[1] The role of disease of the PAL as a cause of lameness is a subject of debate. Thickening of the PAL has been associated with lameness in a number of different diseases of the DFTS and associated structures. All of these diseases have characteristic clinical manifestation of distention of the DFTS and thickening of the PAL in common and have therefore as a group been referred to as *annular ligament syndrome.* Other terms that have been used to describe this clinical presentation include *stenosis of the fetlock canal, annular ligament constriction, fetlock canal dysfunction,* and *stenosing palmar ligament desmitis.* The role of desmitis in this syndrome is supported by histopathological evidence of chronic inflammation and repair tissue in PAL biopsies from horses with clinical disease.[19,20] Undoubtedly, however, the cause of PAL desmitis is multifactorial. Direct external trauma to the PAL may be caused by laceration or by direct impact (e.g., overreach). Overextension of the fetlock at high speed is likely to be associated with high tensile forces in the PAL and may lead to injury and failure of the PAL under tension. Excessive tendon swelling within the DFTS, although uncommon, may result in sustained pressure on and inflammation of the PAL (see Chapter 70). Finally, chronic inflammation of the DFTS (non-infectious or infectious tenosynovitis) leads to fibrosis and thickening of the fibrous part of the DFTS capsule, which includes the PAL. In some horses the subcutaneous connective tissue in the area of the PAL becomes greatly thickened and fibrotic. Once an inflammatory response is initiated, the PAL becomes thick-

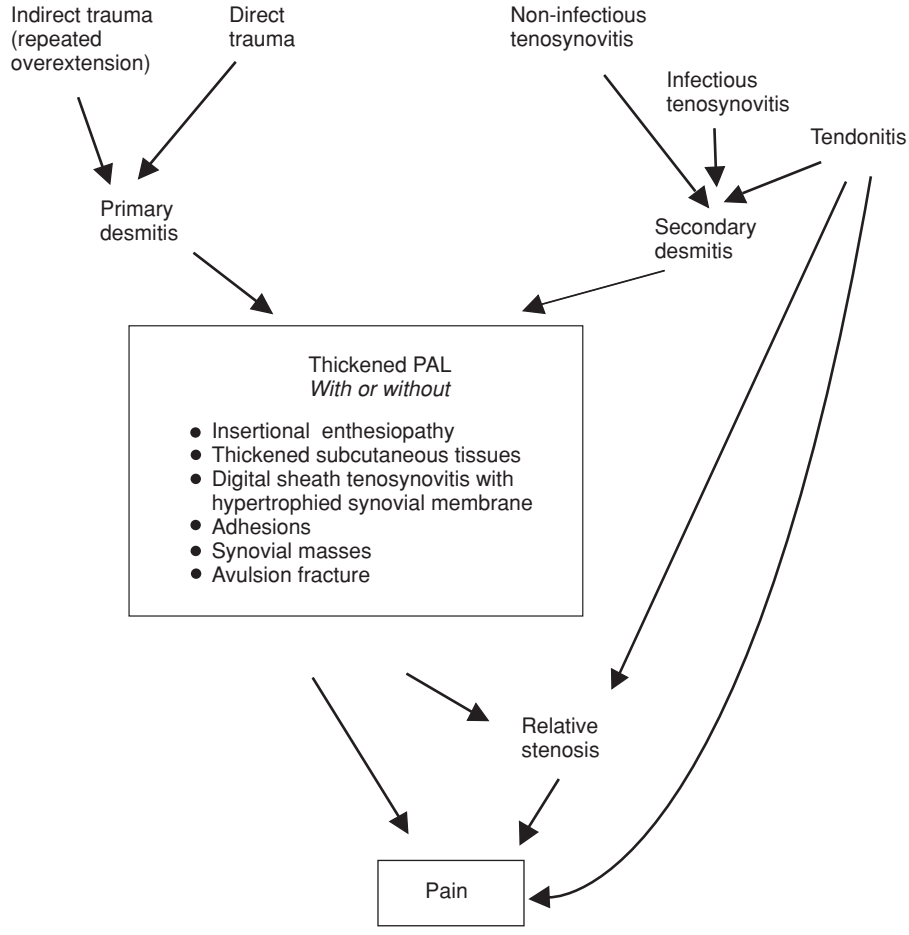

Fig. 75-3 Etiopathogenesis of palmar annular ligament (*PAL*) syndrome.

ened, whatever the cause. Thickening of the PAL reduces the space within the fetlock canal and results in a relative stenosis. The continuous pressure from the tendons associated with this relative stenosis is a source of trauma and perpetuates this sequence of events, leading to further inflammation, fibrosis, and thickening of the PAL and persistent pain (Fig. 75-3).

A positive relationship exists between incidence of desmitis and increasing age of the patient, a predisposition in some breeds (Paso Fino and Warmbloods), and location (especially hindlimbs in the mentioned breeds).[19] Rarely, small avulsion fractures may occur at the site of insertion of the PAL to the palmar border of the PSB after overextension injury or local trauma (see page 666 and Fig. 73-17).[21]

Diagnosis

Occasionally, acute lameness may be caused by acute PAL desmitis without tenosynovitis. Clinical signs include localized heat, swelling, and pain on palpation of the palmar aspect of the fetlock. The clinical signs of the more common chronic PAL syndrome are characteristic. Horses have long-term, persistent, mild to moderate lameness that improves little with rest and worsens with return to exercise. A fetlock flexion test exacerbates lameness. DFTS distention with a notch in the palmar outline of the fetlock is virtually pathognomonic for the syndrome (Fig. 75-4). This notch is caused by the inability of the DFTS to distend at the site of intimate attachment between the SDFT and the dorsal surface of the thickened, inelastic PAL. Sometimes enlargement of the PAL or the overlying subcutaneous tissues results in a localized bulge rather than a notch over the palmar aspect of the fetlock region. Extension of the affected fetlock often is decreased during weight bearing at walk or trot, and occasionally affected horses are reluctant to put the heel of the affected foot to the ground. This splinted fetlock posture is assumed by the affected horse to decrease pressure of the flexor tendons on the inflamed PAL.

Lameness often is improved, but not always completely eliminated by, intrathecal analgesia. The response to a low four- or six-point nerve block is often better. Sometimes an abaxial sesamoid nerve block improves lameness. Fibrosis of the PAL and DFTS may result in mechanical gait restriction, which cannot be abolished totally by regional analgesia.

Various imaging techniques can be used to support the clinical diagnosis of PAL desmitis. In horses with chronic lameness, entheseous new bone may be present at the PAL insertions on the palmar border of one or both PSBs. Air tenography or ultrasonography must be used to demonstrate thickening of the PAL or the overlying subcutis. The ultrasonographic appearance of the diseased PAL differs between affected horses and may be helpful in differentiating the etiopathogenic mechanisms behind each injury. The unaffected PAL can be difficult to identify because it is only 1 to 2 mm thick, and imaging the ligament in the sagittal mid-line is complicated by the vinculum between the PAL and the SDFT, which makes resolution of the ligament difficult. The PAL is identified most easily at its attachment to the PSB by tilting the transversely positioned transducer laterally or medially from the palmar midline. The ligament then can be followed toward the midline. Several measurements of the thickness of the PAL should be made on transverse and longitudinal images to obtain reliable data. Because of the difficulties in consistently

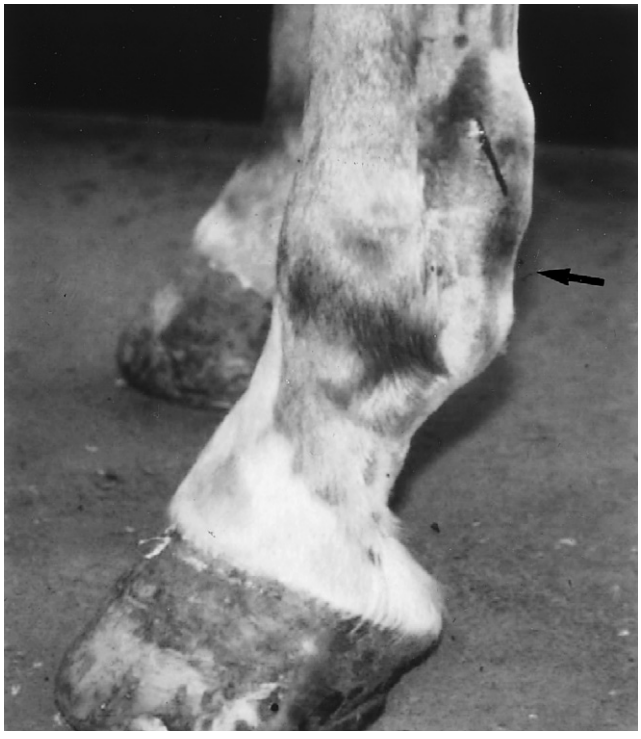

Fig. 75-4 Digital sheath distention with characteristic notch in the plantar outline of the fetlock region (*arrow*) in a horse with plantar annular ligament syndrome.

identifying the PAL, some clinicians have proposed measuring the distance between the palmar surface of the SDFT and the skin surface.[22] Any increase in thickness over 5 mm is considered to be abnormal. However, three different structures are in this region, each of which can be thickened to varying degrees: the subcutaneous tissues, the PAL, and the parietal and visceral layers of the synovial membrane of the DFTS.

Primary Palmar Annular Ligament Thickening

Primary PAL thickening is characterized by ultrasonographic evidence of thickening of the PAL, without evidence of pathological conditions of the structures within the DFTS (Fig. 75-5). Although tenosynovitis is present, the synovial lining of the sheath is not substantially thickened. With acute desmitis the ligament is enlarged and contains focal hypoechoic areas, or it is diffusely hypoechoic.

Primary Tenosynovitis with Secondary Palmar Annular Ligament Thickening

Ultrasonographic differentiation between secondary PAL thickening and primary desmitis is sometimes difficult, but the synovial lining of the DFTS usually is considerably thickened, including the lining at the level of the PAL (Fig. 75-6). Horses with chronic, complex tenosynovitis may have adhesions and synovial masses.

Subcutaneous Fibrosis with a Normal or Minimally Enlarged Palmar Annular Ligament

Subcutaneous fibrosis in the region of the PAL can produce clinical signs similar to those of PAL thickening and often can be distinguished only by careful ultrasonographic imaging (Fig. 75-7). These injuries frequently are caused by focal trauma to the PAL area and can involve portions of the PAL and the surface of the underlying SDFT.

Tendon Injuries with Secondary Palmar Annular Ligament Thickening

Injuries of the SDFT, manica flexoria, or DDFT within the fetlock canal may result in inflammatory tenosynovitis, which also can lead to PAL thickening. Although tendonitis usually

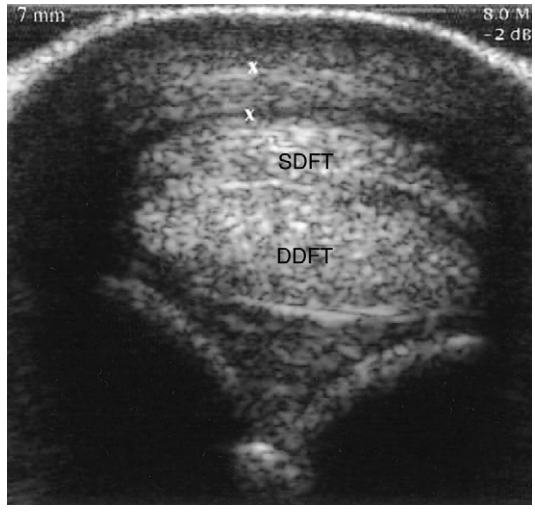

Fig. 75-5 Transverse ultrasonographic image of the distal metacarpal region at the level of the proximal sesamoid bones. There is primary enlargement of the palmar annular ligament. The palmar annular ligament (*X* to *X*) measured 7 mm in thickness. *SDFT,* Superficial digital flexor tendon; *DDFT,* deep digital flexor tendon.

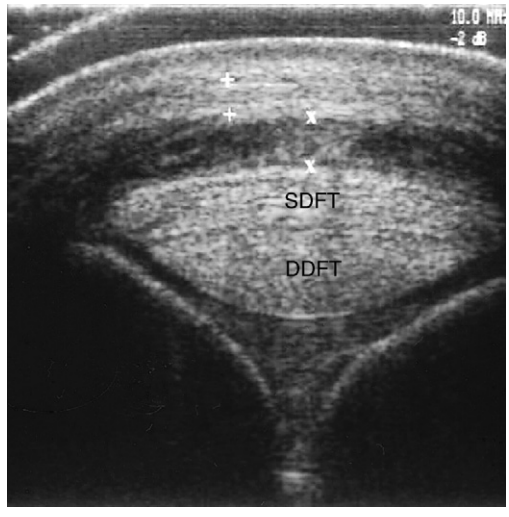

Fig. 75-6 Transverse ultrasonographic image of the distal metacarpal region at the level of the proximal sesamoid bones. There is thickening of the palmar annular ligament (*+* to *+*) and the synovial membrane of the digital sheath (*X* to *X*). *SDFT,* Superficial digital flexor tendon; *DDFT,* deep digital flexor tendon.

can be diagnosed by ultrasonography, many focal longitudinal tears of the SDFT and DDFT remain undetected, especially if they are located at the level of the ultrasonographic blind spot beneath the ergot.

Treatment and Prognosis

Treatment of horses with acute desmitis with rest and topical anti-inflammatory medication often results in rapid resolution of lameness and swelling. In many horses, however, PAL desmitis becomes a chronic condition, treatment of which requires surgical release (desmotomy) of the inflamed and thickened PAL. Desmotomy resolves pain from traction on the ligament during weight bearing and eliminates pressure from structures within the stenotic fetlock canal on the PAL. This can be per-

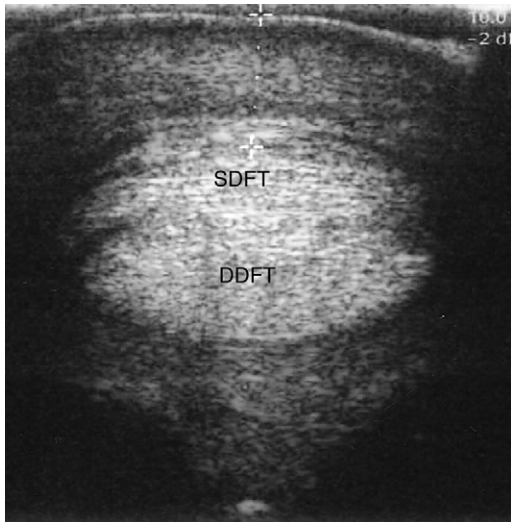

Fig. 75-7 Transverse ultrasonographic image of the distal metacarpal region. There is subcutaneous fibrosis, with minimal thickening of the palmar annular ligament, associated with clinical signs of palmar annular ligament syndrome. Although the distance from the skin to the plantar surface of the superficial digital flexor tendon amounts to 9.19 mm (+ to +), the palmar annular ligament is only 1.59 mm thick. *SDFT,* Superficial digital flexor tendon; *DDFT,* deep digital flexor tendon.

formed via a traditional surgical approach, using an open or a closed technique. A minimally invasive approach through a small skin incision in the proximal recess of the digital sheath is simple and quick, but subcutaneous transection of the PAL is performed blind (see Chapter 70). An alternative tenoscopic technique has been described.[6] Tenoscopically guided PAL desmotomy can be performed with a slotted canula used for carpal tunnel release in people (Smith & Nephew Dyonics Inc, Andover, MA; AM Surgical, Smithtown, NY), or by direct tenoscopic evaluation and use of a freehand, angle-tip knife or Metzenbaum scissors. Tenoscopy offers the advantage of enabling the surgeon to evaluate the entire DFTS and treat any concurrent pathological conditions, such as adhesions, synovial masses, and longitudinal tendon tears, simultaneously. However, passing a 5-mm arthroscope from distal to proximal through a severely constricted fetlock canal can be difficult, especially in smaller breeds. A minimally invasive approach (incisional or tenoscopic) decreases the risk of wound dehiscence and fistulation and allows for earlier, safe return to exercise. If a traditional open approach is used, particular attention should be paid to careful closure of the subcutaneous tissues in two layers, and of the skin, to minimize the risks of synovial fistulation and wound dehiscence. Contrary to previous reports, our opinion is that horses that have undergone an open surgical approach to the DFTS should not be returned to exercise before healing of the incisional wound is complete. Instead the horse should be confined to a stable and the limb enclosed in a firm, padded bandage until the time of suture removal at 2 weeks. After this, a gradually increasing exercise regimen can be initiated (2 weeks walking in hand, 4 weeks lunging) before return to ridden work. Some authors report the need for 4 to 6 months of rest and controlled exercise before soundness can be expected.[22]

Specific recommendations for treating PAL syndrome can be made according to the specific ultrasonographic characteristics. Unresponsive primary desmitis requires PAL desmotomy. In horses with primary tenosynovitis with secondary PAL desmitis, therapeutic planning should be aimed at discovering and treating the primary cause of the synovitis (e.g., infection, traumatic tendon injuries, and hypertrophic synovitis), with or without PAL desmotomy. Tenoscopy is indicated in these horses.

If the stenotic effect is caused by extensive subcutaneous fibrosis over the PAL, conservative management with rest and anti-inflammatory medication is occasionally successful in resolving lameness. However, if this approach fails to bring resolution, surgical release of the PAL and subcutaneous ring of fibrosis may resolve lameness. Lameness associated with small avulsion fractures of the palmar border of the PSB may be relieved by PAL desmotomy near its insertion site. In one study, however, three horses regained soundness without surgery after a prolonged period of 3 to 6 months of controlled exercise.[21] Finally the resolution of tendonitis of the SDFT or DDFT within the DFTS may be assisted by PAL desmotomy, although the benefit of surgical treatment has proved less obvious in these horses.[10,22] In spite of these reports, PAL desmotomy has proved useful in managing SDF tendonitis in Standardbreds (see Chapter 70).[23] The prognosis after surgical desmotomy is generally favorable for soundness in horses with desmitis of the PAL without tendonitis and has ranged from 64% to 87%.[7,22] Factors with a negative influence on outcome are the presence of tendonitis, infection, or adhesions. Although in one study, adhesions reduced the return to soundness to 44% after PAL desmotomy,[22] a recent study showed that 72% of horses with complex tenosynovitis made a full recovery after tenoscopic debridement and PAL desmotomy.[7]

DISEASES OF THE INTERSESAMOIDEAN (PALMAR) LIGAMENT OF THE FETLOCK

Etiopathogenesis

When the fetlock overextends, the proximal scutum slides distally to the metacarpal condyle and its palmar surface undergoes pressure from the flexor tendons. Moreover, in this position the distal and extensor branches of the SL induce high tension on the abaxial surface of the PSBs, thus creating high tension on the intersesamoidean ligament.[24] The effect of these forces could provide a biomechanical explanation for injuries to the intersesamoidean ligament. Post-mortem examination of 305 pairs of PSBs revealed radiological changes in 25.8% and gross abnormalities of the intersesamoidean ligament in 25.9%,[25] although pre-mortem recognition of intersesamoidean ligament pathological conditions causing lameness has been considerably less common.[24] Diseases of the intersesamoidean ligament have been classified by the ultrasonographic and radiographic appearance as rupture of the intersesamoidean ligament, avulsion fracture of the intersesamoidean ligament from the PSBs, desmitis of the intersesamoidean ligament (non-infectious and infectious), and enthesopathy of the intersesamoidean ligament.[24,26-28] Infectious desmitis and axial osteomyelitis of the PSBs usually are associated with infectious arthritis of the fetlock joint or infectious tenosynovitis of the DFTS. Which condition precedes the other is not always obvious. If infectious desmitis or osteomyelitis is the primary condition, a hematogenous route for the infection is likely.

Diagnosis

Injuries of the intersesamoidean ligament usually cause acute-onset, moderate to severe lameness. Enthesopathy with thinning of the intersesamoidean ligament and erosion of the flexor surface of the corresponding PSB may have a more insidious course. Avulsion fractures of the axial border of the PSBs often are accompanied by fetlock effusion. Lameness accompanying these conditions is improved, but not always abolished, by intra-synovial analgesia of the fetlock joint or the DFTS and is eliminated by a low four-point block. Scintigraphy may be required to find the exact site of the

pathological condition. In horses with suspected infectious desmitis or osteomyelitis, cytological examination of synovial fluid of the fetlock joint or DFTS often, but not always, indicates synovial infection.

Diagnosis of injury to the intersesamoidean ligament and associated structures mainly is based on abnormal imaging features. Evidence of osseous fragmentation or lysis along the axial border of the PSB, predominantly centered at the proximal half of the intersesamoidean space, indicates desmitis, enthesopathy with osteitis, avulsion fracture, or osteomyelitis. Evidence of osteolysis also may be observed along the flexor surface of the PSB in association with enthesopathy of the intersesamoidean ligament. Ultrasonographic abnormalities include enlargement of the distance between both PSBs with intersesamoidean ligament rupture (>6 mm), enlargement of the intersesamoidean ligament, and alteration of the echogenicity within the intersesamoidean ligament in case of desmitis, and asymmetrical reduction in the thickness of the intersesamoidean ligament and irregular bony outline to the flexor surface of a PSB in association with enthesopathy.

Treatment and Prognosis

No effective treatment exists for rupture of the intersesamoidean ligament with abaxial displacement of the PSB or for enthesopathy with thinning and degeneration of the intersesamoidean ligament within the DFTS, and the prognosis for soundness in either situation is grave. We have observed complete recovery in a general purpose riding and jumping horse with intersesamoidean desmitis and a small avulsion fracture of the axial border after a prolonged period of confinement (6 months). Some reports also have documented favorable outcome for horses with non-infectious desmitis with or without avulsion fracture, after arthroscopic removal of fracture fragments and debridement of the osteochondral defect and the associated discolored area of the intersesamoidean ligament.[27,28] Histological evaluation of the damaged intersesamoidean ligament in these horses revealed chronic inflammation and granulation tissue, with thrombosis of the microvasculature. All horses with non-infectious desmitis treated surgically in one study returned to previous level of performance, although the horses' intended uses were not mentioned.[27] Recovery time was 7 to 12 months. In the same study, similar treatment was generally unsuccessful for horses with infectious desmitis and osteomyelitis.[27]

DISEASES OF THE DIGITAL ANNULAR LIGAMENTS

Desmitis and avulsion fractures of the proximal insertion sites of the proximal digital annular ligament may occur.

Etiopathogenesis

Hyperextension of the fetlock causes differential movement between the SDFT and the proximal phalanx. The proximal digital annular ligament is adhered intimately to the palmar surface of the SDFT in this region, and the proximal digital annular ligament inserts to the proximal phalanx at two levels, thus this differential movement may result in injury of the proximal digital annular ligament or its insertions. Additionally, external trauma to the palmar aspect of the pastern may result in desmitis of the proximal digital annular ligament. Desmitis of the proximal digital annular ligament also has been described in association with injury of the SDFT or infectious tenosynovitis with thickening of the DFTS in horses with infectious desmitis and osteomyelitis.[29] It has been suggested that thickening of the proximal digital annular ligament or fibrosis of the subcutaneous tissues on the palmar aspect of the pastern may result in functional constriction of the DFTS and the flexor tendons, causing lameness, as is the case with the palmar annular ligament syndrome.[30]

Diagnosis

Characteristic clinical findings in horses with desmitis of the proximal digital annular ligament are soft tissue thickening in the palmar pastern region, prominent palmar protrusion of the DFTS distal to the proximal digital annular ligament, and abolishment of lameness by an abaxial sesamoid nerve block or intra-thecal analgesia of the DFTS. Ultrasonography provides a definitive diagnosis. The normal proximal digital annular ligament is not visible on ultrasonographic examination, but the palmarodorsal thickness of the combined skin–proximal digital annular ligament layer should not exceed 2 mm. In horses with proximal digital annular desmitis, this distance increased to 4 to 5 mm (Fig. 75-8).[30] It is important to rule out pathological conditions of the flexor tendons or distal sesamoidean ligaments as causes of soft tissue swelling in the palmar pastern region (see Chapter 83).

Avulsion fractures of the proximal insertion of the proximal digital annular ligament were seen as a cause of lameness in three horses.[31] There were few localizing signs, and diagnosis relied on response to nerve blocks and radiography. Radiographs showed an avulsion fracture fragment at the insertion site of the proximal digital annular ligament on the palmarolateral or palmaromedial border of the proximal phalanx (Fig. 75-9).

Treatment

Desmitis of the proximal digital annular ligament with constriction of the DFTS was treated successfully in two horses by desmotomy of the proximal digital annular ligament.[30] Two horses with avulsion fractures of the proximal digital annular ligament were treated with 2 months of rest alone and one with 6 weeks of immobilization, followed by 6 weeks of rest. All three horses returned to the previous level of performance.[31]

SYNOVIAL GANGLION OR HERNIA OF THE DIGITAL SYNOVIAL SHEATH

A synovial hernia or ganglion is a thin-walled cyst originating from a tendon sheath or joint capsule.[32] Hernias of the DFTS result from a traumatic defect in the fibrous layer of the

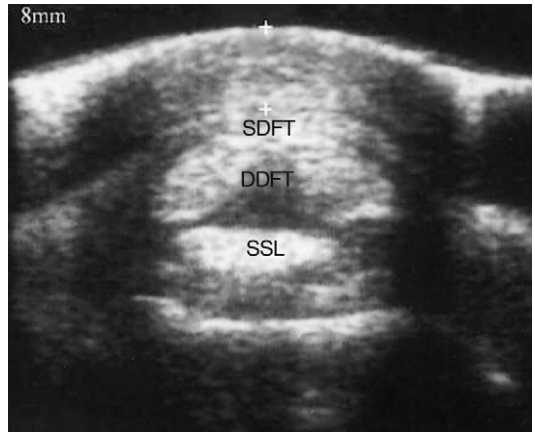

Fig. 75-8 Transverse ultrasonographic image of the proximal pastern region of a hindlimb. The distance from the skin surface to the plantar surface of the SDFT (*+* to *+*) is enlarged to 8 mm in this horse with chronic desmitis of the proximal digital annular ligament. *SDFT,* Superficial digital flexor tendon; *DDFT,* deep digital flexor tendon; *SSL*, straight sesamoidean ligament.

tendon sheath wall, allowing the synovial lining to herniate through it. Subsequent irritation to the hernial sac causes secretion of synovium and filling of the sac. The opening in some hernial sacs or ganglions can act as a one-way valve that prevents movement of synovial fluid back to the digital sheath. Injection of a radiopaque dye into the DFTS has resulted in flow of the dye from the parent synovial cavity into the ganglion (Fig. 75-10), but not vice versa. We have encountered such a ganglion at the level of the proximolateral recess of the DFTS in a hindlimb in two Warmbloods, associated with mild lameness, which was increased by lower limb flexion. Intrathecal analgesia of the DFTS abolished the lameness. Surgical exploration revealed a cystic structure, connected to a small slit-like opening in the DFTS wall. Resection and closure of the DFTS wall defect resulted in complete resolution of lameness.

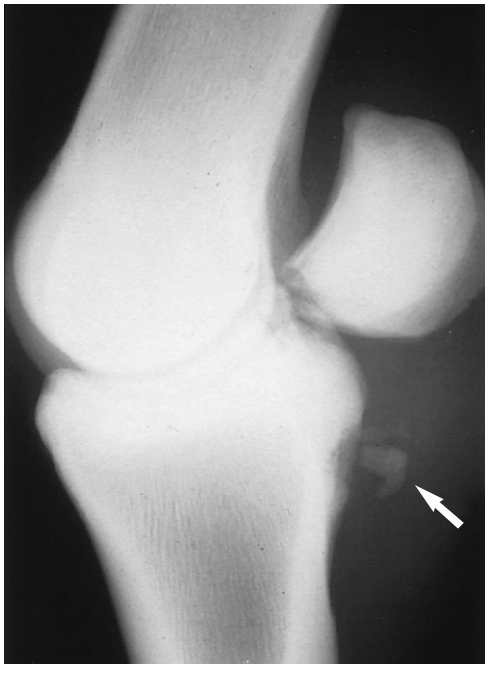

Fig. 75-9 Lateral radiographic view of a metatarsophalangeal joint. An avulsion fracture fragment (*arrow*) involves the proximal insertion of the proximal digital annular ligament to the plantarolateral border of the proximal phalanx.

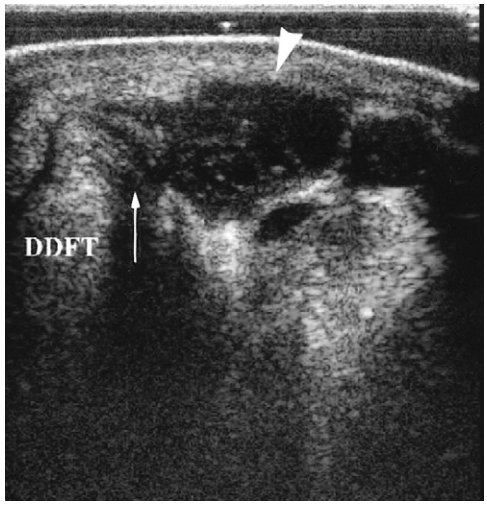

Fig. 75-10 Transverse ultrasonographic image over the lateral aspect of the distal metatarsal region of a horse with a synovial ganglion of the digital flexor tendon sheath. There is subcutaneous fluid collection (*arrowhead*) outside the boundary of, but in communication with, the digital flexor tendon sheath (*arrow*). *DDFT,* Deep digital flexor tendon.

TENDONITIS OF THE SUPERFICIAL AND DEEP DIGITAL FLEXOR TENDONS WITHIN THE DIGITAL SHEATH

Tendonitis of the superficial and deep digital flexor tendons within the digital sheath are discussed in Chapters 70 and 71.

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CHAPTER • 76

The Carpal Canal and Carpal Synovial Sheath

Sue J. Dyson

ANATOMY

The carpal canal encloses the carpal synovial sheath, which contains the superficial (SDFT) and deep (DDFT) digital flexor tendons. The dorsal wall of the carpal canal is formed by the common palmar ligament of the carpus, which is a thickened part of the fibrous joint capsule that extends distally as the accessory ligament of the DDFT (ALDDFT). Proximally the accessory ligament of the SDFT (ALSDFT) forms the medial wall of the canal. Laterally the carpal canal is formed by the accessory carpal bone and the accessorioquartale and accessoriometacarpeum ligaments extending distally. The caudal antebrachial fascia, flexor retinaculum, and palmar metacarpal fascia form the palmar aspect of the canal.

The carpal synovial sheath extends from 7 to 10 cm proximal to the antebrachiocarpal joint to the mid-metacarpal region. The proximal recess is wide and extends between the ulnaris lateralis and lateral digital extensor muscles laterally, but it is firmly supported on the medial aspect by the antebrachial fascia. The distal recess extends between the DDFT and its accessory ligament. If the carpal sheath is distended, swelling can be seen on the lateral aspect of the distal antebrachium and between the DDFT and its accessory ligament, medially or laterally in the metacarpal region.

The ALSDFT arises from the caudomedial aspect of the radius about 10 cm proximal to the antebrachiocarpal joint. The ALSDFT is a fibrous fan-shaped band that merges with the SDFT at the level of the antebrachiocarpal joint and prevents overload of the SDFT muscle during overextension of the metacarpophalangeal joint. After desmotomy of the ALSDFT, load on the SDFT is increased.[1] At the level of the distal radius is an extension from the lateral aspect of the sheath wall between the SDFT and DDFT. At the level of the accessory carpal bone is a mesotendon extending from the lateral aspect of the DDFT to the sheath wall. In clinically normal horses the amount of fluid within the carpal sheath varies, but it is usually the same bilaterally in each horse.

Fluid within the sheath may be seen readily by ultrasonography between the DDFT and its accessory ligament in normal horses, with no palpable distention of the sheath wall.[2] Within the proximal part of the carpal sheath the SDFT and DDFT contain muscular tissue and therefore on ultrasonographic examination have hypoechoic regions within them.[3] However, the ALSDFT is uniform in its echogenicity.[4] The position of the accessory carpal bone prohibits ultrasonographic examination from the caudal aspect of the carpus. The carpal sheath and its contents are evaluated most easily from the caudomedial aspect of the distal antebrachium and carpus and the palmar aspect of the proximal metacarpal region. The

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CHAPTER • 76

The Carpal Canal and Carpal Synovial Sheath

Sue J. Dyson

ANATOMY

The carpal canal encloses the carpal synovial sheath, which contains the superficial (SDFT) and deep (DDFT) digital flexor tendons. The dorsal wall of the carpal canal is formed by the common palmar ligament of the carpus, which is a thickened part of the fibrous joint capsule that extends distally as the accessory ligament of the DDFT (ALDDFT). Proximally the accessory ligament of the SDFT (ALSDFT) forms the medial wall of the canal. Laterally the carpal canal is formed by the accessory carpal bone and the accessorioquartale and accessoriometacarpeum ligaments extending distally. The caudal antebrachial fascia, flexor retinaculum, and palmar metacarpal fascia form the palmar aspect of the canal.

The carpal synovial sheath extends from 7 to 10 cm proximal to the antebrachiocarpal joint to the mid-metacarpal region. The proximal recess is wide and extends between the ulnaris lateralis and lateral digital extensor muscles laterally, but it is firmly supported on the medial aspect by the antebrachial fascia. The distal recess extends between the DDFT and its accessory ligament. If the carpal sheath is distended, swelling can be seen on the lateral aspect of the distal antebrachium and between the DDFT and its accessory ligament, medially or laterally in the metacarpal region.

The ALSDFT arises from the caudomedial aspect of the radius about 10 cm proximal to the antebrachiocarpal joint. The ALSDFT is a fibrous fan-shaped band that merges with the SDFT at the level of the antebrachiocarpal joint and prevents overload of the SDFT muscle during overextension of the metacarpophalangeal joint. After desmotomy of the ALSDFT, load on the SDFT is increased.[1] At the level of the distal radius is an extension from the lateral aspect of the sheath wall between the SDFT and DDFT. At the level of the accessory carpal bone is a mesotendon extending from the lateral aspect of the DDFT to the sheath wall. In clinically normal horses the amount of fluid within the carpal sheath varies, but it is usually the same bilaterally in each horse.

Fluid within the sheath may be seen readily by ultrasonography between the DDFT and its accessory ligament in normal horses, with no palpable distention of the sheath wall.[2] Within the proximal part of the carpal sheath the SDFT and DDFT contain muscular tissue and therefore on ultrasonographic examination have hypoechoic regions within them.[3] However, the ALSDFT is uniform in its echogenicity.[4] The position of the accessory carpal bone prohibits ultrasonographic examination from the caudal aspect of the carpus. The carpal sheath and its contents are evaluated most easily from the caudomedial aspect of the distal antebrachium and carpus and the palmar aspect of the proximal metacarpal region. The

heterogeneous echogenicity of the flexor tendons proximally can make definitive diagnosis of a tendon lesion difficult. Endoscopic evaluation may yield further information and permit surgical debridement of torn fibers (see Chapter 24).

The transverse ridge of the distal radius is at about the same level as the distal physis. Irregular roughening of this ridge may be seen radiographically in normal horses and should not be confused with entheseous new bone associated with tearing of the attachment of the ALSDFT further proximally.

CLINICAL SIGNS

Lameness associated with the carpal synovial sheath almost invariably is associated with some distention of the sheath. The horse may have restricted flexibility of the carpus, with pain on passive flexion. Rarely, increased pressure within the carpal sheath may result in compromised blood flow within the median artery and reduction in arterial pulse amplitudes in the more distal part of the limb.[5] Palpation of the structures within the proximal part of the carpal sheath is not possible, but in the metacarpal region the SDFT, DDFT, and ALDDFT should be assessed carefully. Lameness varies from mild to severe and usually is improved by intrathecal analgesia. Clinical investigation should include radiographic and ultrasonographic examination. Carpal sheath effusion also may occur secondary to cellulitis in the antebrachial or metacarpal regions (see Chapters 14 and 38).

IDIOPATHIC SYNOVITIS

Synovitis of the carpal sheath usually results in acute-onset, moderate to severe unilateral lameness associated with distention of the carpal sheath. No palpable abnormalities of the flexor tendons are apparent. Ultrasonographic examination reveals an abnormal amount of fluid within the sheath but no other structural abnormality. Horses usually respond well to box rest and controlled walking exercise for 4 to 6 weeks, combined with intrathecal administration of corticosteroids and hyaluronan.

INTRATHECAL HEMORRHAGE

Hemorrhage within the carpal sheath may be idiopathic, result from trauma (e.g., a fall), or occur secondary to a fracture of the accessory carpal bone (see pages 391 and 686). The horse may be very lame. Diagnosis is confirmed by synoviocentesis. The fluid within the sheath may appear more echogenic than synovial fluid. Careful ultrasonographic examination of the flexor tendons and the retinaculum should be performed to identify any primary lesion. In the absence of a primary tendon lesion and fracture, some relief of pain may be gained by draining blood from the sheath. This should be followed by administration of hyaluronan to reduce the risks of subsequent adhesion formation.

TRAUMA RESULTING IN CHRONIC ENLARGEMENT OF THE CARPAL SHEATH

A horse may have acute distention of the carpal sheath and moderate to severe lameness after a fall. In the acute stage ultrasonographic examination may reveal only an abnormal amount of fluid within the sheath, but over the next several weeks thickening of the sheath wall and the palmar retinaculum may become apparent, with echogenic fibrous material within the sheath (Fig. 76-1). The margin of the SDFT or DDFT may be poorly demarcated, and either structure may be enlarged. Some horses respond satisfactorily to rest and controlled exercise combined with repeated medication of the sheath with hyaluronan. Early aggressive treatment seems to yield the best results. Passive flexion of the carpus also may be beneficial. If lameness persists for more than 6 weeks, endoscopic evaluation of the sheath and its contents should be considered (see Chapter 24).

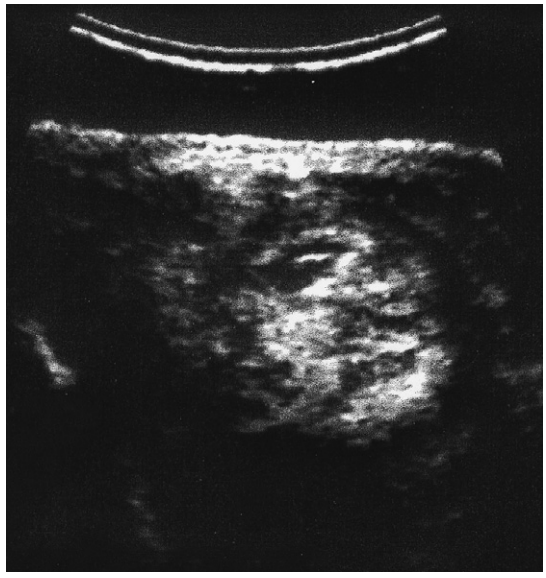

Fig. 76-1 Transverse ultrasonographic image of the carpal sheath of 9-year-old dressage horse with chronic left forelimb lameness of 3 months' duration. Medial is to the left. There is thickening of the sheath wall and enlargement of the superficial digital flexor tendon, but no internal structural abnormality could be defined because of the normal heterogeneous echogencity of the tendon at this level from muscle tissue. The horse failed to respond to intrathecal medication or endoscopic debridement of the proliferative synovial membrane (also see Fig. 21-9.)

DESMITIS OF THE ACCESSORY LIGAMENT OF THE SUPERFICIAL DIGITAL FLEXOR TENDON

Desmitis of the ALSDFT is an unusual injury in show jumpers, event horses, dressage horses, and Thoroughbred racehorses, but it seems to occur more commonly in European Standardbred trotters.[6] The condition is rarely recognized in North American trotters.[7] Lameness is usually sudden in onset and associated with localized swelling.

Diagnosis is based on ultrasonographic examination. Abnormalities of the ALSDFT include enlargement, abnormal fiber pattern, and reduced echogenicity. Enlargement of the ALSDFT results in increased distance between the caudal radius and the median artery. Injuries to the ALSDFT are often seen with other injuries, either in the carpal canal including superficial digital flexor tendonitis or thickening of the flexor retinaculum, or elsewhere.[8,9] These injuries include tenosynovitis of the flexor carpi radialis tendon sheath and suspensory desmitis.

Treatment consists of box rest and controlled exercise for up to 6 months, combined with intrathecal administration of hyaluronan and corticosteroids. The prognosis for horses with simple injuries is fair. Six of eight horses with uncomplicated desmitis of the ALSDFT returned to the former athletic function.[8] However, horses with concurrent injuries were more likely to suffer recurrent lameness.

SUPERFICIAL DIGITAL FLEXOR TENDONITIS

Older horses (>15 years of age) may show acute superficial digital flexor tendonitis within the carpal canal (see Chapter 70).[2] In the acute stage the carpal sheath is distended, but the horse may show no palpable abnormality of the SDFT in the metacarpal region or only slight peritendonous edema proximally. Diagnosis is based on ultrasonographic identification of lesions of the SDFT. In some horses lesions may not be detectable acutely but may become apparent over the next several weeks. Some older horses initially show superficial digital flexor tendonitis, which progresses proximally to involve the SDFT within the carpal sheath. These horses have a poor prognosis regardless of the method of management.

Younger horses may develop superficial digital flexor tendonitis in the proximal metacarpal region. Such lesions may extend proximally into the carpal region, with only slight distention of the carpal sheath. A high proportion of these lesions result in recurrent lameness if treated conservatively. In horses with chronic tendonitis, surgical treatment by desmotomy of the ALSDFT combined with carpal fasciotomy should be considered (see Chapter 70). Moderate results have been achieved in a small number of racehorses and ponies.[7]

DEEP DIGITAL FLEXOR TENDONITIS

Primary deep digital flexor tendonitis within the carpal sheath is unusual, but marginal tears have been identified endoscopically in a small number of horses with persistent lameness associated with carpal sheath distention (Fig. 76-2). Surgical debridement has resulted in clinical improvement. Secondary tears on the cranial aspect of the DDFT may occur with a solitary osteochondroma (see the following section). Carpal sheath distention with incomplete rupture of the cranial head of the DDFT has been reported.[10] Hemorrhagic fluid was aspirated from the sheath, and disruption of the deep digital flexor muscle was identified by ultrasonography.

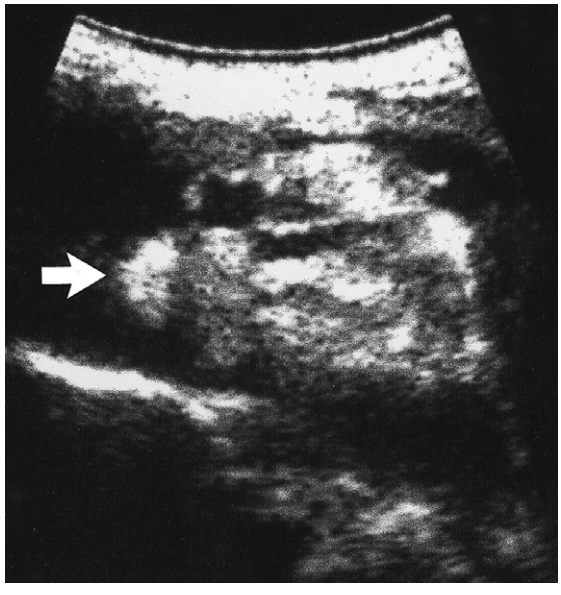

Fig. 76-2 Transverse ultrasonographic image of the carpal sheath of 8-year-old dressage horse with moderate lameness associated with distention of the carpal sheath of 4 months' duration. Medial is to the left. The horse had not responded to intrathecal medication and rest. An echogenic mass appears on the medial aspect of the deep digital flexor tendon. Endoscopic evaluation revealed a large granulomatous structure, which was surgically debrided. Histological examination revealed evidence of a marginal tear of the deep digital flexor tendon, with surrounding fibrous material. The lameness resolved satisfactorily.

SOLITARY OSTEOCHONDROMA

An osteochondroma is an exostosis continuous with the cortex of the bone and is covered by cartilage. The distal caudal radius is a common site, immediately proximal to the distal radial physis. An osteochondroma is readily identifiable by radiography and ultrasonography. Almost invariably an impingement lesion is found on the cranial aspect of the DDFT. Treatment is by surgical removal of the osteochondroma and debridement of any torn fibers of the DDFT. Treatment usually produces an excellent functional and cosmetic result.[2]

FRACTURE OF THE ACCESSORY CARPAL BONE

Fractures of the accessory carpal bone are often the result of a fall and result in acute-onset, moderate to severe lameness (see Chapter 39). The majority have a vertical configuration.[11,12] Reports are conflicting about the incidence of carpal canal syndrome secondary to a fibrous or non-union of the accessory carpal bone.[11-13] Seven of 11 horses returned to full athletic function without complications after a vertical (frontal) fracture of the accessory carpal bone, despite healing by fibrous union in the six horses re-examined radiographically.[12] The four remaining horses were sound: two were retired for breeding, and two were used for pleasure riding. I have had similar experience. However, if lameness associated with thickening of the carpal sheath wall persists, resection of a piece of the carpal retinaculum may be successful.[13] Radiographs should be inspected carefully, because occasionally chip fractures of the articular margin of the accessory carpal bone occur alone or concurrently with a more typical vertical fracture. Horses with such fractures warrant a more guarded prognosis.

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12. Barr A: Fractures of the accessory carpal bone in the horse, *Vet Rec* 126:432, 1990.
13. Mackay-Smith MP, Cushing L, Leslie A: "Carpal canal" syndrome in horses, *J Am Vet Med Assoc* 160:993, 1972.

CHAPTER • 77

Tarsal Sheath

Eddy Cauvin

The tarsal sheath corresponds to the synovial sheath of the lateral digital flexor tendon at the level of the hock. Tenosynovitis of this sheath is a well recognized condition[1-6] and can be caused by a wide range of lesions. Nonpainful, chronic distention in the absence of obvious pathological lesions, often called *idiopathic thoroughpin*, is common and should be distinguished from other debilitating causes of tenosynovitis, many of which can cause persistent, severe lameness.[7,8] Specific lesions within the sheath can be difficult to confirm clinically.[8,9]

FUNCTIONAL ANATOMY

The deep digital flexor tendon (DDFT) in horses is formed by the fusion in the proximal metatarsal region of the thin medial digital flexor tendon and the larger lateral digital flexor tendon.[10-13] The two tendons pass within separate sheaths. The lateral digital flexor muscle covers the caudal aspect of the tibia and is joined by the tibialis caudalis muscle in the distal crus. The tendon starts 2 to 4 cm proximal to the tarsocrural joint and passes medial to the tuber calcanei over a fibrocartilage-covered groove on the plantar aspect of the sustentaculum tali of the calcaneus. The lateral digital flexor tendon passes over the thick distal plantar ligament in the distal tarsus, medial to the superficial digital flexor tendon (SDFT), before being joined by the medial digital flexor tendon 1 to 3 cm distal to the tarsometatarsal joint.

The tarsal sheath is 16 to 20 cm long and starts near the musculotendonous junction of the lateral digital flexor tendon in the distal caudal crus. At this level the tarsal sheath forms a large pouch between the lateral digital flexor muscle and the common calcanean tendon. The distended pouch is largest over the lateral aspect of the crus. At the level of the tarsocrural joint the sheath extends laterally to surround the lateral digital flexor tendon. Cranially a rigid groove is formed by fibrocartilaginous thickening of the tarsocrural joint capsule. The sheath terminates as a recess dorsomedial to the DDFT in the proximal third of the metatarsal region.

The tarsal sheath is enclosed at the level of the sustentaculum tali by a thick, transversely oriented ligament, the plantar retinaculum (Fig. 77-1) and in the distal tarsus by a superficial fascia. The plantar nerves and vessels run within the retinaculum, in the plantar two thirds of its width, that is, plantar and plantaromedial to the lateral digital flexor tendon. The sheath is lined by a parietal synovial membrane with few villi, except distally. This membrane reflects plantarly to wrap around the tendon, leaving a thin but continuous membrane, or mesotenon, along the plantaromedial aspect of the lateral digital flexor tendon. This membrane carries vessels to the tendon and therefore should be preserved during surgery.

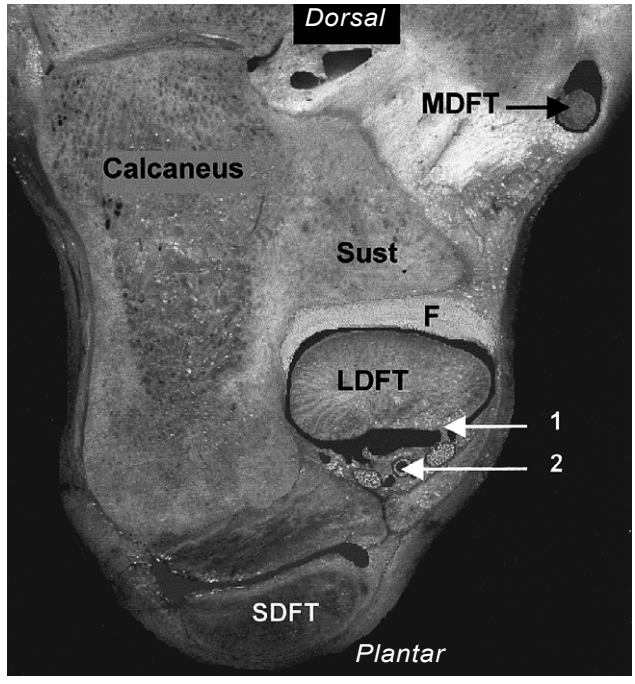

Fig. 77-1 Transverse section of the proximal tarsus, showing the lateral digital flexor tendon (*LDFT*) in the fibrocartilaginous groove (*F*) on the plantar aspect of the sustentaculum tali (*Sust*). Lateral is to the left. Vessels and nerves course within the retinaculum (*2*), lateral to the attachment of the mesotenon (*1*). *MDFT,* Medial digital flexor tendon; *SDFT,* superficial digital flexor tendon.

CAUSES OF TARSAL TENOSYNOVITIS

Distention of the tarsal sheath (Fig. 77-2) is commonly termed *thoroughpin*, or *true thoroughpin*,[14] but the condition may have distinct causes.

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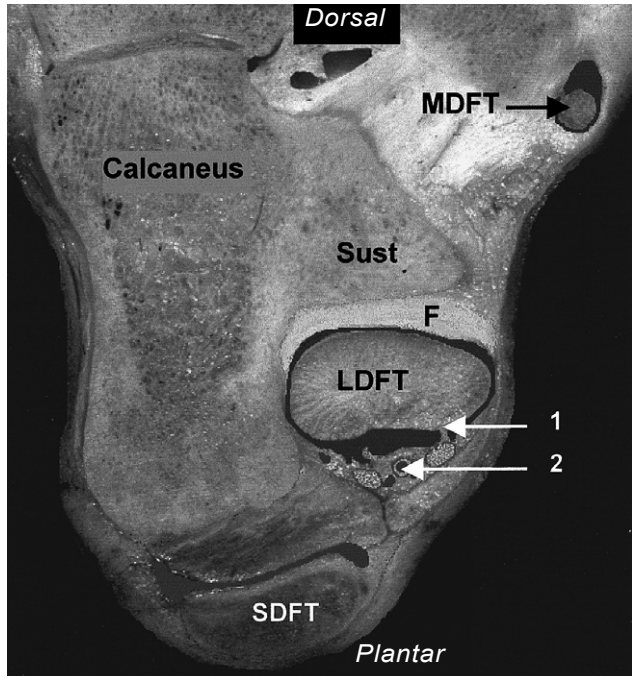

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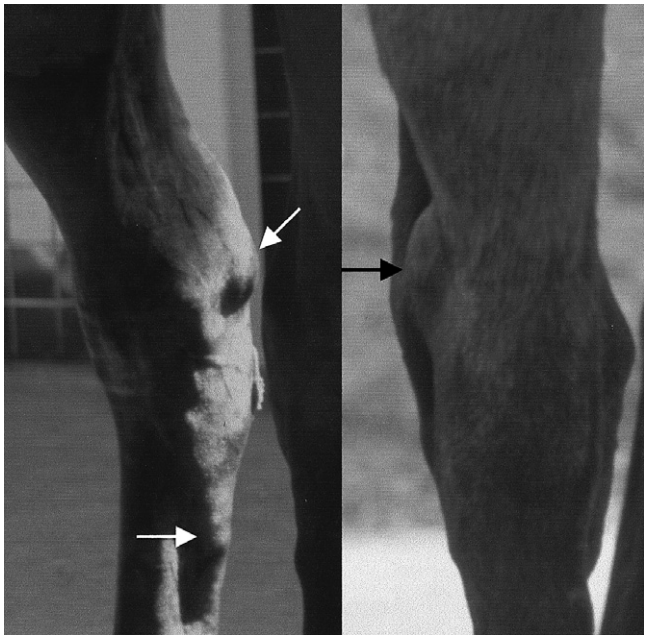

Fig. 77-2 Distention of the tarsal sheath, giving the typical thoroughpin swelling, medially in the distal caudal crus and proximal metatarsal region (*white arrows*) and laterally, caudal to the tibia (*black arrow*).

Idiopathic Thoroughpin

Slight to moderate distention of the tarsal sheath is often seen in young horses.[8,9] It also occurs in adults, particularly in Warmbloods and Western performance horses and horses with a straight hock conformation, after extended box rest or transport. Effusion also may result from acute inflammation in nearby tissues, congestion, and edema in the distal limb (sympathetic effusion). The distention usually is not associated with signs of inflammation or lameness and tends to resolve spontaneously.[8] However, in some horses the distention can become recurrent or persistent.

Traumatic Thoroughpin

Tenosynovitis may be induced by trauma, leading to acute inflammation and hemorrhage in the sheath. In my experience, direct trauma to the medial plantar aspect of the sheath is common and most often results from a kick by another horse. Tenosynovitis also may result from hitting hard objects during jumping or occasionally interference from the contralateral hindfoot. These traumatic injuries may be associated with a chip fracture or fragmentation of the medial edge of the sustentaculum tali at the insertion of the plantar retinaculum.[3,13] Transverse fracture of the calcaneus involving the sustentaculum tali also has been described.[3] In most horses no signs of direct trauma are apparent, and overstretching, or sprain, which may or may not involve the lateral digital flexor tendon, are suspected of causing acute tenosynovitis.[8,9] Intrathecal hemorrhage always causes substantial inflammation and pain and can induce the formation of fibrinous adhesions. The inflammation often leads to chronic distention, with synovial thickening, fibrosis, and fibrous adhesion formation between the lateral digital flexor tendon and parietal sheath lining, which occasionally can cause persistent pain and mechanical lameness. Acute tenosynovitis without overt lameness also has been described.[9]

Primary Lateral Digital Flexor Tendon Injuries

Sprain injuries to the lateral digital flexor tendon do occur in the tarsal sheath region, and they are characterized with ultrasonography by longitudinal fraying and irregular hypoechoic lesions at the level of the sustentaculum tali. These lesions may be caused by overstretching and compression of the tendon over the bone.

Infectious Tenosynovitis

Direct trauma to the medial aspect of the hock can result in breach and contamination of the tarsal sheath.[3,9,13,15] The medial edge of the sustentaculum tali is the most prominent relief on the medial aspect of the tarsus, and a wound at this level often disrupts the retinaculum, thus opening the sheath.[6,13] Puncture wounds are rare in my experience, but they may occur especially in the distal caudal crus. Iatrogenic contamination induced by intrathecal injection is also common and should be suspected in horses with worsening of the lameness and signs of inflammation after such injections.[9] If untreated, suppurative tenosynovitis may lead to infectious tendonitis, destruction of the fibrocartilage, and eventually osteitis or osteomyelitis of the calcaneus.[3,4,6,13] Infection also may result from extension of abscesses in adjacent tissues.

Other Causes

One horse with hemangiosarcoma involving the tarsal sheath has been described.[7] Chondrosarcomata, extending from the tarsocrural joint, and systemic lupus-like synovitis have been reported as rare causes of thoroughpin.[9]

DIAGNOSIS OF TARSAL SHEATH INJURIES

Clinical Signs

Distention of the tarsal sheath is visible in the distal crus, particularly laterally between the common calcanean tendon and the tibia, and to a lesser extent, on the medial aspect of the caudal crus (Fig. 77-2). Such distention should not be confused with swellings associated with the plantar pouch of the tarsocrural joint (see Chapter 45), situated farther distally between the tuber calcanei and distal tibia (bog spavin) or with distention of other bursae; for example, the deep calcanean bursa and calcanean bursa of the SDFT (see Chapter 80).[8,10] False thoroughpin may result from soft tissue masses such as hematomata, tarsal sheath herniation, granulation tissue,[8,9] or abscesses (see Chapter 80). Distention of the tarsal sheath also may be detected in the proximal metatarsal region on the medial aspect of the DDFT.

In horses with idiopathic tenosynovitis the swelling is soft and non-painful and unassociated with lameness.[9,16] In inflammatory conditions swelling is soft, warm, and painful in the acute stage and hard and rarely painful in chronic injuries. The degree of lameness varies.[3,7] The level of pain is not always correlated with the severity of the condition. Usually prominent pain is induced by hock flexion in acute and chronic injuries, and the degree of hock flexion may be greatly decreased. Confirming that the lameness is associated with the sheath may be necessary by using intrathecal injection of 5 to 10 ml of local anesthetic solution and by elimination of other sources of pain. Synoviocentesis may be difficult in horses with acute injuries because of edematous, hypertrophic, and congested synovial membrane.

Confirming infection is not always easy in all horses in the absence of a wound. Wounds over the sustentaculum tali always should be considered suspicious and warrant a contrast tendovaginogram or tendovaginocentesis.

Radiography

Radiographic examination is paramount in all horses with severe tarsal tenosynovitis to rule out fractures or osteitis of the sustentaculum tali and tuber calcanei (see Chapter 45).[2-4,13] Lesions are most visible on dorsoplantar and dorsomedio-

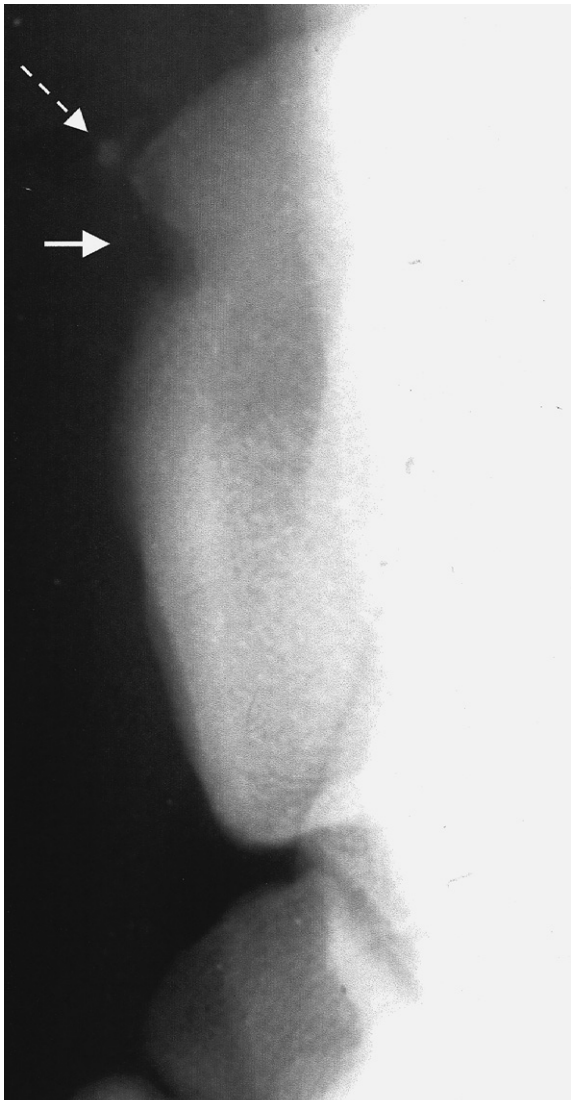

Fig. 77-3 Dorsomedial-plantarolateral oblique radiographic view of the calcaneus, showing erosion (*plain arrow*) and fragmentation (*dotted arrow*) of the edge of the sustentaculum tali.

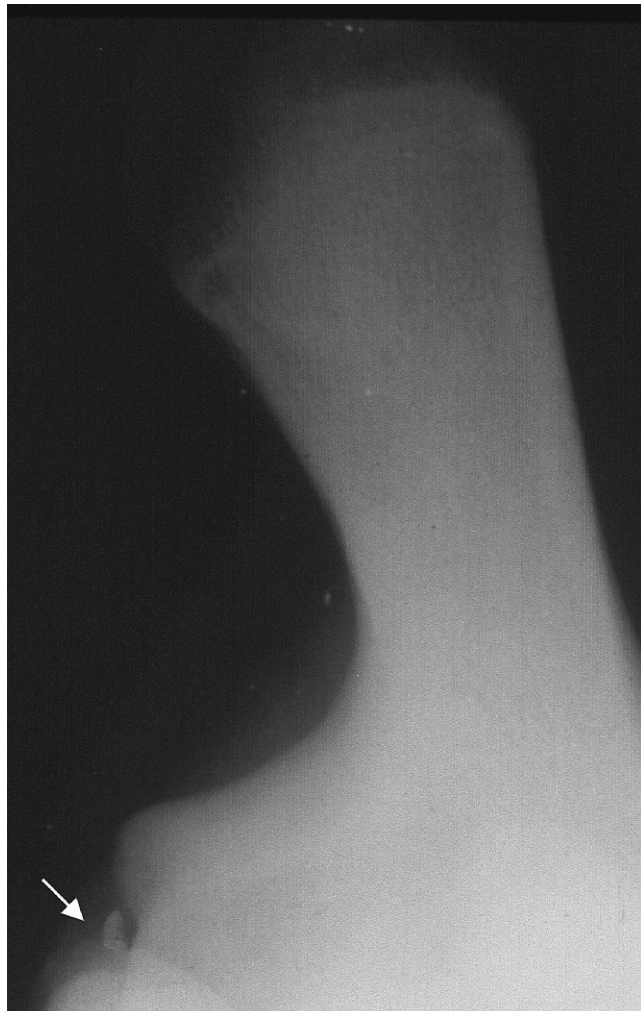

Fig. 77-4 Skyline radiographic view of the calcaneus, highlighting fragmentation of the edge of the sustentaculum tali (*arrow*), medial to the groove.

plantarolateral oblique projections to highlight the medial and plantaromedial aspects of the calcaneus (Fig. 77-3). A flexed skyline projection of the plantar aspect of the sustentaculum tali (flexed proximocaudal-distoplantar projection) is essential to demonstrate some lesions[17] (Fig. 77-4). Contrast radiography is also useful to highlight the surface of the fibrocartilages and the outline of the lateral digital flexor tendon[18] but has been replaced largely by ultrasonography.[12] However, contrast radiography remains a useful technique to confirm puncture wounds into the sheath. After collecting synovial fluid, 5 ml of an iodinated contrast medium mixed with 5 ml of Hartman's solution is injected in the proximal pouch of the sheath, and lateromedial, dorsomedial-plantarolateral oblique, and dorsoplantar radiographs are obtained to highlight a communication with the skin.

Ultrasonography

Ultrasonographic examination of the sheath is best performed with a linear 7.5-MHz or higher-frequency transducer. The lateral digital flexor tendon is best imaged from the medial aspect of the limb.[12,13,19] Accurate imaging requires high-definition equipment and experience. The chestnut can be trimmed and soaked with warm water to improve imaging. In idiopathic distention, the sheath is filled with anechoic fluid, and no evidence of synovial membrane hyperplasia is apparent. In acute tenosynovitis the sheath is distended, and significant thickening of the synovial membrane occurs. Hemorrhage is seen frequently as a hypoechogenic, whorl-like structure in the proximal pouch. Lesions may be seen in the lateral digital flexor tendon, but they are relatively rare. Longitudinal tears and superficial fraying are the most common types of lesions, but they should not be confused with hyperplasia of the visceral synovial membrane covering the tendon. Early fibrinous adhesions may be visible. The integrity of the fibrocartilage groove and retinaculum can be assessed and fragmentation of the edge of the sustentaculum tali may be detected.[8,13] In chronic injuries, significant fibrosis may be seen around the sheath, and often large adhesions are between the lateral digital flexor tendon and parietal sheath, especially in the proximal pouch (Fig. 77-5). The visceral lining is usually thick (several millimeters) and has an increased echogenicity. Horses with chronic lameness and those that have received multiple injections of corticosteroids may have large, nodular fibrocartilaginous or partially mineralized masses, termed *ossicles.*[2,9] Differentiating these from chip fractures or mineralization of the tendon or sheath lining without the use of tenoscopy may be difficult. Ultrasonography can help to differentiate true from

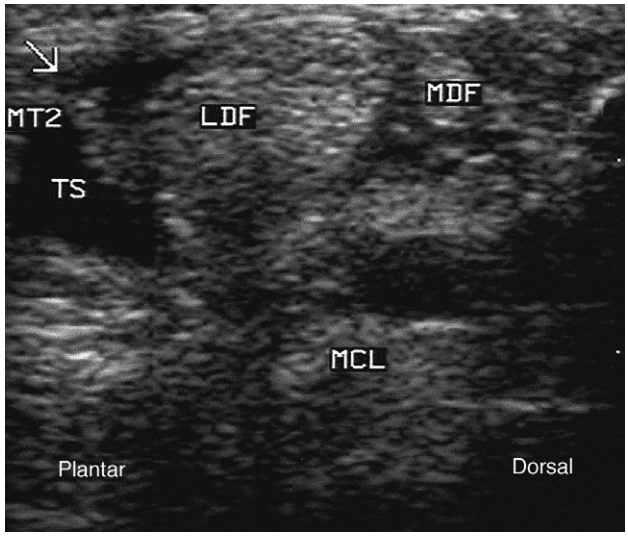

Fig. 77-5 Transverse ultrasonographic image of the tarsal sheath at the level of the tarsometatarsal joint obtained from the plantaromedial aspect. Plantar is to the left. There is hyperplasia of the synovial membrane around the lateral digital flexor tendon (*LDF*) and a well-organized adhesion between the tendon and parietal sheath wall (*arrow*). The tarsal sheath (*TS*) is distended. *MCL*, Medial collateral ligament; *MT2*, plantar border of the second metatarsal bone; *MDF*, medial digital flexor tendon.

false thoroughpin (see Chapter 80). Infection is characterized by severe inflammation of the synovial lining, and the fluid is often echogenic and heterogeneous because of the fibrin clots and debris.[6,13] This may not be obvious early in the condition.

Tenovaginocentesis

Tenovaginocentesis (collecting fluid from the tendon sheath) is useful to rule out infection and confirm inflammation.[9,15] The fluid may be normal in appearance, but it is usually contaminated by blood. A moderate increase of cellularity (less than 8000/µl) may be evident, except in acute traumatic injuries where the nucleated cell count can exceed 10,000/µl. Generally infection is associated with a thin, cloudy to purulent fluid and cell counts well in excess of 10,000/µl. Note that centesis should be carried out after ultrasonography to avoid bleeding and gas artifacts.

Tenoscopy

Tenoscopy (see Chapter 24) is rarely necessary as a purely diagnostic method, because the sheath is evaluated adequately with ultrasonography in most horses. A major exception is in infectious conditions, where it may be useful to assess potential damage to the fibrocartilages, which may not be visible with other diagnostic imaging methods.[13]

MANAGEMENT OF TARSAL TENOSYNOVITIS

Idiopathic and Traumatic Tenosynovitis

Treatment is of little value in horses with idiopathic tenosynovitis, which generally resolves spontaneously.[8,9,16] Reduced (but not interrupted) exercise, sweats, and systemic anti-inflammatory therapy can be helpful.[9] It is not advisable to place a needle in the sheath, because this may cause bleeding and subsequent inflammation.

Treatment of acute tenosynovitis is controversial. In the absence of tendon or bony lesions, rest, systemic or local non-steroidal anti-inflammatory drugs (NSAIDs), local dimethylsulfoxide creams, and cryotherapy (ice packs or cold hosing) are often effective.[9] The horse should be rested in a box for 24 to 48 hours and then walked out in hand to reduce sheath fibrosis. Hyaluronan may be useful for horses with more severe tenosynovitis or in horses in which tendon lesions are present. Intrathecal corticosteroids may be useful in horses with acute tenosynovitis, but they certainly are contraindicated if tendon lesions exist. The latter carry a poor prognosis, except for superficial fraying.[8,9] Bone fragments should be removed by means of tenoscopy, because they can cause chronic inflammation and interference with the tendon.

In horses with more chronic tenosynovitis with associated lameness, corticosteroids such as methylprednisolone acetate (40 mg), triamcinolone acetonide (10 mg), or betamethasone have been used with varying success.[8,9] Fluid retrieval to decrease the volume of the sheath, followed by rest and the application of pressure bandages may produce temporary relief of the distention,[9] but recurrence is common, probably because of chronic proliferative synovitis. Repeated injections have been associated with mineralization of the tendon and synovial lining, and rupture of the lateral digital flexor tendon has been reported,[9] and therefore are not recommended. If possible, the cause of the chronic inflammation should be ascertained. With bony fragments, ossicles, or adhesions, conservative treatment and medical therapy are often disappointing, and surgical debridement appears to be the most effective treatment.[9,13] Debridement is best achieved by tenoscopy (see Chapter 24) (Fig. 77-6). NSAIDs are administered for 1 to 2 weeks post-operatively, and the horse is restricted to box rest for 10 days, after which hand walking starts several times daily. The horse may be turned out in a small paddock or ridden gently after 2 to 3 weeks. Hyaluronan may be injected intra-thecally at least 10 days after surgery.

Infectious Tenosynovitis

Acute infectious tenosynovitis may be treated by thorough lavage of the sheath, with 5 L of sterile, polyionic isotonic fluids, through large-bore catheters or needles placed in the proximal pouch and distal recess of the sheath.[9,15] Lavage may be carried out in the standing horse, but it is best performed with the horse under general anesthesia for accurate needle placement and use of a high-velocity, high-pressure lavage system. This technique is less useful if purulent material, adhesions, or bony lesions are present. These horses are best treated by tenoscopic lavage and debridement.[13] All debris and hypertrophic synovium, frayed areas on the tendon, and lesions on the sustentaculum tali (Fig. 77-7) are debrided, preferably using motorized equipment, and bone fragments are removed. Drains are rarely necessary, except with recurrent infectious tenosynovitis, but in horses with chronic lameness, leaving a 2- to 4-cm incision (the arthroscope portal) open for drainage is advisable. If a wound is present at the level of the sustentaculum tali, this may be used as an endoscopic portal and then enlarged and left to heal by second intention. Drainage can be prolonged, and primary closure and use of a closed suction drain may be preferable. Fragmentation and osteitis of the sustentaculum tali outside the sheath is best approached through a separate incision. Curettage down to healthy bone is performed. Damage to the long medial collateral ligament of the tarsus should be assessed. Aminoglycoside antimicrobial drugs may be inserted into the sheath at the end of surgery. The horse is treated with systemic broad-spectrum antibiotics (see Chapter 66) for at least 2 weeks postoperatively. NSAIDs are used for at least 5 days and may be continued as necessary. A pressure bandage is applied from foot to mid-crus and changed every 1 to 3 days, depending on the amount of exudate produced. The horse should be rested in a box until the wound has healed and then walked out in hand for 2 to 3 weeks before being turned out in a small paddock.

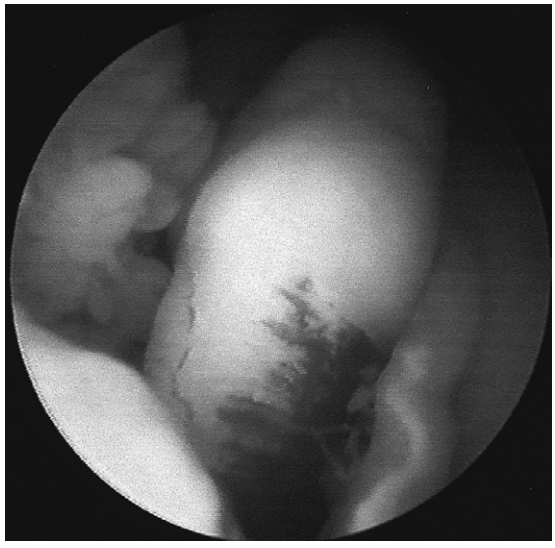

Fig. 77-6 Tenoscopic view of the tarsal sheath at the level of the sustentaculum tali. A large ossicle is seen lateral to the lateral digital flexor tendon.

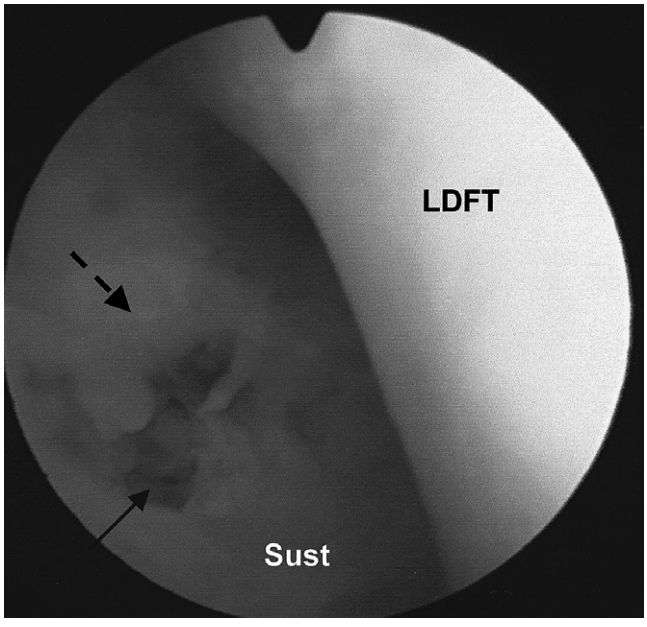

Fig. 77-7 Tenoscopic view of the tarsal sheath at the level of the sustentaculum tali (*Sust*). A small erosion in the fibrocartilage (*plain arrow*), surrounded by fibrinous pannus (*dotted arrow*), is secondary to subacute infectious tenosynovitis. *LDFT,* Lateral digital flexor tendon.

In more chronically infected horses with significant damage to the fibrocartilaginous groove on the sustentaculum tali, or to the lateral digital flexor tendon, open surgery has been advocated.[6,8] The sheath is approached through a desmotomy (longitudinal transection) of the flexor retinaculum to debride all the lesions, followed by the application of a fenestrated drain and repeated lavages for several days. The retinaculum is left unsutured to relieve pressure, and the skin is closed over it.[6] A tenectomy of the damaged tendon within the sheath was performed in one horse unresponsive to this approach. This, however, was associated with significant complications from poor healing of the wound. In another horse, mid-metatarsal tenotomy of the DDFT was carried out, with a heel extension shoe applied to prevent digital hyperextension. This decreased the lameness, presumably by decreasing shearing of the lateral digital flexor tendon on the roughened sustentaculum tali. In my opinion, most horses respond to tenoscopic treatment. More aggressive techniques should be reserved for horses that are unresponsive or those with severe damage to the tendon.

Prognosis

The prognosis is good in horses with idiopathic thoroughpin,[8,9,16] although some horses may have persistent, nonpainful distention of the sheath. In these horses surgery to imbricate the stretched synovial sheath has been advocated,[9] but recurrence is common. The prognosis is fair in horses with acute tenosynovitis treated medically[8,9] and in horses with chip fractures of the sustentaculum tali treated by tenoscopy.[13] In chronic traumatic tenosynovitis, some horses may respond to a single injection of corticosteroids,[3] but most have recurrent distention and lameness caused by adhesion formation and peritendovaginal fibrosis. These may respond well to tenoscopic treatment,[13] but a guarded prognosis should be given if lameness is a prominent feature. In horses with infection the prognosis is fair with prompt drainage and curettage, but poor in horses with severe tendon lesions or extensive damage to the sustentaculum tali.[2,4,9]

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CHAPTER • 78

Extensor Tendon Injury

Jane C. Boswell and Michael C. Schramme

The extensor tendons of the carpus and digit and the respective synovial sheaths are vulnerable to injury because they lie relatively unprotected in the subcutaneous fascia on the dorsal surface of the limb. This section discusses lameness caused by injuries and disorders of the extensor tendons of the carpus and digit. Injuries of the extensor tendons of the hock (gastrocnemius and calcaneal tendons) are discussed elsewhere (see Chapter 81).

ANATOMY

The *extensor carpi radialis* muscle is the largest extensor muscle of the forelimb and has a prominent muscle belly on the cranial aspect of the antebrachium. The extensor carpi radialis muscle extends the carpus, smooths carpal joint movement by dampening oscillations as the hoof strikes the ground, and flexes the elbow.[1,2] The extensor carpi radialis tendon extends through most of the muscle belly and appears on the surface of the muscle in the middle of the antebrachium. The tendon passes through the middle groove at the distal end of the radius, over the dorsal aspect of the carpus, and inserts on the metacarpal tuberosity on the dorsal surface of the proximal end of the third metacarpal bone.[1] As the tendon passes over the carpus, it is bound by the extensor retinaculum and enveloped by a synovial sheath, which extends from 8 to 10 cm proximal to the carpus, to the level of the middle carpal joint. Distal to the sheath the tendon is attached to the carpometacarpal joint capsule. Usually a small bursa is beneath the tendon at the level of the third carpal bone.[1,3]

The *common digital extensor* muscle is a compound muscle with three heads (humeral, radial, and ulnar). It lies lateral to the extensor carpi radialis muscle on the craniolateral aspect of the radius and acts to extend the digit and carpal joints and to flex the elbow. The main common digital extensor tendon appears on the surface of the muscle in the middle of the antebrachium and passes distally through the lateral groove on the cranial aspect of the distal end of the radius and over the capsule of the carpal joints. As the tendon passes distally over the dorsal aspect of the metacarpal region, it courses medially and reaches the dorsal midline just proximal to the fetlock. At the level of the distal end of the proximal phalanx the tendon becomes wider as it is joined by branches of the interosseous tendon (suspensory ligament). The common digital extensor tendon inserts on the extensor process of the distal phalanx and the dorsal surface of the proximal extremities of the proximal and middle phalanges.

The common digital extensor tendon is enveloped by a synovial sheath as it passes over the carpus. The sheath extends from about 8 cm proximal to the carpus to the proximal end of the metacarpal region.[1,3] At the level of the fetlock a bursa occurs between the tendon and the dorsal pouch of the fetlock joint capsule.[1,3,4]

A small tendon also arises from the smaller head (the radial head) of the common digital extensor muscle. This tendon runs through the synovial sheath of the principal tendon and then passes laterally to fuse with the tendon of the lateral digital extensor, or it may continue separately between the common and lateral digital extensor tendons to the fetlock. A small tendon also may arise from the ulnar head of the common digital extensor muscle, which may fuse with the principal tendon, or insert on the joint capsule dorsal to the fetlock joint.

The *lateral digital extensor* muscle is smaller than the other extensor muscles and is situated caudal to the common digital extensor muscle. The action of the lateral digital extensor muscle is to extend the digit and carpus. The lateral digital extensor tendon arises at the level of the distal third of the antebrachium and passes distally through the groove on the lateral styloid process of the radius and over the lateral aspect of the carpus. The tendon becomes flatter and larger distal to the carpus, as it joins the tendon of the radial head of the common digital extensor muscle and a strong band from the accessory carpal bone. The lateral digital extensor tendon runs lateral to the common digital extensor tendon and gradually inclines toward the dorsal aspect of the metacarpal region and inserts on the eminence on the dorsal proximal aspect of the proximal phalanx.[1]

A synovial sheath envelops the lateral digital extensor tendon as it passes over the carpus. It begins 6 to 8 cm proximal to the carpus and extends to the proximal end of the metacarpal region.[1,3] At the fetlock a small bursa lies between the tendon and the joint capsule.[1]

The *extensor carpi obliquus,* or *abductor digitus longus,* is a small muscle that extends the carpus. The tendon of the extensor carpi obliquus arises at the distal end of the radius and courses distally, cranially, and medially over the extensor carpi radialis tendon and then passes through the oblique groove at the distal end of the radius to insert on the head of

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CHAPTER • 78

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The *common digital extensor* muscle is a compound muscle with three heads (humeral, radial, and ulnar). It lies lateral to the extensor carpi radialis muscle on the craniolateral aspect of the radius and acts to extend the digit and carpal joints and to flex the elbow. The main common digital extensor tendon appears on the surface of the muscle in the middle of the antebrachium and passes distally through the lateral groove on the cranial aspect of the distal end of the radius and over the capsule of the carpal joints. As the tendon passes distally over the dorsal aspect of the metacarpal region, it courses medially and reaches the dorsal midline just proximal to the fetlock. At the level of the distal end of the proximal phalanx the tendon becomes wider as it is joined by branches of the interosseous tendon (suspensory ligament). The common digital extensor tendon inserts on the extensor process of the distal phalanx and the dorsal surface of the proximal extremities of the proximal and middle phalanges.

The common digital extensor tendon is enveloped by a synovial sheath as it passes over the carpus. The sheath extends from about 8 cm proximal to the carpus to the proximal end of the metacarpal region.[1,3] At the level of the fetlock a bursa occurs between the tendon and the dorsal pouch of the fetlock joint capsule.[1,3,4]

A small tendon also arises from the smaller head (the radial head) of the common digital extensor muscle. This tendon runs through the synovial sheath of the principal tendon and then passes laterally to fuse with the tendon of the lateral digital extensor, or it may continue separately between the common and lateral digital extensor tendons to the fetlock. A small tendon also may arise from the ulnar head of the common digital extensor muscle, which may fuse with the principal tendon, or insert on the joint capsule dorsal to the fetlock joint.

The *lateral digital extensor* muscle is smaller than the other extensor muscles and is situated caudal to the common digital extensor muscle. The action of the lateral digital extensor muscle is to extend the digit and carpus. The lateral digital extensor tendon arises at the level of the distal third of the antebrachium and passes distally through the groove on the lateral styloid process of the radius and over the lateral aspect of the carpus. The tendon becomes flatter and larger distal to the carpus, as it joins the tendon of the radial head of the common digital extensor muscle and a strong band from the accessory carpal bone. The lateral digital extensor tendon runs lateral to the common digital extensor tendon and gradually inclines toward the dorsal aspect of the metacarpal region and inserts on the eminence on the dorsal proximal aspect of the proximal phalanx.[1]

A synovial sheath envelops the lateral digital extensor tendon as it passes over the carpus. It begins 6 to 8 cm proximal to the carpus and extends to the proximal end of the metacarpal region.[1,3] At the fetlock a small bursa lies between the tendon and the joint capsule.[1]

The *extensor carpi obliquus,* or *abductor digitus longus,* is a small muscle that extends the carpus. The tendon of the extensor carpi obliquus arises at the distal end of the radius and courses distally, cranially, and medially over the extensor carpi radialis tendon and then passes through the oblique groove at the distal end of the radius to insert on the head of

the second metacarpal bone. The tendon is enveloped by a synovial sheath.[1]

In the hindlimb, the *long digital extensor* muscle is situated superficially on the craniolateral aspect of the leg and acts to extend the digit and flex the hock. The tendon of the muscle begins in the middle of the muscle belly and passes distally over the dorsal aspect of the hock, where the tendon is bound by the extensor retinacula and enveloped by a synovial sheath. The sheath begins slightly proximal to the level of the lateral malleolus of the tibia and extends to the proximal third of the metatarsal region. The long digital extensor tendon is joined by the lateral digital extensor tendon about 10 cm distal to the tarsus. In the angle of this union the *extensor digitorium brevis* also joins the principal tendon of the long digital extensor.[1] Distal to this point the arrangement of tendons is the same as in the forelimb.

The lateral digital extensor lies on the lateral surface of the limb caudal to the long digital extensor. The lateral digital extensor tendon runs through the entire length of the muscle belly and emerges at the level of the distal third of the tibia. The tendon descends through the groove on the lateral malleolus of the tibia, where it is bound by the extensor retinacula, and usually blends with the tendon of the long digital extensor. Occasionally the tendon does not insert on the long digital extensor tendon but passes separately lateral to the long digital extensor tendon and inserts on the eminence on the dorsal proximal aspect of the proximal phalanx, like the corresponding tendon in the forelimb.[1] The tendon is surrounded by a synovial sheath that extends from 4 to 6 cm above the lateral malleolus of the tibia to the proximal third of the metatarsal region.[1,3]

DIAGNOSTIC TECHNIQUES

Many disorders of the extensor tendons, particularly acute injuries, maybe diagnosed by careful clinical examination, gait analysis, and palpation. Diagnostic perineural or intrathecal analgesia may be useful in horses with chronic injuries to the extensor tendons to determine the importance of clinical findings. Synoviocentesis of distended tendon sheaths or bursae also may be helpful to distinguish between tenosynovitis caused by injury or sepsis.

Plain radiography is of little value in identifying soft tissue injuries of the extensor tendons. However, radiography may be useful to evaluate mineralization within the tendons or the synovial sheaths, enthesopathy, and associated osseous abnormalities. Contrast radiography may demonstrate a penetrating tract, intrathecal adhesions, or synovial fistulae between the extensor tendon sheaths and carpal joints.[5,6]

Ultrasonography is a safe, non-invasive method of evaluating the extensor tendons, the synovial sheaths, and bursae. Because of a superficial position, a 7.5-MHz or 10-MHz transducer and standoff pad are used to image the extensor tendons and their respective tendon sheaths. Careful evaluation of transverse and longitudinal images allows the clinician to determine the extent of tendon damage, the presence of foreign bodies within the tendon or tendon sheaths, synovial membrane hyperplasia, and intrathecal adhesions.[7]

CONDITIONS AFFECTING THE EXTENSOR TENDONS

Laceration of the Digital Extensor Tendon

Lacerations of the extensor tendons commonly occur in the metacarpal or metatarsal regions of the limbs because of the superficial location at these sites (see Chapter 82). Extensor tendon lacerations are more common in the hindlimb[8-10] and are frequently associated with considerable soft tissue damage and opening of the extensor tendon sheaths.

Transection of an extensor tendon below the carpus or tarsus leads to reduced ability to extend the digit, which results in an exaggerated, rapid flip of the hoof at the end of the swing phase of the stride, intermittent knuckling over at the fetlock, or tripping at the walk. The horse, however, will bear weight in a normal posture if the foot is placed flat on the ground. This gait abnormality is more obvious in horses with hindlimb injuries and when the laceration is near the fetlock, because remaining peritendonous fascial attachments provide some support to the distal part of the tendon in more proximal injuries.

Transection of the extensor tendons proximal to the carpus, and at or just proximal to the tarsus, is also common. Transection of the common digital extensor and extensor carpi radialis tendons commonly occurs proximal to the carpus and often results in pain on flexion of the carpus. Transection of the long digital extensor tendon proximal to or at the tarsus results in greater tarsal flexion during the swing phase of the stride and intermittent knuckling of the digit. Within a few days of transection of an extensor tendon the horse learns to adapt its gait, and tripping and knuckling of the fetlock become less frequent.

The diagnosis of extensor tendon laceration is often apparent from the gait deficit. If necessary, the diagnosis may be confirmed by exploration of the wound with a sterile, gloved finger after aseptic preparation and wound lavage. Plain and contrast radiography should be used to evaluate concomitant joint or bone damage and to help to identify foreign bodies.

After aseptic preparation, wounds over the extensor tendons should be debrided and lavaged. It is important to debride any exposed or obviously devitalized bone and, where possible, to cover bone with skin to reduce the risk of sequestrum formation. Primary apposition of the lacerated tendon is unnecessary, even if a large gap has formed between the distracted tendon ends. Progressive ultrasonographic evaluations show that fibrous tissue develops between the transected tendon ends, and gradually becomes more organized, and regains the linear arrangement of collagen along the line of the original tendon. This fibrous tissue provides an adequate mechanical link between the tendon ends, allowing extensor function of the digit to return.[11]

Although lacerations of extensor tendons may heal without external coaptation, wound healing is facilitated and exuberant granulation tissue formation controlled if limb immobilization is used during the first 3 to 6 weeks after injury. Immobilization may be achieved using a polyvinyl chloride (PVC) splint or cast. Full-limb PVC splints may be applied to the dorsal or palmar aspect of the forelimb to prevent knuckling of the fetlock and carpal flexion. In the hindlimb a distal limb splint applied to the dorsal or plantar aspect of the limb can be used. Application of a shoe with a toe extension also may be applied to help with flat foot placement and to prevent tripping or knuckling over of the fetlock in the early postoperative period. Casts provide an inexpensive and efficient means of immobilizing the limb and tendon ends, and are especially useful when extensor tendon lacerations occur with extensive, contaminated wounds, in which primary wound closure cannot be achieved.

After removal of the cast or splint, the horse should remain confined to a box stall for another 4 to 6 weeks. After cast removal, the toe should be trimmed short so that the toe does not catch to cause knuckling at the fetlock and disruption of the organizing fibrous tissue. A controlled exercise program of in-hand walking exercise may be initiated at this time to strengthen the tendon and improve gliding function. Passive range of movement exercises also may be beneficial. If no signs of knuckling are present after 10 to 12 weeks of controlled exercise, a gradual return to athletic use may begin.

The prognosis for return to athletic function after extensor laceration is good.[8-10] In a retrospective study of 50 horses with extensor tendon lacerations, 73% returned to athletic function, 18% were pasture sound, and 3% remained lame.[10] Complications in that study included wound infection and dehiscence, exuberant granulation tissue, and sequestrum formation. Stringhalt also may occur as a complication of lacerations or injury to the extensor tendons in the dorsoproximal aspect of the metatarsal region. It is speculated that stringhalt may either result from adhesion formation, which decreases the ability of the long or lateral digital extensor tendons to stretch during tarsocrural flexion, or from abnormalities in the myotactic reflex that governs extensor muscle tone and relaxation.[12]

Rupture of Common Digital Extensor Tendon

Rupture of the common digital extensor tendon occurs in foals, and may be present at birth, or develops within the first few weeks of life. This condition may be primary or secondary to flexural deformities of the carpus or metacarpophalangeal joint that result in increased tension in the common digital extensor tendon.[13] It has been suggested that rupture of the common digital extensor tendon may be an inheritable condition,[14] because Arabians and Quarter Horses were overrepresented in one retrospective study.[15]

Because rupture of the common digital extensor tendon always occurs within the synovial sheath,[13] affected foals have a characteristic fluctuant swelling over the dorsolateral aspect of the carpus at the level of the distal carpal joints (Fig. 78-1).

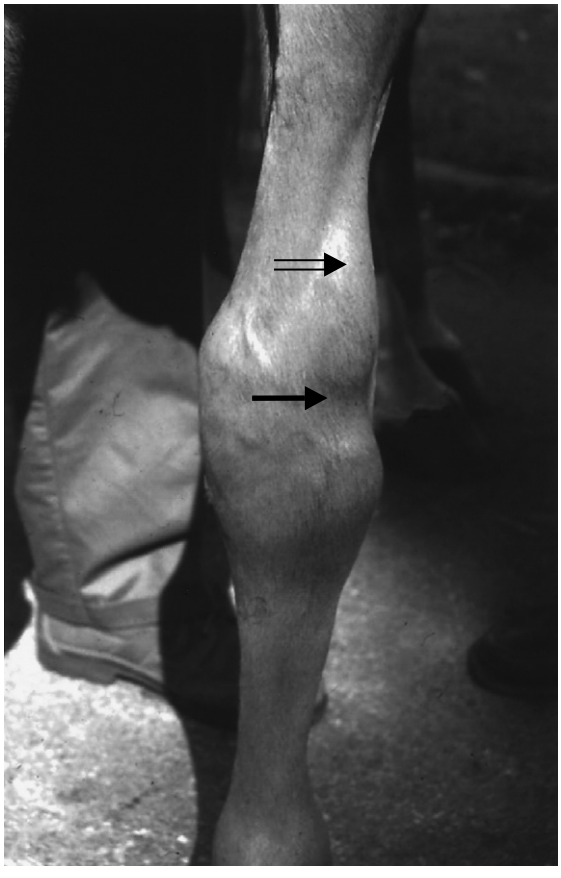

Fig. 78-1 Fluctuant swelling over the dorsolateral aspect of the carpus in a foal with rupture of the common digital extensor tendon. The injury shows the typical transverse depression in the sheath where the common digital extensor tendon has ruptured *(solid arrow)* and the bulge in the sheath where the transected distal part of the tendon is situated *(open arrow)*.

Palpation reveals fluid distention of the tendon sheath, and the ruptured ends of the common digital extensor tendon can usually be identified. Affected foals may have a normal stance, but often stand with the carpus slightly flexed (over-at-the-knee appearance), and may frequently knuckle forward on the fetlock. Most affected foals, however, quickly accommodate the gait and learn to flip the distal limb during the swing phase of the stride.

Diagnosis of rupture of the common digital extensor tendon is based on careful palpation and is confirmed by ultrasonography. Radiography of the carpus should be performed to rule out cuboidal bone malformation.

In foals with primary injuries, those without associated flexural deformities, treatment usually involves box rest and the application of well-padded bandages to support the carpus and prevent abrasion of the dorsum of the fetlock. For foals that exhibit frequent knuckling over on the fetlock, or foals with concurrent flexural deformities of the carpus, a PVC splint should be applied to the palmar aspect of the limb extending from the elbow to the fetlock. For foals with concurrent cuboidal bone malformation, tube casts from the elbow to the fetlock should be used to provide carpal support. Foals with splints usually walk comfortably, and sufficient fibrosis usually has developed after 2 to 4 weeks to allow removal of the splints. However, the foal should be maintained in padded bandages for several more weeks to maintain pressure on the area of tendon rupture to minimize the resulting blemish.

It has been suggested that the ruptured ends of the common digital extensor tendon are unlikely to reunite but adhere to the tendon sheath and that the lateral digital extensor tendon assumes the function of the common digital extensor tendon.[14] However, anecdotal evidence indicates that the tendon ends may rejoin in some horses.[16] Consequently, foals with primary rupture of the common digital extensor tendon, without concurrent cuboidal bone malformation, usually have an excellent prognosis for athletic performance with minimal blemish. Foals with concurrent cuboidal bone malformation or flexural deformities have a more guarded prognosis for athletic performance.[17]

Rupture of Extensor Carpi Radialis Tendon

Rupture of the extensor carpi radialis tendon is a rare condition of adult horses.[14] Partial rupture also may occur rarely, particularly in horses that are used for jumping.[18] Complete and partial rupture of the tendon usually occurs within the synovial sheath on the dorsal aspect of the carpus. The injury is believed to result from trauma associated with repeated overflexion of the carpus.[14] Partial rupture of the tendon has been reported to be a consequence of tendon damage caused by exostoses on the distal radius.[18]

Acute rupture of the extensor carpi radialis tendon is characterized by pain and distention of the tendon sheath. Extension and flexion of the carpus are limited, which may result in dragging of the toe.[19] With chronic injuries of the extensor carpi radialis tendon, distention of the tendon sheath persists, but this condition is usually painless unless an associated inflammatory tenosynovitis occurs.

Complete rupture of the extensor carpi radialis tendon results in excessive carpal flexion during limb protraction and consequently a higher arc of foot flight in the affected limb. Limb extension may be characterized by a double movement as the carpus suddenly snaps into extension.[20,21] Atrophy of the extensor carpi radialis muscle may be evident in chronic injuries. Partial rupture of the extensor carpi radialis tendon may be associated with mild lameness and restricted carpal flexion.[18]

Diagnosis of rupture of the extensor carpi radialis tendon may be confirmed by careful palpation, ultrasonography, and

contrast radiography.[6,7] Plain radiography should be used to evaluate associated osseous lesions.

However, horses with acute, complete rupture of the extensor carpi radialis tendon may be treated surgically. Tenosynovotomy and primary suturing of the tendon ends, or substitution of the extensor carpi obliquus tendon by tendon anastomosis, has been described.[14] However, other authors believe that surgical apposition of the tendon ends is unnecessary and advocate tenoscopy that allows characterization of the extent of the injury, debridement of the damaged tendon ends, tenolysis, and lavage of the sheath. Postoperatively the limb should be immobilized in a tube cast or splint for 2 to 4 weeks. Once the cast and sutures have been removed, passive range of movement exercises should be instituted to prevent the formation of intrathecal adhesions and to reduce carpal joint capsule fibrosis that result in a limited range of carpal flexion. The prognosis for return to athletic performance for horses with complete rupture of the extensor carpi radialis tendon is unfavorable. The prognosis for return to athletic performance for horses with partial rupture of the extensor carpi radialis tendon is also guarded to fair, but tenolysis and partial tenosynovectomy may benefit some horses.[14,19]

Enthesopathy of the Insertion of the Extensor Carpi Radialis Tendon

In the racing Standardbred, tearing of the extensor carpi radialis tendon attachment at its insertion on the proximodorsal aspect of the third metacarpal bone can cause lameness, and these tears may play a role in the cause of fractures of the dorsomedial aspect of the proximal end of the third metacarpal bone (see Chapter 38).[22]

CONDITIONS AFFECTING THE EXTENSOR TENDON SHEATHS

Tenosynovitis

Idiopathic Tenosynovitis

Idiopathic tenosynovitis may be defined as tenosynovitis with synovial effusion, but without inflammation, pain, or lameness.[14] Idiopathic tenosynovitis of the extensor tendon sheaths has been reported in foals at birth[23]; in adults the condition tends to develop insidiously. The pathogenesis in newborn foals is unknown, but in adult horses the pathogenesis is presumed to be caused by chronic trauma.[14] However, distention of the long digital extensor sheath in hindlimbs often occurs bilaterally, unassociated with any history of trauma, and is of unknown cause.

Ultrasonographic examination usually reveals anechoic fluid within the sheath. Occasionally, hypoechoic lesions are identified within the enclosed tendon, the significance of which is unknown, because lameness is rarely present.

Because idiopathic tenosynovitis usually is not associated with pain or lameness, treatment is unnecessary unless the owner is concerned about cosmesis. Treatment by injection of corticosteroids (10 to 20 mg triamcinolone acetonide, or 40 to 100 mg methylprednisolone acetate) and pressure bandaging often only provide a temporary resolution of tendon sheath effusion.[14] Anecdotal reports suggest that intrathecal injection of 4 to 15 mg of atropine sulfate, alone or with corticosteroids and hyaluronan, may cause permanent resolution of the effusion. Atropine sulfate is purported to reduce the production of synovial fluid from synoviocytes; however, its anticholinergic effects means its use has been associated with transient signs of mild colic and depression.

Acute Traumatic Tenosynovitis

Acute tenosynovitis is characterized by a rapidly developing effusion of the tendon sheath, accompanied by heat, pain, and variable lameness. Acute tenosynovitis of the extensor tendon sheaths is often caused by trauma, such as a fall, or hitting a jump with the carpus.[18] The injury is common in event horses, in which it often is not associated with lameness.[16]

Diagnosis of acute tenosynovitis is based on clinical signs and ultrasonography. Ultrasonography is useful to evaluate concurrent tendonitis of the extensor tendons and to differentiate acute tenosynovitis from other conditions that are associated with soft tissue swelling over the dorsal aspect of the carpus, including hygroma, cellulitis, synovial hernia, and effusion of the carpal joints (see Chapter 39).

Horses with acute tenosynovitis of an extensor tendon sheath are treated by rest, cold hydrotherapy, and nonsteroidal anti-inflammatory drugs. Aspiration of fluid and injection of corticosteroids is reserved for horses that do not respond to more than 1 week of conservative treatment.[24]

Infectious Tenosynovitis

Infectious tenosynovitis is characterized by dramatic synovial effusion, heat, pain, swelling, and severe lameness. Contamination from a penetrating wound is the most common cause of infectious tenosynovitis, but infection also may arise from hematogenous spread of bacteria and iatrogenic infection.[25] Infection of the common digital extensor tendon sheath also has been reported as a complication of hemicircumferential periosteal transaction.[26] After penetration of a tendon sheath, severe lameness does not become evident unless infection becomes established. As long as the tendon sheath is open and draining, then the lameness may be less severe.[27]

Once infection is established, rapid and aggressive treatment is necessary to prevent synovial membrane hyperplasia, fibrosis of the tendon sheath, intrathecal adhesion formation, and damage to the extensor tendons. Prompt recognition of infectious tenosynovitis is essential for a successful outcome. Diagnosis of infectious tenosynovitis is based on clinical signs and must be confirmed by synovial fluid aspiration and analysis. Infected synovial fluid is typically turbid and has a low viscosity, an elevated total nucleated cell count ($>30 \times 10^9$/L, with more than 90% neutrophils), and a total protein concentration of more than 40 g/L. Contrast radiography (Fig. 78-2) or ultrasonography may be useful to confirm communication between a penetrating wound and the tendon sheath, especially if horses are presented soon after injury.

The primary aim of treatment of horses with infectious tenosynovitis is the rapid elimination of bacteria and rapid return of the normal synovial environment. This is best achieved by wound debridement, lavage of the tendon sheath with copious amounts of sterile isotonic fluids, and the provision of bactericidal levels of appropriate antibiotics within the sheath.

Appropriate systemic and intra-thecal antibiotic therapy should be initiated immediately. The presence of Enterobacteriaceae most commonly is associated with tendon sheath infection caused by a penetrating wound, whereas staphylococci are most commonly identified as the cause of iatrogenic infections of the tendon sheath.[25] The most effective combination of antibiotics for the treatment of infectious tenosynovitis is amikacin and cephalosporin (>85% effective).[28] However, the cost of these drugs may be prohibitive, and other drug combinations, such as penicillin and gentamicin, may be considered. The initial selection of antibiotic may be altered according to bacterial sensitivity, if a positive culture is obtained from the synovial fluid.

Wound debridement and lavage of the affected tendon sheath with copious amounts of sterile isotonic fluids is important to reduce concentrations of bacteria and inflammatory mediators. Early in the infection, effective lavage maybe achieved by through-and-through lavage using 16-gauge needles or arthroscopic egress cannulae. In more established infectious tenosynovitis, leukocytes and fibrin accumulate

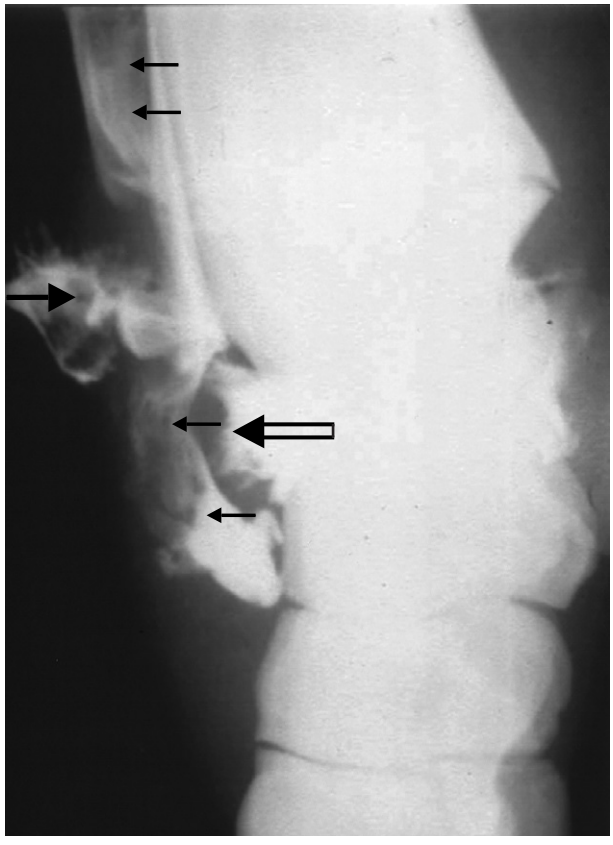

Fig. 78-2 Lateromedial radiographic view of a carpus. Dorsal is to the left. Positive-contrast medium has been injected into the extensor carpi radialis tendon sheath and antebrachiocarpal joint in a horse that had sustained a deep puncture wound over the dorsal aspect of the carpus. Radiopaque contrast agent is near the skin surface *(solid arrow)*, outside the tendon sheath *(small arrows)*, confirming penetration of the extensor carpi radialis sheath. The antebrachiocarpal joint *(open arrow)* was not involved.

within the sheath, necessitating tenoscopic debridement. If satisfactory debridement and lavage have been achieved, primary closure of the sheath may be performed. The horse should be monitored closely for lameness, and repeated synovial fluid samples should be taken at 2- to 3-day intervals to monitor for return of infection. Alternatively the sheath may be left open, or a closed suction drain system may be inserted into the sheath for further elimination of inflammatory mediators and fibrin. The drain should be left in place for 3 to 5 days, but careful management is important to prevent ascending suprainfection.

Systemic antibiotic therapy should be continued for at least 2 weeks after the resolution of clinical signs. Once the infection has been eliminated, intrathecal injection of corticosteroids or hyaluronan may be used, with physiotherapy, to reduce adhesion formation and restore the normal gliding movement of the tendon within its sheath. However, both drugs may cause immunosuppression within the sheath and may potentiate dormant infection. Early return to controlled exercise is also important to reduce intrathecal adhesion formation.

If chronic infectious tenosynovitis becomes established, exploration of the tendon sheath and radical synovectomy may be required (see the following discussion). Successful outcome has been reported after complete resection of the intrasynovial part of the common digital extensor tendon in a horse with chronic infectious tenosynovitis and tendonitis.[29]

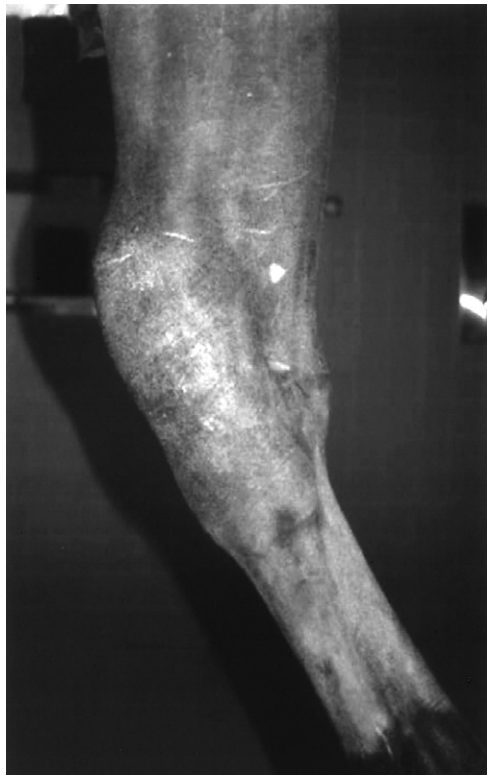

Fig. 78-3 Chronic distention of the tendon sheath of the extensor carpi radialis in a horse with chronic tenosynovitis.

The prognosis for return to soundness after infectious extensor tenosynovitis appears to be more favorable than for infectious flexor tenosynovitis,[25,27] possibly because extensor tendons are non–weight bearing compared with flexor tendons. The prognosis for return to soundness is generally good after early surgical intervention and appropriate antibiotic therapy for horses with infectious extensor tenosynovitis.

Chronic Tenosynovitis

Chronic tenosynovitis of the extensor tendon sheaths is characterized by persistent synovial effusion, fibrous thickening of the sheath, and subcutaneous edema.[30,31] Chronic tenosynovitis results in variable, sometimes severe, lameness; restricted carpal flexion; and a gait characterized by circumduction of the affected limb during protraction.[31] However, in some horses there is no gait abnormality.

Chronic tenosynovitis commonly arises after penetrating injuries to the carpal extensor sheaths, which may result in the inoculation of foreign material or bacteria into the synovial cavity, and establishment of an infectious or non-infectious chronic tenosynovitis. The condition commonly occurs in horses jumping natural fences, because of penetration of the sheath by thorns.[31] Chronic tenosynovitis also may occur after acute tenosynovitis and may be associated with partial tendon rupture.[21,30] Chronic inflammation of the tendon sheath causes granulomatous proliferation of the synovial membrane, connective tissue deposition in the fibrous capsule, and fibrous adhesion formation between the tendon and its sheath, resulting in restriction of movement and pain on carpal flexion.[30,31]

Diagnosis of chronic tenosynovitis is based on clinical signs of effusion and thickening of the affected sheath (Fig. 78-3)

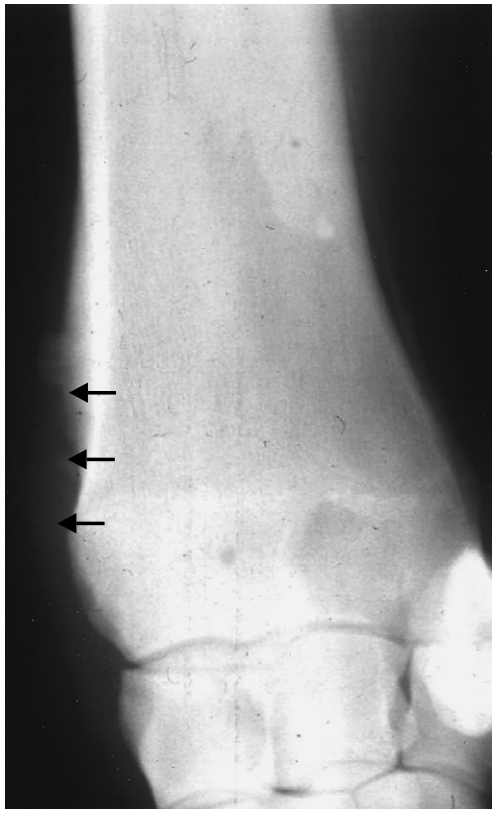

Fig. 78-4 A craniolateral-caudomedial oblique radiographic view of the distal radius in a horse with chronic tenosynovitis of the extensor carpi radialis tendon sheath. There is palisading new bone formation *(arrows)* on the craniomediodistal ridge of the radius adjacent to the tendon sheath.

and restricted carpal flexion. Radiography of the carpus often reveals palisading new bone formation on the craniodistal ridges of the radius adjacent to the affected sheaths (Fig. 78-4) and entheseous new bone formation on the dorsal aspect of the carpal bones.[31] Synovial fluid aspirates vary from serosanguineous to turbid, with an increased nucleated cell count and total protein concentration. Ultrasonography may demonstrate tendon damage, synovial membrane hypertrophy, foreign bodies, and intrathecal adhesions.

Conservative treatment with antibiotics, analgesics, intrathecal corticosteroids, and symptomatic physiotherapeutic procedures such as bandaging, cold hosing, massage, and forced exercise is usually unrewarding.[18,21,30,31] The results of surgical treatment have been good, however. Surgical treatment involves exposing the entire length of the tendon within its sheath, radical synovectomy of the hyperplastic synovial membrane, removal of intrathecal adhesions, debridement of damaged tendon, and primary closure of the fibrous layer of the tendon sheath and skin. Postoperatively the carpus should be kept bandaged and the horse confined to its box for 10 days. After suture removal on the fourteenth post-operative day, an intensive program of physiotherapy involving manual flexion and extension of the carpus and an ascending program of in-hand walking exercise should be initiated. Early passive motion to stimulate cavitation and reformation of the synovial lining and prevent adhesion formation is considered an essential part of the treatment.[31] Initially, carpal flexion is resented and administration of non-steroidal anti-inflammatory drugs and sedatives may be required. Full flexion of the carpus should be achieved by 30 days after surgery.

The prognosis for soundness after surgical exploration and radical synovectomy is excellent, but the resultant scar may cause a substantial blemish. In a retrospective study of 15 horses with chronic tenosynovitis treated by surgical exploration and radical synovectomy, all were sound and all but one horse returned to the former use.[31]

Osteochondromatosis

Synovial osteochondromatosis of the extensor carpi radialis tendon sheath has been reported. Synovial osteochondromatosis is characterized by the formation of multiple small osseous bodies within a synovial-lined structure. The cause is unclear, but it may be associated with trauma. Clinical signs include swelling on the dorsal surface of the carpus characterized by multiple, firm subcutaneous nodules and crepitus during joint movement, and lameness is usually evident. Diagnosis is confirmed by radiography and ultrasonography.[32] Although no information is available for the surgical treatment of osteochondromatosis of the extensor tendon sheaths in horses, partial synovectomy and arthroscopic removal of the osteochondral bodies has been used successfully in other synovial cavities in horses and other species.[33]

Intersynovial Fistulae

Intersynovial fistulae are uncommon, but they have been documented between the common digital extensor tendon sheath and antebrachiocarpal joint,[34] the common digital extensor tendon sheath and middle carpal joint,[14] and the extensor carpi radialis tendon sheath and middle carpal joint.[5] The cause of these fistulae is unclear, but they are considered to be traumatic in origin and may occur with carpitis or carpal fractures.[5,34] Typically, horses with intersynovial fistulae have chronic lameness and distention of the affected tendon sheath. Synovial fluid can be massaged from the joint to the tendon sheath. Diagnosis may be confirmed by contrast radiography[6] and intrasynovial analgesia. Surgical treatment is advocated in the management of intersynovial fistulae,[14] but little information concerning the prognosis of affected horses is available, because reports of this condition are rare. Surgical treatment involves exposure of the fistula, removal of redundant synovial membrane, and closure of the fibrous layers of the joint and tendon sheath.

Infectious Bursitis

Infectious bursitis may occur in any of the bursae associated with the extensor tendons over the dorsal aspect of the fetlock. The condition is seen most commonly in horses that jump natural obstacles (e.g., eventers and National Hunt horses). Typically these horses have swelling of the dorsal aspect of the fetlock (Fig. 78-5) and mild to severe lameness. Infectious bursitis may affect the subtendonous bursae or more commonly affects an acquired subcutaneous (supratendonous) bursa on the dorsal aspect of the long digital extensor tendon in the fetlock region.[35] Occasionally both bursae may communicate around the lateral or medial aspect of the long digital extensor tendon. Some horses have a severe, non–weight-bearing lameness, and this condition frequently is confused with infectious arthritis of the metacarpophalangeal or metatarsophalangeal joints. Diagnosis is confirmed by ultrasonographic examination or contrast radiography[6] and synoviocentesis. Synoviocentesis from the palmar or plantar pouch of the adjacent metacarpophalangeal or metatarsophalangeal joint should be used to rule out joint infection. Treatment consists of surgical drainage and debridement and appropriate antibiotic therapy. The prognosis for return to soundness is good.

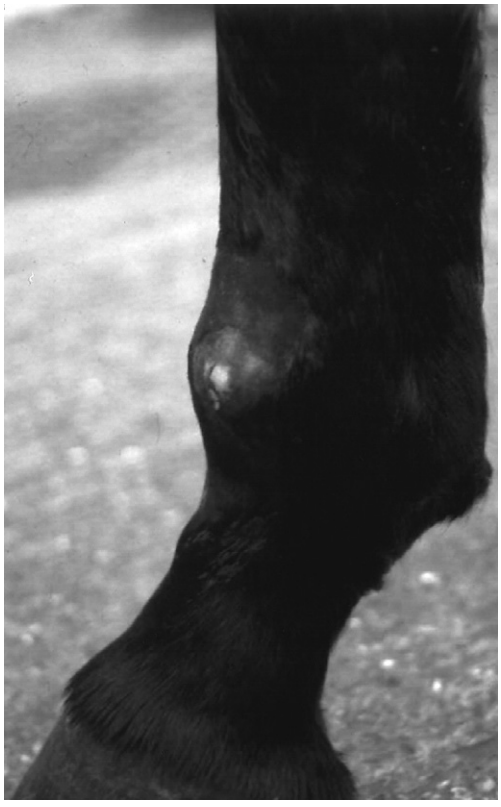

Fig. 78-5 Prominent soft tissue swelling over the dorsal aspect of the fetlock region in the hindlimb of a horse with severe lameness. This is a supratendonous infectious bursitis and should not be confused with infectious arthritis of the metatarsophalangeal joint. Arthrocentesis of the metatarsophalangeal joint through a dorsal approach may result in iatrogenic infectious arthritis.

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CHAPTER • 79

Curb

Mike W. Ross and Ronald L. Genovese

The historical definition of curb is enlargement on the plantar aspect of the fibular tarsal bone (calcaneus), caused by inflammation and thickening of the (long) plantar ligament.[1] However, by ultrasonographic evaluation, we have redefined curb as a complex of soft tissue injuries that occurs on the distal plantar aspect of the tarsus. Long plantar desmitis is only one of many injuries that causes curb. However, the term *curb* is useful to describe swelling of the distal, plantar aspect of the tarsus (excluding the calcaneal bursa and proximal aspect of the calcaneus). In the rest of this chapter, curb is used specifically to mean soft tissue swelling of the plantar aspect of the tarsus. Conformational abnormalities or bony exostoses can mimic or contribute eventually to formation of curb.

CLINICAL APPEARANCE OF CURB

The convex profile typical of curb is best seen from the side (Fig. 79-1). Careful evaluation of swelling from all perspectives, palpation, and thorough lameness examination are critical. Curb must be differentiated from other swellings of the hock, including capped hock, effusion, edema and fibrosis of the calcaneal bursa (see Chapter 80), tarsal tenosynovitis (see Chapter 77), thoroughpin (with or without involvement of the tarsal sheath), and bony enlargements of the distal hock region (see Chapter 45). Injuries of the deep digital flexor tendon (DDFT) as it courses along the plantaromedial aspect of the hock within the tarsal sheath can produce typical signs of curb, but they account for only a small percentage injuries in horses with curb.

Horses with sickle-hock conformation are said to be curby (see Chapter 4). Sickle-hock and in-at-the-hock conformation lead directly to curb, a finding most common in the Standardbred (STB) and Thoroughbred (TB) racehorse. Prognosis for STB racehorses with sickle-hock conformation and curb is worse in the trotter than in the pacer. Trotters with sickle-hock conformation are usually fast early in training and racing, but this conformation is often career limiting. Sickle-hock conformation is also undesirable in the TB racehorse. Horses with sickle-hock conformation often develop curb first, but they then independently or concomitantly develop other lameness associated with the distal hock joints. Tarsal region lameness begins with curb in 2- and 3-year-olds and progresses to osteoarthritis of the centrodistal and tarsometatarsal joints or fractures of the central tarsal bone, or more commonly the third tarsal bone.

Horses can have curby conformation without developing curb, and horses with normal hindlimb conformation can develop curb. The proximal aspect of the fourth metatarsal bone (MtIV) is often prominent in horses with sickle-hock conformation. The most dramatic example of altered joint morphology occurs in young foals with tarsal crush syndrome, the result of delayed or incomplete ossification of the tarsal cuboidal bones (see Chapter 45).

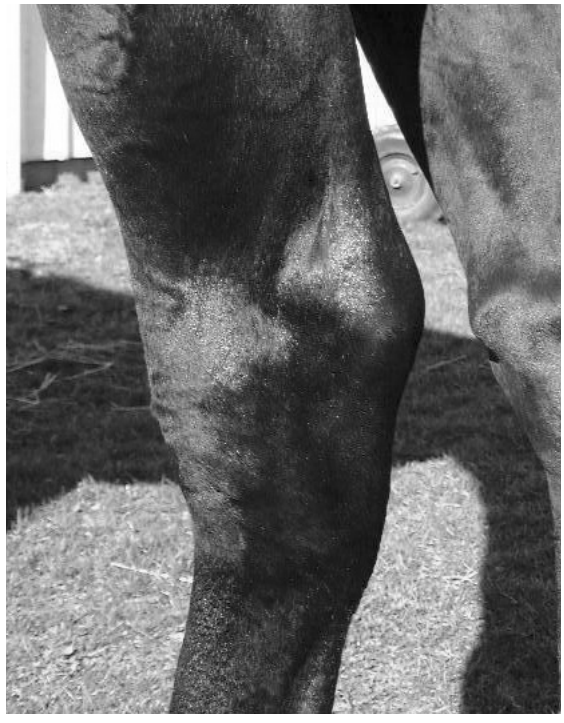

Fig. 79-1 A Standardbred racehorse with typically appearing curb. Swelling associated with the distal, plantar tarsus is centered over the centrodistal and tarsometatarsal joints. In this horse swelling was caused by superficial digital flexor tendonitis.

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34. Johnson JE, Ryan GD: Intersynovial fistula in the carpus of a horse, *Cornell Vet* 65:84, 1975.
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CHAPTER • 79

Curb

Mike W. Ross and Ronald L. Genovese

The historical definition of curb is enlargement on the plantar aspect of the fibular tarsal bone (calcaneus), caused by inflammation and thickening of the (long) plantar ligament.[1] However, by ultrasonographic evaluation, we have redefined curb as a complex of soft tissue injuries that occurs on the distal plantar aspect of the tarsus. Long plantar desmitis is only one of many injuries that causes curb. However, the term *curb* is useful to describe swelling of the distal, plantar aspect of the tarsus (excluding the calcaneal bursa and proximal aspect of the calcaneus). In the rest of this chapter, curb is used specifically to mean soft tissue swelling of the plantar aspect of the tarsus. Conformational abnormalities or bony exostoses can mimic or contribute eventually to formation of curb.

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Horses with sickle-hock conformation are said to be curby (see Chapter 4). Sickle-hock and in-at-the-hock conformation lead directly to curb, a finding most common in the Standardbred (STB) and Thoroughbred (TB) racehorse. Prognosis for STB racehorses with sickle-hock conformation and curb is worse in the trotter than in the pacer. Trotters with sickle-hock conformation are usually fast early in training and racing, but this conformation is often career limiting. Sickle-hock conformation is also undesirable in the TB racehorse. Horses with sickle-hock conformation often develop curb first, but they then independently or concomitantly develop other lameness associated with the distal hock joints. Tarsal region lameness begins with curb in 2- and 3-year-olds and progresses to osteoarthritis of the centrodistal and tarsometatarsal joints or fractures of the central tarsal bone, or more commonly the third tarsal bone.

Horses can have curby conformation without developing curb, and horses with normal hindlimb conformation can develop curb. The proximal aspect of the fourth metatarsal bone (MtIV) is often prominent in horses with sickle-hock conformation. The most dramatic example of altered joint morphology occurs in young foals with tarsal crush syndrome, the result of delayed or incomplete ossification of the tarsal cuboidal bones (see Chapter 45).

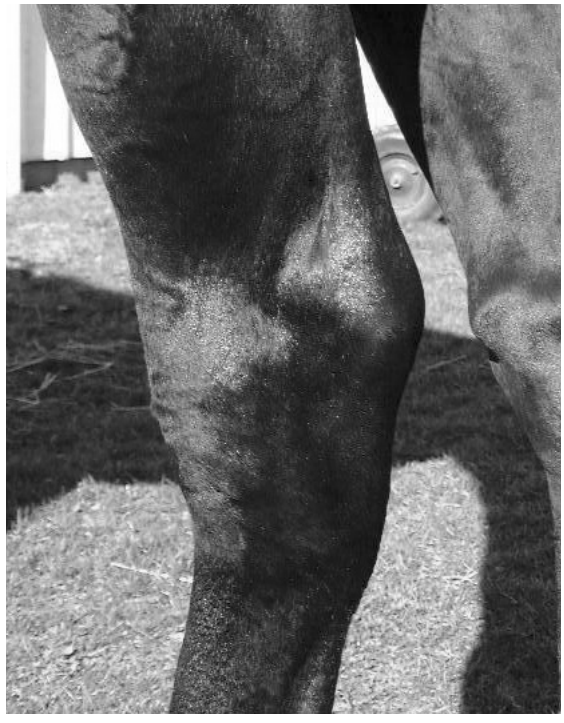

Fig. 79-1 A Standardbred racehorse with typically appearing curb. Swelling associated with the distal, plantar tarsus is centered over the centrodistal and tarsometatarsal joints. In this horse swelling was caused by superficial digital flexor tendonitis.

Firm, fibrous soft tissue swelling can develop just proximal to MtIV as a sequela to injection or local analgesia of the tarsometatarsal joint, presumably from local trauma, hemorrhage, or leakage into the subcutaneous tissues. Mild bony proliferation or fragmentation of proximal MtIV, or of the fourth tarsal bone, can cause focal swelling easily mistaken for curb.

Considerable variation in clinical signs occurs, and the injury cannot be categorized, or a management program and prognosis established, without thorough clinical and ultrasonographic examinations. Historically, owners and trainers consider curb to be an annoying, self-limiting problem that rarely causes lameness or poor performance, that responds to a single treatment that is uniformly effective, and that is cured once curb is treated. Most racehorse trainers are opposed to resting a horse with curb unless lameness is performance limiting, so veterinarians often are faced with management decisions without an option for even short-term rest or a reduction in training intensity. Many traditional therapies have no data to support efficacy.

Lameness ranges from none, mild, or severe depending on the structure involved and extent of injury. Lameness tends to be worse if the soft tissue structure involved is located dorsal to the superficial digital flexor tendon (SDFT) in the plantar tarsus (DDFT, long plantar ligament), if SDFT injury is diffuse, or if a mixed injury involves more than one structure. Diagnosis is straightforward in horses with obvious lameness seen at a trot in hand and painful swelling, but lameness may be evident only as a slight loss of performance or unlevelness when performing at maximum and may be perceived only by trainers, drivers, or riders. A horse with chronic curb may not exhibit signs of pain during palpation, or lameness at a trot in hand, but can show lameness at speed, and convincing a trainer that the long-term swelling is a source of pain may be difficult. The area should be palpated carefully with the limb bearing weight and flexed. Swelling may be firm and fibrous, with few signs of active inflammation, or may be warm, painful, and edematous. Acute, compliant or mushy swelling is associated with hemorrhage or other subcutaneous fluid accumulation and sometimes deeper soft tissue injury. Horses with this form of curb usually have acute, moderate to severe lameness. Horses with distal hock joint pain often exhibit a painful response when direct pressure is placed on plantar hock structures, including the SDFT, proximal aspect of MtIV, second metatarsal bone, and proximal aspect of the suspensory ligament. Often swelling is not detected in these horses. Response to upper limb flexion varies and is non-specific. Direct digital palpation followed by trotting is useful, because horses with active curbs show increased lameness.

Because horses with curb can have concomitant osteoarthritis or other problems of the lower hock joints, differentiation of the source of pain is important but difficult. Diagnostic analgesia is useful but not foolproof. If horses are lame at a trot in hand, local infiltration of local anesthetic solution subcutaneously over the curb is effective. A minimum of 20 to 30 ml of local anesthetic solution should be infiltrated along the lateral, plantar, and medial aspects of the curb. Small-gauge needles should be avoided (to avoid needle breakage), and the injection should be performed with the limb in flexion, because horses may object to several injections. If horses are not visibly lame at a trot in hand, examination at the track or under saddle should be performed. Selective intra-articular analgesia of the lower hock joint and sequential perineural analgesia to rule out the lower limb are essential. A tibial nerve block alleviates pain with curb, but it is seldom done because other common sources of pain are abolished similarly.

Radiography and scintigraphy help differentiate other sources of pain, but ultrasonography is the imaging method of choice to determine which structures are involved and the extent of damage.

APPLIED ANATOMY AND NORMAL ULTRASONOGRAPHIC EXAMINATION OF THE PLANTAR TARSUS

Plantar to the calcaneus are skin, subcutaneous tissues, a thin fibrous tissue layer, the SDFT, and the long plantar ligament. Medially the DDFT courses distally over the sustentaculum tali, within the tarsal sheath. Normally the tarsal sheath has a small amount of fluid that can be seen during ultrasonographic examination, but it is not felt. The long plantar ligament originates from the calcaneus, closely adheres to this bone, and inserts distally on the plantar surface of the fourth tarsal bone and MtIV. The plantar tarsus can be divided into zones to classify findings (Fig. 79-2) or the distance measured from the proximal aspect of the calcaneus (point of hock). Transverse and longitudinal images of both limbs should be obtained from the plantar midline, plantaromedial (to evaluate the DDFT), and slightly plantarolateral (to evaluate the distal aspect of the long plantar ligament). Measurement of cross-sectional area (CSA) is important to confirm lesions in which enlargement has occurred but with no overt fiber damage. Precise placement of the ultrasound transducer is important since the long plantar ligament changes size and shape as it courses distally. Knowledge of normal ultrasonographic anatomy is crucial (Figs. 79-3 to 79-6).

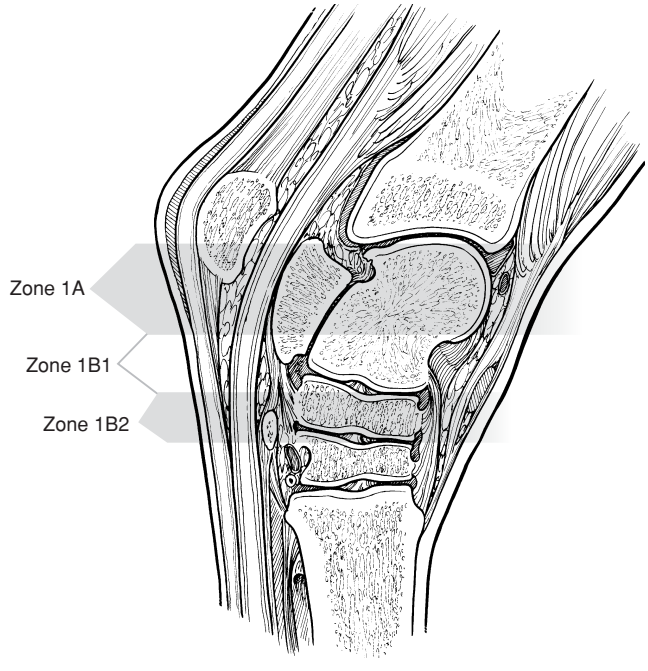

Fig. 79-2 The plantar aspect of the tarsus is divided into zones lA and lB. Because zone lB is rather large and important, the zone is sometimes subdivided into 1B1 and 1B2. An alternative technique for recording level of injury is to measure distally from the proximal aspect of the calcaneus.

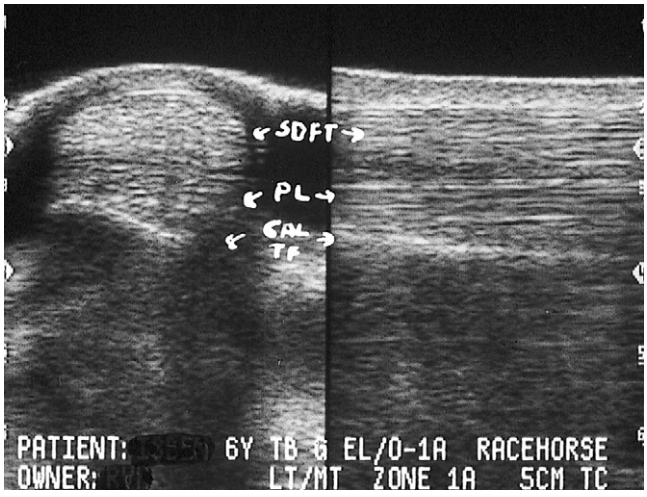

Fig. 79-3 Transverse (*left*) and longitudinal (*right*) midline ultrasonographic images at 5 cm distal to the point of the hock. A thin subcutaneous fibrous tissue layer runs along the plantar surface of the superficial digital flexor tendon. The superficial digital flexor tendon (*SDFT,* crescent shape) is narrower in a medial to lateral direction and somewhat thickened from that seen proximally. In the longitudinal scan the normal SDFT has a dense parallel fiber pattern. Deep to the SDFT the long plantar ligament *(PL)* is at full thickness (plantar to dorsal direction), is rectangular in shape, and is attached firmly to the calcaneus *(CAL)*. At this level the deep digital flexor tendon is out of view medially and must be evaluated by placing the transducer plantaromedially.

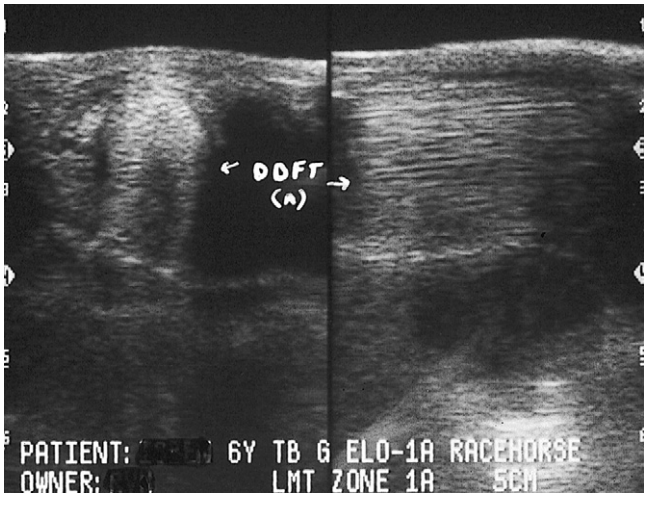

Fig. 79-4 Transverse (*left*) and longitudinal (*right*) ultrasonographic images from the plantaromedial aspect of the left hock 5 cm distal to the point of the hock. In the transverse image the tarsal sheath surrounds the deep digital flexor tendon *(DDFT)*. The tendon is oval and has a large eccentric hypoechoic region composed of residual muscle tissue, but this defect could be caused by incident angle artifact.

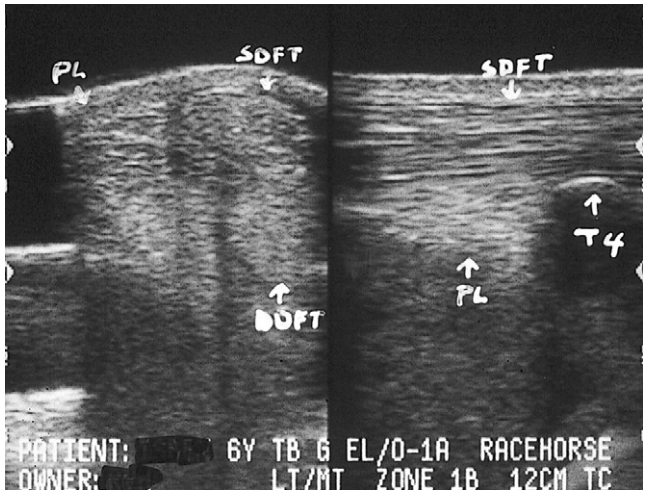

Fig. 79-5 Transverse (*left*) (medial is to the right) and longitudinal (proximal is to the left) (*right*) plantarolateral ultrasonographic images 9 cm distal to the point of the hock at the level of the fourth tarsal bone (*T4*). The long plantar ligament *(PL)* is a multi-septated ligamentous structure, and large plantar fiber bundles are not normally perfectly aligned with dorsal bundles. In transverse images the long plantar ligament normally may appear to lack echogenicity, and the size and shape changes at the insertion on the fourth tarsal bone. The long plantar ligament is wide at the insertion on the fourth tarsal bone (longitudinal scan).

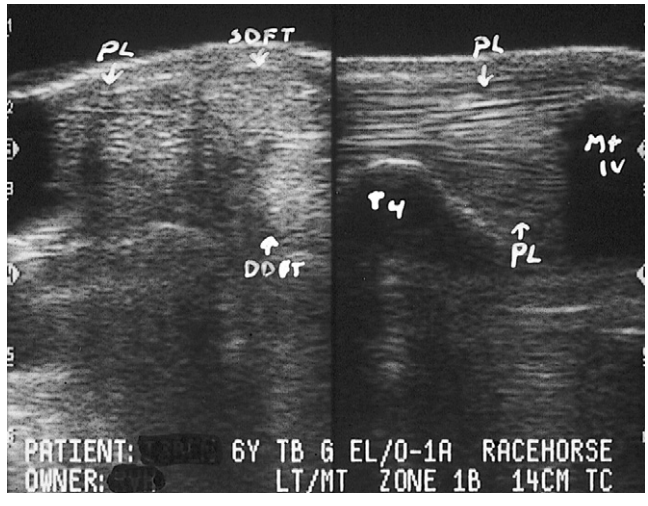

Fig. 79-6 Transverse (*left*) (medial is to the right) and longitudinal (*right*) (proximal is to the left) plantarolateral ultrasonographic images 14 cm distal to the point of the hock, just distal to the insertion of the long plantar ligament *(PL)* on the fourth tarsal bone *(T4)*. The long plantar ligament is thick in the plantar to dorsal direction. A midline image would show the superficial digital flexor tendon *(SDFT)* and deep digital flexor tendon *(DDFT)*, but at this level a plantarolateral transducer placement is needed to assess the long plantar ligament. In the longitudinal image the long plantar ligament is seen attaching to the fourth tarsal bone proximally and fourth metatarsal bone *(MtIV)* distally.

CURB: A COLLECTION OF SOFT TISSUE INJURIES

Seventy-three horses with a curb were examined using ultrasonography, comprising 58 (80%) racehorses (48 STB and 10 TB) and 15 non-racehorse sport horses (hunters, jumpers, Tennessee walking horses, reining horses, Three-Day Event horses, barrel racing, and general-use horses).[2] Twenty-two horses (30%) had thickening, inflammation, swelling, or fluid accumulation in peritendonous and periligamentous tissues, without obvious injury of the SDFT, DDFT, and long plantar ligament. Eighteen horses (25%) had a combination of fluid accumulation of peri-tendonous and periligamentous tissues with superficial digital flexor tendonitis, and 25 (34%) had a combination of fluid accumulation of peritendonous and periligamentous tissue and long plantar ligament desmitis. Six horses (8%) had fluid accumulation of peritendonous and periligamentous tissues and DDFT injury, and two horses (3%) had peritendonous and periligamentous tissue infection. This incidence of injury is considered typical.

Curb is primarily an injury of racehorses, especially STBs. Gait, speed, and training methods differ between racing breeds, and STB racehorses have a higher prevalence of conformational abnormalities, such as sickle hock and in at the hock. Weight and load distribution are considerably different. Curb develops frequently in STB racehorses that train and race on a thin, near-hard surface, contrary to many soft tissue injuries that result from work on deep surfaces. Many curbs develop in young STB racehorses early in training. In some instances track surfaces are inconsistent and perhaps slippery, since early training is done in the winter months. Non-racehorse sport horses develop curb, but only sporadically, and lameness is often moderate to severe and most commonly is associated with peritendonous and periligamentous tissue swelling, although other structures are sometimes involved.

Peritendonous and Periligamentous Inflammation

Peritendonous and periligamentous tissue swelling occurs alone or with abnormalities of one or more of the SDFT, DDFT, or long plantar ligament. Peritendonous and periligamentous tissue injury can occur secondary to direct trauma from horses kicking a wall or trailer door, or rarely from a direct kick or interference injury from another horse, resulting in acute, large, painful swelling. Ultrasonographic examination most often reveals frank hemorrhage and edema. More commonly the horse has neither history of trauma nor clinical findings suggesting trauma. We suspect that peritendonous and periligamentous tissue injury reflects excess loading or strain of the plantar tarsal soft tissue structures from race training. Extensive jogging of young STBs early in training may cause dramatic increase in hock loading, and tension and overstretching of thin peritendonous and periligamentous tissue occurs. Peritendonous and periligamentous tissue injury may be an accumulated overload injury and may develop secondarily to other lameness. The peritendonous and periligamentous tissue is most plantar in location, is thin, may be most vulnerable to injury from abnormal strain, and may be the first tissue in progression to be injured. Conformational abnormalities may predispose the horse to such injuries. The cause of such soft tissue injury in mature non-racehorses is unknown, and although swelling can develop with relatively mild lameness, more often lameness is moderate to severe.

Clinical examination reveals localized soft tissue swelling, often with heat and pain on palpation, with or without lameness. Previous application of liniments or blisters, or pin firing may create sore skin and considerable soft tissue swelling. Ultrasonographic findings depend on the duration of the injury. Acute lesions have an accumulation of anechoic fluid subcutaneously; in more chronic injuries, swelling is from subcutaneous echogenic material (Figs. 79-7 to 79-9). The SDFT, DDFT, and long plantar ligament should be inspected carefully, but they are frequently normal.

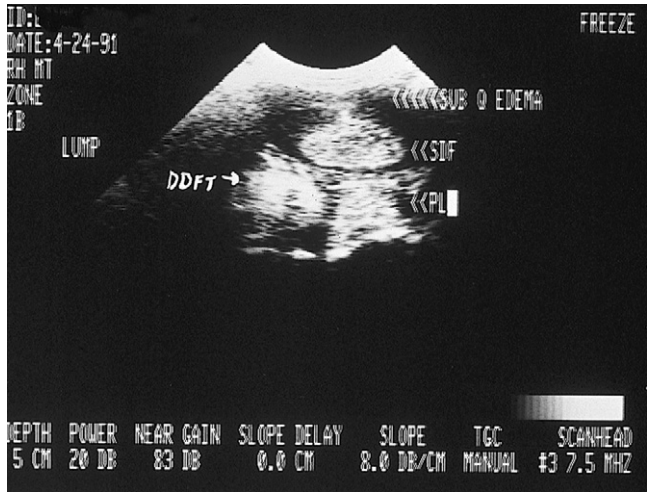

Fig. 79-7 Transverse midline ultrasonographic image (medial is to the left) of the plantar aspect of the hock of 2-year-old Standardbred gelding obtained in zone lB. There is subcutaneous edema *(arrows)* and thickening. The superficial digital flexor tendon *(SDFT)*, deep digital flexor tendon *(DDFT)*, and long plantar ligament *(PL)* were normal. This horse was managed using a peritendonous injection of corticosteroids.

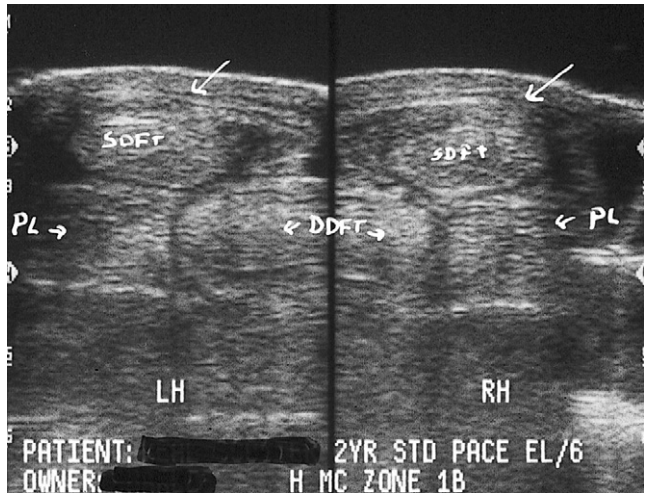

Fig. 79-8 Transverse midline ultrasonographic images of the left hindlimb (*left*) and right hindlimb taken in zone lB in 2-year-old Standardbred colt pacer with bilateral curb showing subcutaneous fibrosis *(arrows)* (*LH*, *RH*) and mild fluid accumulation (*RH*) typical of that seen in curb from peritendonous and periligamentous tissue injury.

Management depends on the degree of lameness, the stage of training, the race or competition schedule and the owner's or trainer's wishes. Blistering is used widely, but we question its value. Although not supported by scientific evidence, thermocautery (pin firing) appears to be an effective management tool, perhaps because it enforces rest. However, prolonged rest is rarely necessary in racehorses, and many horses can be managed by local injection of corticosteroids (triamcinolone acetonide, 9 mg) without significant interruption of training. More than one treatment may be required if the swelling and

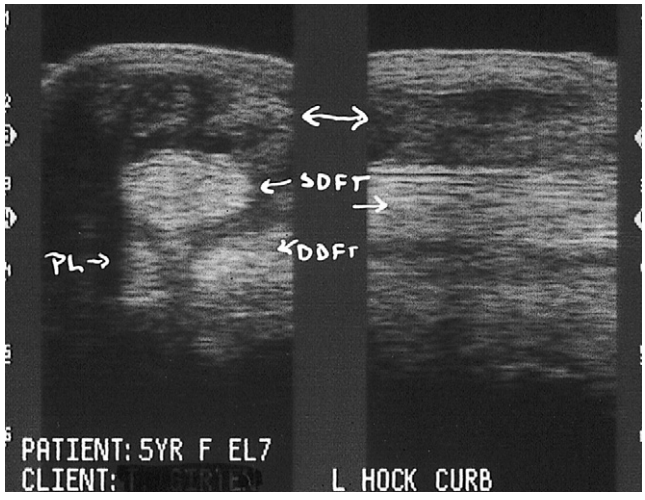

Fig. 79-9 Transverse (*left*) (medial is to the right) and longitudinal midline ultrasonographic images of the hock in zone lB in a 5-year-old Thoroughbred racehorse. There is soft tissue swelling of heterogeneous echogenicity of the peritendonous and periligamentous tissues and fibrous adhesion *(double-headed arrow)* plantaromedial to the superficial digital flexor tendon *(SDFT)*. The SDFT, deep digital flexor tendon *(DDFT)*, and long plantar ligament *(PL)* are normal.

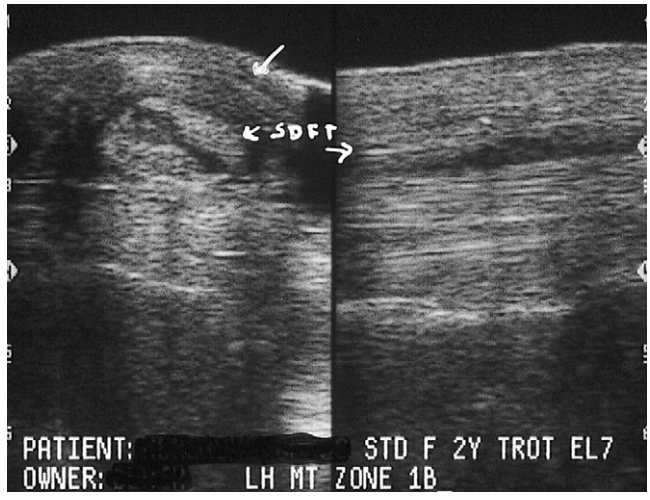

Fig. 79-10 Transverse (*left*) and longitudinal midline ultrasonographic images of the plantar aspect of the hock of 2-year-old Standardbred racehorse with curb resulting from peri-tendonous and peri-ligamentous tissue injury and superficial digital flexor tendonitis. Subcutaneous fibrous tissue accumulation *(top arrow)* plantar to the superficial digital flexor tendon *(SDFT)* and a central anechoic lesion of the SDFT can be seen, findings verified in the longitudinal view. Cross-sectional area of the affected superficial digital flexor tendon was 33% greater than the normal tendon.

lameness do not resolve. A lame horse must be rested to prevent injury to deeper structures. In non-racehorses with moderate to severe lameness, lameness may take several weeks to resolve. If the swelling becomes firm, fibrous, and pain free but lameness recurs, further investigation is warranted, because other causes of tarsal pain often develop in STB and TB racehorses with curb.

Infection can occur from direct trauma with skin penetration, previous injection, or severe topical counterirritation. If infection is suspected, cytological examination and culture are indicated. If a hematoma resulting from trauma is large or recurrent, or the curb is infected, establishing drainage is often necessary. A distal incision is made to provide drainage and fibrin and debris are removed. Care must be taken to avoid penetration of the tarsal sheath when creating the incision. A drain is inserted if necessary and the hock is bandaged. Culture usually reveals *Staphylococcus* species and appropriate antimicrobial and non-steroidal anti-inflammatory drug (NSAID) therapy is instituted. Horses are rested for 2 to 3 weeks to allow the tissues to heal, even though lameness from peritendonous and periligamentous tissue injury was not present before infection developed. Prognosis in horses with infection of peritendonous and periligamentous tissues is excellent but is far worse in those with infection of the SDFT, DDFT, or tarsal sheath.

Superficial Digital Flexor Tendonitis

A common finding in horses with curb is superficial digital flexor tendonitis. Sickle-hock conformation may predispose the horse to tendonitis, which can occur alone, but rarely is seen without concomitant inflammation of the peritendonous and periligamentous tissues. Pathogenesis likely involves progressive or accumulated overload injury of first the thin, fragile peritendonous and periligamentous tissues and later the SDFT.

Previous peritendonous and periligamentous tissue injury appears to predispose to subsequent superficial digital flexor tendonitis if the training level is increased quickly. Peritendonous and periligamentous injury may simply be an early stage of a progressive legion that eventually involves the SDFT or long plantar ligament. Progressive injury occurs frequently if horses with peritendonous and periligamentous tissues injury are treated with cryotherapy, internal blisters, or corticosteroids, and training intensity is accelerated before mature fibrous tissue can form. In horses with peritendonous and periligamentous tissue injury and superficial digital flexor tendonitis lameness varies, but it is much more likely to be observed than in horses with only peritendonous and periligamentous tissue injury. Lameness may be acute in onset and often is seen at fast speeds, but it can be seen in some horses at a trot in hand. Superficial digital flexor tendonitis occurs commonly in young STB racehorses but usually at a later stage of training than peritendonous and periligamentous tissue injury. Ultrasonographic examination reveals enlargement of the CSA of the SDFT, with a variable change in echogenicity and fiber pattern, depending on the severity of the injury (Fig. 79-10). Often subcutaneous edema or fibrosis occur, depending on the chronicity of the injury.

In horses with superficial digital flexor tendonitis, rest is an important part of management. Lesions usually are localized to the plantar tarsus, and those extending farther distally are associated with more severe lameness and horses have a poorer prognosis. Horses with localized lesions have a fair prognosis, although the prognosis is worse in trotters than in pacers. Horses with mild acute superficial digital flexor tendonitis, with enlargement of the tendon without fiber tearing, should be rested for 3 to 4 weeks. Without rest, progressive fiber damage may occur, resulting in prolonged recovery. Horses with more severe injuries may require up to 4 months of stall rest and controlled walking exercise.

In some STB racehorses, superficial digital flexor tendonitis in horses actively racing can be managed symptomatically, without giving rest, if the lesion is well localized and mild. Mild lameness may be observed at speed or while the horse is trotting in hand, but severe lameness should not be evident. Ultrasonographic examination often reveals peritendonous and periligamentous tissue injury with enlargement of the SDFT, but core lesions are not present. In most horses local

therapy using cold water hosing and poultice application, NSAID therapy, and subcutaneous injection of a corticosteroid preparation is successful. Subcutaneous injection of methylprednisolone acetate (200 mg) and Sarapin (25 ml) medial, plantar, and lateral to the SDFT is often done by practitioners with apparent success. Horses are given 10 to 14 days of jogging and light training before racing again. Non-racehorses with localized lesions usually respond well to rest for up to 3 months and have a favorable prognosis.

Horses with severe superficial digital flexor tendonitis have severe mushy swelling. If not given rest, these horses develop progressive tearing of the SDFT distal to the hock in the metatarsal region and lose support of the hock. Long-term rest (9 to 12 months) is recommended, but prognosis for return to previous race class is poor and in trotters, grave.

Deep Digital Flexor Tendonitis

Deep digital flexor tendonitis is a rare cause of curb. Horses with curb resulting from deep digital flexor tendonitis are usually acutely lame and have substantial swelling. Mixed injury with deep digital flexor tendonitis accompanying superficial digital flexor tendonitis and long plantar ligament desmitis occurs, but it is unusual. Horses with deep digital flexor tendonitis have concomitant peritendonous and periligamentous tissue inflammation and effusion of the tarsal sheath (tenosynovitis). The DDFT simply can be enlarged compared with the contralateral limb or have anechoic or hypoechoic core lesions. During ultrasonographic examination, the DDFT should be evaluated carefully from the midline and plantaromedial aspects. Lameness often is pronounced, and rest is recommended for a minimum of 4 to 6 months, but prognosis is guarded because lameness can recur. Serial ultrasonographic examination, corrective shoeing, and controlled exercise are given.

Long Plantar Desmitis

Long plantar ligament injury usually causes acute lameness, but chronic soft tissue swelling and progressive lameness can occur. Soft tissue swelling often is pronounced. Although long plantar desmitis can occur in racehorses, this form of curb appears to be equally common in other types of horses, such as Western performance horses. Long plantar desmitis can be well localized or diffuse. Cross-sectional measurements of the long plantar ligament are critical, because desmitis typically is often manifested as ligament enlargement rather than overt fiber tearing. Subtle thickening of the long plantar ligament may cause high-speed lameness, and evaluation of the cross-sectional area may be the only method to identify early lesions in these horses. Lesions can occur at any level within zones 1A and 1B, and injury may involve the insertion of the long plantar ligament on MtIV (Fig. 79-11).

Conservative management is best for horses with long plantar desmitis, because lameness and swelling often are pronounced. Owners and trainers of non-racehorses are often open to a conservative approach involving ample rest (3 months) to rehabilitate horses with curb properly. Intervening with therapy to enforce rest is not necessary. Controlled return to exercise is straightforward in this type of horse, because walking and trotting under saddle can be given easily. Graded exercise programs are not administered as easily or desired in the STB racehorse compared with non-racehorse sport horses. Lunging and walking and trotting under saddle are usually not practical,

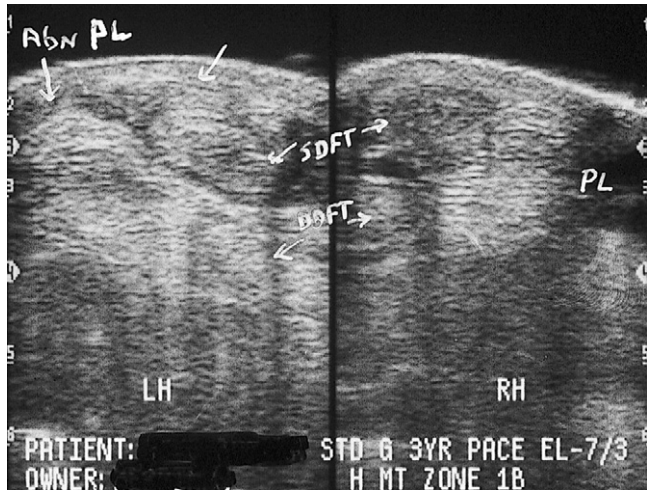

Fig. 79-11 Transverse plantarolateral ultrasonographic images of the left hindlimb *(LH)* (abnormal) and right hindlimb *(RH)* (normal) plantar tarsi in zone lB of 3-year-old Standardbred pacer. The left long plantar ligament *(PL)* is enlarged, with an indistinct central hypoechoic lesion. *SDFT,* Superficial digital flexor tendon; *DDFT,* deep digital flexor tendon.

although riding trotters is popular among trainers originally from Europe. Walking and light jogging in the jog cart is the best way to give graded exercise in the STB racehorse. We do not recommend turnout exercise in any horse with soft tissue injury, because we feel strongly this prolongs recovery and may lead to re-injury, but we realize our recommendations may not be followed. Cryotherapy, topical counterirritants, subcutaneous injections, and thermocautery are less likely to influence inflammation and healing of the long plantar ligament than more superficial causes of curb, but these treatments are sometimes requested.

Curb can result from long plantar desmitis at its insertion on MtIV. These horses do not have extensive swelling but focal thickening just proximal to MtIV. Mild soft tissue swelling must be differentiated from a prominent but normal MtIV seen in yearlings with sickle-hock conformation. Horses with curb resulting from distally located long plantar desmitis show lameness and mild, focal swelling and are managed with rest.

Mixed Soft Tissue Injuries

Ultrasonographic examination of curb nearly always identifies peritendonous and periligamentous tissue inflammation and in many horses an abnormality of the SDFT, DDFT, or the long plantar ligament. Occasionally, however, simultaneous injury of the SDFT and long plantar ligament occurs in addition to peritendonous and periligamentous tissue inflammation. This is most common in non-racehorse sport horses, in which lameness and swelling are severe.

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CHAPTER • 80

Bursae and Other Soft Tissue Swellings

Sue J. Dyson

A bursa is a flattened, closed sac interposed between structures subject to friction or at points of unusual pressure, such as bony prominences and tendons. Bursae are lined with a cellular membrane resembling synovium and are classified according to position (subcutaneous, subligamentous, submuscular, and subtendonous) and according to the method of formation (congenital or acquired).

Acquired bursae develop because of pressure and friction over bony prominences. Tearing of the subcutaneous tissues results in accumulation of transudative fluid, which becomes encapsulated by a fibrous tissue. In chronic injuries fibrous bands may develop within the capsule.

SUPRASPINOUS BURSA

The supraspinous bursa overlies the summits of the dorsal spinous process of the second to fifth thoracic vertebrae, under the funicular part of the nuchal ligament. Inflammation of the supraspinous bursa and surrounding soft tissues, so-called fistulous withers, is usually infectious in origin and may be a sequel to trauma. *Streptococcus* and *Staphylococcus* species, *Brucella abortus,* and *Onchocerca cervicalis* have been considered important causative agents.[1-3]

Clinical signs of supraspinous bursitis are generalized soft tissue swelling, heat and pain, and often draining tract(s). Osteitis or osteomyelitis of the dorsal spinous processes of the cranial thoracic vertebrae may be concurrent. Care must be taken not to misinterpret the normal granular radiopaque appearance of normal, incompletely ossified summits of the dorsal spinous processes.[4] Diagnostic ultrasonography and radiography are useful for determining the extent of the infection, for identifying a foreign body, and for evaluating signs of osteitis or osteomyelitis.

Treatment is by aggressive surgical debridement of all infected tissue and establishment of adequate drainage, taking care not to penetrate the dorsoscapular ligament. Several surgical procedures may be required to resolve the infection successfully.[1-3]

INTERTUBERCULAR (BICIPITAL) BURSA

The intertubercular (bicipital) bursa is discussed in Chapter 41.

HYGROMA

Hygroma is discussed in Chapters 39 and 68.

NAVICULAR BURSA

The navicular bursa is discussed in Chapters 24 and 30.

TROCHANTERIC BURSA

The Editors have no clinical experience of trochanteric bursitis. This condition is discussed further in Chapter 48.

CALCANEAL BURSA

The calcaneal bursa lies between the tendons of the gastrocnemius and the superficial digital flexor muscles, proximal to the hock, and extends distally on the plantar aspect of the calcaneus to the distal aspect of the hock (Fig. 80-1). In some horses a communication exists between the calcaneal bursa and the gastrocnemius bursa.

Injuries of the calcaneal bursa are not common and are usually the result of trauma. However, mild distention of the calcaneal bursa often is seen with gastrocnemius tendonitis (see page 709). Mild distention also may be seen unilaterally or bilaterally, as an incidental finding unassociated with lameness.[5] Primary inflammation of the bursa results in acute onset lameness associated with distention of the bursa. Hemorrhage into the bursa also may occur. Conservative management by rest, with or without injection of short-acting corticosteroids, usually results in resolution of lameness, although enlargement of the bursa may persist.

More commonly infection of the bursa is caused by a penetrating injury or is secondary to infectious osteitis of the calcaneus[6] (see Chapter 45).

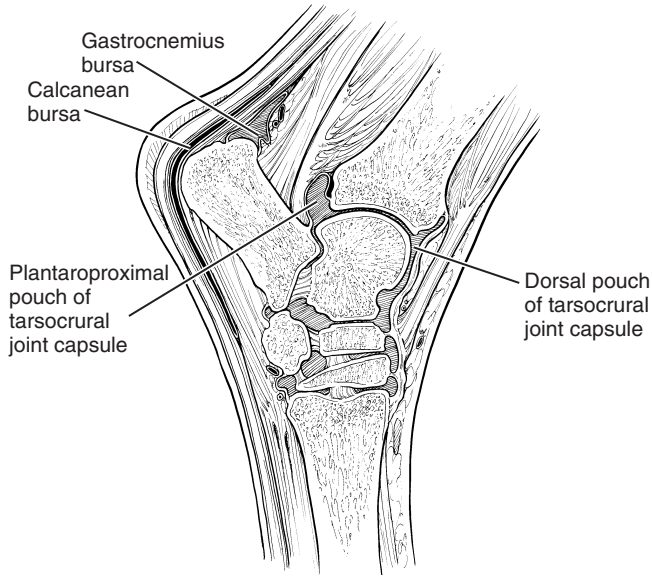

Fig. 80-1 Diagram of sagittal section of the hock region showing the relative positions of the calcaneal and gastrocnemius bursae.

Chronic distention of the calcaneal bursa also has been seen with well-circumscribed osteolytic lesions on the tuber calcanei, which were thought to represent enthesopathy at the insertion of gastrocnemius.[7]

Further investigation should include diagnostic ultrasonography, radiography of the calcaneus, and synoviocentesis. Radiographic examination should include a flexed skyline view of the calcaneus.[8] The internal structure of the bursa may be evaluated endoscopically,[9] but some underlying bony lesions may not be visible if the insertion of gastrocnemius is intact.[7] Endoscopy has been used diagnostically and therapeutically in horses with infectious osteitis, or bursitis, and in those with osteolytic lesions.

Horses with primary infection of the calcaneal bursa should be treated by debridement and lavage of the bursa and broad-spectrum antimicrobial therapy. Horses with infectious and non-infectious lesions of the calcaneus have been managed conservatively and surgically, with rather disappointing results.[6,7] A substantial number of treated horses have persistent lameness.

GASTROCNEMIUS BURSA

The gastrocnemius bursa lies between the superficial digital flexor tendon and the tuber calcanei. Mild distention may be present, unassociated with lameness. Primary injuries of the gastrocnemius bursa are rare. The bursa may be distended in association with gastrocnemius tendonitis, resulting in a capped hock appearance (see page 709). Occasionally, infection may occur because of a puncture wound or extension of infection from the calcaneal bursa.

CUNEAN BURSA

The cunean bursa lies underneath the cunean tendon on the medial aspect of the hock. Inadvertently penetrating the bursa is easy if one is inexperienced in injecting the centrodistal joint. Although the potential for primary bursitis exists, neither of the Editors of this text recognizes primary bursitis as a cause of lameness in racehorses or other sports horses. Gabel recognized a syndrome, "cunean tendonitis and bursitis-distal tarsitis syndrome of harness racehorses," but appreciated that horses showed substantially better improvement in gait after local analgesia of the distal hock joints than after infiltration of the cunean bursa alone.[10] Nonetheless, a component of peri-articular soft tissue pain may occur in Standardbred trotters and pacers with distal hock joint pain, and treatment of the cunean bursa with cortiscosteroids frequently is used as part of management. Cunean tenectomy is practiced by some, but it has largely fallen from favor. Subcutaneous injection of corticosteroids and Sarapin over the proximal aspect of the second metatarsal bone may yield better results than medication of the cunean bursa.[11]

CAPPED ELBOW

A capped elbow is an acquired bursa that develops over the olecranon of the ulna. The bursa results from repeated trauma from the heel of the shoe on the ipsilateral forelimb when the horse is lying down. The condition generally is not associated with lameness and is merely a cosmetic blemish. The use of a sausage boot around the pastern prevents trauma from the shoe, and usually the swelling diminishes significantly and rapidly in size. Chronic injuries have been treated by injection of corticosteroids, orgotein, or dysprosium-165, with disappointing results, or surgically, with better cosmetic results.[12]

CAPPED HOCK

A capped hock appearance may be caused by distention of the gastrocnemius bursa or by development of an acquired bursa over the tuber calcanei. An acquired bursa develops because of repetitive trauma, such as the horse kicking the stable walls or leaning backward on its hindlimbs when traveling. Capped hock usually has no associated lameness. Protection of the hocks with hock boots may help to prevent deterioration.

ACQUIRED BURSA ON THE DORSAL ASPECT OF A HIND FETLOCK

Firm swelling on the dorsal aspect of the hind fetlocks of horses that jump fixed fences is common. These lesions are false bursae that overlie the extensor tendon and are usually of no consequence, except cosmetically. However, a puncture wound may result in infection, resulting in enlargement of the bursa and surrounding soft tissue swelling, localized heat, and pain on palpation.[5] In contrast to infection in a joint or tendon sheath, lameness is usually only mild to moderate. Ultrasonography is useful to identify better the causes of the soft tissue swelling, and diagnosis is confirmed by synoviocentesis and identification of many nucleated cells. Surgical treatment is required and the prognosis is good.

FALSE THOROUGHPIN

A thoroughpin is the colloquial name for distention of the tarsal sheath, but more commonly the term is misused to describe a variety of swellings that may develop in the distal crus cranial to the gastrocnemius tendon. These conditions are otherwise called *false thoroughpins* and should be differentiated from distention of the tarsal sheath, tarsocrural joint capsule (see Chapter 45), or calcaneal bursa (see page 705). A false thoroughpin occurs laterally more commonly than medially and may develop unilaterally or bilaterally. A false thoroughpin varies in size from small to large. In contrast to distention of the tarsal sheath or the plantar outpouching of the tarsocrural joint capsule, these swellings cannot be balloted from laterally to medially and do not extend distal to the hock (Fig. 80-2). They may be sudden or insidious in onset and may or may not be associated with lameness.

The causes vary and are poorly understood. False thoroughpins are usually solitary, fluid-filled sacs (Fig. 80-3), unilocular or multilocular, with a wall of variable thickness, with or without large echogenic fibrous bands traversing them (Fig. 80-4). They may develop secondary to local hemorrhage or because of herniation of the tarsal sheath or the calcaneal or gastrocnemius bursae.[13-15]

Diagnostic ultrasonography is useful to identify the nature and extent of the swelling (Fig. 80-4). Unlike the tarsal sheath, no tendon is within the cavity. Positive-contrast radiography can be used to demonstrate whether any communication exists between adjacent structures (Fig. 80-3).

A false thoroughpin may be an incidental clinical finding unassociated with lameness. They are seen commonly in horses with a base-narrow hindlimb conformation.[5] Other causes should be excluded before one concludes that a false thoroughpin is the cause of lameness. Even if the swelling is acute in onset, in the absence of lameness I have maintained horses in work with no deleterious effects. Sometimes such swellings spontaneously reduce in size, but some swelling is likely to persist.

Long-term lameness associated with a false thoroughpin has been seen in a number of horses with chronic hindlimb

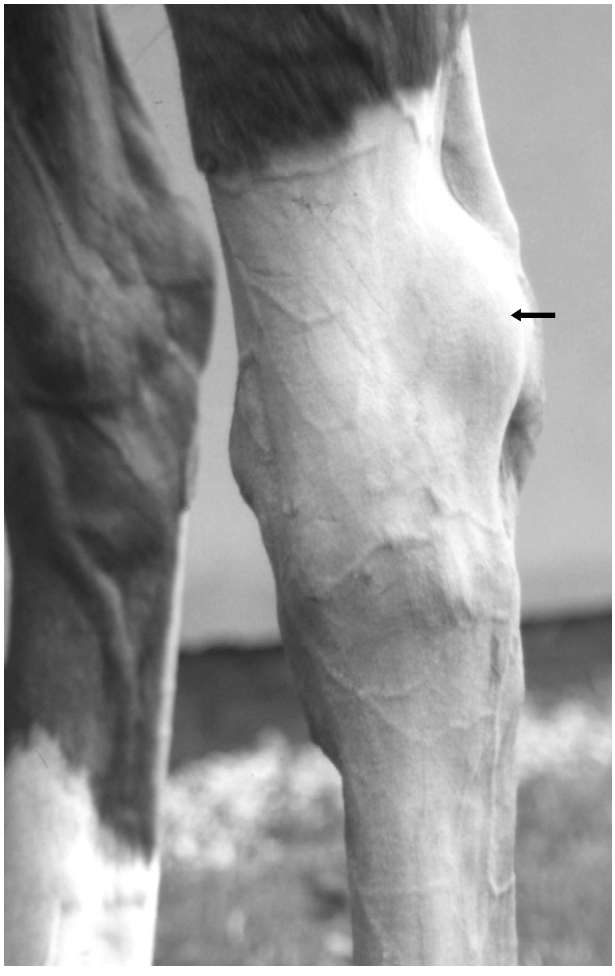

Fig. 80-2 A false thoroughpin (*arrow*). The swelling in this horse was acute in onset, but it was unassociated with lameness. The horse was maintained in full work, and despite this the swelling reduced in size. Ultrasonographic evaluation revealed a unilocular fluid-filled cavity.

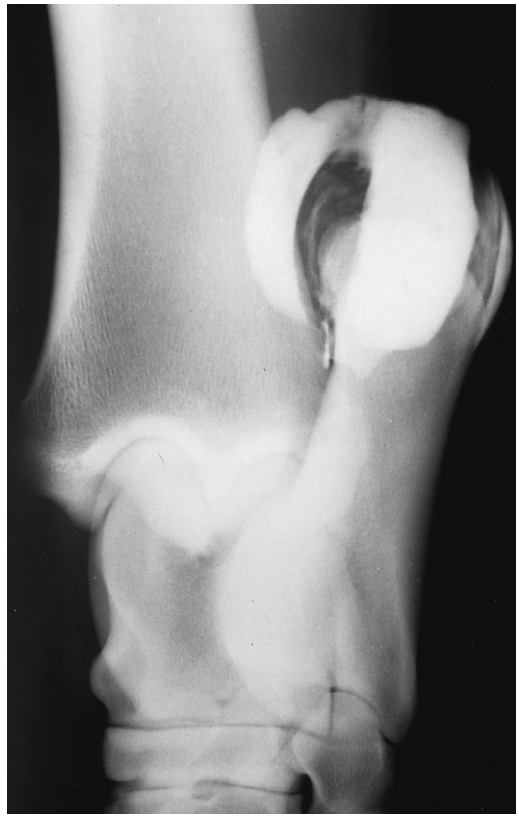

Fig. 80-3 Dorsolateral-plantaromedial oblique radiographic view of a hock. A positive-contrast radiographic study of false thoroughpin. This fluid-filled cavity did not communicate with the tarsal sheath.

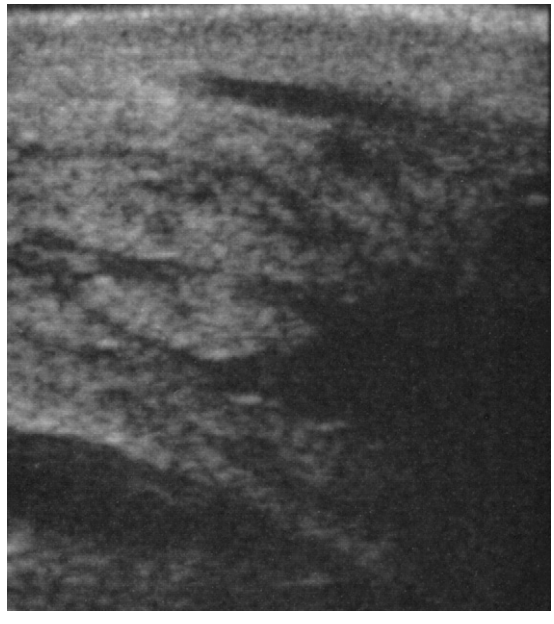

Fig. 80-4 Longitudinal ultrasonographic image of false thoroughpin. Proximal is left. There is a thick-walled fluid-filled cavity with some echogenic bands.

lameness that has not responded to conservative management. Surgical excision of large multiloculated cyst-like lesions has resolved lameness successfully in some horses,[5] although cosmetic results may be disappointing.

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CHAPTER • 81

Other Soft Tissue Injuries

Sue J. Dyson

RUPTURE OF FIBULARIS (PERONEUS) TERTIUS

Anatomy

Fibularis (peroneus) tertius is an entirely tendonous muscle that lies in the craniolateral muscle group of the crus, between the subcutaneous long digital extensor and the cranialis tibialis, which cover the craniolateral aspect of the tibia. The muscle originates from the extensor fossa of the femur. Distally the fibularis tertius divides into branches that enfold the tendon of insertion of tibialis cranialis and insert on the dorsoproximal aspect of the third metatarsal bone, the calcaneus, and the third and fourth tarsal bones. The tendon is an important part of the reciprocal apparatus of the hindlimb, which coordinates flexion of the stifle and hock.

Fibularis tertius is the most echogenic structure on the craniolateral aspect of the crus and is identified readily by ultrasonography as a well-demarcated hyperechoic structure relative to the surrounding muscles (Fig. 81-1).

History and Clinical Signs

Rupture of fibularis tertius invariably is caused by trauma resulting in hyperextension of the limb; for example, a horse trying to jump out of a stable and getting one hindlimb caught on the top of the stable door. This usually results in rupture of the tendon in the middle of the crus but occasionally farther distally. Alternatively, rupture may be caused by a laceration on the dorsal aspect of the tarsus, resulting in transection of the tendon. Occasionally, partial tearing of the tendon occurs, usually at the level of the tarsocurual joint, with prominent swelling. Occasionally the reciprocal apparatus is partially but not totally disrupted. Avulsion injuries of the origin of the tendon rarely occur in young foals.

The clinical signs are pathognomonic, because rupture of this tendon allows the hock to extend while the stifle is flexed. When standing at rest, the horse may appear clinically normal, although with acute injury careful palpation may reveal some muscle swelling on the craniolateral aspect of the crus or further distally. When the horse walks, it should be viewed carefully from behind and from the side. The hock may extend more than usual. The tendons of gastrocnemius and the superficial digital flexor tendon may appear unusually flaccid, and a dimple is seen on the caudal aspect of the crus about one hand's breadth proximal to the tuber calcanei. At the trot the horse appears severely lame, with apparent delayed protraction of the limb because of over-extension of the hock.

If the limb is picked up and pulled backward, the hock can be extended gradually and "clunks" into complete extension while the stifle remains flexed. A characteristic dimple appears in the contour of the caudal distal aspect of the crus (Fig. 81-2). If rupture is only partial, or if lameness is chronic and some repair has taken place, clinical signs may be less severe and the diagnosis less obvious.

Presumably, strain of this tendon can occur, resulting in lameness, but I have no experience of this, and to my knowledge this condition has not been documented.

Diagnosis

The diagnosis of rupture of fibularis tertius is based on the pathognomonic clinical signs. The site of rupture can be iden-

lameness that has not responded to conservative management. Surgical excision of large multiloculated cyst-like lesions has resolved lameness successfully in some horses,[5] although cosmetic results may be disappointing.

REFERENCES

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Diagnosis

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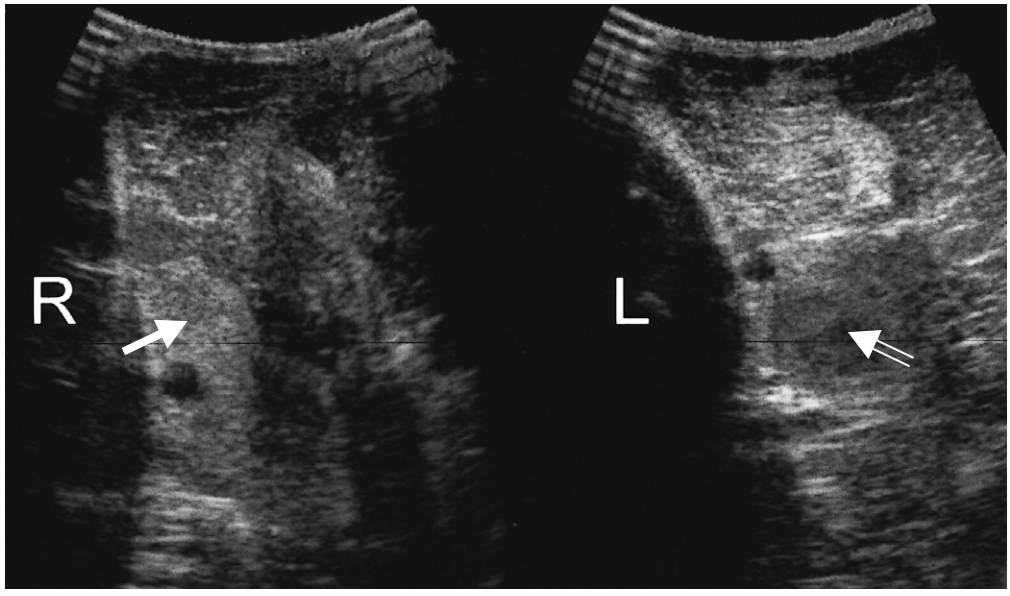

Fig. 81-1 Transverse ultrasonographic images of the craniolateral aspect of the mid-crus of 7-year-old horse with left hindlimb lameness of 2 months' duration. Fibularis tertius is the most echogenic structure in the center of the image of the right (*R*) hindlimb *(solid arrow)*. Compare this with the image of the left (*L*) hindlimb in which fibularis tertius is markedly hypoechoic *(open arrow)*. Note also that the overlying muscle is increased in echogenicity compared with the right hindlimb.

Fig. 81-2 A horse with rupture of the fibularis tertius. The hock can be extended while the stifle is flexed. Note also the characteristic dimple in the contour of the caudodistal aspect of the crus. Clinical signs developed after the horse had attempted to jump out of its stable and had got hung up on the door. The horse made a complete recovery.

tified with ultrasonography (Fig. 81-1). The normally echogenic structure is not clearly identifiable and may be replaced by a region hypoechoic relative to the surrounding muscles. In chronic injuries the surrounding muscles may become hypertrophied. Usually no associated radiological abnormalities are apparent in adult horses, although avulsion fracture of the origin has been described in foals.

Treatment

Confinement to box rest for 3 months, followed by a slow resumption of work usually results in total resolution of clinical signs. Most horses are able to return to full athletic function without recurrence of clinical signs. However, delayed recognition of the clinical signs and failure to confine the horse may result in a chronic lesion, which fails to heal satisfactorily. However, compensatory hypertrophy and or fibrosis of surrounding muscles may permit functional recovery.[1]

COMMON CALCANEAL TENDONITIS

The common calcaneal tendon comprises components from the superficial digital flexor and gastrocnemius tendons and from biceps femoris, soleus, semimembranosus, and semitendinosus muscles. Contributions from the latter two muscles are called the axial and medial tarsal tendons. So-called common calcaneal tendonitis has been described as resulting from a kick in the hock region.[2] Unfortunately the horse was not examined with ultrasonography until 5 months after injury, at which time soft tissue swelling was marked. Ultrasonographic examination revealed that the superficial digital flexor and gastrocnemius tendons appeared normal, but the axial and medial tarsal tendons were enlarged. The horse made a complete functional recovery.

GASTROCNEMIUS TENDONITIS

Tendonitis of gastrocnemius is a relatively unusual cause of hindlimb lameness in the horse.[3-5]

Anatomy

The gastrocnemius muscle arises from two heads that terminate in the mid-crus in a common tendon. Proximally the tendon lies caudal to the superficial digital flexor tendon (SDFT); farther distally the tendon lies laterally and is ultimately cranial, inserting on the tuber calcanei. The SDFT and gastrocnemius tendons are separated by a bursa, the calcaneal bursa, that extends to the mid-tarsal region. A small bursa, the gastrocnemius bursa, also lies cranial to the insertion of the gastrocnemius tendon on the tuber calcanei. A communication may exist between these bursae.

The tendon of gastrocnemius may still contain some muscular tissue as far distally as the level at which it lies lateral to the SDFT. This results in hypoechoic regions within the tendon.

Gastrocnemius tendonitis usually occurs distally, distal to the musculotendonous junction. Rarely, damage occurs at the musculotendonous junction.[6] Occasionally, injuries occur at the origin of the gastrocnemius on the femur.[7]

History and Clinical Signs

Lameness may be acute or gradual in onset and varies from mild to severe. Distention of the calcaneal bursa frequently is associated with gastrocnemius tendonitis, and the horse often develops a capped-hock appearance also because of distention of the gastrocnemius bursa (Fig. 81-3). However, these swellings can occur without lameness or detectable pathological conditions of the gastrocnemius or SDFT (see page 706). Mild enlargement of the gastrocnemius tendon may occur, but this can be difficult to appreciate. Eliciting pain by palpation usually is not possible.

Severe lameness is characterized by a reduced height of arc of foot flight, shortened cranial phase of the stride, and a tendency to hop off the caudal phase of the stride. Horses with less severe lameness have no specific gait characteristics. Lameness often is accentuated by proximal or distal limb flexion. Two of four horses with injury to the origin of the gastrocnemius muscle had an unusual gait characterized by internal rotation of the affected limb (outward movement of the calcaneus).[7]

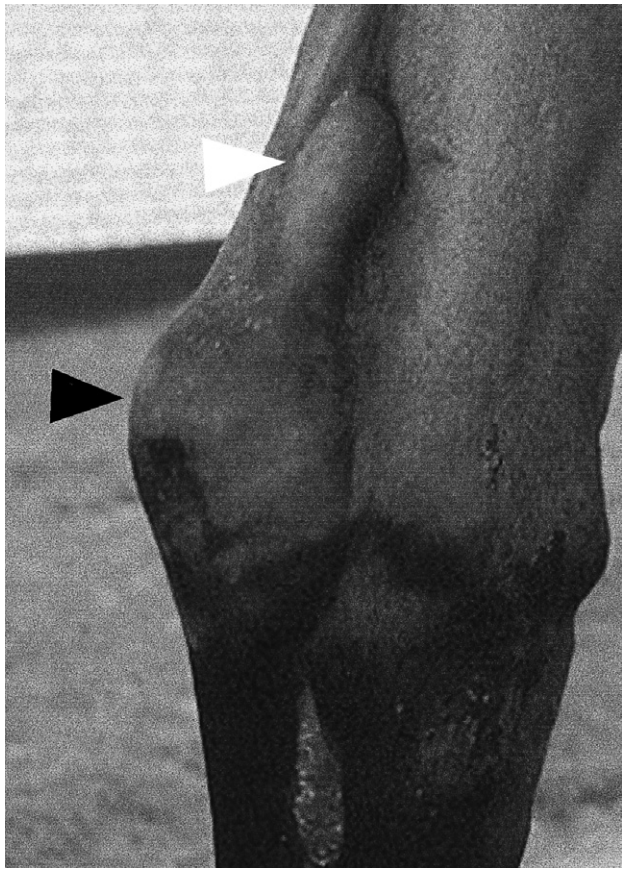

Fig. 81-3 Medial view of the left hock of 7-year-old Thoroughbred with acute-onset lameness associated with gastrocnemius tendonitis. Note the capped-hock appearance (*black arrowhead*) and the distention of the calcaneal bursa (*white arrowhead*).

Diagnosis

Lameness is improved substantially by perineural analgesia of the tibial nerve, possibly because of local diffusion of the local anesthetic solution, unless the injury is at the origin. Diagnosis is confirmed by ultrasonographic examination. Comparison with the contralateral limb is useful.

The tendon usually is damaged in the distal aspect of the crus, where it lies cranial to the SDFT. Ultrasonographic abnormalities include enlargement of the tendon, poor definition of the margins, and focal or diffuse hypoechoic or anechoic regions (Fig. 81-4). Usually no detectable radiographic abnormalities are apparent. Radiographic (proliferative changes) and scintigraphic (increased radiopharmaceutical uptake) abnormalities may exist in horses with chronic injury at the origin of the gastrocnemius muscle on the caudal femur.[7]

Treatment and Prognosis

Conservative treatment with box rest and controlled exercise for up to 12 months generally has resulted in progressive improvement in lameness and improvement in the ultrasonographic appearance of the tendon. Horses with mild lesions have been able to return to full athletic function without recurrent lameness, but more severe lesions have a more guarded prognosis.[4,5] Three of four horses with injury of the origin returned to athletic use, but recurrent injury occurred in the fourth horse.[7]

SUBLUXATION AND LUXATION OF THE SUPERFICIAL DIGITAL FLEXOR TENDON FROM THE TUBER CALCANEI

Lateral (see Fig. 6-29), or less commonly medial, luxation or subluxation of the SDFT from the point of hock may occur along with damage to or rupture of the tendonous bands that insert medially and laterally on the tuber calcanei. Although usually a unilateral injury, the condition can occur bilaterally.

Lateral displacement of the SDFT occasionally occurs secondarily to hyperextension of the hind fetlock associated with

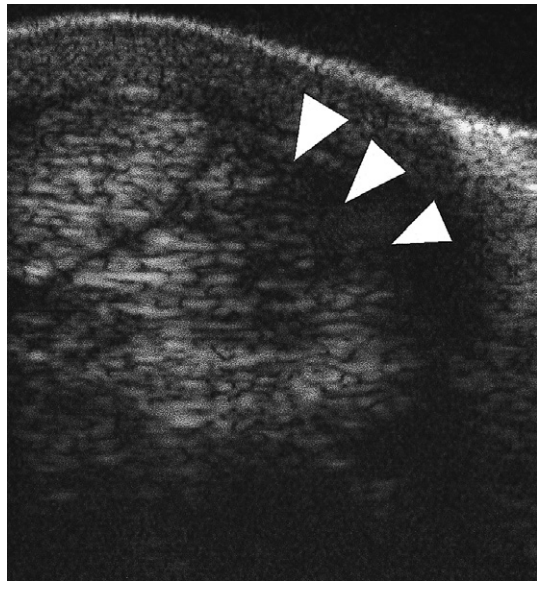

Fig. 81-4 Transverse ultrasonographic image of the caudal aspect of the distal crus of 6-year-old mare with left hindlimb lameness that was alleviated by perineural analgesia of the tibial nerve. Medial is to the left. The gastrocnemius tendon is enlarged, its plantar lateral aspect is poorly defined (*arrowheads*), and there is a large hypoechoic region consistent with gastrocnemius tendonitis.

progressive breakdown of the suspensory apparatus (see Fig. 73-24 and page 671).

Anatomy

The SDFT lies caudally in the distal crus and broadens to form a cap over the tuber calcanei. At this level, broad tendonous bands extend medially and laterally to insert on the tuber calcanei. The calcaneal bursa is interposed between the SDFT and the tendon of gastrocnemius.

History and Clinical Signs

Partial or complete disruption of one of the retinacular bands that attach the SDFT to the tuber calcanei can result in subluxation, or more commonly, luxation of the SDFT laterally or medially. Lameness is usually sudden in onset and severe, although occasionally mild lameness precedes this, associated with soft tissue swelling in the region of the point of hock. Frequently no history of trauma is apparent, and the injury often occurs as the horse is being worked. The horse may suddenly stop and may become extremely distressed, especially if the tendon repeatedly moves on and off the tuber calcanei. The horse may kick out repeatedly with the limb. The tendon may return to its normal position when the horse bears weight. Soft tissue swelling rapidly ensues, making accurate palpation difficult. If the horse is kicking repeatedly when moving, one may conclude wrongly that the soft tissue swelling developed as the result of trauma caused by kicking.

With subluxation of the SDFT, the tendon is usually positioned normally at rest. Careful observation of the tendon as the horse moves may reveal instability. With luxation it may be possible to see that the SDFT has been displaced laterally or, less commonly, medially. If the tendon remains luxated, then the horse tends to be less agitated, although obviously in pain in the acute stage. Careful palpation may reveal instability of the tendon or its displacement to an abnormal position.

In horses in which lateral displacement occurs secondary to hyperextension of the fetlock, the condition may be insidious in onset and slowly progressive and unassociated with acute lameness.[8]

Diagnosis

Ultrasonographic examination is helpful if the tendon is displaced by confirming its abnormal position, but such examination can add to confusion if the tendon is in the normal position when the horse stands still.

Treatment

In the acute stage pain relief is essential and tranquilization may be necessary to calm the horse. If the SDFT is unstable and is moving constantly on and off the tuber calcanei, management in the acute and chronic phases may be difficult. If the tendon is permanently dislocated laterally or medially, the distress usually resolves rapidly. Anti-inflammatory drugs are best avoided, because the surrounding soft tissue swelling helps stabilize the tendon. If the tendon has luxated laterally, prolonged rest (6 months) usually results in resolution of pain, although a mechanical lameness may persist. This limits the horse's function for dressage, but these horses may be able to race or show jump at a high level. The prognosis associated with medial luxation is more guarded and tends to be associated with a greater degree of mechanical lameness.

If the SDFT is unstable initially, the tendon may with time and progressive further disruption of the attaching retinacular bands become more stable in a luxated position. Peritendonous injection of a sclerosing agent, P2G (Martindale Pharmaceuticals, Romford, Essex, England), has been helpful in stabilizing the luxated tendon in a limited number of horses.[8] Surgical transection of a partially torn band has helped in chronic subluxation.[9] Attempts at surgical stabilization of the SDFT in its normal position often have been disappointing, although successful results have been reported.[10,11] Surgical stabilization is only worth considering if the horse is temperamentally suited to a full-limb cast. Prognosis is influenced by the ease of reconstruction of the torn retinaculum, which depends on the site of the tear (close to the tendon, close to the bone, or mid-way) and its age. Only four of nine horses were sound.[12]

BICEPS BRACHII TENDONITIS

Biceps brachii tendonitis is discussed in Chapter 41.

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CHAPTER • 82

Tendon Lacerations

Sue J. Dyson and Alicia L. Bertone

Tendon lacerations are serious injuries in horses because of the loss of the biomechanical function of the tendon, the slow return of tendon strength, the immediate strenuous loading demanded by the equine patient, and the complications of scarring. Nonetheless, early diagnosis, wound management, limb support, and long-term surgical and medical management have resulted in a good prognosis for most extensor tendon lacerations and a fair prognosis for most flexor tendon lacerations.[1,2] The extensor (dorsal) aspect of the limb is often damaged by wire or a sharp object over which the horse has jumped. The flexor (palmar or plantar) aspect of the limb may be traumatized by circumferential wire injuries, landing on a sharp object, or being struck. The latter may be self-inflicted or from another horse.

DIAGNOSIS

Gross Appearance of the Wound

Any laceration over the dorsal or palmar/plantar surface of the limb distal to the stifle or elbow, especially across the dorsal tarsus, distal dorsal tarsus, dorsal metatarsal region, distal radius, dorsal metacarpal region, and dorsal fetlock region may involve a tendon (Fig. 82-1). Extensor tendons and the superficial digital flexor tendon (SDFT) are positioned directly under the skin; therefore minor-appearing wounds can transect these tendons completely. Direct visual inspection may reveal transected tendon fibers protruding from the wound. However, injuries sustained at the gallop may result in a skin wound removed from the site of tendon damage, because of the movement of the skin during exercise. The position of the wound relative to synovial structures should be evaluated with care, because concurrent synovial contamination or sepsis reduces the prognosis and necessitates specific emergency treatment.

Evaluation of Gait

Each tendon serves a biomechanical function. Complete severance of a tendon results in a posture or gait change, which may be pathognomonic for disruption of the tendon integrity.

Extensor Tendons

Transection of an extensor tendon below the carpus produces a reduced ability to extend the digit, which is detected as an exaggerated, rapid (uncontrolled) dorsal flip of the hoof at the walk. This subtle change is easiest to detect if the lateral and common (or long) digital extensor tendons are transected completely. Intermittently the horse knuckles at the fetlock joint and places the digit on the dorsal surface of the pastern and fetlock joint. The gait abnormality is more obvious in the hindlimb and with lacerations in close proximity to the fetlock. Remaining peritendonous fascial attachments provide some support in the more proximal injuries. Horses with extensor tendon lacerations fully bear weight in a normal posture, unless other aspects of the wound create lameness and pain.

Transection of extensor tendons proximal to the carpus and at, or just proximal to, the tarsus also commonly occurs. Proximal to the carpus, transection of the extensor carpi radialis and common digital extensor tendons is most frequent. Flexion of the carpus may cause pain. The tendon sheath often is often involved.

Proximal or dorsal to the tarsus, transection of the long digital extensor, cranial tibialis, and fibularis (peroneus) tertius tendons is most frequent. If the fibularis tertius is disrupted, the hock can be extended while the stifle is flexed, indicating loss of the reciprocal apparatus. The gastrocnemius tendon develops a characteristic wrinkle in this extended position (see Fig. 81-2 and page 708). The degree of gait abnormality may be mild. Transection of all the extensors over the tarsus still allows full weight bearing with the foot flat on the ground. A greater tarsal extension during the swing phase of the stride and intermittent knuckling of the digit can be detected.

Flexor Tendons

Transection of flexor tendons below the carpus or tarsus produces pain on weight bearing and therefore lameness and gait abnormality. Transection of the SDFT is most common, because it has the most superficial position of the two flexor tendons. The suspensory ligament (SL) is deep to the deep digital flexor tendon (DDFT) and is therefore least commonly injured with lacerations. A horse with complete transection of the SDFT may stand normally, or it may bear weight on the toe of the hoof to minimize movement of the tendon ends with fetlock joint extension. Administration of phenylbutazone for pain may eliminate lameness, and a gait abnormality may become hard to detect. The DDFT and SL support the fetlock joint together with the SDFT. Therefore slight hyperextension of the fetlock because of disruption of the SDFT may be difficult to detect unless the contralateral limb is picked up. The greater the number of structures transected, the less support to the fetlock and other distal joints and the greater the likelihood of vessel and nerve transection. Elevation of the toe is pathognomonic for transection of the DDFT.

Digital Palpation of the Wound

Digital palpation is a simple and direct way to determine the extent of damage to structures below the skin. Integrity of the tendons is readily determined by feel. Partial tears can be distinguished from complete tears, and this affects treatment (see "Partial Tendon Lacerations"). The tendon ends are often palpable beneath the skin proximal and distal to the wound, but they may be removed from the wound if the injury was sustained while the horse was galloping. The muscular attachment to the proximal end pulls the proximal tendon end farther from the wound. The wound should be shaved around the edges and cleansed thoroughly with a dilute antiseptic solution such as chlorhexidine before exploration. Gross debris can be debrided manually from the wound. Sterile gloves should be worn for the digital exploration after the wound is clean. Digital palpation may reveal involvement of a tendon sheath or joint capsule; however, small tears of these synovial structures may not be palpable. Sterile preparation of the skin and injection of a balanced electrolyte solution into the synovial structure in question at a site distant from the

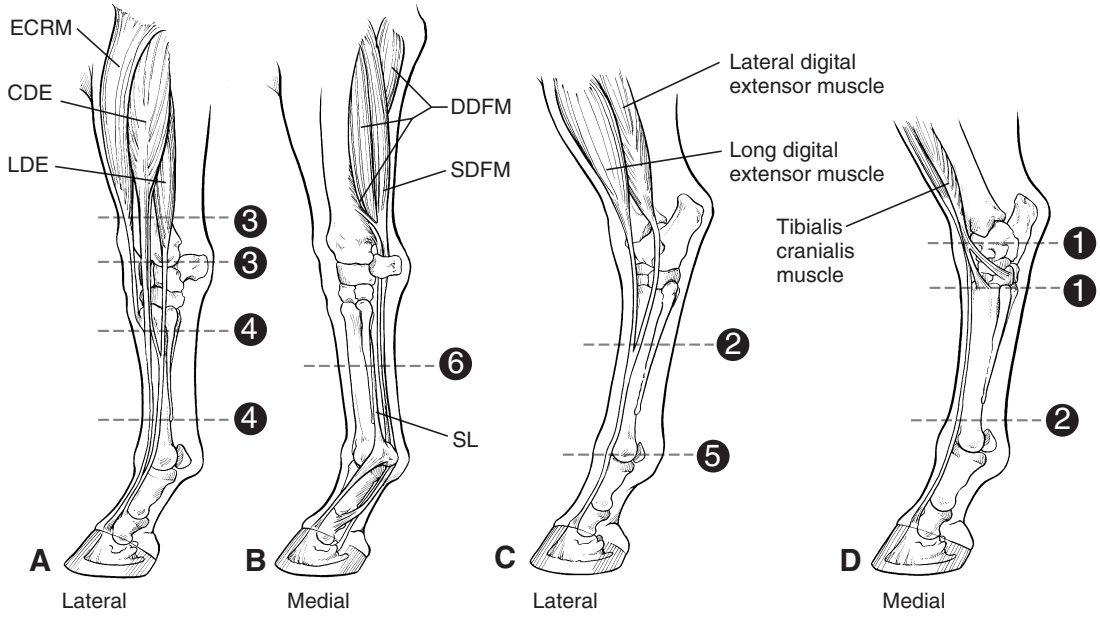

Fig. 82-1 Diagram illustrating common sites of tendon injury. **A,** Lateral forelimb. **B,** Medial forelimb. **C,** Lateral hindlimb. **D,** Medial hindlimb. *1*, Dorsal tarsus; *2*, dorsal metatarsal region; *3*, distal radius; *4*, dorsal metacarpal region; *5*, dorsal fetlock region; *6*, palmar metacarpal region; *ECRM*, Extensor carpi radialis muscle; *CDE*, common digital extensor muscle; *LDE*, lateral digital extensor muscle; *DDFM*, deep digital flexor muscle; *SDFM*, superficial digital flexor muscle; *SL*, suspensory ligament. (Adapted from Bertone AL: Tendon laceration. In Tendon and ligament injuries. Part II, *Vet Clin North Am Equine Pract* 11:293, 1995.)

wound may demonstrate communication if solution exits the wound. Synovial fluid also can be obtained and submitted for cytological evaluation. Hemorrhage or inflammation is usually present in the synovial fluid if the sheath has been penetrated. Mild inflammatory changes can be seen in synovial structures adjacent to tendon injuries, even if no actual communication with the wound occurs.

Ultrasonographic Evaluation

Ultrasonographic evaluation of the tendons can quantitate the degree of tendon damage, particularly in horses with partial tears. Ultrasonographic examination is not necessary for diagnosing complete tears and therefore is performed rarely.

Ultrasonographic evaluation can be useful during the healing phases of horses with partial or complete tendon lacerations.[3] The amount of repair tissue should increase early in repair and then decrease as the tissue matures and gains strength. In flexor tendons, about 6 weeks are required for the tendon to gain the strength to support 450 kg, and the strength of the repair tissue is largely because of an increase in tissue mass. The repair tissue cross-sectional area is greater than the original tendon area, but the strength of that tissue per unit area is reduced.[4] The quality of the repair tissue can be monitored with ultrasonography. Ultrasonographic examinations should show progressively increased homogeneity of echogenicity, reduction in the hypoechoic areas of damaged tendon or early immature repair tissue, and appearance of parallel arrangements of fibers.

EMERGENCY MANAGEMENT

Treatment of Shock

Trauma is often severe in horses with lacerated tendons, particularly flexor tendons. Some horses may be trapped for hours in wire or entangled in equipment. Blood loss may be extensive if major arteries to the distal limbs have been transected. The loss of the function of a limb is painful and stressful. Initial management of these horses can be lifesaving. If possible, the horse should be caught and brought into a warm, clean area for examination and further treatment. If the horse has severe tachycardia (>100 beats/min), with pale mucous membranes and is reluctant to move, initial treatment should be performed on site. If the wound is still bleeding, a clean pressure bandage should be applied to stop the bleeding and provide some support to the limb. If the function of the limb is impaired mechanically, a splint should be applied over the bandage to minimize further damage with movement. Use of tranquilizers and sedatives should be kept to a minimum until the degree of blood loss and shock can be assessed and treated. Most sedatives are peripheral vasodilators and may produce significant hypotension in a hypovolemic patient. Some horses may be in stress-induced shock from pain, with extreme catecholamine release. The effect of tranquilizers may be unpredictable and potentially worsen the bleeding. Securing the horse in a familiar, warm, clean environment and stabilizing the injured limb may resolve the stress-related shock and allow assessment of hemorrhagic shock by evaluating peripheral pulse strength and quality, heart rate, and mucous membrane color. If hemorrhagic shock is severe, the most important treatment is intravenous fluid volume replacement, which can be in the form of high-volume isotonic fluids (20 to 60 L minimum per 450 kg of horse), or hypertonic saline (9% NaCl; 1 L per 450 kg of horse), followed by isotonic fluid replacement. Hypertonic fluid therapy can be effective for rapid expansion of the vascular space in hemorrhagic shock, but hypertonic solutions dehydrate the interstitium and induce a profound renal diuresis. Therefore it is critical that isotonic fluid therapy begins within 30 minutes to 1 hour after hypertonic fluid administration. Hypertonic fluid therapy is practical because of the convenience of the small volumes necessary and works well if the horse is referred or transported to a facility that has access for fluid administration.

Transportation

For transportation the injured limb should be placed toward the back of the trailer. The horse's weight shifts to the front of the trailer during braking, which is often less controlled than acceleration. The horse's head should not be tied tightly, so the head and neck can be used for balance. The limb should be supported with a padded pressure bandage and a splint for transport.

MEDICAL MANAGEMENT

All horses with tendon lacerations need medical therapy, whether surgery to re-appose tendon ends is elected or not. If tendon laceration, partial or complete, is diagnosed, a more thorough aseptic preparation of the wound should be performed. These procedures require sedation, restraint of the patient, and local or regional analgesia.

Wound Cleansing and Debridement

The hair should be clipped circumferentially around the limb from above the wound (to the estimated top of the bandage) and the entire limb distal to the wound. Drainage of serum and exudate from the wound is often voluminous, and removal of the hair makes subsequent cleaning of the limb easier and more thorough, thereby minimizing bacterial growth and contamination. A 10-minute scrub of the wound with an antiseptic solution should be performed. If bone and tendon are exposed, care must be taken to minimize trauma to these tissues. A minor sterile instrument pack may be helpful in trimming heavily contaminated tissues, macerated tendon ends and removing hair and debris from deep in the wound. Lacerated tendons should be trimmed at the edges to remove traumatized tissue that is expected to become necrotic. Debridement of tendon ends should be most conservative in horses with flexor tendon lacerations, for which apposition of tendon ends with suture is recommended.

Systemic and Local Medications

Tetanus toxoid should be administered to any horse with a tendon laceration, and tetanus antitoxin if no vaccination history exists. Because all wounds are contaminated at injury and compound wounds have extensive soft tissue injury, broad-spectrum antimicrobial drugs should be administered systemically for a minimum of 3 days. Metronidazole should be considered in horses with grossly contaminated distal extremity wounds. The duration of antimicrobial therapy may be longer in horses with heavily contaminated wounds, wounds healing by second intention, infected wounds, wounds involving a tendon sheath, or wounds with delayed treatment (>24 hours). Wound lavage should be copious, usually with a minimum of 5 L of a balanced electrolyte solution.

SURGICAL TREATMENT

Surgery in the form of wound closure is performed in most horses with compound wounds involving tendons. Primary closure is preferred, if possible, to provide the best success of obtaining primary wound healing, minimal scar formation, and the fewest complications associated with the transected tendon. However, wounds that are heavily contaminated, older than 24 hours, or heavily traumatized should have a delayed closure (1- to 3-day delay) performed to reduce the contamination and necrotic debris before closure. The decision to close a wound older than 24 hours must be made based on the condition of the wound and surrounding structures. Wounds in horses that have been managed appropriately from the time of injury until surgery can be closed at any time, if tissue loss is minimal and infection is not present.

Extensor Tendons

Transected extensor tendons heal well without primary suturing of the tendon, even if a large gap has formed between the tendon ends. Serial ultrasonographic evaluations show fibrosis occurring between the tendon ends that eventually becomes more organized and regains the linear arrangement of collagen along the pattern of the original tendon. This fibrous tissue seems to provide a mechanical link between the tendon ends, because extensor function of the digit returns in most horses. In our experience, a palpable thinning of the new tendon and an enlargement at the old tendon ends usually remains, even after 1 year.

Horses with lacerated extensor tendons have a good prognosis, with 73% of injured horses returning to athletic soundness and 18% to pasture soundness.[2] In that study, 62% of the affected limbs were treated with a 3-layer cotton bandage, 23% with a splint and bandage, and 10% with fiberglass casts.

It is important that the horse is confined to box rest for at least 6 weeks so that lameness does not ensue. With lameness the hoof may be positioned on the toe, and the force of the flexor tendons maintains this position, particularly without the counter-force of the extensor tendon. Chronic flexor pull may result in permanent flexor deformity and lameness. If flexor dominance is noted, a splint or cast should be applied (see Chapter 87).

In our experience, the best cosmetic outcome, and chances of achieving primary wound closure, occur with using a fiberglass cast for 3 to 6 weeks. The cast provides the most immobility to the limb and the lacerated tendon ends. Early fibrosis matures more quickly, without disruption of the early granulation tissue.

Flexor Tendons

Horses with complete laceration of one or more flexor tendons are best treated with tendon suturing, wound closure, and post-operative immobilization for about 6 weeks. Flexor tendons support the weight of the horse on loading. Thus healing and return to full strength is a slow process, one that does not return to normal for at least 6 months. In studies investigating methods of tendon repair, immobilization of the limb in a cast without suturing produced a significantly weaker repair that resulted in the clinical sequela of a hyperextended fetlock joint.[4,5] The amount of scar tissue filling the tendon gap was significantly less in this group compared with the sutured groups and was the reason for the reduced strength. Suturing of flexor tendon injuries is therefore recommended. Monofilament suture (nylon or polyglyconate) produced the greatest strength of repair compared with carbon fiber suture, when placed in a double-locking loop pattern (nylon) or three-loop pulley pattern (polyglyconate) for apposition of tendon ends, or for spanning tendon gaps.[4-6] The limb should be cast for at least 6 weeks, with the fetlock joint in mild flexion, by building a heel support with casting tape or plaster to provide a level weight-bearing surface with the ground.

Repairs of flexor tendon lacerations above the hock (i.e., gastrocnemius tendon, DDFT, or SDFT) should follow the same principles, but the prognosis is decreased because of the greater difficulty in maintaining a full-limb cast, larger size of the tendons at this location, and greater biomechanical stresses to support the hock with weight bearing.

Partial Tendon Lacerations

Tendons that are partially transected can be treated successfully without suturing but with wound closure and limb immobilization in most horses. Partial transection of flexor tendons usually involves the SDFT only or the axial margin (medial or lateral) of the SDFT and DDFT. If the limb is immobilized, the remaining fibers of tendon provide the stability for the torn tendon ends to remain in apposition and

provide the strength to prevent further tendon tearing during healing. Anecdotally, if greater than 75% of the SDFT is lacerated, tendon suturing may provide a reduced gap and faster healing and improve the repair. If the SDFT is completely transected along with a partial laceration of the DDFT, the SDFT should be treated with suturing as previously described and the DDFT left unsutured.

Lacerations in Tendon Sheaths

If a laceration enters a tendon sheath, then therapy is altered to include aggressive lavage of the sheath and wound, intrathecal administration of antibiotics, close monitoring of sheath fluid cytological condition, longer use of systemic antibiotics, and limb immobilization. If tissue loss is minimal, wounds entering tendon sheaths should be closed primarily. Primary closure and fiberglass cast application offer the best chance of early healing and minimize the potential for the complications of ascending infection, chronic drainage and fistulae formation, and fibrosis.

CONVALESCENT THERAPY

Shoeing

After removal of a cast or splint for extensor tendon lacerations, a gradual return to full weight bearing is recommended. Shoeing recommendations are simple and include trimming or shoeing level, without toe extensions that may catch and produce knuckling. For flexor tendon lacerations, an elevated and extended heel shoe can be applied and the heel lowered sequentially over the next 6 weeks to a flat position. For severe lacerations involving the DDFT, SDFT, or SL, an extended heel shoe may always be required for additional flexor support.[7]

Graduated Exercise

Horses with extensor tendon injuries have not been evaluated as closely during the healing process to assess tissue maturation and return of strength as those with flexor tendons injuries Because the function of the extensor tendon is to extend the digit and not endure a load on weight bearing, return to full strength may occur sooner than for flexor tendons. Horses should remain in a box stall or a confined area during the wound healing phases and early fibroblastic repair phases of tendon healing (3 to 6 weeks). After this time, hand walking and controlled exercise such as swimming can begin to strengthen the tendon and improve gliding function. After 10 to 12 weeks of controlled exercise, and if no signs of knuckling or flexor dominance are present, gradual return to athletic use can begin.

Horses with flexor tendon lacerations require a more gradual convalescent period. After the first 6 weeks of immobilization, the next 6 weeks should be spent in confinement and regaining a normal foot posture and full weight bearing. Heel support shoes are applied during this time. After 12 weeks, hand walking can begin and gradually increase in frequency and duration over the next 3 to 6 weeks. Controlled exercise with walking, lunging, swimming, or ponying (leading from another horse) is preferred to turnout. Turnout can be given after the horse is sound at the walk and trot and ultrasonographic examination demonstrates extensive fibrosis and mature scar tissue. Heavy athletic use should not begin until 8 to 12 months after the injury.

PROGNOSIS

Successful outcome (soundness) occurs in about 75% of horses with extensor tendon lacerations and up to 54% of horses with flexor tendon lacerations.[1,2,8] The prognosis for horses with partial disruption of the SDFT, or the DDFT, or both is better than those with complete lacerations.[8] Long-term failures are attributable to continued pain from extensive adhesions, joint pain, other injury at the time of laceration, tendon sheath adhesions, tendon contracture, annular ligament constriction, re-injury, and failure of the repair to regain adequate strength to support the joint, leading to breakdown. Re-injury to a damaged tendon may occur during healing, but such a tendon can heal successfully, although convalescence is prolonged. In general the prognosis is better for pleasure riding horses than for sports horses, but especially with injuries involving the flexor tendons of a hindlimb, complete function may be restored. An association exists between lacerations of either or both the long and lateral digital extensor tendons in the proximal part of the metatarsal region and the subsequent development of stringhalt several months later.[9]

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CHAPTER • 83

Soft Tissue Injuries of the Pastern

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Injuries to the flexor tendons and ligaments in the pastern are a common cause of lameness in horses.[1-6] Injuries to the collateral ligaments or the palmar or plantar ligaments of the proximal interphalangeal joint are a less frequent cause of lameness.[1,7-9] Swelling, heat, and sensitivity of the affected tendon or ligament to palpation often accompany lameness. Ultrasonographic evaluation of the pastern is indicated when local swelling, heat, and sensitivity are detected, or when effusion occurs in the digital flexor tendon sheath (DFTS). The cause of the swelling can be determined by ultrasonography, and the severity of the injury can be characterized. If injury occurs to the superficial or deep digital flexor tendons (SDFT, DDFT) in the metacarpal or metatarsal region, ultrasonographic examination should include an evaluation of these structures in the pastern. Lameness associated with soft tissue injuries of the pastern also can occur without localized soft tissue swelling, and ultrasonographic examination is indicated if pain is localized to the region using diagnostic analgesia and no radiographic abnormality is detected, or entheseous new bone is seen. The clinician should bear in mind that intra-articular analgesia of the metacarpophalangeal or metatarsophalangeal joints and the proximal interphalangeal joint is not necessarily specific and may influence closely related structures such as the distal sesamoidean ligaments and the palmar ligaments of the proximal interphalangeal joint. It also may be important to use diagnostic analgesia to determine whether any injury causing soft tissue swelling in the pastern region is the source of pain causing lameness. Perineural analgesia of the palmar nerves at the level of the base of the proximal sesamoid bones (PSBs) should remove lameness if the soft tissue swelling is the sole cause of lameness. Nuclear scintigraphy may help to determine if entheseous new bone is active, and magnetic resonance imaging has the potential to provide additional information.

ANATOMY

Most of the soft tissue structures in the pastern are on the palmar or plantar aspects and are similar in the forelimbs and hindlimbs. The following describes the forelimb but applies equally to the hindlimb. The SDFT forms a thin ring around the DDFT at the ergot and in the proximal most portion of the pastern and then bifurcates into medial and lateral branches. The origin of the branches have a teardrop shape. The cross-sectional area (CSA) of each SDFT branch gradually enlarges as the branch extends distally along the palmarolateral and palmaromedial aspects of the pastern, until the branches insert on the distal aspect of the proximal phalanx and on the proximal aspect of the middle phalanx. The DDFT lies immediately dorsal to the SDFT and extends along the midline to its insertion on the distal phalanx.[1-6,10-14] The DDFT has a bilobed shape in the pastern and is surrounded by the DFTS. The oblique (middle) distal sesamoidean ligaments originate from the base of the lateral and medial PSBs as two large, round to oval branches. These branches become smaller in CSA as they extend distally. The branches join in the proximal to mid aspect of the proximal phalanx and insert as a broad band on the palmar aspect of the middle of the proximal phalanx. The straight distal sesamoidean ligament also has its origin at the base of the PSBs and the palmar ligament and extends distally in the midline, palmar to the oblique distal sesamoidean ligament, to insert on the scutum medium of the middle phalanx.[1-6,10-12] The straight distal sesamoidean ligament lies dorsal to the DDFT and has an hourglass shape, larger proximally and distally, and narrowest in the middle.

The DFTS surrounds the SDFT and DDFT throughout the proximal aspect of the proximal phalanx to the bifurcation of the SDFT.[1-6,10-15] The entire length of the DDFT is included in the DFTS, except for a small area in the distal palmar pastern, just proximal to the bulbs of the heel. The dorsal aspect of the DFTS extends farther distally than its palmar aspect. The proximal digital annular ligament is adhered closely to the palmar aspect of the SDFT in the proximal pastern.[1,3-5,16] The distal digital annular ligament forms a sling over the distal part of the DDFT. These two structures are thin in normal horses.

The palmar ligaments of the proximal interphalangeal joint originate in pairs from the medial and lateral aspects of the proximal phalanx, medial or lateral (respectively) to the SDFT branches, and insert on the scutum medium between the straight distal sesamoidean ligament and the branches of the SDFT. These are large, round to oval ligaments that extend in a diagonal direction from the origin to the insertion. The collateral ligaments of the proximal interphalangeal joint originate from a small eminence on the lateral and medial aspects of the proximal phalanx, distal to the origin of the palmar ligaments, and arc across the joint to insert on a small eminence on the lateral and medial aspects, respectively, of the proximal aspect of the middle phalanx.[1,3,7,10] The proximal interphalangeal joint has a closely adhered joint capsule.

The common digital extensor tendon is located on the dorsal aspect of the pastern.[1] The extensor branch of the suspensory ligament (SL) joins the common digital extensor tendon in the distal part of the proximal phalanx. The main insertion of the common digital extensor tendon is on the extensor process of the distal phalanx, but there are also areas of insertion onto the proximal and middle phalanges. A bursa is present between the tendon and the proximal interphalangeal joint.

ULTRASONOGRAPHIC ANATOMY

The pastern has been divided into five zones: three zones for the proximal phalanx and two zones for the shorter middle phalanx [1-6,11-14] (see Chapter16).

Superficial Digital Flexor Tendon

In the proximal pastern (zone PlA) the SDFT is imaged from the palmar aspect and is homogeneously echogenic and has a

thin, half-moon shape in the transverse plane.[1-6,11-13] In longitudinal images the SDFT has a parallel fiber pattern and a triangular shape along the midline in zone PlA, because its thickness decreases distally. In normal horses, distinguishing the proximal digital annular ligament from the DFTS and the palmar border of the SDFT is difficult. The body of the SDFT ranges in thickness (palmar to dorsal) from 2 to 6 mm in PlA to 1 to 4 mm over the middle of the proximal phalanx.[3] The teardrop-shaped branches in the proximal to mid-pastern region (at the junction of zones PlA and P1B) are imaged from the palmaromedial and palmarolateral aspects and are followed individually to their triangular-shaped insertions. The SDFT branches are similarly homogeneously echogenic, with a parallel fiber pattern throughout. The branches of the SDFT range in thickness from 4 to 7 mm in the proximal pastern to 7 to 12 mm distally.[14] The CSA of the two SDFT branches ranges from 0.3 to 0.4 cm^2 in the distal portion of zone PlA where the branch begins, increases to 0.4 to 0.6 cm^2 in zone PlB, and further enlarges to 0.6 to 0.8 cm^2 near the insertion.

Deep Digital Flexor Tendon

The DDFT has an oval to bilobed appearance in the pastern and is imaged on the palmar midline of the pastern until the DDFT is lost from view distally.[1-6,11-14] The two lobes are symmetrical. The fibers of the DDFT extend obliquely from a deeper to a more superficial position in the more distal portion of the pastern and are separated from the straight distal sesamoidean ligament by an anechogenic space. The dorsopalmar thickness and lateral to medial width of the DDFT decrease in the mid-pastern and increase again in the distal pastern. The DDFT measures 5 to 10 mm (palmar to dorsal) in the proximal pastern, slightly less in the mid-pastern, and 7 to 12 mm in the distal pastern region. The width of the DDFT in a lateral-to-medial direction ranges from 18 to 33 mm in the proximal pastern, decreasing to 15 to 23 mm in the mid-pastern, and increasing in the distal pastern to 23 to 32 mm.[3] Along the dorsal aspect of the DDFT in the mid-pastern region is a synovial fold of the DFTS that is imaged readily, surrounded by a small amount of anechogenic synovial fluid. In the distal pastern region the palmar aspect of the DDFT adheres to the synovial membrane of the DFTS.

Oblique Distal Sesamoidean Ligaments

The origin of the medial or lateral branch of the oblique distal sesamoidean ligament is best found by placing the ultrasound transducer over the medial or lateral PSB and scanning distally over the bone to its base.[1-6,11,12] Alternatively, the origin of the oblique distal sesamoidean ligament can be found by following the SL branches distally over the respective PSBs to the base. Immediately distal to the base of the PSB is the origin of the oblique distal sesamoidean ligament, best located initially in its transverse section as a large, round to oval structure. The branches of the oblique distal sesamoidean ligament merge in the distal part of zone PlA into a broad, rectangular band dorsal to the DDFT. The oblique distal sesamoidean ligament then inserts on the palmar or plantar aspect of the proximal phalanx in zone P1B. The oblique distal sesamoidean ligament is the most difficult to follow to its insertion, because the ligament extends diagonally from its origin to its insertion. Following the medial or lateral branch from its origin to the main body of the oblique distal sesamoidean ligament requires a transducer angle of about 45° from the base of the PSBs to the palmar midline of the proximal phalanx. Properly aligning the transducer and eliminating off-normal incidence artifact is difficult. The oblique distal sesamoidean ligament branches may appear less echogenic because of an oblique orientation. The branches are thickest in the medial to lateral direction proximally. The oblique distal sesamoidean ligament branches measure 12 to 20 mm (lateral to medial) in proximal PlA, decreasing to 9 to 17 mm just before their convergence, and 0 to 9mm (one side only) at their insertion. The palmar to dorsal thickness of the oblique distal sesamoidean ligament branches is 5 to 12 mm in PlA, decreasing to 2 to 6 mm just proximal to the convergence, and decreasing again to 0 to 3 mm at the insertion.

Straight Distal Sesamoidean Ligament

The origin of the straight distal sesamoidean ligament is found by angling the transducer in a proximal and dorsal direction from the proximal most aspect of the pastern, just underneath the ergot, to image the ligament and the base of the PSBs. The straight distal sesamoidean ligament becomes a more oval-shaped structure and is palmar to the oblique distal sesamoidean ligament in zone P1B. The straight distal sesamoidean ligament remains dorsal to the DDFT as it inserts on the scutum medium.

The dorsal-to-palmar thickness of the straight distal sesamoidean ligament gradually increases as the medial to lateral width decreases. The straight distal sesamoidean ligament measures 5 to 9 mm (palmar to dorsal) proximally, increasing slightly over the distal aspect of the proximal phalanx to 6 to 12 mm, and increasing again to 8 to 14 mm at the scutum medium. The medial-to-lateral thickness of the straight distal sesamoidean ligament ranges from 17 to 30 mm in zone P1A, decreases to 10 to 15 mm, and then widens over the scutum medium to 45 to 65 mm.[3] The straight distal sesamoidean ligament is echogenic with normal parallel fiber alignment throughout. Care must be taken to be sure that lesions are not created at the insertion of the straight distal sesamoidean ligament because of the difficulty in aligning the transducer perpendicular to the ligamentous fibers because of the horse's heel. A hypoechoic area of dropout is detected at this location with most ultrasound transducers, because the shape of the heel bulbs precludes obtaining a 90° angle between the transducer and the ligament. If proper angulation of the transducer can be achieved, however, this off-incidence artifact is not imaged.

Cruciate Distal Sesamoidean Ligaments

The cruciate distal sesamoidean ligaments are imaged only in the proximal most portion of the pastern and measure 2 to 4 mm in a palmar to dorsal direction.[3]

Collateral Ligaments

The collateral ligaments of the proximal interphalangeal joint are easiest to examine by imaging them longitudinally and then evaluating them in the transverse plane. The collateral ligaments of the proximal interphalangeal joint are homogeneously echogenic structures, with a parallel fiber pattern from their origin on the distal aspect of the proximal phalanx to their insertion on the proximal half of the middle phalanx.[1,2]

Proximal Interphalangeal Joint

The proximal interphalangeal joint is easiest to image initially in longitudinal plane by identifying the joint space, and then a transverse evaluation of the joint can be made.[7] Fluid normally is not imaged in the proximal interphalangeal joint.

Palmar/Plantar Ligaments of the Proximal Interphalangeal Joint

The palmar ligaments of the proximal interphalangeal joint can be imaged from the origin on the middle of the proximal phalanx to the insertion on the proximal palmar aspect of the middle phalanx. These ligaments are paired on the medial and lateral aspects of the pastern and are located by placing the

transducer dorsal to the branch of the SDFT. The more abaxial branch originates first and is easier to follow than the more axial branch. Each branch has a round to oval shape, is homogeneously echogenic with a parallel fiber pattern, and must be followed individually from origin to insertion.

Digital Nerves

The digital nerves are located dorsal to the lateral and medial aspects of the SDFT, adjacent to the lateral or medial palmar digital arteries.[1] The nerves are found most easily by identifying the digital vein and artery, looking immediately adjacent to the digital vein and the adjacent SDFT. The nerves are tiny, homogeneously echogenic circular structures. The normal thickness of the palmar or plantar digital nerves is 2 to 3 mm, with a CSA of 0.5 to 1 mm^2.[1]

TENDON AND LIGAMENT INJURIES

In the fore pastern the SDFT is the most frequently injured tendon or ligament in all performance horses. The oblique distal sesamoidean ligament is the second most commonly injured structure in the fore pastern, followed by injuries to the DDFT and straight distal sesamoidean ligament.[1-6] In the hind pastern, injuries to the DDFT are most common, with a low incidence of injuries to the other tendonous and ligamentous structures.[1-5] Injuries to the DDFT are accompanied most often by tenosynovitis of the DFTS.[1-6] Injuries to the collateral ligaments of the proximal interphalangeal joint occur infrequently and are more common in the forelimb.[1,3,7] Injuries to the palmar/plantar ligaments of the proximal interphalangeal joint are also uncommon and occur in forelimbs (most common) and hindlimbs. The tendonous and ligamentous structures in the pastern have little covering, and thus they are vulnerable to injury with puncture wounds and lacerations. Ultrasonographic evaluation of the pastern in a horse with an acute laceration or puncture wound to the pastern should be performed aseptically and is an integral part of the evaluation of these soft tissue structures to determine if injury occurred and the severity of the injury.

Superficial Digital Flexor Tendonitis

Injuries to the branches of the SDFT are more common in the forelimb.[1-6] Injury in the pastern may occur in isolation, without an injury to the SDFT in the metacarpal or metatarsal region, or may be an extension of a more proximal tendon injury. Extension of the SDFT injury into the proximal pastern region, and less frequently into the mid- and distal pastern, is more common in the forelimb. Abnormal conformation such as a long pastern, an underrun heel, or an axially displaced heel may predispose the horse to injury of an SDFT branch.

Lameness usually occurs at the onset of injury, is more common with SDFT injuries in the pastern than with those in the metacarpal or metatarsal region, and may persist longer, lasting for 1 to 4 weeks. Longitudinal swelling that extends in a proximal-to-distal direction along the lateral or medial aspect of the pastern throughout its length is often characteristic.[1,2] Focal heat and sensitivity usually accompany this swelling. However, in horses with acute injuries, no localizing clinical signs may be apparent, but lameness is alleviated by palmar (abaxial sesamoid) nerve blocks. Generally, swelling develops within 3 to 4 days. Ultrasonographic examination in the absence of swelling may result in false-negative results. Subluxation of the proximal interphalangeal joint can occur in horses with severe injury to or complete rupture of the SDFT in the pastern. Severe dropping of the fetlock joint with weight bearing can occur in horses with severe SDFT injury.

Core injuries are the most common detected by ultrasonography (Fig. 83-1), followed by diffuse injury to the affected branch.[1,2] Complete ruptures or near complete ruptures of the branches do occur, but they are less frequent. These injuries can be unilateral or bilateral and uniaxial or biaxial, although uniaxial injuries are most common. The medial SDFT branch appears to be more frequently injured than the lateral branch. Peritendonous soft tissue swelling is common. Avulsion fracture of the insertion of the SDFT branch occurs infrequently. Ultrasonographic evaluation of the more proximal SDFT in the metacarpal or metatarsal region is indicated to determine if the injury is an extension of a more proximal injury (see Chapter 70) (Fig. 83-2). Ultrasonographic evaluation of the contralateral fore- or hindpastern is recommended, because bilateral disease may be present, more frequently in the forelimb. Radiographic evaluation of the pastern is indicated for all horses with subluxation of the proximal interphalangeal joint, avulsion fractures at the insertion of the SDFT branch, or a ruptured SDFT branch.

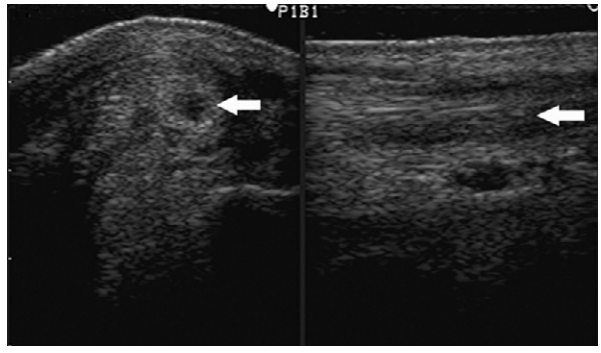

Fig. 83-1 Ultrasonographic images of the left fore lateral branch of the superficial digital flexor tendon obtained in the proximal part of zone P1B in a horse with acute injury. The anechoic to hypoechoic core lesion is apparent within the branch (*arrows*) in the transverse (*left image*) and longitudinal (*right image*) views. Fiber disruption and short linear fibers are imaged within the lesion in the longitudinal view, consistent with a recent injury and early healing. The horse was 1 out of 5 degrees lame, with focal swelling, heat, and sensitivity.

Treatment for horses with acute superficial digital flexor tendonitis in the pastern is similar to that in the metacarpal or metatarsal region.[1,2] Horses with SDFT injuries in the pastern may have a poorer prognosis for returning to racing than those with injuries in the metacarpal region, with a more frequent recurrence of injury.[1,2,17] However, successful return to performance does occur for horses with SDFT branch injuries. Rare horses are able to continue to compete with SDFT branch injuries, without a period of rest, but these are the exceptions rather than the rule. Healing of the SDFT occurs similarly to that in the metacarpal region, with an increase in the echogenicity of the lesion and the subsequent appearance of short, usually randomly aligned linear echoes. Rehabilitation of horses with injuries to the SDFT is similar to that described for bowed tendons and is based on the injury severity (see Chapter 70). A minimum of 6 months in a controlled exercise program is needed for horses with mild SDFT branch injury, whereas 12 months or more are indicated for those with severe injury to the SDFT branch to maximize the horse's chance of returning to its previous level of competition. Ultrasonographic monitoring of tendon healing is an important part of the rehabilitation program. A central echogenic scar surrounded by a hypoechoic halo may be detected in the SDFT branch with a healed core lesion (Fig. 83-3). Peritendonous echogenic tissue representing immature and maturing fibrous tissue often is imaged adjacent to the injured SDFT branch and can result in adhesions between the branch and the surrounding tendonous and peri-

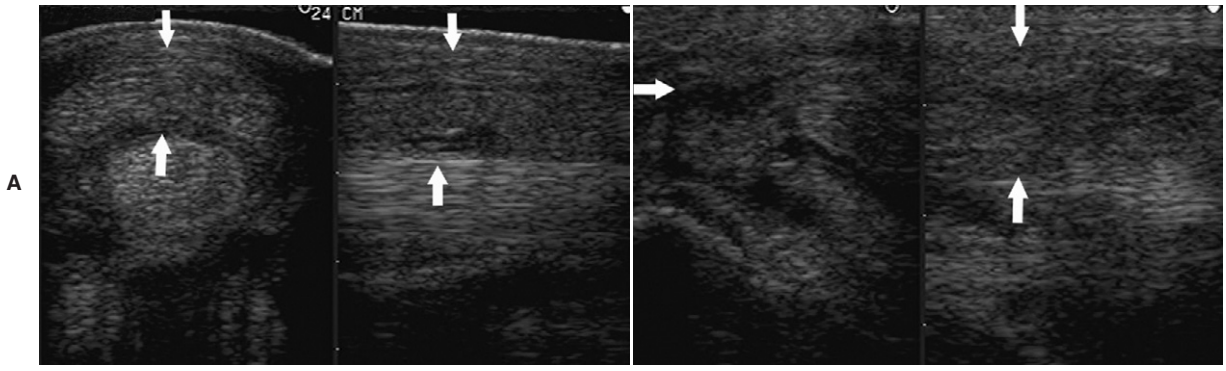

Fig. 83-2 Ultrasonographic images of the right superficial digital flexor tendon in the metacarpal region (**A**) and pastern (**B**) obtained from a horse with an acute severe injury to that tendon. This horse was lame at the walk, with substantial swelling of the superficial digital flexor tendon in the metacarpal and pastern regions and heat and sensitivity of the tendon on palpation. The metacarpophalageal joint dropped on weight bearing, and mild subluxation of the proximal interphalangeal joint was apparent. The transverse images are on the left, and the longitudinal images are on the right. **A,** The superficial digital flexor tendon is enlarged and slightly hypoechoic and was injured from 5 to 32 cm distal to the accessory carpal bone. The superficial digital flexor tendon in zone 3A is surrounded by a thickened hypoechoic digital flexor tendon sheath at the proximal reflection of the sheath (*arrows*). Although the superficial digital flexor tendon still appears relatively echogenic in the transverse image, moderate fiber disruption is visible in the longitudinal image (*arrows*). **B,** The medial branch of the superficial digital flexor tendon in the distal portion of zone P1A is hypoechoic with an abaxially located anechoic lesion *(arrow)* in the branching portion of the tendon in the transverse view (on the left). The surrounding digital sheath and peritendonous tissues are greatly thickened and hypoechoic, and the distinction between the abaxial margin of the superficial digital flexor tendon branch and the peritendonous structures is difficult to discern, particularly in the longitudinal image (on the right). The medial branch of the superficial digital flexor tendon (outlined by *arrows*) has nearly complete fiber disruption in the longitudinal image.

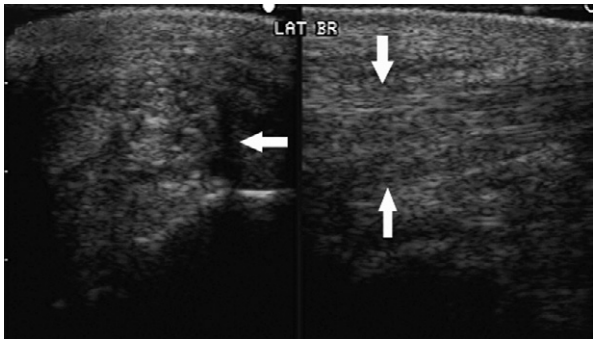

Fig. 83-3 Ultrasonographic images of the left fore lateral branch of the superficial digital flexor tendon obtained in zone P1B of a horse with a chronic healed core injury. The horse had sustained the original injury more than 5 years earlier, and after a long, controlled exercise program the horse had raced successfully several times a year, although some small areas of re-injury were detected periodically, necessitating short periods of downtime from race training. The original central core lesion is still visible in this branch as an echogenic central area, with a thin hypoechoic rim (*arrow*) in the transverse view (*left image*) and longitudinal *(right image)* views. In the longitudinal view the central area of the tendon has a more random fiber pattern than the periphery.

tendonous structures. New areas of fiber disruption often occur adjacent to the previously healed area (see Fig. 83-2) or are associated with adhesions to the surrounding tendonous or peri-tendonous structures.

Deep Digital Flexor Tendonitis

Deep digital flexor tendonitis is discussed in Chapter 71.

Distal Sesamoidean Desmitis

Oblique Distal Sesamoidean Desmitis

Desmitis of the oblique distal sesamoidean ligament is most common and has been seen in all types of performance horses.[1-6] Horses with a valgus or varus limb conformation or a long sloping pastern may be at increased risk for oblique distal sesamoidean ligament injuries. Swelling in the pastern region in horses with oblique distal sesamoidean ligament injuries is characteristic, because this ligament runs diagonally across the proximal to mid-pastern and swelling of the pastern usually occurs in this direction. Most horses have local swelling, heat, and pain detected on palpation of the affected ligament and lameness in the affected leg. Subluxation of the proximal interphalangeal joint can occur in horses with complete rupture of the oblique distal sesamoidean ligaments. Complete biaxial rupture of the oblique distal sesamoidean ligaments is more common in Thoroughbreds and can have catastrophic implications. Distal sesamoidean ligament injury involving the medial branch of the oblique distal sesamoidean ligament is more common than injury involving the lateral branch and is more common in the forelimb than in the hindlimb.[3] Hindlimb oblique distal sesamoidean ligament injuries are more common in horses that are not used for racing. Horses with SL injury are also at increased risk of injuring the oblique distal sesamoidean ligaments. Therefore ultrasonographic evaluation of the SL is recommended for all horses with oblique distal sesamoidean desmitis.

Discrete core lesions often are seen in the medial and lateral branches of the oblique distal sesamoidean ligament, although diffuse areas of fiber damage and splits also occur (Fig. 83-4). Injuries to the insertion of the oblique distal sesamoidean ligament are usually diffuse (Fig. 83-5). Periligamentous soft tissue thickening is often seen. Comparison of the ultrasonographic findings in the affected limb with the

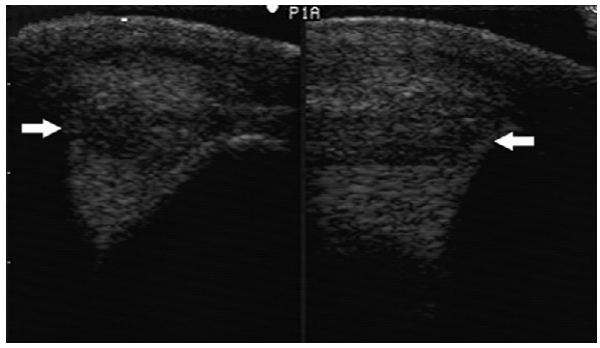

Fig. 83-4 Ultrasonographic images of the origin of the left fore medial branch of the oblique distal sesamoidean ligament obtained in zone PlA. The abaxial portion of the oblique distal sesamoidean ligament is diffusely hypoechoic (*arrow*) in the transverse (*left image*) and longitudinal (*right image*) views. Substantial fiber disruption is visible in the longitudinal view, beginning at the base of the proximal sesamoid bone *(arrow)*. Some periligamentous echogenic soft tissue thickening surrounds the branch. The stallion was sound, but had local heat, swelling, and mild sensitivity of the medial oblique distal sesamoidean ligament.

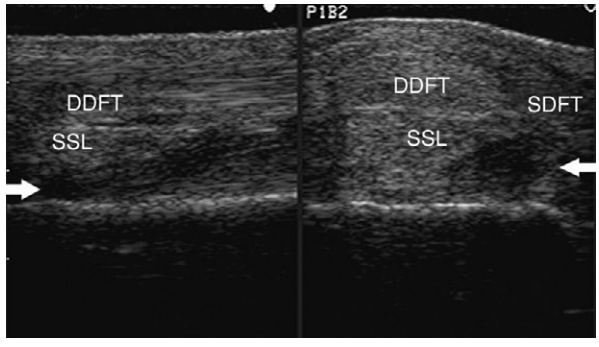

Fig. 83-5 Transverse *(on the right)* and longitudinal *(on the left)* ultrasonographic images of the lateral branch of the right fore oblique distal sesamoidean ligament obtained where the two branches join. The lateral aspect of the transverse view is on the right side of the image. This horse had sustained an acute injury to the lateral branch of the oblique distal sesamoidean ligament from the base of the lateral proximal sesamoid bone to its insertion. A large anechoic to hypoechoic core lesion (*arrows*) is visible. The horse was lame at the walk, with mild subluxation of the proximal interphalangeal joint and local swelling, heat, and sensitivity along the entire lateral branch of the oblique distal sesamoidean ligament. *DDFT,* Deep digital flexor tendon; *SSL,* straight sesamoidean ligament; *SDFT,* superficial digital flexor tendon.

contralateral limb is recommended to be sure that subtle or early injuries are not missed. The origin and insertion of the oblique distal sesamoidean ligaments should be carefully evaluated for avulsion fractures.[1,2] Avulsion fractures usually occur in association with fiber tearing in the distal sesamoidean ligaments and occur from the base of the PSBs (Fig. 83-6) and the insertion on the proximal phalanx. Avulsion fractures remain visible for years after the original injury, long after the associated desmitis in the distal sesamoidean ligament has resolved. Radiographs of the fetlock (particularly the PSBs) and the pastern regions should be obtained in all horses with oblique distal sesamoidean desmitis, paying careful attention to the base of the PSBs. However, it is important to recognize that entheseous new bone on the base of one or both of the

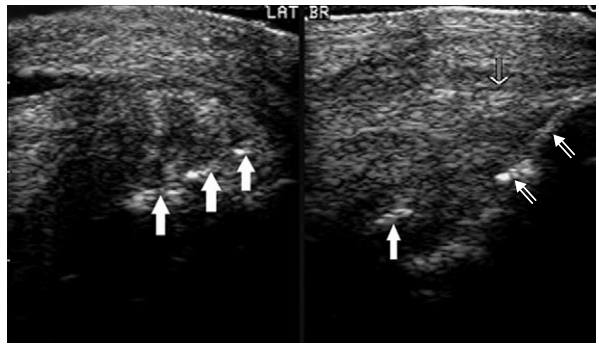

Fig. 83-6 Ultrasonographic images of the lateral branch of the oblique distal sesamoidean ligament obtained in zone PlA of the left forelimb in a horse with chronic active desmitis of the oblique distal sesamoidean ligament and small basilar sesamoid fractures. The hyperechoic bony fragments *(arrows)* are distracted away from the base of the proximal sesamoid bone in the transverse view (*left image*) and the longitudinal view (*right image*). Hypoechoic to anechoic areas are visible in the transverse view, abaxial to the fracture fragments, and areas of amorphous and random fiber pattern are imaged in the longitudinal view, distal to the fracture fragments (to the left of the image). The basilar surface of the lateral proximal sesamoid bone is irregular *(open arrows)*. The horse was 1 out of 5 degrees lame, with thickening of the base of the lateral proximal sesamoid bone, but no heat or local sensitivity.

PSBs or on the mid-palmar aspect of the proximal phalanx can be seen as incidental radiographic abnormalities, unassociated with lameness or active desmitis. Homogeneously radiopaque mineralized bodies are also sometimes seen distal to the PSBs as incidental findings.

Horses with acute injuries to the oblique distal sesamoidean ligament should be managed in the same way as those with other tendon and ligament injuries, with initial anti-inflammatory therapy and exercise restriction.[1,2] A controlled exercise program with incremental increases in the exercise level should be based on ultrasonographic monitoring. As the injury heals, the CSA of the ligament usually decreases, the echogenicity of the lesion increases, and linear echoes are imaged in the area of previous fiber tearing (Fig. 83-7). A long recuperative period usually is indicated for horses with oblique distal sesamoidean desmitis to maximize the chance of return to athletic function.

Prognosis for horses with oblique distal sesamoidean ligament injury is guarded to grave for returning successfully to racing and other competitive athletic activities and depends on the severity of the injury. Horses with coexisting suspensory desmitis, basilar fractures of the PSBs, or subchondral palmar third metacarpal or plantar third metatarsal bone disease have a poorer prognosis for return to athletic function. The incidence of recurrence of oblique distal sesamoidean ligament injury is high. Prognosis is grave for athletic horses with subluxation of the proximal interphalangeal joint associated with distal sesamoidean desmitis.

Straight Distal Sesamoidean Desmitis

Injuries to the straight distal sesamoidean ligament occur infrequently and may occur alone or with other soft tissue injuries.[1-6] These injuries usually are associated with lameness, but focal heat, swelling, and sensitivity are not always detected. Lameness is usually acute in onset and may be severe. Some horses, especially those with proximal lesions, never develop localizing soft tissue swelling, and diagnosis depends on localizing pain to the pastern region by diagnostic analgesia and subsequent ultrasonographic identification of a

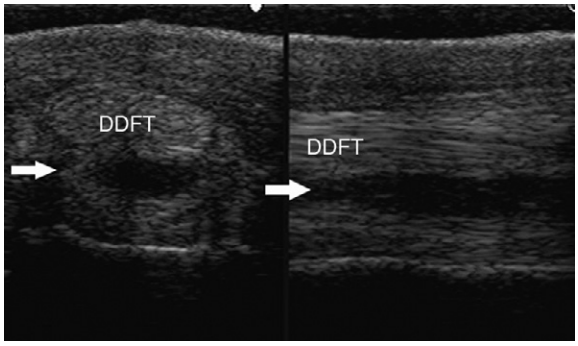

Fig. 83-7 Ultrasonographic images of the right fore pastern obtained in zone P1B from a horse with an acute injury to the straight distal sesamoidean ligament. The horse was lame at the walk, with swelling of the palmar aspect of the pastern and local heat and sensitivity. The large anechoic core lesion (*arrows*) in the palmar aspect of the straight distal sesamoidean ligament is visible in the transverse view (*left image*), with complete fiber disruption in this region that is best imaged on the longitudinal view (*right image*). The straight distal sesamoidean ligament is enlarged in dorsal to palmar and medial to lateral directions. Note also the thickened subcutaneous tissues. *DDFT,* Deep digital flexor tendon.

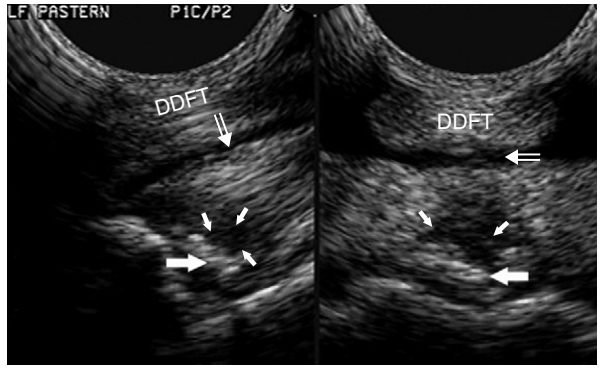

Fig. 83-8 Ultrasonographic images of the left fore straight distal sesamoidean ligament obtained in zone PlC-P2A from a horse with an acute severe injury to the straight distal sesamoidean ligament and an avulsion of its insertion onto the scutum medium. The horse was lame at the walk, with subluxation of the proximal interphalangeal joint and substantial soft tissue swelling of the palmar pastern, with local heat and pain on palpation of the oblique distal sesamoidean ligament and straight distal sesamoidean ligament. The hypoechoic lesion (*small arrows*) in the distal most portion of the oblique distal sesamoidean ligament and the hyperechoic fragment distracted away from the middle phalanx (*large arrows*) are visible in the transverse (*right image*) and longitudinal (*left image*) views. There is anechogenic effusion *(open arrows)* in the digital flexor tendon sheath. *DDFT,* Deep digital flexor tendon.

lesion. The ease with which the most proximal aspect of the ligament can be seen depends on the conformation of the horse and the position of the ergot. Ultrasonographic viewing of the straight distal sesamoidean ligament is most difficult in horse with short pasterns and easiest in those with relatively long, upright pasterns.

Small splits or core lesions may be seen in the straight distal sesamoidean ligament.[1-6] Large areas of fiber disruption in the straight distal sesamoidean ligament are uncommon (Fig. 83-7). Areas of periosteal proliferative change or avulsion fractures at the insertion of the straight distal sesamoidean ligament on the proximal aspect of the middle phalanx may be seen (Fig. 83-8). Avulsion fractures of the origin of the straight distal sesamoidean ligament are less common than with oblique distal sesamoidean ligament desmitis, but the base of the PSBs should be evaluated carefully.

Treatment for horses with desmitis of the straight distal sesamoidean desmitis is similar to that recommended for horses with oblique distal sesamoidean desmitis.[1-6] Horses with mild injuries have returned successfully to racing, but the prognosis for horses with more severe lesions is guarded for any form of competitive athletic function, because recurrent injury is common. Horses with multiple tendonous or ligamentous injuries in the pastern have a guarded to grave prognosis for returning to full athletic function.

Cruciate Distal Sesamoidean Desmitis

Desmitis of the cruciate distal sesamoidean ligament is rare and difficult to diagnose by ultrasonography because of the location of these ligaments.[1,2]

Proximal Digital Annular Desmitis

Desmitis of the proximal digital annular ligament or proximal digital annular ligament constriction occurs infrequently.[1,3] Affected horses usually have chronic moderate to severe lameness. Distention of the palmar pouch of the DFTS is usually present, in addition to subtle distention proximal to the palmar annular ligament of the fetlock. Thickening of the proximal digital annular ligament and skin usually is substantial, with a combined thickness of 4 to 5 mm (normal thickness is 1 to 2 mm) and distention of the DFTS.[17]

SOFT TISSUE SWELLING

Soft tissue swelling in the pastern, without tendonous or ligamentous injury, can result from skin irritation caused by liniments, blisters, local therapeutic ultrasound or cold laser treatment, local trauma from a blow, bandaging, or bell boots, or from a skin infection. Ultrasonographic findings of thickened anechogenic to echogenic subcutaneous tissues, with normal tendonous and ligamentous structures, are typical for injury or inflammation to the skin and subcutaneous tissues. Thickening of the skin also may be seen in horses with skin irritation or infection. These horses usually respond well to local or systemic anti-inflammatory therapy.

TENOSYNOVITIS OF THE DIGITAL FLEXOR TENDON SHEATH

Tenosynovitis of the digital flexor tendon sheath is discussed in Chapter 75.

ABNORMALITIES OF THE PASTERN JOINT

Lameness and local swelling are two common findings in horses with injuries of the collateral or palmar ligaments of the proximal interphalangeal joint.[1,3-5,7-9] Swelling is usually primarily medial and lateral, although it can be circumferential. Acute desmitis of the collateral ligaments may be confirmed by ultrasonography, with decreased echogenicity and loss of fiber pattern, with or without an associated avulsion fracture (Fig. 83-9).[1,3-5,7] In horses with more chronic injuries enthesophyte formation at the origin and the insertion of the collateral ligaments usually is detected. A smoothing of these areas of insertional injury occurs as the desmitis becomes inactive. Similar ultrasonographic findings may be detected in horses with acute (Fig. 83-10) and chronic injury (Fig. 83-11) to the palmar ligaments of the pastern. These

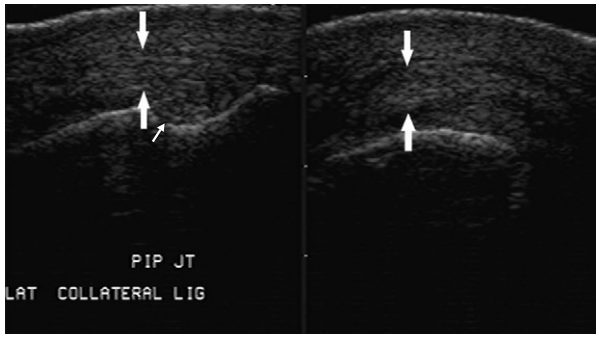

Fig. 83-9 Ultrasonographic images of the lateral collateral ligament of the proximal interphalangeal joint of the right forelimb of horse with moderate lameness and localized swelling. The thickening of the lateral collateral ligament (*arrows*) and the short random fiber pattern seen in the longitudinal view (*left image*) are consistent with desmitis. The distal portion of the proximal phalanx is on the right side of the longitudinal view. The small anechoic slit between the proximal and middle phalanges represents the joint space (*small arrow*). Some echogenic subcutaneous thickening is viewed best superficial to the collateral ligament in the transverse view (*right image*).

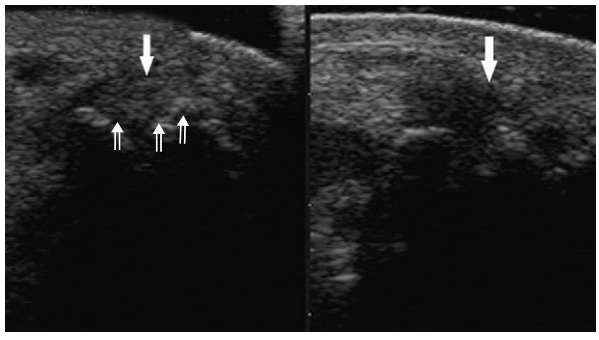

Fig. 83-11 Ultrasonographic images of the lateral palmar ligament of the proximal interphalangeal joint in a horse with chronic severe desmitis and severe enthesopathy. The bony proliferative changes of the proximal phalanx in the transverse (*left image*) and longitudinal views (*right image*) make imaging the ligament in its entirety in the longitudinal plane impossible. The visible portion of the ligament appears homogeneously echogenic in the transverse image (*arrows*), but there is a hypoechoic area within the ligament adjacent to the bony proliferative change in the longitudinal view (*arrow*). The mare was 2 out of 5 degrees lame, with thickening over the lateral and medial aspects of the proximal phalanx, but no heat or local sensitivity was detected.

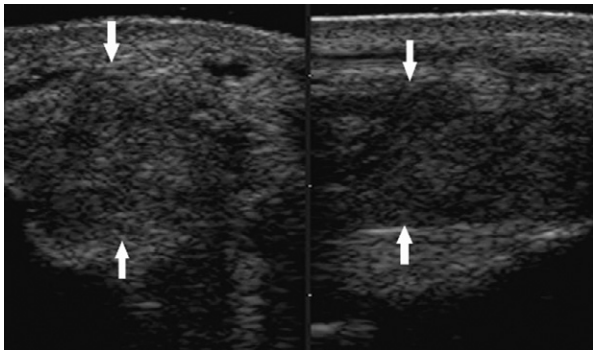

Fig. 83-10 Ultrasonographic images of the left abaxial plantar ligament of the proximal interphalangeal joint in a horse with severe desmitis associated with mild lameness and localized swelling. There is enlargement of the ligament. It is hypoechoic and circular to oval in the transverse view (*left image*), and has a random fiber pattern in the longitudinal view (*right image*). The *arrows* outline the margins of the ligament. A small amount of echogenic peritendonous subcutaneous tissue is visible.

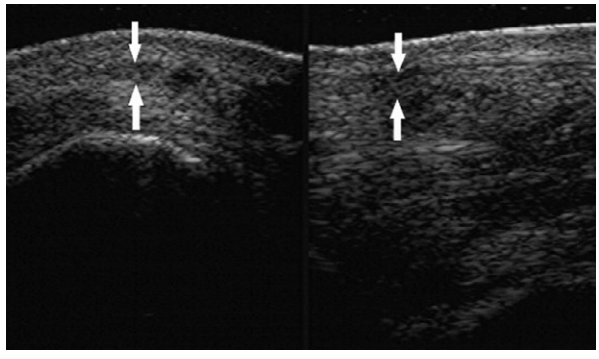

Fig. 83-12 Ultrasonographic images of the left hind lateral plantar digital nerve obtained from a horse with neuritis resulting in acute onset of moderate lameness with local swelling, heat, and exquisite sensitivity to palpation. The enlarged nerve *(arrows)*, oval to circular shape, is located plantar to the digital artery in the transverse view (*left image*). The cross-sectional area of the nerve is increased to 0.17 cm^2. A hypoechoic area disrupts part of the nerve (*arrows*) in the longitudinal view (*right image*).

horses have a guarded prognosis for return to full athletic function.

NEURITIS/NEUROMA

Neuritis of the palmar (plantar) digital nerves results in acute lameness associated with exquisite pain on palpation of the nerves and localized heat and swelling. Ultrasonography shows swelling and decreased echogenicity of the nerve (Fig. 83-12). Neuromas following palmar digital neurectomy initially appear as focal painful swellings over the stump of the digital nerve. With ultrasonography the nerve appears enlarged and hypoechoic, with perineural soft tissue swelling in horses with an acute neuroma. The neuroma becomes more echogenic and heterogeneous with increasing chronicity of injury. A large amount of perineural echogenic tissue may be present in horses with chronic neuromas.

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CHAPTER • 84

Skeletal Muscle and Lameness

Stephanie J. Valberg and Sue J. Dyson

DIAGNOSIS OF SPECIFIC MUSCLE DISORDERS

Diagnosis of a particular muscle disorder is accomplished best with a thorough neuromuscular examination. The key components of the examination include the items mentioned in this section.

History

A history of stiffness, muscle cramping, pain, muscle fasciculations, exercise intolerance, weakness, or muscle atrophy may indicate a muscle disorder. Further characterization requires a detailed account of the horse's exercise schedule; diet; vaccination history; signs of respiratory disease; duration, severity, and frequency of muscle problems; any factors that initiate the muscle problem; and all medications with which the horse is being treated.

Physical Examination

A detailed evaluation of the muscular system includes inspection of the horse while standing with the forelimbs and hindlimbs exactly square to assess symmetry of muscle mass. Any evidence of fine tremors or fasciculations should be noted before palpating the horse. The entire muscle mass of the horse should be palpated for heat, pain, swelling, or atrophy, comparing contralateral muscle groups. Firm, deep palpation of the lumbar, gluteal, and semimembranosus and semitendinosus muscles may reveal pain, cramps, or fibrosis. The triceps, pectoral, gluteal, and semitendinosus muscles should be tapped with a fist or percussion hammer and observed for a prolonged contracture suggestive of myotonia. Running a blunt instrument, such as artery forceps, a needle cap, or a pen, over the lumbar and gluteal muscles should illicit extension (swayback), followed by flexion (hogback), in healthy animals. Guarding against movement may reflect abnormalities in the pelvic or thoracolumbar muscles or pain associated with the thoracolumbar spine (see Chapter 54) or sacroiliac joints (see Chapters 52 and 53). The horse should be observed at a walk or trot for any gait abnormalities and some horses should be ridden.

Ancillary Diagnostic Tests

Muscle Enzymes

Skeletal muscle necrosis may be identified by determining the activity in blood of serum enzymes or proteins that are normally present in high concentration within intact muscle cells but leak out into the blood stream following cell damage.[1,2] Three enzymes are used routinely to assess muscle necrosis: creatine kinase (CK), aspartate aminotransferase (AST), and lactate dehydrogenase (LDH). Carbonic anhydrase III and serum myoglobin also have been suggested as markers of equine muscle necrosis.[3,4] The permeability of the muscle cell membrane, rate of enzyme production, alternate tissue sources of the enzyme, and rate of enzyme excretion and degradation also may influence serum enzyme activities.[1]

Serum creatine kinase. Isoforms of CK are found in skeletal muscle, cardiac muscle, and nervous tissue. CK is a relatively low molecular weight protein (80,000 d) that is involved intimately in energy production within the cell cytoplasm. CK is liberated within hours of muscle damage or increased cell membrane permeability into the extracellular fluid and usually peaks at 4 to 6 hours after muscle injury (half-life [$t_{1/2}$] is 108 minutes).[5] A threefold to fivefold increase in serum CK from normal values is believed to represent necrosis of about 20 g of muscle tissue.[6] Rhabdomyolysis results in a proportionately greater increase in the skeletal muscle isoform than the cardiac muscle isoform, although some studies disagree with the tissue

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specificity of serum CK isoforms in the horse.[7] Limited elevations in CK (<1000 U/L) may accompany training or transport.[8] Extreme fatiguing exercise (e.g., endurance rides, or the cross country phase of a Three Day Event) may result in CK activities being increased to more than 1000 U/L, but usually less than 5000 U/L. Under these circumstances, serum CK activities rapidly return to baseline (i.e., <350 U/L in 24 to 48 hours). Recumbent animals also may have slightly elevated CK activities that are usually less than 3000 U/L. In contrast, more substantial elevations (from several thousand to hundreds of thousands of units per liter) in the activity of this enzyme may occur with rhabdomyolysis.[9]

Serum aspartate aminotransferase Serum AST, previously known as serum glutamic-oxaloacetic transaminase, is a larger molecular weight protein that has high activity in skeletal and cardiac muscle and liver, red blood cells, and other tissues.[1] Elevations in AST are not specific for myonecrosis and increases could result from hemolysis and muscle, liver, or other organ damage. AST activity rises more slowly in response to myonecrosis than does CK, often peaking between 12 to 24 hours after the insult. In addition, AST is cleared slowly by the reticuloendothelial system and may persist for 2 to 3 weeks after rhabdomyolysis ($t_{½}$ is 7 to 10 days).[5]

By comparing serial activities of CK and AST, information concerning the progression of myonecrosis or muscle cell membrane permeability may be derived. Elevations in CK and AST reflect relatively recent or active myonecrosis or muscle cell stress. Persistently elevated serum CK indicates that myonecrosis or muscle stress is likely to be continuing. Elevated AST activity accompanied by decreasing or normal CK activity indicates that myonecrosis has ceased. The degree of elevation of CK and AST does not necessarily reflect the severity of clinical signs.

Serum lactate dehydrogenase LDH is a tetramer made of combinations of the M (muscle) and H (heart) subunits, with five isoenzyme forms found in various organs within the body. Electrophoretic separation suggests that the M_4 (LDH_5) and M_3H (LDH_4) isoforms are found predominantly in skeletal muscle. Elevations in LDH may be detected in horses with rhabdomyolysis, myocardial necrosis, or hepatic necrosis.[8] Therefore concurrent measurement of serum CK is necessary to establish that rhabdomyolysis is present.

Myoglobin Elevation in plasma and serum myoglobin concentrations indicate acute muscle damage. Myoglobin is a low molecular weight protein (16,500 MW) that leaks into plasma immediately after muscle damage and is rapidly cleared in the urine by the kidney. About 200 g of muscle or more must be damaged before myoglobin is detectable in the urine in people.[10] Normal serum concentrations in resting horses have been determined by nephelometry (range 0 to 9 μg/L), with measured concentrations with rhabdomyolysis ranging from 10,000 to 800,000 μg/L.[3,11]

Exercise Response Test

Diagnosing chronic exertional rhabdomyolysis may be problematic in horses that do not have acute clinical signs and have normal serum AST and CK concentrations at rest. In such horses, an exercise challenge can be helpful in detecting subclinical exertional rhabdomyolysis. In addition, quantifying the extent of rhabdomyolysis during mild exercise is helpful in deciding how rapidly to put a horse back into training. Blood samples should be taken before exercise and about 4 to 6 hours after exercise to evaluate peak changes in CK level. Serum CK activity measured immediately after exercise will not reflect the amount of damage occurring during the exercise test. Small fluctuations in serum CK activity may occur with exercise because of enhanced muscle membrane permeability, particularly if exercise is prolonged or strenuous and the horse is untrained.[12] A submaximal exercise test is often valuable for detecting rhabdomyolysis because it provides more consistent evidence of subclinical rhabdomyolysis than maximal exercise tests.[9,13] Fifteen minutes of trotting is often sufficient to produce subclinical muscle damage in horses prone to exertional myopathies.[13] If signs of stiffness develop before this, exercise should be concluded. A normal response would be less than a three- to fourfold increase from basal CK concentration.

Thermography

Thermography (see Chapter 25) may be useful for identifying superficial abnormal temperature changes caused by muscle damage, but it has little value in horses with deeper injuries. However, there are many potentially confusing issues, such as recent removal of a rug or tack. Careful comparisons of the left and right sides should be made. Muscle inflammation is seen as an area of increased temperature in the skin directly overlying the affected muscle. The most common sites of muscle strain identified thermographically include the longissimus dorsi, the origin or body of the middle gluteal, the insertion of the gluteals on the greater and third trochanters of the femur, biceps femoris, semitendinosus, semimembranosus, and adductor.[14]

Nuclear Scintigraphy

Nuclear scintigraphy is useful for identifying some forms of muscle damage and may alert the clinician to an area of deep muscle damage that had not been suspected based on clinical examination. In human athletes ^{99m}Tc stannous pyrophosphate has been used to assess the degree of skeletal muscle damage and to delineate areas of damage.[15,16] It is thought that abnormal radiopharmaceutical uptake reflects an early stage of muscle damage caused by episodic ischemia, which is reversible in some fibers, but may lead to muscle necrosis in others.[15]

^{99m}Tc methylene diphosphonate is taken up in some damaged muscle in the horse and is best seen in bone-phase images, that is, 3 hours after radiopharmaceutical injection. Scintigraphy has been used most commonly in horses with a history of poor performance, with or without stiffness after exercise, to confirm a diagnosis of exertional rhabdomyolysis.[17] Some, but not all, horses with recurrent exertional rhabdomyolis have abnormal radiopharmaceutical uptake into the affected muscles. The mechanism of ^{99m}Tc-MDP binding is unknown, but the release of large amounts of calcium from damaged muscle or the exposure of calcium binding sites on protein macromolecules in the damaged muscle may be responsible.

The use of scintigraphy for diagnosing other muscle injuries has not been documented in the horse, but in one author's experience (S.J.D.) can be helpful in some horses with proximal forelimb or hindlimb muscle injury. Radiopharmaceutical uptake tends to be much more focal and much less intense than in horses with exertional rhabdomyolysis. In some, but not all, horses the region of increased radiopharmaceutical uptake has correlated with a region of increased echogenicity identified by ultrasonography.

Ultrasonography

Diagnostic ultrasonography is potentially useful for identifying muscle trauma and fibrosis, provided that physical disruption of the muscle occurs and assuming that one knows where to look. Muscles have a rather typical striated echogenic pattern,[18,19] but this varies by muscle group, and careful comparisons must be made between similar sites in contralateral limbs in transverse and longitudinal images. The appearance of muscle is also sensitive to how the horse is standing and whether the muscle is under tension, so it is important that the horse is standing squarely and bearing weight evenly. Muscle fascia appears as well-defined, relatively echogenic bands. Care must be taken in identifying large vessels and artifacts created by them.

In an acute injury, muscle fiber disruption is seen as relatively hypoechoic areas within muscle, with loss of the normal muscle fiber striation. The jagged edge of the margin of the torn muscle may be increased in echogenicity. Tears in

the muscle fascia may be identified. The defect in the muscle may be filled by a loculated hematoma, which is slowly replaced by granulation tissue that is hypoechogenic. As the muscle fiber repairs, echogenicity progressively increases. Relatively hyperechoic regions may develop because of fibrous scarring, which may result in long-term gait abnormalities. Hyperechoic regions causing shadowing artifacts reflect mineralization.

Muscle Biopsy

The routine examination of muscle biopsies from horses with various myopathies has resulted in identification of a number of specific equine myopathies. To characterize fully a neuromuscular disorder and its rate of progression, muscle fiber sizes and shapes, fiber type distribution, mitochondrial distribution, polysaccharide staining pattern, neuromuscular junctions, nerve branches, connective tissue, and blood vessels should be examined in frozen sections using a battery of tinctorial and histochemical stains.[20]

A number of basic pathological responses of muscle can be identified in formalin-fixed, paraffin-embedded sections. These include inflammation, muscle fiber necrosis, muscle fiber regeneration, variations in muscle fiber sizes and shapes, vacuolar change, and proliferation of connective tissue. However, many pathological alterations cannot be detected in formalin-fixed tissue, but they can be seen readily in histochemical stains of fresh-frozen biopsy samples.[21] These alterations include muscle fiber types and the respective pattern of distribution to differentiate neurogenic atrophy from myogenic atrophy, characterization of vacuolar storage material, characterization of inclusion bodies, assessment of mitochondrial density, and biochemical analysis of substrate concentrations and enzyme activities.

When considering collection of muscle biopsy specimens, some general guidelines are applicable. Samples preferably should be collected from what is considered abnormal or diseased muscle. A 6-mm outer diameter (Carl Mortensen, Baerskov, Denmark) percutaneous needle biopsy technique can be used to obtain small muscle samples through a 1.5-cm skin incision using subcutaneous local analgesia. If this technique is used, at least 1.5 cm^2 of tissue should be obtained. These samples do not tolerate transport well to an outside laboratory. The optimum biopsy for transport of histopathological tissues to a laboratory is collected using surgical or open techniques, performed with the horse under local analgesia. Care must be exercised to infiltrate only the subcutaneous tissues, not the muscle, with the local anesthetic agent. The objective is to obtain about a 2-cm cube of tissue, and a suitably long skin incision is required. Subsequently two parallel incisions 2 cm apart should be made with a scalpel longitudinal to the muscle fibers. The muscle should only be handled in one corner using forceps. The muscle sample is then excised by transverse incisions 2 cm apart, and the tissue is fixed appropriately.

Routine histopathological samples can be placed in formalin. Samples for histochemical analysis require fixation in isopentane (methylbutane), chilled in liquid nitrogen to ensure rapid freezing and minimization of freeze artifact. In the field, where freezing is not possible, fresh samples wrapped in gauze moistened with saline solution can be shipped in a watertight hard container on ice-packs to specialized laboratories. Samples that potentially may be used for biochemical analysis should be frozen immediately in liquid nitrogen. Samples for electron microscopy (EM) require appropriate fixation in glutaraldehyde preparations. Ideally, thin sections of muscle for electron microscopy should be clamped in vivo to maintain fibers at a resting length before they are excised. However, if pathological conditions other than the alignment of thick and thin myofilaments are to be investigated, small muscle pieces can be excised and placed directly in appropriate EM fixative.

Responses of strips of fresh muscle to stimuli such as caffeine, halothane, and a variety of other agents can be performed on site by specialized laboratories.[22,23]

Electromyography

A specific diagnosis of the cause of muscle atrophy, muscle fasciculations, or myotonic dimpling after tapping the muscle can be aided by performing electromyography. Electromyography of normal skeletal muscle shows a brief burst of electrical activity when the needle is inserted in muscle and then quiescence, unless motor units are recruited (motor unit action potentials) or the needle is close to a motor end plate (miniature end plate potentials). Normal muscle shows little spontaneous electrical activity, unless the muscle contracts or the horse moves. Horses with abnormalities in the electrical conduction system of muscle, or denervation of motor units, show abnormal spontaneous electrical activity in the form of fibrillation potentials, positive sharp waves, myotonic discharges, or complex repetitive discharges.[24]

Classification of Muscle Disease

Based on the information obtained on neuromuscular examination and muscle biopsy a diagnosis usually can be obtained. The following classification system may be helpful in narrowing down causes of muscle disease in horses:

I. Non-exercise associated rhabdomyolysis
  A. Inflammatory myopathies
    1. Clostridial myositis
    2. Influenza myositis
    3. Sarcocystis myositis
    4. Immune-mediated myopathy
  B. Nutritional myopathy
    1. Vitamin E and selenium deficiency
  C. Toxic myopathy
    1. Ionophore toxicity
    2. Pasture myopathies
      a. Rayless golden rod or white snake root
      b. *Cassia occidentalis*
      c. Atypical myoglobinuria
  D. Traumatic myopathy
    1. Compressive anesthetic myopathy
    2. Trauma
  E. Metabolic myopathy
    1. Glycogen branching enzyme deficiency in Quarter Horses
    2. Polysaccharide storage myopathy

II. Exertional rhabdomyolysis
  A. Focal muscle strain
  B. Sporadic tying-up (historically first episode; normal AST)
  C. Chronic tying-up
    1. Dietary imbalances, vitamins, minerals, electrolytes
    2. Polysaccharide storage myopathy (biopsy diagnosis)
    3. Recurrent exertional rhabdomyolysis (biopsy diagnosis)
    4. Idiopathic chronic exertional rhabdomyolysis

III. Exertional myopathy with normal CK
  A. Mitochondrial myopathy

IV. Muscle atrophy
  A. Myogenic atrophy
    1. Severe rhabdomyolysis
    2. Disuse
    3. Cushing's disease
    4. Immune-mediated myositis (rapid atrophy)
  B. Neurogenic atrophy
    1. Equine protozoal myelitis
    2. Local nerve trauma
    3. Equine motor neuron disease

V. Muscle fasciculations
  A. Pain, fear

B. Electrolyte abnormalities
C. Hyperkalemic periodic paralysis
D. *Otobius megnini* ear tick infestation
E. Myotonic dystrophy
F. Stiff horse syndrome

MUSCULAR PAIN, STRAIN, AND TEARS

The role of muscle pain and injury in lameness and poor performance in the horse is poorly recognized. In human athletes muscle fatigue, muscle stiffness, and muscle soreness are well recognized entities, although the pathological processes in the absence of detectable structural abnormalities are not understood completely. Raised intramuscular pressure may be associated with muscle pain after prolonged vigorous exercise in human athletes.[15]

Delayed-onset muscular stiffness or soreness is recognized in people as pain that develops 24 to 48 hours after unaccustomed use of certain muscles and usually resolves spontaneously, assuming the muscles are not overworked again.[25] Continued overstress may result in structural muscle damage. However, specific training involving the activity that provoked the original delayed-onset muscular stiffness or soreness decreases the amount of soreness associated with that condition.

Muscle soreness in the pectoral region after repeated jumping efforts is commonly recognized, especially in event horses several hours after completing the cross country phase of a Three Day Event (S.J.D.).[26] Massage seems to improve the soreness.

Muscle fiber tearing and hemorrhage can result in acute muscular pain in human athletes. A palpable defect or swelling can be detected in superficial muscles. For deeper muscles ultrasonography is required for accurate diagnosis.[27,28]

Muscle fibrosis and mineralization have been well documented in the horse after tearing of the semimembranosus and semitendinosus muscles (see Fibrotic Myopathy, Chapter 81), but acute lesions elsewhere in the limbs have been documented

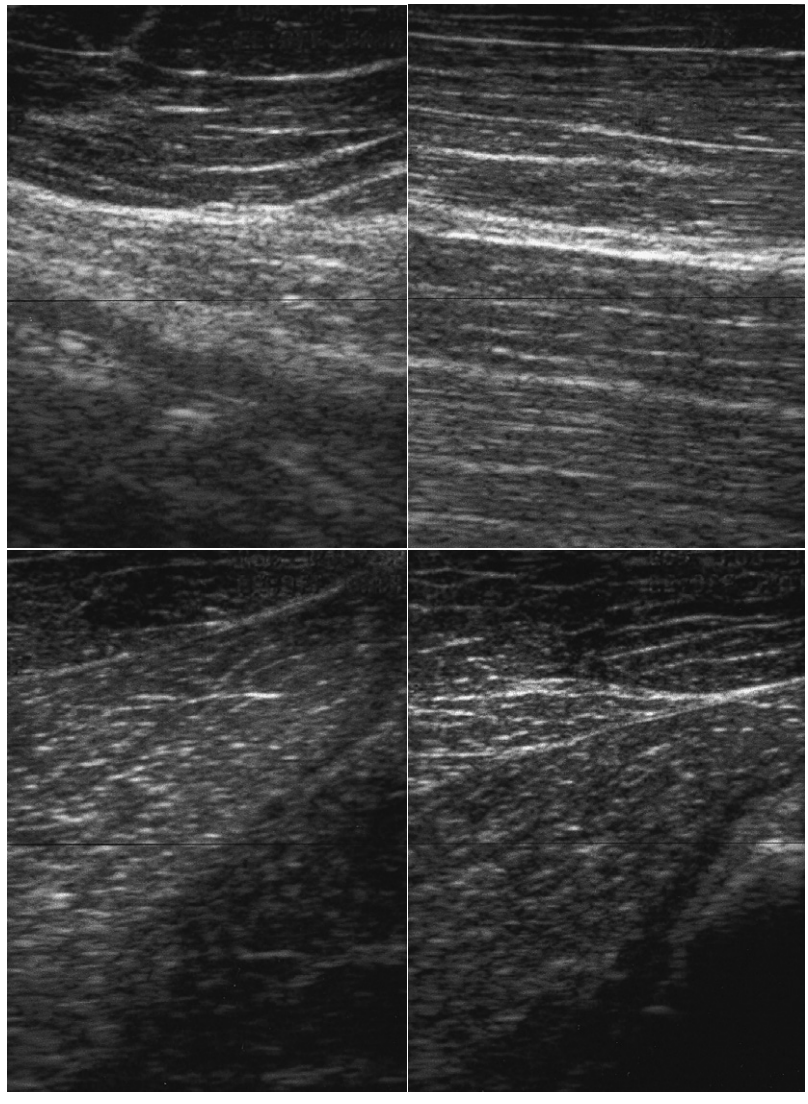

Fig. 84-1 A and B, Longitudinal ultrasonographic images of the muscles at the base of the neck on the left and right sides of an advanced-level event horse with restricted forelimb gait (*left* images indicate left side; *right* images indicate right side). Caudal is to the left. The horse had severe pain and tension in the strap muscles at the base of the neck on the left side and slight muscle atrophy. Note the increased echogenicity of the deeper muscle on the left side compared with the right. The horse was treated by H-wave stimulation that resulted in progressive relief of the muscle spasm and significant improvement in gait and ability to jump.

poorly.[25,29,30] Diagnostic ultrasonography[18,19] has helped in the diagnosis of acute and more chronic muscle lesions, but diagnosis often remains a challenge because of the deep location of some affected muscles and the lack of localizing clinical signs.

In one author's (S.J.D.) experience the most commonly recognized muscle injury sites in the forelimb include biceps brachii, braciocephalicus, the pectorals, and the musculotendonous junction of the superficial digital flexor muscle (Figs. 84-1 to 84-4). In the hindlimb, semimembranosus and semitendinosus, adductor, gluteal, and gastrocnemius muscle injuries have been recognized most frequently.

Muscle tension and spasm in the thoracolumbar region are well documented as sources of pain contributing to poor performance in association with a primary hindlimb lameness, but primary muscle pain in this region often has tended to be overlooked by many clinicians but recognized by physiotherapists. Localized muscle soreness and the interpretation of abnormal sensitivity of acupuncture points are potentially confusing. Protective muscle spasm also may develop secondary to a primary lesion of the thoracolumbar spine or sacroiliac region (see Chapters 52 through 54).

Jeffcott et al.[31] demonstrated that injection of lactic acid into the left longissimus dorsi muscles could significantly diminish performance of Standardbred (STB) trotters worked at speed on a treadmill, although changes in gait were subtle.

One of the authors (S.J.D.) has seen a number of horses that suddenly had lost performance during competition or training, after a particularly extravagant jump or after an awkward jump. The horses had subsequently become reluctant to jump or to gallop downhill. Clinical examination revealed intense muscle spasm in the caudal thoracic and lumbar regions with associated pain. Manipulation to release muscle spasm resulted in relief of pain and rapid restoration of normal performance. Acute back muscle pain also may be induced by a fall.

Localized back muscle soreness is induced readily by a poorly fitting saddle and also may be caused by a rider who sits crookedly or is unable to ride completely in balance with the horse. Poor riding may be due to the ineptitude of the rider, or to the shape of the saddle and the way in which it sits on a particular horse and so positions the rider. Such muscle pain usually is localized to the saddle area and may be associated with slight soft tissue swelling. Thermographic examination may be helpful to demonstrate to an owner the associated

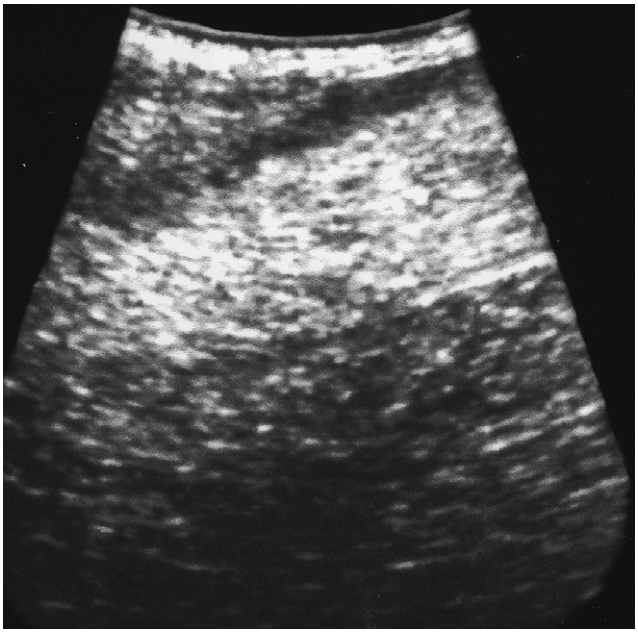

Fig. 84-2 Longitudinal ultrasonographic image of the right semitendinosus muscle of 6-year-old Thoroughbred with acute-onset right hindlimb lameness, with slight swelling of the muscle and pain on palpation. A focal region of increased echogenicity is caused by muscle fiber tearing and hemorrhage.

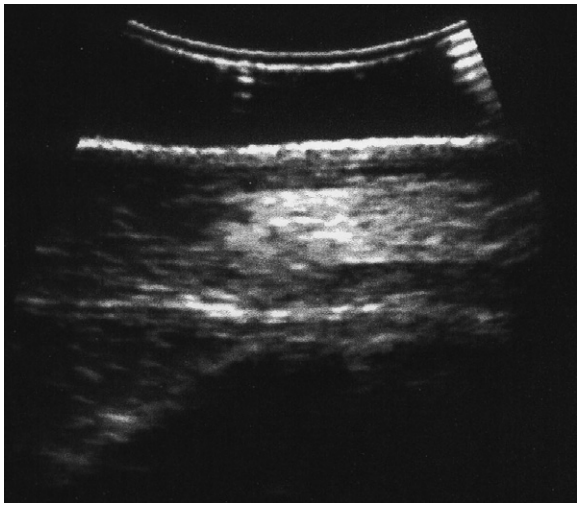

Fig. 84-3 Longitudinal ultrasonographic image of the left brachiocephalicus muscle of a Grand Prix dressage horse that showed left forelimb lameness only when performing lateral movements, such as half pass. The lameness was not altered by any local analgesic technique. There is a focal area of increased echogenicity, caused by muscle fibrosis.

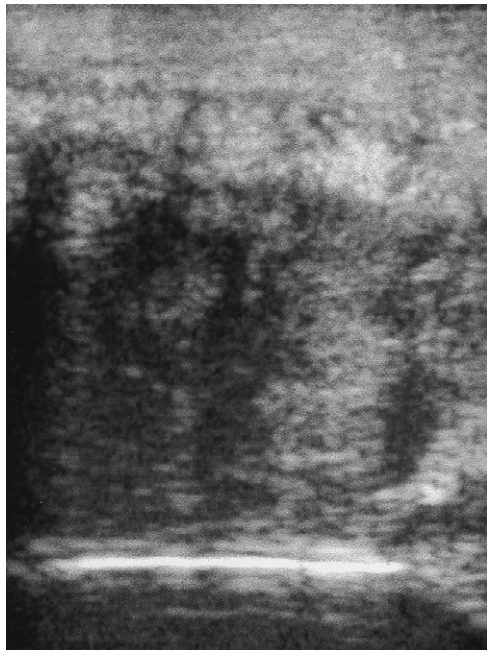

Fig. 84-4 Longitudinal ultrasonographic image of the cranial aspect of the antebrachium of 7-year-old hunter. The horse had developed acute-onset lameness 3 months previously, associated with substantial soft tissue swelling on the cranial aspect of the antebrachium. The extensor carpi radialis muscle is enlarged greatly and has hypoechoic and hyperechoic regions, with little normal muscle architecture.

localized inflammation. Pressure measurements can also be used (see Fig. 118-6).

Diagnosis

History and Clinical Examination

It is important to establish whether the horse has a history of a fall or other traumatic event, the duration of clinical signs, whether swelling had been noted, and whether the horse had exhibited lameness or had performed poorly.

The detection of muscle swelling caused by recent trauma, or loss of muscle bulk caused by either fibrosis associated with a chronic injury or atrophy, often requires the horse to stand completely squarely, bearing weight evenly on all limbs and looking straight ahead. The horse should be appraised visually and by careful, systematic palpation, looking for defects in the muscle, muscle swelling, areas of abnormal muscle firmness caused by fibrosis, muscle tension or spasm, and areas of pain.

In an acute injury resulting in muscle tearing or rupture, in the early stages palpating a defect may be possible, but the defect will become filled with hemorrhage, inflammatory exudates, and edema. Careful palpation should enable detection of most acute superficial muscle injuries, but localization of deeper muscle injury may be more difficult. Identification of chronic muscle strain is more challenging, because the clinical signs are more subtle. The horse must be as relaxed as possible to assess properly the response to firm and deep palpation. If the muscle is sore, the horse may react by increasing tension in anticipation of pain or by pulling away. The area of damaged muscle may have knots.

The neck, limbs, and thoracolumbar region should be moved passively to detect any limitations in movement or pain induced by movement. The horse should be observed moving at the walk and trot to identify any characteristic gait abnormalities such as an abnormal hind foot placement caused by fibrotic myopathy (see Chapter 81) or sinking of a front fetlock because of rupture at the musculotendonous junction of the superficial digital flexor tendon (see Chapter 13). However, one must bear in mind that muscle soreness resulting in compromise of performance may not result in overt lameness, because pain may be induced only when the muscle contracts strongly or is stretched maximally. Pain associated with a braciocephalicus muscle in a dressage horse may be evident only in particular movements such as half pass (Fig. 84-3). A show jumper with sore gluteal muscles may not push off as strongly with the affected limb, resulting in the hindquarters drifting toward the ipsilateral side as the horse jumps.

Muscle Stimulation

Muscle-stimulating machines can be helpful in identifying superficial muscle injuries. Intermittent electrical stimulation of focal areas within a muscle results in muscle contraction and relaxation. The strength of stimulus can be varied. Horses vary in the sensitivity and tolerance of the procedure, so careful comparisons must be made between the left and right sides. Damaged muscle tends to respond greater to a lower stimulus strength, and contraction and relaxation are less smooth and may induce pain.

Thermography, Nuclear Scintigraphy, and Ultrasonography

Thermography, nuclear scintigraphy, and ultrasonography are discussed on page 724.

Muscle Enzymes

Determination of serum muscle enzyme concentrations is rarely of value in identifying muscle soreness or trauma, but it is useful for detecting horses with rhabdomyolysis (see page 723).

Treatment

The aims of treatment include healing of damaged muscle, relief of muscle spasm and pain, restoration of normal circulation, minimization of fibrous scar formation, and remobilization of muscles. The precise mode of treatment depends on the type of muscle injury and the stage of injury and repair. Treatment modalities include laser,[32] therapeutic ultrasound,[33] H wave, transcutaneous electrical stimulation, electromagnetic therapy,[34] massage[26] or passive stretching, combined with box rest, and a graduated, controlled exercise program. Relief of acute muscle spasm may require chiropractic manipulation. During return to work the exercise program must be moderated carefully according to the site of the injury to avoid over-stress in the early stages of repair but encourage a progressive increase in strength. These subjects are discussed more fully elsewhere (see Chapters 94 through 97).

Prevention

Because of the poor recognition of muscle injury in the horse, little work has been done on its prevention. Recently, however, evidence documenting the beneficial effects of warm-up before strenuous exercise has become available.[35-37] Warm-up enhances blood flow to active muscle and increases muscle temperature, which results in better oxygen delivery to exercising muscle, improved enzyme function, and increased range of motion. The best warm-up program for each type of exercise remains poorly defined. However, warm-up should aim to prepare the physiological systems, without contributing to excessive heat generation or fatigue.

EXERTIONAL RHABDOMYOLYSIS

Exertional rhabdomyolysis (ER), literally the dissolution of muscle with exercise, is a common muscle disorder in horses and is a frequent cause of poor performance in a variety of breeds, including Draft breeds, Warmbloods, Thoroughbreds (TBs), STBs, Arabians, Morgans, Quarter Horses, Appaloosas, and American Paint horses.[38] ER was common in Draft horses before the advent of the automotive engine and was called *Monday morning disease, azoturia*, or *paralytic myoglobinuria*.[39,40] Horses often died from myonecrosis or subsequent renal failure. A milder syndrome was noted in lighter breeds and the terms *tying up, set fast, myositis,* and *chronic intermittent rhabdomyolysis* have been used to describe muscle necrosis or muscle cell stress after any form of exercise in the lighter horse breeds.[41,42]

In the past the tendency has been to assume a common cause for all exercise-related myopathies in horses. However, over the past decade it has become apparent that ER is a complex syndrome that has numerous causes. Dietary imbalances, concurrent respiratory infections, and inappropriate training regimens all may produce a sporadic episode of rhabdomyolysis in otherwise successful athletic horses.[40,43-45] ER may occur as a single event in a horse, triggered by a specific event with no tendency for recurrence, or may be a recurrent problem.

Sporadic Exertional Rhabdomyolysis

The most common cause of sporadic ER is exercise that exceeds the horse's underlying state of training.[42] Horses that are advanced too quickly in training, horses that are only ridden sporadically while being fed full rations continually, and horses performing strenuous exercise such as racing or endurance riding without sufficient conditioning commonly develop rhabdomyolysis. In addition, rhabdomyolysis may be more common in horses exercising during an outbreak of respiratory disease. Equine herpes virus I and equine influenza virus have been implicated as causative agents.[29,35]

Clinical Signs

Classically, horses loose impulsion during exercise and develop a stiff, stilted gait, particularly involving the hindquarters. Horses sweat excessively and have a high respiratory rate

because of pain. The horse may be unable to walk forward after resting because of firm, painful muscle contractures involving the back and hindquarters. Signs are most commonly seen after 15 to 30 minutes of light exercise. A horse with severe rhabdomyolysis may show signs of colic, become recumbent, and develop occult myoglobinuria. The urine may be discolored and have an abnormal smell. Attempts to move more severely affected animals may result in extreme pain, obvious anxiety, and exacerbation of the condition.

ER is often symmetrical, involving gluteal, biceps femoris, semitendinosus, and semimembranosus muscles.[46] ER may accompany the exhaustion syndrome in endurance horses, with concurrent evidence of a rapid heart rate, dehydration, hyperthermia, synchronous diaphragmatic flutter, and collapse.[37] Muscle contractures are not always consistent in endurance horses with ER.

Diagnosis is usually obvious based on the clinical signs, but in equivocal situations measurement of serum muscle enzyme concentrations is useful. The degree of elevation of muscle enzyme concentrations does not reflect necessarily the severity of clinical signs.

Treatment of Acute Rhabdomyolysis

If the attack has occurred during exercise some distance from where the horse normally is stabled, the horse should not be made to walk home, but it should be transported back home or left in a nearby stable. If an attack has occurred at a competition, the horse should be treated there and should not be transported home over a long distance until at least 24 to 48 hours later.

The objectives of treatment are to relieve anxiety and muscle pain and to correct fluid and acid-base deficits. Acetylpromazine (0.04 to 0.07 mg/kg), an α-adrenergic antagonist, is helpful in relieving anxiety and may increase muscle blood flow. Its use is contraindicated in dehydrated horses. In horses with extreme pain, detomidine (0.02 to 0.04 μg/kg) combined with butorphanol (0.01 to 0.04 mg/kg) provides excellent sedation and analgesia. Non-steroidal anti-inflammatory drugs (NSAIDs) such as ketoprofen (2.2 mg/kg), phenylbutazone (2.2 to 4.4. mg/kg), or flunixin meglumine (1.1 mg/kg) successfully resolve pain in less severely affected horses. Analgesic treatment is continued to effect, but most horses are relatively free of pain within 18 to 24 hours.

Intravenous or intragastric dimethylsulfoxide (as a <20% solution) can be used as an antioxidant, anti-inflammatory, and osmotic diuretic in severely affected horses. Corticosteroid administration has been advocated by some clinicians in the acute stage. If the horse is recumbent methylprednisolone succinate (2 to 4 mg/kg IV) should be given once. Muscle relaxants such as methocarbamol (5 to 22 mg/kg, IV slowly) seem to produce variable results, possibly depending on the dosage used. The administration of dantrolene sodium (2 to 4 mg/kg PO) in severely affected horses may decrease muscle contractures and possibly prevent further activation of muscle necrosis.

Severe rhabdomyolysis can lead to renal compromise because of the ischemic and combined nephrotoxic effects of myoglobinuria, dehydration, and NSAIDs. The first priority in horses with hemoconcentration or myoglobinuria is to re-establish fluid balance and induce diuresis. In horses with mild rhabdomyolysis administration of fluids via a nasogastric tube may be adequate, but generally fluids are best given intravenously. Balanced polyionic electrolyte solutions are best. If severe rhabdomyolysis is present, then isotonic saline solution or 2.5% dextrose in 0.45% saline solution may be necessary, because horses often have hyponatremia, hypochloremia, and hyperkalemia. If hypocalcemia is present, then supplementing intravenous fluids with 100 to 200 ml of 24% calcium borogluconate is recommended, but serum calcium should not exceed a low normal range. Affected animals are usually alkalotic, making bicarbonate therapy inappropriate.[47]

Ten liters of fluids may be given rapidly. The total fluid replacement is based on an estimation of the degree of dehydration and the clinical response. If the horse is mildly dehydrated (5%), 10 L may be given fast and then 15 L over the next 4 to 6 hours. If dehydration is severe (20%), 10 L may be given fast and then 50 L at 4 L/hr. If the horse is recumbent, the veterinarian should consider using at least two intravenous lines and infusing into the jugular and cephalic veins. The catheters should be sutured in place.

Ideally, reassessment of the packed cell volume, total plasma protein, and serum electrolytes after the initial period of therapy should provide a successful guide for the therapeutic regimen. However, in the practical situation the clinical response to therapy is usually an adequate indicator. In severely affected animals, regular monitoring of blood urea nitrogen and serum creatinine is advised to assess the extent of renal damage. Diuretics usually are contraindicated unless the horse is in oliguric renal failure.

Horses should be rested in a stall on a hay diet for a few days. Small paddock turnout in a quiet area for a few hours twice a day is then helpful. Horses may be hand walked at this time, but not for more than 5 to 10 minutes at a time. Rest with regular access to a paddock should continue until serum muscle enzyme concentrations are normal. Training should be resumed gradually and a regular exercise schedule that matches the degree of exertion to the horse's underlying state of training should be established. Lunging exercise should be avoided until the horse is back in normal work. If the horse has a day or several days off, the dietary energy concentrations should be reduced accordingly.

Chronic Exertional Rhabdomyolysis

Horses that have repeated episodes of ER from a young age, from the time of purchase, or when they are put back into training after a long period of rest may have an underlying abnormality of muscle function. Many of these horses have repeated episodes of rhabdomyolysis with minimal exercise, even when the dietary and training recommendations for sporadic ER are followed. Forms of chronic ER are seen in many breeds of horses including Draft horses, Warmbloods, Quarter Horses, American Paint horses, Appaloosas, TBs, Arabians, STBs, and Morgans.[41,42,45,48,49] Inclement weather has been cited as a trigger of ER, and rhabdomyolysis is reported more commonly in the autumn and winter in the United Kingdom.[50,51] Two specific heritable causes of ER have been identified to date: recurrent ER and polysaccharide storage myopathy.[52-54] Possibly other specific causes exist that have yet to be identified (idiopathic chronic ER). In all of these forms of chronic rhabdomyolysis, apparently specific environmental stimuli are necessary to trigger muscle necrosis in genetically susceptible animals.[54] Animals cannot be cured of the susceptibility to this condition, but if the specific disease is identified, changes in management can be implemented to minimize episodes of rhabdomyolysis.

About 5% of TB racehorses develop ER during a racing season, and 75% of these horses have more than 4 episodes in 4 months.[55] Horses with a nervous disposition, especially fillies, are highly predisposed. Recent research suggests that a subset of these horses has a specific form of ER denoted as recurrent exertional rhabdomyolysis (RER).[23] RER appears to be an inherited, intermittent, stress-induced defect in the regulation of muscle contraction.[52,55] Analysis of pedigrees of TB horses from across the United States suggests that RER is an autosomal dominant trait.[52] Small breeding trials using TB horses with RER confirm that the characteristic abnormality in muscle contracture is inherited in an autosomal dominant fashion (S.V.). Recurrent episodes of rhabdomyolysis in STBs and Arabian horses may be caused by a similar abnormality. A heritable basis for RER in STBs was supported by equine lymphocyte antigen profiles in affected horses.[56]

Factors that trigger rhabdomyolysis in susceptible horses include gender, temperament, excitement, stress, dietary starch, exercise duration and intensity, season, and lameness.[50,51,55] Females are most commonly afflicted with RER (67% female; 33% male), particularly those that are 2 years of age and in race training. Nervous horses are five times more likely to develop RER, and horses with lameness are four times more likely to develop rhabdomyolysis. Susceptible horses receiving more than 5 kg of concentrate feed (oats, corn, and molasses mix) are more likely to develop rhabdomyolysis than those receiving 2.5 kg of concentrate feed per day.[57]

Electrolyte depletion in horses can occur because of dietary deficiency and losses in sweat with strenuous exercise.[46] Sodium, potassium, magnesium, and calcium play key roles in muscle fiber contractility. With severe, acute electrolyte depletion, such as that after endurance exercise, serum electrolytes may be below normal ranges.[46] With chronic dietary depletion, however, serum concentrations may not reflect total body electrolyte imbalances.[44,58] Work by Harris and Colles established renal fractional excretions as a technique to evaluate electrolyte concentrations in horses with chronic ER.[44,58] Blood and urine samples are obtained concurrently, and creatinine and electrolyte concentrations measured in both. Serum creatinine concentration divided by urine creatinine concentration, multiplied by the reciprocal for urine and serum electrolyte concentrations, multiplied by 100 provides the fractional excretion of a particular electrolyte. In the United Kingdom a number of horses with chronic ER had low fractional excretions of sodium, and daily dietary supplementation of 60 g (2 oz) of NaCl resulted in abatement of clinical signs. Other horses had high phosphorus excretion suggesting a dietary calcium/phosphorus imbalance, and decreasing bran while providing a daily calcium supplement (60 g of $CaCO_3$) was helpful in reducing clinical signs of ER.[44] Hypokalemia also has been suggested to play a role in chronic ER, although it is not a common finding in horses consuming adequate quantities of forage.[59,60] Supplementation with good quality forage, or 30 g of KCl per day is recommended for horses with low renal fraction excretion of potassium.

The increased oxidative metabolism associated with exercise results in the generation of free radicals. Selenium, acting through the enzyme glutathione peroxidase, and vitamin E, acting within the lipid component of cell membranes, scavenge free radicals and prevent lipid peroxidation of cell membranes. Primary selenium or vitamin E deficiency is common in young animals living in areas with selenium-deficient soil (see page 736); however, such deficiency rarely has been demonstrated as a cause of ER.[51] In fact, many horses with chronic ER have higher concentrations of selenium and vitamin E because of zealous dietary supplementation by the owner.[61] Whether horses that experience repeated episodes of ER may generate more free radicals than normal horses is not known. A higher generation of free radicals in horses with chronic ER may explain the perceived benefit of repeated administration of selenium and vitamin E in TB horses with RER.[43] Adequate values for blood selenium are greater than 0.07 μg/ml and for serum vitamin E greater than 1.1 μg/ml.

Horses consuming a high-grain diet appear to be more likely to develop ER than horses fed a low-grain or low-fat diet.[57,62,63] The reason for this is unclear and may differ between different forms of chronic ER. For example, in horses with polysaccharide storage myopathy, high soluble carbohydrate diets may enhance glycogen storage. In horses with recurrent ER, however, glycogen storage does not increase substantially even though serum CK activities are highest on high-grain diets.[63] Dietary effects in RER may in part be related to the psychogenic effects of grain on excitability.

A contribution of reproductive hormones to triggering ER has been postulated, because the incidence of chronic ER appears to be highest in mares.[51,55,63,64] Many owners report that episodes of rhabdomyolysis occur most commonly during estrus, but in one study of racehorses no direct correlation was shown between progesterone fluctuations and serum CK activity.[65] The estrus cycle is likely one of many factors that combine to trigger ER in susceptible horses. In some mares where episodes of ER coincide with estrus, suppression of estrus using progesterone implants or injections may be helpful. This should be done with dietary and training alterations. Hypothyroidism and lactic acidosis also have been suggested as a cause of ER but never truly substantiated.[45,49,63,66]

Clinical Signs

Horses with RER have intermittent elevations in serum CK activity.[67,68] Episodes of muscle stiffness, sweating, and firm muscle contractures often occur in horses once they become fit and are frequently associated with excitement at the time of exercise. In TBs, rhabdomyolysis occurs most frequently during training when horses are held to a slow gallop.[55] In STBs, rhabdomyolysis often occurs 15 to 30 minutes into jogging (slow trotting).[67] Obvious clinical signs are rare after racing. A history of poor performance and elevated serum AST and CK may be the only presenting complaints in some horses. Older TBs used as riding horses may have intermittent episodes of rhabdomyolysis associated with lay up of fit horses. Event horses often develop clinical signs after the steeplechase phase of a Three Day Event, or in the 10-minute box, or less commonly during the cross country phase. Muscle stiffness and reluctance to collect may be present on a continual basis between episodes in some of these older horses. Arabian horses often develop clinical signs with little exertion, frequently in association with excitement.

In some riding horses the clinical signs of a mild attack may be subtle and are missed easily by the rider. The horse is maintained in work and actually may be experiencing daily episodes resulting in cumulative muscle damage (Fig. 84-5).

Diagnosis

A submaximal exercise test (see page 724) is useful for identifying RER in a horse that has a history of poor performance unrelated to signs typical of tying-up. Nuclear scintigraphy (see page 724) may demonstrate increased uptake of radiopharmaceutical in the affected muscles of some, but not all, affected horses.

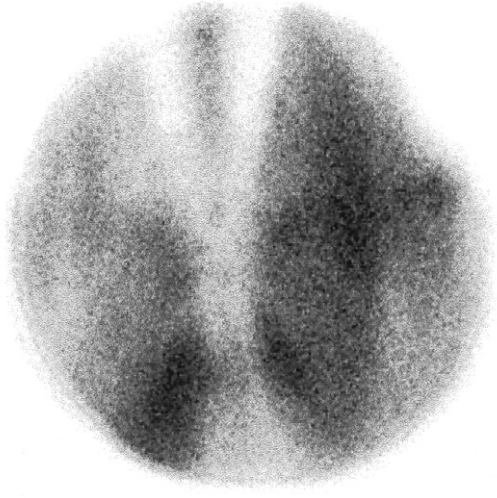

Fig. 84-5 Dorsal scintigraphic image of the pelvic region of an 8-year-old hunter mare with a history of poor performance and reluctance to work. There is intense radiopharmaceutical uptake in the muscles, associated with chronic, repetitive exertional rhabdomyolysis.

Muscle biopsies from horses with RER that are in training have a characteristic histological appearance. These horses have increased numbers of centrally located nuclei in mature muscle fibers (Fig. 84-6). They may have evidence of varying stages of muscle degeneration and regeneration and normal to slightly increased muscle glycogen staining.[68] In addition, RER is characterized by abnormal sensitivity of intact muscle bundles to contractures induced by the addition of caffeine or halothane to a muscle bath.[22,23] Elevated myoplasmic calcium concentration has been reported with ER.[69] Physiological similarities exist between the contracture results of RER and contractures tests for malignant hyperthermia. Biochemical studies of isolated muscle cell membranes have not identified, however, the same defect in the function of the calcium release channel in horses with RER.[70] The increased halothane sensitivity of the muscles in RER horses may explain why many TBs with RER develop rhabdomyolysis under halothane anesthesia.[71]

Management

In the past, horses have been rested in box stalls for several weeks after an episode of RER. Our opinion is that this is counterproductive and increases the likelihood that the horse will develop rhabdomyolysis when put back into training. The initial muscle pain usually subsides within 24 hours of acute RER and daily turnout in a small paddock can be provided at this time. Subsequently a gradual return to performance is recommended once serum CK is within the normal range.

Prevention

Prevention of further episodes of RER in susceptible horses includes standardized daily routines and providing an environment that minimizes stress. This should include desensitizing horses to stressful situations, moving to a quiet area of the barn, regular turnout, and positioning near compatible horses. Daily exercise is essential, whether in the form of turnout, walking on a horse walker, lunging, or riding. The horse should have a long, slow warm-up period.

The diet should be adjusted to include a balanced vitamin and mineral supplements, high-quality hay (but not alfalfa), and a minimum of carbohydrates (<3 to 5 kg). Post-exercise serum CK activity in horses with RER is influenced by the amount of energy horses are fed on a daily basis and the composition of the diet. When 500-kg TBs with RER were fed 88 MJ (21 Mcal) of energy and exercised for 30 minutes a day, serum CK activity after exercise was within the normal range. No measurable effect of feeding a starch-based (2.5 kg of an oat, corn, and molasses mix) or a fat-based (2.3 kg of rice bran; 20% digestible energy from fat) supplement with hay was found on muscle necrosis.[66] However, when the energy level was increased to a level that was closer to that which is normally fed to racehorses (117 MJ or 28 MCal) the source of energy had a significant effect on post-exercise CK activity. Horses fed a high proportion of energy in the form of starch (5 kg of oat, corn, and molasses mix) had significantly higher CK levels after exercise than horses fed 25% of total digestible energy as fat (Releve, Hallway Feeds, Lexington, KY).[72] Apparently, therefore, TBs susceptible to RER that are in moderate- to high-intensity training should be fed concentrate feeds that are relatively low in starch (<20% digestible energy) and high in fat (20% digestible energy or a feed that is 10% to 12.5% fat by weight) to minimize post-exercise muscle damage. Achieving this level of fat supplementation without additional starch can be done by adding corn oil to the diet (a maximum of 600 ml per day, with 800 U of vitamin E), adding rice bran (up to 2.5 kg combined with grains), or purchasing specially designed diets such as Releve. Corn and barley should be avoided because they are particularly high in starch. No more than half of the total of forage should be fed as alfalfa; lower starch hays such as grass or meadow, timothy, brome, or oat hay are preferable.

The use of low doses of acetylpromazine tranquilizers (0.005 to 0.01 mg/kg) 30 minutes before exercise in excitable horses is believed to help some horses. Dantrolene (2 to 4 mg/kg PO) given 1 hour before exercise may be effective in preventing RER in some horses.[73] Dantrolene is used to prevent malignant hyperthermia in people and swine by decreasing the release of calcium from the calcium release channel. Phenytoin also has been advocated as a treatment for horses with RER.[48] Dosages are adjusted in horses to maintain serum levels of 8 to 10 μg/ml. Initial doses begin at 6 to 8 mg/kg orally for 3 to 5 days. Doses can be increased by 1-mg/kg increments every 3 days until rhabdomyolysis is prevented, but they should be cut back if horses appear drowsy. If possible, serum phenytoin concentrations should be assessed regularly at the initiation of treatment. Phenytoin acts on a number of ion channels in muscle and nerves, including sodium and calcium channels. Unfortunately, long-term treatment with dantrolene or phenytoin is expensive, and efficacy has not been established.

The described management is effective in reducing episodes of rhabdomyolysis in many horses, but some remain intractable. Nervous young racing fillies and older riding horses, especially event horses, present the greatest challenge in management.

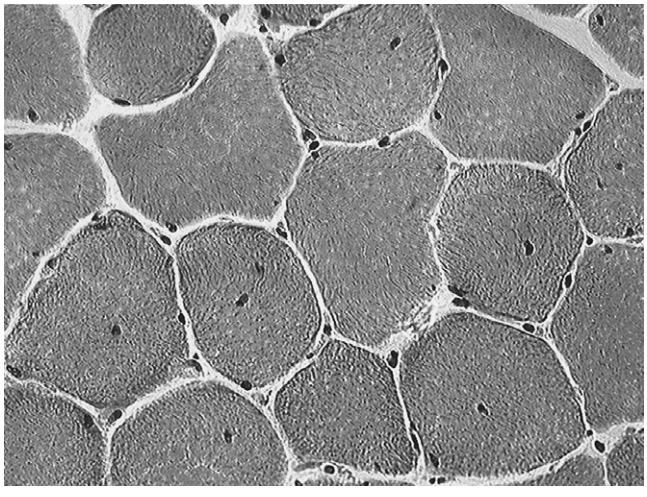

Fig. 84-6 Cryostat cut transverse sections of a biopsy of the semitendinosus muscle of a Thoroughbred with recurrent exertional rhabdomyolysis. Centrally located nuclei are present in mature muscle fibers, which is a common feature of biopsies of horses with recurrent exertional rhabdomyolysis. (Hematoxylin-eosin stain.)

Polysaccharide Storage Myopathy

A subset of horses with chronic ER have a glycogen storage disorder characterized by the accumulation of glycogen and an abnormal polysaccharide in skeletal muscle.[53] Abnormal polysaccharide accumulation occurs in Quarter Horses, American Paint horses, Appaloosas, Draft breeds, Draft crossbreeds, Warmbloods, and a small number of TB riding horses that have a history of muscle stiffness and increased serum CK activity after exercise.[42,62,74,75]

Pedigree analysis has been performed in affected Quarter Horses, Paint horses, and Appaloosas.[54] The sires and dams for almost all of these horses with polysaccharide storage myopathy (PSSM) traced back to one of two related sires within 7 to 9 generations in the pedigree. An inherited trait in Quarter Horse–related breeds is further supported by a small breeding trial where mares with PSSM, bred to a related stallion, produced PSSM-affected foals.[76]

Clinical Signs

Horses with PSSM often have a calm and sedate demeanor. Clinical signs of muscle stiffness, reluctance to exercise, exercise intolerance, or overt muscle contractures and reluctance to move usually are apparent at the commencement of training. Most horses have numerous episodes of ER or a consistent history of poor performance. However, some mildly affected horses have only one or two episodes of ER per year.[62] Serum CK activities are often elevated, even when horses are rested, and increase by 1000 U/L or more 4 hours after 15 minutes of exercise at a trot. Muscle atrophy, renal failure, and severe colic-like pain are less common complaints. More recently, PSSM has been identified in 4-month-old to weanling Quarter Horses and Paint horses with severe non-ER (see pages 732 and 733) and concurrent pneumonia.[77]

A variety of other clinical signs have been reported in Belgian and Percheron horses that have PSSM. These include a generalized decrease in muscle mass, overt muscle atrophy, weakness in the hindlimbs with difficulty rising, reluctance to back up, and gait abnormalities such as shivers[74,75,78] (see Chapter 49). Such horses often have only a mild elevation in serum CK and AST. Muscle biopsies from these Draft horses show, in addition to accumulation of abnormal polysaccharide, more extensive atrophy, centrally located nuclei, and basophilic sarcoplasmic masses, not seen in Quarter Horse breeds with PSSM. Whether Draft horses have a cause for PSSM identical to that described for Quarter Horses in the following discussion is unclear.

Diagnosis

A diagnosis of PSSM is based on histopathological examination of muscle biopsies (Fig. 84-7). The distinctive features of PSSM are subsarcolemmal vacuoles, dark periodic acid–Schiff (PAS) stains for glycogen in snap-frozen samples, and abnormal PAS-positive inclusions in fast twitch fibers.[53,62] Other features that may be present include muscle necrosis, macrophage infiltration of myofibers, regenerative fibers, rimmed vacuoles, and occasionally atrophied type II fibers. Pre-incubation of muscle sections with amylase should result in complete digestion of glycogen in normal horses. The PAS-positive inclusions in horses with PSSM are slow to digest, leaving distinct residues that indicate an abnormal polysaccharide is present. Analysis of the structure of polysaccharide in PSSM muscle by iodine spectra absorption indicates that the polysaccharide is less highly branched than normal muscle glycogen. A combination of β-glycogen particles and a filamentous form of glycogen also are found on electron microscopy of PSSM-affected muscle.

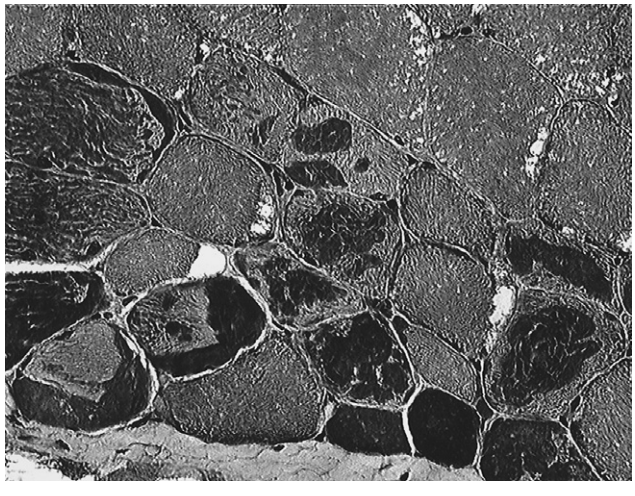

Fig. 84-7 Cryostat cut transverse sections of semitendinosus biopsy of a horse with polysaccharide storage myopathy. White subsarcolemmal vacuoles are present in some fibers, and the dark periodic acid–Schiff-positive granular inclusions represent abnormal polysaccharide in numerous other muscle fibers. (Periodic acid–Schiff stain; hematoxylin counterstain.)

Causes

PSSM appears to be an inherited abnormality associated with increased skeletal muscle uptake of glucose from the blood stream and increased synthesis of glycogen and abnormal polysaccharide.[79] Muscle glycogen concentrations in PSSM horses are 1.5 to 4 times normal, and glucose-6-phosphate concentrations are up to 10 times normal.[80] Glycogen accumulation in horses with PSSM does not appear to be caused by an inability to metabolize glycogen.[80,81] Rather, horses with PSSM appear to have a novel defect characterized by enhanced insulin sensitivity of skeletal muscle. After an intravenous or oral bolus of glucose, blood glucose clearance is 1.5 times faster in horses with PSSM than in normal horses, despite a lower increase in insulin.[76] Administration of insulin to horses with PSSM results in a much faster decline in blood glucose, which persists for 4 hours compared with 2 hours in healthy horses, even when fed grain. In addition, although glucose clearance is enhanced by exercise in healthy horses, it appears to be depressed by exercise in horses with PSSM.[82] The link between enhanced glycogen storage and rhabdomyolysis is not readily apparent. Potentially, altered intra-cellular glucose homeostasis may transiently affect hexokinase binding to mitochondria, leading to insufficient adenosine triphosphate production and rhabdomyolysis.

Management

Horses with acute rhabdomyolysis can be managed in a fashion similar to that described for sporadic rhabdomyolysis[83] (see page 729). Corticosteroids (1 mg/kg of prednisone or prednisolone orally per day) may be of additional benefit in temporarily decreasing insulin sensitivity in horses with acute signs. Muscle glycogen concentrations may be lowered by feeding a diet that is low in starch, supplemented with fat and high in fiber.[84] Such diets consist of a good-quality grass hay, no concentrate feed, and a fat supplement, such as 1 to 3 kg of rice bran (a by-product of rice processing) or 250 to 500 ml (1 to 2 cups) of vegetable oil.[62,85] Rice bran is 20% fat and when compared with isocaloric amounts of grain, no postprandial elevations in blood glucose or insulin occur. Daily exercise is essential to return horses with PSSM to athletic endeavors. As much daily turnout as possible, combined with a gradual training program, has a significant impact on decreasing serum CK activity within 30 days.[84] Box stall rest for more than 12 hours per day appears to increase the incidence of rhabdomyolysis in these horses.

NON-EXERTIONAL RHABDOMYOLYSIS

Although tying-up after exertion is the most common muscle disorder in horses, many horses can suffer severe rhabdomyolysis without any preceding exertion. Causes for non-ER include metabolic, immune-mediated, infectious, nutritional, and toxic factors.

Metabolic Myopathies

Polysaccharide Storage Myopathy

Clinical signs. The most common presentation for PSSM is the development of muscle necrosis, pain, and firm muscles in adult horses with the onset of training (see the previous discussion). A subset of horses with PSSM and a concurrent illness may develop rhabdomyolysis without any preceding exercise. Rhabomyolysis may be so severe that horses become recumbent and unable to rise. This form of PSSM has been described in weanling and yearling Quarter Horses with various concurrent bacterial pneumonias.[77] In addition to serum CK

concentration greater than 200,000 U/L, electrolyte abnormalities, such as hyponatremia, hypochloremia, hyocalcemia, hyperphosphatemia, and hyperkalemia, are common results of renal compromise and the loss of partitioning between extracellular and the large intra-muscular fluid compartments.[86]

Diagnosis A diagnosis of PSSM can be challenging in young horses. The standard diagnostic criteria of abnormal polysaccharide in PAS stains of muscle biopsies may be obscured by the extensive loss of membrane integrity, causing glycogen to leach from tissues. In addition, a diagnosis may be complicated because rhabdomyolysis in weanlings with PSSM may precede the later stage of accumulation of abnormal polysaccharide. Subclinical elevations in serum CK have been noted in foals at 1 month of age that, when serially followed, did not show abnormal polysaccharide accumulation until 18 months of age.[76] The early accumulation of abnormal polysaccharide in the young foals may indicate a particularly severe affliction with PSSM. The diagnosis of PSSM in a weanling may be supported by identifying PSSM in a muscle biopsy from the dam.

Many other differential diagnoses are possible for acute rhabdomyolysis in foals. Neonatal foals may have increased CK at birth with a history of placentitis or dystocia and elevated creatinine concentration.[87] Septicemia could potentially cause an inflammatory reaction in skeletal muscle of neonatal foals. Most commonly, myodegeneration is caused by vitamin E and selenium deficiency with subsequent peroxidation of cell membranes by oxygen free radicals[88] (see page 736). Weanling and older Quarter Horses have developed an immune-mediated myositis on exposure to *Streptococcus equi*.[89]

Treatment Treatment is similar to that for the adult form of PSSM (see page 732). However, the prognosis for weanlings with non-ER is guarded because of the high likelihood of recurrence of the disorder.

Glycogen Branching Enzyme Deficiency

Clinical signs A fatal glycogen storage disorder distinct from PSSM recently has been identified in Quarter Horse foals.[90] The disorder is caused by a deficiency in the glycogen branching enzyme (GBE) responsible for producing a normally configured glycogen molecule in numerous tissues. Clinical signs appear to be caused by a lack of intracellular glucose stores for normal tissue metabolism. Foals may be stillborn in the last third of gestation, weak after birth, or live to up to 7 weeks of age. Death may be sudden when exercised on pasture, associated with weak respiratory muscles, or the result of euthanasia because of persistent recumbency. Treatable flexural deformities of all limbs and recurrent hypoglycemia occur in some affected foals. Persistent leukopenia, intermittent hypoglycemia, and high serum CK, AST, and γ-glutamyltransferase activities are common laboratory findings.

Diagnosis Routine post-mortem examination usually reveals few abnormalities, apart from pulmonary edema in some foals, and basophilic inclusions in skeletal muscle and cardiac tissues in foals older than 1 month of age. PAS staining of muscle, heart, and sometimes liver shows notable lack of normal PAS staining for glycogen and a variable amount of abnormal PAS-positive globular or crystalline intracellular inclusions. Electron microscopy and iodine spectra absorption indicated that the polysaccharide is filamentous, with a minimally branched structure. Reduced GBE activity in the red blood cells of the dams of the affected foals and several siblings support an autosomal recessive mode of inheritance. Because the seven affected foals identified to date have more than 2000 half siblings, GBE deficiency may be a common cause of neonatal mortality in Quarter Horses. A diagnosis of GBE deficiency should be suspected in foals from Quarter Horse–related breeds that have weakness and contracture of all limbs at birth and have a combination of persistent hypoglycemia, leukopenia, and elevated CK (1000 to 15,000 U/L), AST, and γ-glutamyltransferase levels.

Inflammatory Myopathies

Immune-Mediated Myopathies

Severe, fatal rhabdomyolysis has been seen in horses with *S. equi* infections. The reason for the massive rhabdomyolysis is not completely clear, but a toxic shock-like syndrome may occur in some of these horses.

Vascular infarction Mild elevations in serum CK activity have been observed with purpura hemorrhagica in horses.[91] Rarely, some horses vaccinated for or exposed to *S. equi* within the last month develop high serum CK activity, acute colic, firm swellings within muscle and under the skin, and unilateral lameness, without the extensive edema associated with purpura.[89]

Infarction of skeletal muscle, subcutaneous tissue, focal areas of the gastrointestinal tract, and lungs resulting from a severe vasculopathy have been identified in these horses. Such horses often have leukocytosis, hyperfibrinogenemia, hypoproteinemia, and extremely elevated serum CK and AST. Treatment with intravenous penicillin and dexamethasone (0.12 to 0.2 mg/kg), followed by tapering doses of prednisone at an initial dose of 1 mg/kg have been successful in a few hoses. Without aggressive corticosteroid therapy, horses usually die of intestinal infarction.

Immune-mediated myositis Myocarditis in people with rheumatic fever is believed to result from antigenic similarities between the M protein of some streptococcal organisms and the contractile protein myosin in the heart. A form of immune-mediated myositis in Quarter Horses and related breeds also appears to be related to exposure to streptococcal organisms.[88,92] The primary clinical manifestation of this myopathy is rapid atrophy of lumbar and gluteal muscles. Horses often are exposed to a respiratory infection, especially *S. equi*. However, affected horses usually have few or no signs of respiratory disease. Malaise and a persistent elevation in serum CK and AST (1000 to 40,000 U/L) are common. Muscle mass in untreated horses may decrease by 50% within 5 days and the horses become weak.

The characteristic features of muscle biopsies taken during the first week of atrophy are a lymphocytic vasculitis, atrophy of type II fibers, intense lysosomal staining with an acid phosphatase stain, occasional lymphocytic myofiber infiltration, and waves of rhabdomyolysis and regeneration[93] (Fig. 84-8). Untreated horses may show substantial fibrosis around blood vessels. A diagnosis is made on the basis of a history of exposure to strangles, elevated serum CK activity, and muscle biopsy results.

Corticosteroid therapy, such as dexamethasone (0.05 mg/kg) for 3 days followed by prednisone (1 mg/kg for 7 to 10 days) tapered by 100 mg per week over 1 month, is usually successful in halting the atrophy and restoring appetite. Concurrent antibiotic therapy is recommended if leukocytosis, hyperfibrinogenemia, or lymphadenopathy is present. Muscle mass gradually returns over 2 to 3 months. Some horses may show signs of recrudescence of the syndrome on re-exposure to respiratory infections.

Virus-Associated Myopathy

Necrosis of skeletal and cardiac muscle may occur with viral diseases such as equine influenza and equine infectious anemia. In most situations, virus-induced muscle damage represents a component of systemic multiple organ system involvement. Equine influenza A2 has been found to cause severe rhabdomyolysis. Equine herpes virus has been reported to induce primary muscle stiffness and clinical signs resembling ER.[38,94]

Clostridial Myonecrosis

Various species of clostridial organisms cause acute myonecrosis in horses. Infections are characterized by a rapid clinical course,

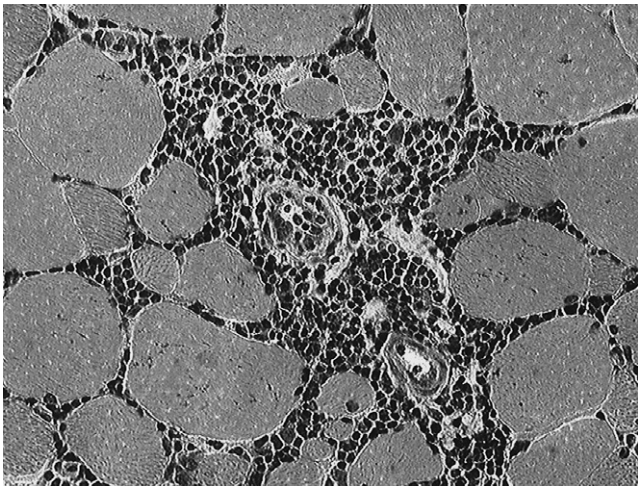

Fig. 84-8 Immune-mediated myositis: cryostat cut transverse sections of biopsy of the longissimus dorsi from horse with rapid muscle atrophy. Prominent cuffing of the blood vessels with mononuclear cell infiltrates, and the lymphocytes surrounding a number of muscle fibers, are characteristic of immune-mediated myopathy in Quarter Horses. (Hematoxylin-eosin stain.)

fever, systemic toxemia, and high mortality.[95,96] Specific bacteria associated with clostridial myonecrosis include *Clostridium septicum*, *Clostridium perfringens (welchii)*, *Clostridium chauvoei*, *Clostridium novyi* type B, and occasionally *Clostridium fallax*. Mixed infections involving several agents are common.

Clinical signs Horses often have a prior condition such as colic, exertional myopathy, or laminitis for which they have received intra-muscular injections in the preceding 48 hours.[97] Affected horses usually rapidly develop depression, swelling, and crepitus at the injection site, and a fever, toxemia, and tachypnea. Tremors, ataxia, dyspnea, recumbency, coma, and death may occur within the next 12 to 24 hours. Mortality may approach 100%. There is usually only one primary site of infection that is associated with an injection or deep wound.[96] Direct clostridial spore deposition into the tissue may occur with penetration, and if suitable necrotic conditions exist, the spores convert to the vegetative, toxin-producing form of the organism. Alternatively, clostridial spores may lie dormant in muscle tissues until necrosis ensues after infection. Powerful exotoxins responsible for the local necrotizing myositis and systemic toxemia are released by proliferating clostridia. Hematological and serum biochemical analyses usually reflect a generalized state of debilitation and toxemia (e.g., hemoconcentration and a stress/toxic leukogram may be present). Elevations in the activities of serum CK and serum AST usually occur; however, they often do not reflect the toxicity of clostridial myonecrosis.

Diagnosis Aspirates from the affected tissues can yield diagnostic information. Obtaining tissue specimens for direct smear examination and fluorescent antibody testing and for anaerobic bacterial culture from affected tissues is preferable. Frequently a diagnosis is made post mortem. Swelling and autolysis are rapid and blood-stained fluid often is observed discharging from body orifices. Extreme swelling and crepitus may be noted over the affected body area. Tissue cut from the affected area may reveal abundant malodorous, serosanguineous fluid. The carcass usually has a foul odor similar to that of rancid butter.

Treatment Although clostridial myonecrosis is often fatal, aggressive specific therapy combined with supportive care may be successful in individual horses.[96] Clostridial myonecrosis resulting from infections with C. *perfringens* seems to be most amenable to treatment and horses with this form have the best prognosis for survival, although extensive skin sloughing over the affected area is common. Antibiotic therapy, aggressive surgical debridement including fasciotomy, and supportive care are the hallmarks of successful treatment. Sufficient fenestrations should be made to establish drainage and aeration over the entire affected area. High doses of intravenous potassium penicillin every 2 to 4 hours are given until the animal is stable (1 to 5 days) and are followed by metronidazole given orally.

Supportive fluid therapy and anti-inflammatory agents for control of pain and swelling are recommended. Short-acting corticosteroids, such as prednisolone or hydrocortisone, may be used for initial therapy of systemic and toxic shock, but continued use is contraindicated in the face of overwhelming sepsis.

Post-Injection Muscle Soreness and Abscessation

Intramuscular injection of a variety of drugs and vaccines may be followed by severe localized muscle soreness. with or without swelling. Injections in the neck may result in neck stiffness and a bilaterally shortened forelimb stride. Injections in the gluteal muscle mass or thigh region can cause unilateral hindlimb stiffness and lameness. These reactions are usually transitory, but occasionally muscle abscessation is a sequel.

A muscle abscess results in localized swelling, heat, and pain. The extent of the abscess, its depth, and the thickness of the walls may be determined by ultrasonography. Surgical drainage is required.

Other Muscle Abscesses

Suppurative myositis may arise through penetrating injuries, hematogenous spread of infection, or local spread of infection. Initially an ill-defined cellulitis occurs, which may heal or progress to a well-defined abscess (Fig. 84-9). An abscess may heal, expand, or fistulate, usually to the skin surface. Once fistulated the abscess may collapse and heal or result in a chronic granuloma with intermittent discharge. *Staphylococcus aureus, S. equi*, and C*orynebacterium pseudotuberculosis* are common causes of skeletal muscle abscessation.

The effect of an abscess on the horse's gait depends on its location and varies from mild stiffness to severe lameness.

Diagnosis is confirmed by ultrasonography or by culture of aspirated fluid. Abscesses lying deep within muscles can be difficult to diagnose. Fibrinogen level and nucleated white blood cell count may be raised. The synergistic hemolysin inhibition test, detecting antibodies to C. *pseudotuberculosis*, can be helpful for detecting internal abscesses.[98]

Treatment consists of poulticing, lancing, flushing, and draining. Occasionally, surgical removal may be required for complete excision. The use of antimicrobial drugs is controversial but may be recommended for corynebacterial abscesses. Commonly recommended drugs include procaine penicillin (20,000 IU/kg IM bid) and crystalline penicillin (20,000 to 40,000 IU/kg IV qid) alone or in combination with rifampin (5 mg/kg PO bid). If antimicrobial therapy is used, it should be continued for several weeks.

Prognosis is usually good for horses with superficial abscesses. Deep abscesses are more difficult to manage successfully. Prolonged resolution or recrudescence often occurs with corynebacterial abscesses.

Sarcocytosis

Cysts of the sporozoan parasite *Sarcocystis* are seen commonly in routine histological sections of the heart, esophageal, and skeletal muscles.[99] More than 90% of horses older than 8 years of age have sarcocysts in the esophageal muscles. Cysts may pose no problem, but with heavy infestations multisystemic dysfunction occurs. Horses with heavy infestation show signs of fever, anorexia, stiffness, weight loss, muscle fasciculations, atrophy, and weakness.[100] Diagnosis of sarcocystosis requires history, clinical signs, laboratory evaluation, and the demon-

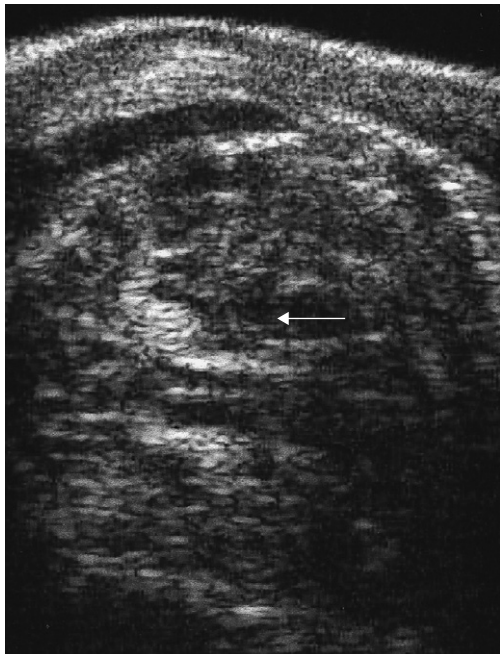

Fig. 84-9 Transverse ultrasonographic image of the lateral aspect of the antebrachium of a pony with cellulitis previously unresponsive to antimicrobial treatment. There was extensive soft tissue swelling throughout the antebrachium and carpal regions, with effusion in the carpal sheath and the tendon sheaths of extensor carpi radialis and the long and lateral digital extensor tendons. The pony was severely lame. There is a loculated, partly fluid-filled developing abscess *(arrow)*.

stration of an inflammatory reaction to immature cysts in muscle biopsies. Infection occurs from contamination of feed by infected canine feces. Successful treatment of one horse with sarcocystosis using phenylbutazone, trimethoprim sulfa, and pyrimethamine is reported.

Traumatic Myopathies

Post-Anesthetic Myopathy

Post-anesthetic myopathy occurs as a unilateral problem such as triceps myopathy, a bilateral problem such as hindlimb adductor myopathies, or as a generalized condition.[101] In horses anesthetized in lateral recumbency the dependent triceps muscle is most commonly affected, whereas the longissimus dorsi and gluteal muscles usually are affected in horses that have been in dorsal recumbency.

Hypoperfusion of muscle groups is the single most important causative factor, often resulting from a raised intracompartmental pressure. Positioning of the horse on the operating table is critical. Compressing muscles against a hard, unyielding surface, venous drainage obstruction, and hypotension may all be contributory factors. Generalized myopathies may occur independent of positioning and hypotension may play a greater role in these horses. The longer the duration of anesthesia, the higher the risk.

Clinical signs Clinical signs are usually evident as soon as the horse tries to stand, but they may be delayed for up to 2 hours. If clinical signs are severe, the horse is unable to stand. Triceps myopathy often is characterized by a dropped elbow stance typical of radial nerve paralysis. Involvement of the gluteal muscles may result in unwillingness to bear weight on the hindlimbs. Horses may appear distressed, with profuse sweating, tachycardia, and tachypnea. The degree of distress depends on the severity of the muscle damage. Affected muscles may feel hard, and localized swelling may occur.

Diagnosis Diagnosis is confirmed by measuring serum CK. The concentration often is increased after general anesthesia, but levels greater than 2000 U/L are significant. The CK concentration may be normal immediately after the horse stands, but as blood flow returns to the affected muscles, the concentration rises.

Treatment and prognosis Treatment aims to relieve distress and pain, to encourage perfusion of muscles, and to keep the horse standing if at all possible. Pain relief is provided by NSAIDs (phenylbutazone, 4.4 mg/kg; flunixin meglumine, 1 mg/kg), combined with opiate analgesics (e.g., butorphanol, 0.1 mg/kg) if necessary. Tranquilization with acepromazine (0.05 mg/kg) and sedation with α_2-agonists may be required. Fluid therapy is important to maintain renal perfusion and urine output and to ensure adequate muscle perfusion. A balanced polyionic electrolyte solution should be infused at up to 20 ml/kg per hour for the first several hours.

The prognosis for horses with unilateral myopathy is usually good, although with severe damage residual muscle fibrosis may develop, which may compromise function. With persistent clinical signs, ultrasonographic examination may be useful to determine the extent and severity of muscle damage.[102] The prognosis for horses with generalized myopathy is more guarded.

Prevention Prevention requires careful pre-operative planning to minimize the time under general anesthesia, careful positioning of the horse on the operating table, and maintenance of arterial blood pressure above 60 mm Hg using fluid therapy and inotropic agents such as dobutamine (1 to 5 μg/kg per minute). The position of the horse on the operating table is in part dictated by the surgical procedure. In lateral recumbency the dependent forelimb should be pulled forward and the upper limb should be supported at least parallel to the table. The hindlimbs should be supported parallel to, or above parallel, and adequately separated to permit venous drainage. In horses in dorsal recumbency, support of the forelimbs or hindlimbs in a semi-flexed position with the help of an overhead hoist can be helpful.

Fibrotic Myopathy

Fibrotic myopathy is discussed in Chapter 81.

Compartment-like Syndrome in the Antebrachium

Compartmental syndrome is a condition in which high pressure within a closed fascial space reduces capillary blood perfusion below a level necessary for tissue viability. The syndrome develops in skeletal muscle enclosed by relatively non-compliant osseofascial boundaries.[103] Elevated intracompartmental pressure may result from increased intracompartmental fluid or from decreased compartment size. Increased pressure because of fluid accumulation may result from muscle trauma and hemorrhage, or it may be a sequel to prolonged ischemia and subsequent reperfusion and edema. Prolonged raised pressure above 30 mm Hg resulted in myonecrosis in the dog.[104]

A compartment-like syndrome has been described in the horse involving the caudolateral muscle compartment of the antebrachium.[105] This encloses the lateral digital extensor, ulnaris lateralis, superficial and deep digital flexor and flexor carpi ulnaris muscles, and also the median artery, vein, and nerve.

Lameness is a sequel to trauma and is acute and severe in onset, with the horse reluctant to bear weight on the limb, associated with firm swelling on the caudolateral aspect of the antebrachium. Digital pulse amplitudes in the more distal part of the limb may be reduced if the limb is flexed or extended. With time the affected muscle mass may become cold.

Treatment is by fasciotomy. Incising the superficial fascia over ulnaris lateralis resulted in immediate separation of the fascia by 3 to 4 cm, and rapid relief of clinical signs.[105]

NUTRITIONAL MYODEGENERATION

• John Maas and Stephanie J. Valberg

Causes Nutritional myodegeneration (NMD) (white muscle disease, nutritional muscular dystrophy) is an acute degenerative disease of cardiac and skeletal muscle caused by a dietary deficiency of selenium or vitamin E.[88,106] Marginally to severely selenium-deficient areas occur in regions of numerous countries.[107,108] This syndrome occurs in young, rapidly growing foals born to dams that consumed selenium-deficient diets during gestation. The disease also has been implicated in masseter muscle myopathy and occasionally non-ER in adult horses. Selenium and vitamin E appear to be synergistic in preventing NMD. However, on the basis of prophylaxis and response to treatment, selenium deficiency appears to be the most important contributor.

Clinical signs Foals with primary necrosis of myocardium and respiratory muscles show dyspnea, a rapid irregular heartbeat, profound weakness, and recumbency.[86,88] Death often occurs in less than 24 hours despite medical therapy. If an animal responds to treatment, it often fails to thrive because of residual myocardial damage. Animals with predominantly cardiac signs also may manifest mild skeletal muscle problems associated with NMD.

The skeletal muscle form of NMD frequently has a slower onset, characterized by muscular weakness or stiffness. Animals may be recumbent and unable to stand. Animals that are able to rise without assistance, or with assistance, show muscle weakness, trembling of limb muscles, or stiffness. Stiffness is more pronounced as fibrosis occurs after extensive degeneration and calcification of muscle fibers. Most affected animals are able to stand for only short periods. Supporting muscle groups of the forelimbs and hindlimbs may appear swollen and may be hard and painful on palpation. Commonly affected muscle groups include the gastrocnemius, semitendinosus, semimembranosus, and biceps femoris, and muscles of the lumbar, gluteal, and neck regions. If the diaphragm and intercostal muscles are affected, the animal may show respiratory distress and evidence of increased abdominal effort when breathing. Dysphagia, because of necrosis in the tongue, may be the only sign in some affected foals and frequently is accompanied by aspiration pneumonia.

In the western United States we have seen some selenium-responsive NMD in adult horses that were extremely deficient in selenium in the winter time (snow on the ground), and the only clinical signs have been myoglobinuria (red snow) and mild stiffness in the morning. These mainly have been work horses and Quarter Horses. Some animals exhibit what appears to be abdominal pain, with violent thrashing. Myocardial damage and signs consistent with cardiac dysfunction may be present in horses with skeletal NMD. Animals with primary skeletal NMD often respond favorably to treatment and rest. Improvement is evident after a few days, and within 3 to 5 days animals can often stand and walk.

Clinical pathological findings Obvious elevation in CK, AST, and LDH activities occur during myodegeneration. CK activities often will be in the thousands to hundreds of thousands of units per liter. Progressively decreasing CK activity can be used as a prognostic indicator of cessation of myodegeneration. Electrolyte derangements are common in horses with severe rhabdomyolysis.[86] Hyperkalemia, hyperphosphatemia, hyponatremia, hypochloremia, and hypocalcemia can occur when the normal distinction between extracellular and intracellular compartments is destroyed by massive tissue necrosis. Myoglobinuria is common. Elevated serum protein concentrations and hemoconcentration commonly reflect dehydration in foals unable to nurse or drink water and reflect fluid shifts into damaged tissues. Neutrophilia is common because of the high incidence of aspiration pneumonia.

Diagnosis Whole blood selenium and plasma vitamin E samples provide satisfactory information for assessing the intermediate to long-term nutritional status. Whole blood selenium analysis is preferred over plasma and serum.[109] Whole blood selenium concentrations ranging from 0.07 to greater than 0.1 ppm are considered normal. Short-term oral or intra-muscular supplementation of selenium can confuse interpretation of circulating selenium concentration. Selenium-dependent glutathione peroxidase (GSH-Px), formed in the red blood cells during erythropoiesis, also provides an index of body selenium status. Adequate GSH-Px activities are greater than 20 to 50 units per milligram of hemoglobin per minute in horses. However, GSH-Px reference values are only specific to the laboratory where the analysis is performed and must be validated by comparison with blood selenium concentration. The activity of GSH-Px in red blood cells of domestic species remains constant for 4 to 6 days when maintained at 39° F (4° C); after this time significant decreases occur. The critical concentration of vitamin E (α-tocopherol) in plasma is 1.1 to 2 ppm. Vitamin E deteriorates rapidly in plasma samples. Therefore, plasma samples for α-tocopherol analysis need to be put on ice immediately, protected from the light by wrapping in tin foil, and stored frozen (–21° F, –70° C) if analysis is to be delayed.

Pathological findings Bilaterally symmetrical myodegeneration is a consistent finding in NMD. Skeletal muscle degeneration is characterized by pale discoloration and a dry appearance of affected muscle, white streaks in muscle bundles, calcification, and intramuscular edema. The white streaks in muscle bundles represent bands of coagulation necrosis or in horses with chronic disease where insults may have occurred weeks before may represent fibrosis and calcification. Affected muscle bundles are often adjacent to apparently normal or minimally affected muscle. Histologically, affected muscle fibers may be hypercontracted and fragmented, with some mineralization of muscle fibers and macrophage infiltration. Tissue biopsies and tissue specimens obtained at necropsy can be assayed for selenium content. Normal liver concentrations of selenium are 1.05 to 3.5 ppm dry matter.

Pathophysiological findings During normal cellular metabolism, highly reactive forms of oxygen (free radicals) are produced. These include hydrogen peroxide, hydroperoxides, lipoperoxides, superoxide, various hydroxy radicals, and singlet oxygen. Vitamin E is active within the cell membrane as a lipid-soluble antioxidant that scavenges free radicals that otherwise might react with unsaturated fatty acids to form lipid hydroperoxides. In contrast, GSH-Px destroys hydrogen peroxide and lipoperoxides that have already been formed and converts them to water or relatively harmless alcohols. Other enzymes, such as catalase and superoxide dismutase, also are involved in this protective process. It is believed that a deficiency of selenium results in rhabdomyolysis through oxidant damage to muscle cell membranes. The precise interrelationships between selenium, vitamin E, other metabolic factors and triggering mechanisms in NMD are not fully understood, because many animals deficient in selenium or vitamin E have no evidence of muscle disease. In certain situations deficiencies of selenium and vitamin E are necessary for disease to occur. In other animals NMD can occur when a deficiency of only one of the nutrients is present and the other is normal in blood and tissues.

Treatment and prognosis Myocardial damage is often extensive and incompatible with life with the cardiac form of NMD. Only rarely is treatment successful. In contrast, the skeletal muscle form of NMD is more generally amenable to treatment, although the prognosis for clinical recovery from the skeletal form of NMD is guarded and depends often on whether secondary complications, such as respiratory disease, develop. In all horses with NMD, therapy should involve specific supplementation with selenium and vitamin E and general supportive care.

Alleviation of selenium-responsive NMD requires use of injectable selenium products, available with selenium concentrations varying from 1 mg of selenium per milliliter to 5 mg/ml. All products contain 50 mg/ml (68 IU) of vitamin E as *dl*-α-tocopherol acetate. The label dose for selenium is 0.055 to 0.067 mg/kg (2.5 to 3 mg/45 kg) body mass given intramuscularly or subcutaneously. Dosage should not be increased greatly above the label dose to prevent an inadvertent selenium toxicosis. Absorption and distribution of injectable selenium occurs rapidly and may account for the rapid improvement in clinical signs seen in horses with reversible clinical signs.

The amount of vitamin E in vitamin E/selenium combinations is insufficient for vitamin E supplementation. Injectable vitamin E products are now available that contain 300 IU vitamin E per milliliter as *d*-α-tocopherol (Vital E; Schering-Plough Animal Health, Kenilworth, NJ). Administration increases the tissue and plasma level of vitamin E activity for about 3 weeks. Bioavailability of vitamin E from injectable products depends on the form of vitamin E (the alcohol form, *d*-α-tocopherol, is the most active) and amount and quality of the solution emulsifier used. Oral vitamin E supplementation is a good approach to supplement dietary levels. Daily recommended levels of supplementation for horses range from 600 to 1800 mg of *dl*-α-tocopherol acetate.[110] Oral α-tocopherol is now available for all species and contains 500 IU vitamin E per milliliter (Vita/E, Schering-Plough Animal Health). The recommended dosage of this product is 0.5 to 1.5 IU/kg body mass.

Supportive therapy may include administration of antibiotics to help combat secondary pneumonia and infected decubital lesions, which are common in recumbent patients. When dysphagia is present, feeding by nasogastric tube, provision of adequate energy intake, and attention to fluid and electrolyte balance are critical if recovery is to be successful. Hyperkalemia may be life threatening in affected foals. Mineralocorticoids, alkalinizing fluids, dextrose, and insulin may be used to reduce circulating potassium concentrations.[86]

Prevention and control The prevention and control of NMD is achieved through supplementation of selenium and vitamin E. Oral supplementation for horses at 1 mg of selenium per day increases blood selenium concentrations above levels known to be associated with NMD.[109] Supplementation of pregnant mares is advised in areas known to be selenium deficient. However, only limited selenium may cross the placenta.[106] Supplementation during lactation increases levels of selenium in milk and thus provides a potential means of selenium supplementation in foals. However, evidence in cattle indicates that this increased level of selenium in milk may not meet the foal's nutrient requirements.[111] Supplementation by injectable selenium products alone would require treatment every 30 to 45 days and would provide only partial amounts of the recommended level of selenium.[112]

Regardless of the supplementation method, periodic blood (or tissue) sampling of animals at risk is necessary to ensure maintenance of desired levels of selenium. In high-risk areas, samples should be taken every 60 to 90 days to determine selenium status in susceptible animals and every 6 to 12 months to monitor supplementation. Based on these assessments, adjustments to the rate or extent of selenium supplementation may be made. Feeding animals properly prepared and stored hay and grain or allowing them access to high-quality green forage should ensure adequate vitamin E intake.

TOXIC MYOPATHIES

• Stephanie J. Valberg and Sue J. Dyson

Ingestion of a number of toxic substances in feed or forage may cause of rhabdomyolysis in horses.

TOXIC SUBSTANCES

Ionophores

Ionophores are commonly added to ruminant feeds for growth promotion and coccidiostat properties. Horses, however, are 10 times more sensitive than cattle to the toxic effects of ionophores in feed. When equine feeds are inadvertently contaminated with ionophores or horses eat cattle feed, cardiomyopathy is the most common chronic sequela. However, some animals may die acutely with colic-like signs, myoglobinuria, hypokalemia, cardiac arrhythmia, and tachypnea.[113]

Pasture Myopathies

Trematones Horses ingesting 0.5% to 2% body weight of trematone-containing plants are likely to die from skeletal muscle and cardiac muscle necrosis. Horses show depression, weakness, low head posture, and increased cardiac and respiratory rates. Serum AST and CK often are elevated greatly, and serum electrolyte abnormalities such as hypocalcemia, hyponatremia, hypochloremia, hyperkalemia, and hyperphosphatemia may be present. Treatment is generally supportive as described in the section on acute ER. Trematone has been identified in white snakeroot (*Eupatorium rugosum*) and rayless goldenrod (*Haplopappus heterophyllus*). White snakeroot grows in shaded areas of the eastern and central United States.[114-116] Rayless goldenrod is common in the southwestern United States on open pastures. Trematone remains active in the hay and in the stalks of the dead plants on pasture, so the fresh and dried forms of the plants should be kept from horses.[115,116]

Cassia occidentalis Muscle necrosis also may occur in horses ingesting *Cassia occidentalis* seeds, prevalent in the southeastern United States.[117] Horses develop incoordination and recumbency and die. Gross skeletal muscle lesions are not present, but histopathological lesions include segmental myonecrosis.

Blister Beetles One of 70 horses poisoned with blister beetles developed muscle necrosis.[118]

Atypical Myoglobinuria Atypical myoglobinuria occurs sporadically in horses kept at pasture, usually with no supplemental feeding.[119] The condition has been recognized most commonly in the United Kingdom. The cause is unknown. Atypical myoglobinuria occurs most often in spring and autumn and often is associated with a sudden deterioration in weather conditions. The clinical signs are sudden in onset and rapidly progressive, frequently resulting in death. Several horses in a group may be affected, although some may remain without clinical signs. Affected horses are reluctant to move, have muscle weakness, and may become recumbent. Gut sounds may be reduced, with reduction in fecal production, although appetite may be unaffected. The horses do not show signs of pain, despite post-mortem evidence of widespread myopathy. Typically myoglobinuria and substantial increases in serum CK and AST occur.

Post-mortem examination reveals widespread myodegeneration in skeletal muscles and the myocardium. Supportive therapy has resulted in recovery of some horses although, being low value pleasure horses, it is difficult to assess what level of work they were subsequently able to cope with.

Disorders Associated with Muscle Fasciculations or Myotonia

A variety of muscle disorders exist in the horse that can cause muscle fasciculations, muscle cramping, and sometimes recumbency. These include metabolic alkalosis combined with hypocalcemia, hypomagnesemia, infestation with ear ticks, hyperkalemic periodic paralysis, myotonia congenita, and dystrophic myotonia. With the exception of ear ticks, these disorders are not associated with a significant elevation in serum CK activity.

Hypocalcemia in Horses

Hypocalcemia (lactation tetany, transport tetany, idiopathic hypocalcemia, and eclampsia) is a rare disorder in horses. Clinical signs of increased muscle tone may resemble tetanus. Horses may show a stiff, stilted gait; hindlimb ataxia; muscle fasciculations (especially temporal, masseter, and triceps muscles); trismus; dysphagia; salivation; anxiety; profuse sweating; tachycardia; elevated body temperature; cardiac dysrhythmias; synchronous diaphragmatic flutter; convulsions; coma; and death.[120-122] Clinical signs of excitability usually are seen when serum calcium values are below normal but above 8 mg/dl. Values of 5 to 8 mg/dl usually produce tetanic spasms and incoordination. Concentrations below 5 mg/dl usually result in recumbency and stupor. This disorder often progresses and may cause death within 48 hours, particularly in lactating mares. Metabolic alkalosis, hypomagnesemia/hypermagnesemia, and hyperphosphatemia/hypophosphatemia have all been found in association with hypocalcemia in horses.[121,122]

Treatment involves the intravenous administration of calcium solutions, such as 20% calcium borogluconate, or those recommended for the treatment of parturient paresis in cattle. Administration of these solutions at the rate of 250 to 500 ml/500 kg, diluted 1:4 with saline solution or dextrose, often results in full recovery, although in some horses recovery may take several days.[120] Relapses do occur. If no response to an initial infusion occurs, a second dose may be given 15 to 30 minutes later. Cardiac rate and rhythm should be monitored closely when calcium-containing solutions are infused intravenously.

Ear Tick–Associated Muscle Cramping

Intermittent painful muscle cramps, not associated with exercise, have been described in horses with severe *Otobius megnini* infestations.[123] These horses show intermittent signs of severe muscle cramping of pectoral, triceps, abdominal, or semitendinosus/semimembranosus muscles, lasting from minutes to a few hours, with severe pain that often resembles colic. Horses may fall over when stimulated. Between muscle cramps, horses appear to be normal. Percussion of triceps, pectoral, or semitendinosus muscles results in a typical myotonic cramp. Horses have serum CK activities ranging from 4000 to 170,000 U/L. Numerous ear ticks, *O. megnini*, can be identified in the external ear canal of affected horses. Without treatment for ear ticks, the spasms continue. However, local treatment of the ear ticks, using pyrethrins and piperonal butoxide, results in recovery within 12 to 36 hours. Acepromazine is helpful to relieve painful cramping.

Myotonia

Myotonic muscle disorders represent a heterogeneous group of clinically similar diseases that share the feature of delayed relaxation of muscle after mechanical stimulation or voluntary contraction. Skeletal muscle ion channel dysfunction, producing abnormal muscle membrane excitability, appears to be the shared abnormality among myotonias in many species.[124,125]

Foals with myotonia congenita usually have conspicuously well-developed musculature and mild hindlimb stiffness.[126,127] Gait abnormalities usually are most pronounced when exercise begins and frequently diminish as exercise continues. Bilateral bulging (dimpling) of the thigh and rump muscles is often obvious and exacerbated by stimulation of affected muscles by percussion. Affected muscles may remain contracted for up to a minute or more, with subsequent slow relaxation.

A diagnosis is best made by electromyography. High-frequency crescendo-decrescendo action potentials, with a classic dive-bomber pattern, are characteristic for myotonia. Muscle biopsies may reveal an increased proportion of type I fibers, fiber hypertrophy, fiber splitting, and centrally located nuclei.

Foals with myotonia congenita usually do not demonstrate progression of clinical signs beyond 6 to 12 months of age. A variety of breeds of horses have been described with this disorder, but an inherited basis for myotonia has not been established.[128,129] Phenytoin may diminish clinical signs, but no long-term treatment is available.[130,131]

A more severe, progressive form of myotonia that eventually results in debilitating muscle atrophy, fibrosis, and stiffness has been observed in Quarter Horse foals. At birth, foals appear well muscled and slight stiffness disappears with exercise. As the condition progresses, muscle hypertrophy is less obvious and exercise may produce debilitating muscle contractures. Foals may become so stiff that they are unable to stand. This condition resembles myotonia dystrophica in people, in that numerous organ systems may be involved.[131] Retinal dysplasia, lenticular opacities, and gonadal hypoplasia have been reported in one such Quarter Horse foal.[131,132] In people, abnormal sodium channel regulation has been identified in myotonic dystrophy, with reduced protein kinase activity.[125] A diagnosis is established by electromyography and muscle biopsy. Electromyography reveals increased numbers of high-frequency dive-bomber–like electrical discharges. Muscle biopsies show fiber-type grouping, increased numbers of cells with centrally located nuclei, sarcoplasmic bodies, ring fibers, and fibrosis. The prognosis is extremely poor and most foals are humanely destroyed by 1 year of age.

Stiff Horse Syndrome

A condition called *stiff horse syndrome* has been described in Belgium, characterized by stiffness and muscle spasms, associated with an abnormally straddled posture.[133] The muscle spasms typically were triggered by voluntary movements, such as going to eat from the manger, or being led from the stable. Electromyography showed persistent motor unit activity in the axial and gluteal muscles. The clinical signs were responsive to oral prednisolone therapy but recurred after treatment stopped. Several similar horses have been seen in the United Kingdom.[134,135] The cause of the condition is currently unknown. Similar conditions have been recognized in people, some of which are thought to be autoimmune.

HYPERKALEMIC PERIODIC PARALYSIS

• Sharon J. Spier

Hyperkalemic periodic paralysis (HyPP) is an autosomal dominant trait affecting Quarter Horses, American Paint horses, Appaloosas, and Quarter Horse crossbreed animals worldwide.[136-138] The genetic disease has been linked to a prolific Quarter Horse sire named Impressive. Current estimates indicate that 4% of the Quarter Horse breed may be affected.[139] Affected horses may have been selected preferentially as breeding stock because of their pronounced muscle development, and evidence exists of selection of HyPP-affected horses as superior halter horses by show judges.[140]

Clinical Signs

Clinical signs among horses carrying the same mutation range from asymptomatic to daily muscle fasciculations and weakness. In most horses intermittent clinical signs begin by 2 to 3 years of age, with no apparent abnormalities between episodes.[136,137] Ingestion of diets high in potassium (>1.1%)—such as those containing alfalfa hay, molasses, electrolyte supplements, and kelp-based supplements—or sudden dietary changes commonly trigger episodes.[141] Fasting, anesthesia or heavy sedation, trailer rides, and stress also may precipitate clinical signs. However, the onset of signs is often unpredictable, without a definable cause. Other possible precipitating factors that have been noted in people and horses are exposure to cold, fasting, pregnancy, concurrent disease, and rest after exercise. Exercise itself does not appear to stimulate clinical signs, and serum CK shows no change to modest increases during episodic fasciculations and weakness.

In most horses clinical episodes begin with a brief period of myotonia, with some horses showing prolapse of the third eyelid. Sweating and muscular fasciculations are observed commonly in the flanks, neck, and shoulders. The muscle fasciculations become more generalized as additional muscle groups are involved. Stimulation and attempts to move may exacerbate muscular fasciculations. Some horses may develop severe muscle cramping. Muscular weakness during episodes is a common characteristic of HyPP. Horses remain standing during mild attacks. In more severe attacks clinical signs may progress to apparent weakness, with swaying, staggering, sitting on the haunches, or recumbency within a few minutes. Heart and respiratory rates may be elevated and horses may show manifestations of stress, yet remain relatively bright and alert during episodes. Affected horses usually respond to noise and painful stimuli during clinical manifestations of the disorder. Episodes last for variable periods, usually from 15 to 60 minutes. Several horses have died during acute episodes.[136,137] Respiratory distress occurs in some animals as a result of paralysis of upper respiratory muscles, and a tracheostomy may be necessary. In addition, young horses that are homozygous for the HyPP trait have been observed to manifest a respiratory stridor and periodically may develop obstruction of the upper respiratory tract.[141,142] Horses homozygous for HyPP may show dysphagia or respiratory distress. Endoscopic findings include pharyngeal collapse and edema, laryngopalatal dislocation, and laryngeal paralysis. Once the episode subsides, horses show no apparent or minimal gait abnormalities. Although HyPP horses appear normal between attacks, electromyographic examination of affected horses reveals abnormal fibrillation potentials, complex repetitive discharges with occasional myotonic potentials, and trains of doublets between episodes.[137]

Cause

HyPP is caused by a point mutation that results in a phenylalanine/leucine substitution in a key part of the voltage-dependent skeletal muscle sodium channel alpha subunit.[138] In horses with HyPP, the resting membrane potential is closer to firing than normal horses. Sodium channels normally are activated briefly during the initial phase of the muscle action potential. The HyPP mutation results in a failure of a subpopulation of sodium channels to inactivate when serum potassium concentrations are increased. As a result, an excessive inward flux of sodium and outward flux of potassium ensues, resulting in persistent depolarization of muscle cells and temporary weakness.

Diagnosis

Descent from the stallion Impressive on the sire's or dam's side in a horse with episodic muscle tremors is strongly suggestive of HyPP. Quarter Horse foals born after 1998 that are offspring of an affected parent have a statement recommending DNA testing for HyPP on the Certificate of Registration. In most horses with hyperkalemia (6 to 9 mEq/L), hemoconcentration and hyponatremia occur during clinical manifestations of the disease, with normal acid-base balance.[136,137,142] Serum potassium concentration returns to normal after abatement of clinical signs. Some affected horses may have normal serum potassium concentrations during minor episodes of muscle fasciculations. Differential diagnoses for hyperkalemia include delay before serum centrifugation, hemolysis, chronic renal failure, and severe rhabdomyolysis.

Because clinicians may not be present during acute episodes, the definitive test for identifying HyPP is demonstration of the base-pair sequence substitution in the abnormal segment of the DNA encoding for the alpha subunit of the sodium channel.[140] Submission of mane or tail hair should be made to appropriate genetic laboratories.*

*Readers are referred to the Web site http://www/vgl.ucdavis.edu/tsthypp.htm; information on international submissions is included on this site.

Treatment

Low-grade exercise can sometimes abort an episode of mild disease or if horses are just beginning to exhibit clinical signs. Feeding grain or corn syrup to stimulate insulin-mediated movement of potassium across cell membranes also may be helpful. Other treatment options that may abort an episode include intra-muscular administration of epinephrine (3 ml of 1:1000/500 kg) and administration of acetazolamide (3 mg/kg orally every 8 to 12 hours). Many horses experience spontaneous recovery from episodes of paralysis and appear normal by the time a veterinarian arrives.

With severe clinical signs, administration of calcium gluconate (0.2 to 0.4 ml/kg of a 23% solution diluted in 1 L of 5% dextrose) often provides immediate improvement. An increase in extracellular calcium concentration raises the muscle membrane threshold potential, which decreases membrane hyperexcitability. To reduce the serum potassium, intravenous dextrose (6 ml/kg of a 5% solution) alone, or combined with sodium bicarbonate (1 to 2 mEq/kg), can be used to enhance intracellular movement of potassium. With severe respiratory obstruction, a tracheostomy may be necessary.

Control

Decreasing dietary potassium and increasing renal losses of potassium are the primary steps taken to prevent HyPP episodes.[143,144] Feedstuffs to avoid include high-potassium feeds, such as alfalfa hay, brome hay, canola oil, soybean meal or oil, and sugar molasses and beet molasses. Optimally, later cuts of timothy or bermuda grass hay; grains such as oats, corn, wheat, and barley; and beet pulp should be fed in small meals several times a day. Regular exercise and frequent access to a large paddock or yard are also beneficial. Pasture works well for horses with HyPP, because the high water content of pasture grass makes it unlikely that horses will consume large amounts of potassium in a short period of time. Ideally, horses with recurrent episodes of HyPP should be fed a diet containing between 0.6% and 1.1% total potassium concentration. Because of the wide variation in potassium concentration of forages depending on maturity and soils, it is advisable to have feeds analyzed for potassium concentrations and other nutrient requirements.

For horses with recurrent episodes of muscle fasciculations despite dietary alterations, acetazolamide (2 to 4 mg/kg orally every 8 to 12 hours) or hydrochlorothiazide (0.5 to 1 mg/kg orally every 12 hours) may be helpful.[142-144] Effects of these agents are exerted through different mechanisms; however, both cause increased renal potassium adenosine triphosphatase activity. In addition, acetazolamide stabilizes blood glucose and potassium levels by stimulating insulin secretion. Breed registries and other associations have restrictions on the use of these drugs during competitions.

Prognosis

In most horses, HyPP is a manageable disorder, although recurrent bouts may occur and severe episodes may be fatal. Owners of affected horses should be strongly discouraged from breeding these animals for the long-term health of the Quarter Horse and other breeds. Breeding of an affected horse to a normal horse results in a 50% chance of producing a foal with HyPP. Owners of affected horses should advise veterinarians of HyPP status before anesthesia or procedures requiring heavy sedation.

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SECTION • 1
Traditional Therapy

CHAPTER • 85

Principles and Practices of Joint Disease Treatment

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Recent advances reveal that osteoarthritis is a dynamic process, unlike previous concepts of a passive wear-and-tear phenomenon. Although many questions remain unanswered, our understanding of the pathophysiology of osteoarthritis and biological effects of therapeutic agents has expanded. Technical improvements in imaging modalities and arthroscopic surgical techniques allow early diagnosis and have broadened our perspective, paving the way for future progress in managing equine osteoarthritis.

MEDICAL TREATMENT

Pain (lameness) is the main reason most horses are examined, and rapid resolution of signs is the principal concern of most owners and trainers. Ideally, medical management of osteoarthritis would serve to arrest or at least slow the progression of lesions, a treatment objective termed *chondroprotection* or *disease modification*. Several factors influence therapeutic decisions in the medical management of osteoarthritis in horses, including the following:

1. The specific joint involved
2. The stage of osteoarthritis
3. The current and intended use of the horse
4. The age of the horse
5. Regulations governing anti-inflammatory medication use in competition
6. Treatment cost
7. Response to therapy

Therapeutic decision making also is complicated by the inherent nature of osteoarthritis, a condition that progresses at a variable rate and with signs difficult to quantify. Also, important degenerative changes and structural damage of cartilage matrix may precede the development of clinical signs, and late diagnosis precludes the possibility for complete resolution. Variability in response of horses with similar lesions supports the contention that osteoarthritis is a spectrum of disease. Despite recent progress, established osteoarthritis is incurable and frequently limits the serviceability of affected horses.

Non-Steroidal Anti-Inflammatory Drugs

Non-steroidal anti-inflammatory drugs (NSAIDs) are agents that inhibit one or more reactions involved in the production of prostaglandins and thromboxanes. Prostaglandins, particularly those of the E series, are associated with synovial inflammation and cartilage matrix depletion,[1,2] and although the specific effects of prostaglandins on joint metabolism are unclear, it is widely believed that prostaglandin E_2 plays a role in processes leading to joint degeneration. Prostaglandin E_2 has been demonstrated in the synovial fluid of horses with osteoarthritis,[3] and in vitro studies indicate that synoviocytes and chondrocytes synthesize prostaglandin E_2 after exposure to other inflammatory mediators.[4-6] Prostaglandin E_2 was implicated in the erosion of cartilage and adjacent bone,[7] and other studies suggest that prostaglandins may modulate latent metalloproteinase release and subsequent cartilage matrix degradation.[8,9] Paradoxically, reports suggest that matrix metalloproteinase expression in human synovial fibroblasts is inhibited by E-series prostaglandins.[10] These and other data suggest that prostaglandin E_2 may serve a regulatory function in inflamed joints and inhibition may not produce uniformly favorable results.[11] Thus recent evidence suggests that prostaglandin inhibition, although effective in providing symptomatic relief, may have deleterious effects on cartilage metabolism in the long term.[12] Establishing the specific biological effects of prostaglandin E_2 and the consequences of its inhibition in the equine joint is important, given the widespread use of NSAIDs in osteoarthritis treatment and the recognized toxic side effects that can result.

The principal action of most NSAIDs is the inhibition of cyclooxygenase (COX), the first in a series of enzymes responsible for converting arachidonic acid to prostaglandins (Fig. 85-1). Importantly, two forms of COX exist: the first produces physiological levels of prostaglandins in a constitutive manner (COX 1) and the second, an inducible form of the enzyme (COX 2), is responsible for the elevated levels of

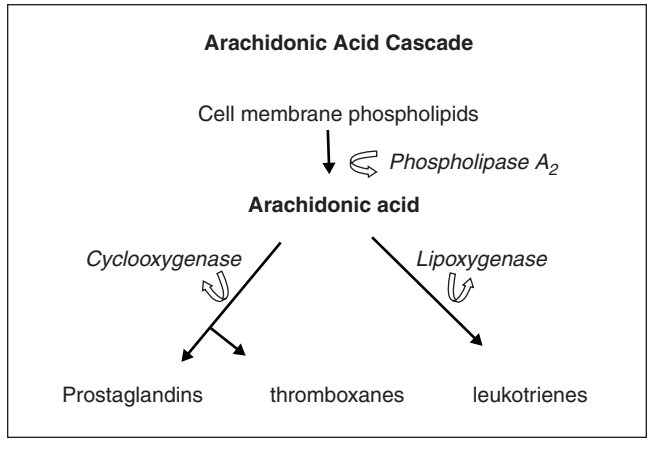

Fig. 85-1 Arachidonic acid cascade. Common therapeutic targets include phospholipase A_2 (corticosteroids) and cyclooxygenase (corticosteroids and non-steroidal anti-inflammatory drugs). Prostaglandins, particularly those of the E series, are associated with synovial inflammation and cartilage matrix depletion; however, accumulating evidence indicates that prostaglandin E_2 plays a role in regulating over-production of proteolytic enzymes by articular chondrocytes.

prostaglandins observed during inflammatory events.[13,14] The constitutive activity of COX 1 is responsible for many of the homeostatic properties ascribed to prostaglandins and toxicity is related to sustained COX 1 inhibition.[15,16] Most currently available NSAIDs inhibit the activities of both COX isoforms. However, the proportionate inhibition of COX 1 to COX 2 varies among compounds, and many aspects of the specific anti-inflammatory profiles of most NSAIDs used in horses remain to be explored in detail. A number of selective COX 2 inhibitors have been approved recently for use in people and to date have similar clinical efficacy to that of non-specific COX inhibitors, largely unaccompanied by typical NSAID side effects.[17-19] COX 2 inhibitors have been introduced to companion animal practice and likely will soon find use in horses, particularly when more affordable generic forms become available. This development may address existing problems with toxicity and potential unfavorable effects on cartilage metabolism of currently used NSAIDs.[20-22]

In addition to COX inhibition, NSAIDs have other anti-inflammatory effects. For example, carprofen reduces edema and effusion in experimental osteoarthritis in horses by a non–COX-mediated pathway.[23] Ketoprofen inhibits lipoxygenase and COX.[24] Moreover, at least some NSAIDs are capable of inhibiting elements of cellular inflammation.[25,26] Thus it is clear that at least some NSAIDs possess anti-inflammatory actions other than COX inhibition, although the biological significance of these effects is uncertain and may occur only at tissue concentrations achieved by exceeding normal dosages. These side actions may complicate the premise that selective COX 2 inhibitors are devoid of side effects, particularly when used at higher dosages where effects unrelated to COX inhibition are evident.

Pain relief from NSAIDs is mainly, but not exclusively, related to COX inhibition. Prostaglandins do not produce pain, except when present in large quantity.[27] Prostaglandin E_2 sensitizes peripheral nerve endings to mechanical stimuli and amplifies the chemical activation of pain receptors by other inflammatory mediators such as bradykinin and histamine, both of which act to lower the pain threshold.[28,29] Reducing prostaglandin levels also appears to modulate pain perception centrally, at the level of spinal receptors distant from sites of inflammation.[30] In addition to COX inhibition, certain NSAIDs possess other mechanisms of analgesia, because COX inhibitory activity of some compounds is not correlated closely with analgesic potency.[31] For example, the R-enantiomer of flurbiprofen is a weak COX inhibitor compared with its D-enantiomer, but it has comparable analgesic potency.[32] Among other ancillary mechanisms, NSAIDs may contribute to analgesia by inhibiting sensory neurotransmitter synthesis at a spinal level.[33]

At present, phenylbutazone is the least expensive and most popular agent used in horses, and its clinical efficacy appears to compare favorably with other NSAIDs.[34] (Table 85-1). Other choices include flunixin meglumine, meclofenamic acid, naproxen, ketoprofen, and carprofen, for which the basic pharmacology in horses was recently reviewed.[35,36] It should be noted that considerable variation exists in the pharmacokinetic profiles of NSAIDs among horses, and clearance is influenced by a variety of factors such as dose, the presence or absence local inflammatory conditions, and feeding schedule (for orally administered drugs).[37-40] The latter effect can be a consideration in estimating withdrawal times, given that the peak plasma concentration and apparent half-life can be substantially delayed when NSAIDs are given to horses with

Table • **85-1**

Non-Steroidal Anti-Inflammatory Drugs Used to Treat Musculoskeletal Pain

GENERIC NAME	TRADE NAME(S)	FORMULATION(S)	RECOMMENDED DOSE (mg/kg)	RELATIVE PRICE (per day)
Phenylbutazone*	Phenylbutazone injection Bizolin 200 Equipalazone Equiphen paste etc.	Tablets Paste Granules/powder Injectable (IV only)	4.4 bid 1, 2.2 bid (4 days), then 2.2 sid (IV, PO)	(Dose is 2.2. mg/kg bid) Tablets (1 g) = 1.0 Paste (12 g) = 2.8 Injectable = 6.9
Flunixin meglumine†	Banamine Citation Meflosyl, etc.	Granules Paste	1.1 daily for 5 days (IV, IM, PO)	(Dose is 1.1 mg/kg sid) Granules (500 mg envelope) = 5.0 Paste (1500 mg tube) = 12.4 Injectable (50 mg/ml) = 6.8
Meclofenamic acid†	Arquel	Granules	2.2 sid (5-7 days), then 2.2 sid or less (PO)	(Dose is 2.2 mg/kg sid) Granules = 1.6
Naproxen†	NM	Tablets	10 sid for up to 14 days (PO)	(Dose is 10 mg/kg sid) Tablets (500 mg) = 7.1
Ketoprofen†	Ketofen	Injectable	2.2 sid (IV)	(Dose is 2.2 mg/kg sid) Injectable (100 mg/ml) = 27.4
Carprofen†	Rimadyl‡	Injectable	0.7 sid (IV)	(Dose is 0.7 mg/kg sid) Tablets (100 mg) = 4.2

NM, Not currently marketed by veterinary distributors in the United States.
*Enolic acid.
†Carboxylic acid.
‡Only available in tablet form in the United States.

access to hay. Dose rates and schedules may have to be adjusted for individual horses to accommodate these effects.

The chondroprotective (and potential deleterious) effects of NSAIDs have been investigated in vitro, although the clinical relevance of the results is not clear. Certain NSAIDs are known to inhibit anabolic activities in chondrocytes, whereas others stimulate matrix synthesis.[41-44] Concern over the potential unfavorable side effects of these agents began with the observation that aspirin inhibited proteoglycan synthesis and could encourage cartilage destruction[45,46] and that ibuprofen was implicated in accelerated joint destruction in people with osteoarthritis.[47,48] Fears of enhanced rates of cartilage degradation with NSAID use were not borne out in a number of clinical and experimental studies.[49,50] In vitro data may not parallel ex vivo data in the same species. For example, indomethacin reduces proteoglycan synthesis in canine cartilage explants; however, cartilage obtained from indomethacin-treated and control dogs had similar synthetic rates.[44] Indeed, certain NSAIDs have shown to be chondroprotective in some osteoarthritis models.[51-53]

Similar to the effects on cartilage matrix synthesis, NSAIDs vary in the ability to inhibit both catabolic events in cartilage and the effects of degradative enzyme activity.[54] Although many studies have focused on direct inhibition of degradative enzymes, NSAIDs may be of benefit by suppressing other mediators.[55,56] For example, in one of the few equine studies, phenylbutazone limited proteoglycan depletion that accompanies in vitro culture of articular cartilage,[57] but the effect was not mediated by stromelysin inhibition.[58]

Corticosteroids

Corticosteroids are the most potent anti-inflammatory agents used to treat osteoarthritis. Normally injected directly into affected joints, corticosteroids depress numerous inflammatory processes, including capillary dilatation; margination, migration, and accumulation of inflammatory cells; and liberation of enzymes, cytokines, and other inflammatory mediators.[59,60] Notably corticosteroids also inhibit prostaglandin production by inhibiting phospholipase A_2 and COX 2 (but not COX 1).[61] The consequence of inhibiting arachidonic acid metabolism is a rapid and pronounced reduction in pain.

A long-standing controversy surrounds the use of corticosteroids in treating osteoarthritis because of the concern that overuse of a pain-free joint could result in accelerated degeneration.[62-64] This impression has been compounded by demonstrated negative effects of corticosteroids on chondrocyte metabolism. Particularly at high concentrations, corticosteroids inhibit proteoglycan synthesis and unfavorably influence the structural organization of cartilage collagens, even in normal cartilage.[65-70] Conversely, other studies have demonstrated that corticosteroids have certain chondroprotective properties. At low doses, they are capable of inhibiting the process of cartilage degradation by inhibitory effects on the synthesis of matrix metalloproteinases (MMPs) and cytokines[71-73] These in vitro data are supported by results in animal models demonstrating cartilage-sparing effects of low-dose corticosteroids, without significant effects on chondrocyte health.[74-76] It is well established that corticosteroids inhibit the gene expression of MMPs; however, this effect must be weighed against a similar effect on the expression of the natural inhibitory protein of these enzymes (tissue inhibitor of matrix metalloproteinase, or TIMP) and matrix collagen and proteoglycan synthesis.[77,78] The magnitude and relative contributions of these inhibitory effects need to be defined more fully, although it may be possible to determine a concentration of drug that produces chondroprotective effects while minimizing negative consequences on chondrocyte anabolic activity.

Although proteoglycan depletion with high doses of corticosteroids is an incontestable phenomenon, its importance as a clinical entity is contentious.[79-81] Noteworthy are recent studies using horses with iatrogenic osteochondral lesions of the carpus treated with betamethasone and triamcinolone acetonide. After treatment horses were exercised on a treadmill. Lesions of cartilage and bone in treated horses were comparable to those in controls.[82,83] To investigate the possibility that different corticosteroid preparations may exert varied effects on joint metabolism, a similar protocol was conducted by the same research group, using methylprednisolone acetate. In this study, morphological lesions were observed that the authors considered related to corticosteroids.[84] It should be noted that, as in other studies evincing unfavorable effects of methylprednisolone acetate, the dose used (100 mg the first and fourteenth days) would be considered generous by current standards. Additionally, the effects of methylprednisolone acetate on chondrocyte metabolism differ between normal and inflamed joints. In inflamed joints modest concentrations of methylprednisolone acetate may have less dramatic effects on matrix synthesis than in normal joints.[85] In summary, although intra-articular use of corticosteroids may potentially aggravate existing cartilaginous lesions, the potential deleterious effects are overemphasized.

The type, dose, and frequency of corticosteroid administration remain subjective. Common choices and dose ranges are given in Table 85-2. In general, pharmacokinetic data suggest that corticosteroid suspensions have short intra-articular half-lives.[86-88] Considerable variation in the clearance of corticosteroid suspensions exists. For example, the active moiety of methylprednisolone acetate can be liberated for up to 1 month.[89] The specific reasons for divergent pharmacokinetic profiles and the duration of anti-inflammatory effect far exceeding intra-articular half-life of corticosteroids are unclear.

Table • **85-2**

Corticosteroid Suspensions Used Intra-Articularly

CORTICOSTEROID	TRADE NAME	MANUFACTURER	CONCENTRATION (mg/ml)	DOSE (mg)*	POTENCY RELATIVE TO HYDROCORTISONE
Betamethasone sulfate	Celestone Soluspan	Schering-Plough	6†	3-18	30
Triamcinolone acetonide	Vetalog	Solvay	6	6-18	5
Flumethasone	Flucort	Syntex	0.5	1.25-2.5	120
Isoflupredone acetate	Predef 2X	Pharmacia and Upjohn	2	5-20	50
Methylprednisolone acetate	Depo Medrol	Pharmacia and Upjohn	40	40-120	5

*Dose ranges are somewhat arbitrary. Magnitude and duration of clinical effects vary widely.
†Each milliliter contains betamethasone acetate (3 mg) and betamethasone sodium phosphate (3 mg).

Most clinicians currently use lower doses of corticosteroids than their predecessors, because the dose-dependent deleterious effects are now recognized and most are now aware that favorable response is achieved using lower doses. For example, intra-articular injection of 120 to 200 mg of methylprednisolone acetate was commonplace years ago, but now it is performed using 10 to 40 mg, depending on the specific joint involved. Another popular and potentially beneficial practice is the co-administration of a low dose of a depo-corticosteroid and hyaluronan. Therapeutic synergy was reported for people with osteoarthritis, and a similar effect in horses is possible.[90] Many veterinarians (and trainers) find the idea of combination therapy appealing, because the corticosteroid dose can be reduced and it can be combined with the putative cartilage-sparing effect of hyaluronan. The minor risk of infectious arthritis exists with any intra-articular injection, and after intra-articular corticosteroids, signs of infection may be delayed from the potent anti-inflammatory effects of these preparations.[91]

Hyaluronan (Sodium Hyaluronate)

Hyaluronan is a glycosaminoglycan composed of the disaccharides D-glucuronic acid and *N*-acetyl-*D*-glucosamine. This glycosaminoglycan is an important component of articular cartilage, where it plays a major role in forming proteoglycan aggregates. The viscoelasticity of synovial fluid is due to its rich hyaluronan content and this molecule serves as the principal lubricant of synovial soft tissues. These physical properties, along with other incompletely characterized pharmacological effects, help maintain synovial homeostasis. The biological characteristics and therapeutic use of hyaluronan in equine osteoarthritis have been reviewed.[92] Characteristics of hyaluronan formulations are summarized in Table 85-3.

Despite not knowing the specific mode(s) of action, hyaluronan frequently is used to treat osteoarthritis in people and horses. Documented clinical benefits after intra-articular injection in people include reduced pain and improved joint mobility,[93,94] and effects compare favorably with NSAIDs and corticosteroids.[90,95] Most equine studies report beneficial effects, but most lack appropriate controls and outcome criteria and are subjective.[96-98]

Hyaluronan has modest analgesic effects,[99] but it has mostly anti-inflammatory effects that may be physical (steric hindrance) or pharmacological (inhibition of inflammatory cells and mediators).[100-104] Symptomatic improvement can be dramatic. In an ovine osteoarthritis model, cartilage lesions in hyaluronan-treated animals were attributed to excessive limb use.[105-107] Hyaluronan may improve range of motion by improving synovial fluid viscosity and soft tissue lubrication. In osteoarthritis, reduced joint excursion may be caused in part by the dilution and depolymerization of endogenous hyaluronan that accompanies synovitis.[108-110]

The specific effects of hyaluronan on articular metabolism remain unclear. Exogenous hyaluronan may stimulate synthesis of a highly polymerized endogenous form of hyaluronan by synoviocytes, but evidence was lacking in studies using cultured synovial fibroblasts derived from normal equine joints or those from joints with osteochondral fragments.[111] Hyaluronan stimulates proteoglycan synthesis by equine chondrocytes.[112] In vitro, hyaluronan is protective for cartilage matrix loss induced by interleukin-1.[113-115] Recent studies have revealed that hyaluronan regulates the production of prostaglandins by articular cells and can protect cartilage against the effects of oxygen-derived free radicals and degradative enzymes.[102,103,116-118] However, compelling evidence for the ability of hyaluronan to inhibit the activity of MMPs has not been reported to date.[111,119] Interestingly, a number of studies have demonstrated that several inflammatory mediators, including prostaglandins, interferon, and certain protein factors, can augment the production of hyaluronan by synovial fibroblasts in vitro.[120,121] Therefore elevated synthesis of hyaluronan in early osteoarthritis may constitute a protective response by the synovium to joint inflammation, providing a rationale for exogenous administration. Like corticosteroids, the protracted pharmacodynamic

Table • **85-3**

Common Hyaluronan Preparations Used in Horses

TRADE NAME	MANUFACTURER	CONCENTRATION	MOLECULAR WEIGHT (IN DALTONS, FROM MANUFACTURER)	HOW SUPPLIED	RECOMMENDED DOSE*
Hylartin V (Hylartil Vet)	Pharmacia and Upjohn	10 mg/ml	3.5×10^6	2-ml syringe	20 mg
MAP-5	Vetrepharm	10.3 mg/ml (2 ml) 5 mg/ml (10 ml)	7.5×10^5	2-ml vial 10-ml vial	20 mg†
Hycoat	Neogen	5 mg/ml	$>1.0 \times 10^6$	6-ml vial	30 mg‡
Hyvisc	Vetmedica	11 mg/ml	2.1×10^6	2-ml syringe	20 mg
HY-50	Bexco Pharma	17 mg/ml		3-ml syringe	51 mg
Equron	Solvay Animal Health	5 mg/ml	$1.5\text{-}2.0 \times 10^6$	2-ml syringe	10 mg
Equiflex	Chesapeake Biological	5 mg/ml	1×10^6	5-ml vial	10 mg
Synacid	Schering-Plough	10 mg/ml	$0.15\text{-}0.20 \times 10^6$	5-ml vial	50 mg
Hyalovet (Hyalovet-20)	Fort Dodge/ Vetrepharm	10 mg/ml	$4\text{-}7 \times 10^5$	2-ml syringe	20 mg
Legend (Hyonate)§	Bayer Corporation	10 mg/ml	3×10^5	4-ml vial	40 mg (IV)

*Intra-articular dosages are those recommended for small- to medium-size joints (e.g., metacarpophalangeal). Some manufacturers recommend twice the dose for larger joints (e.g., tibiotarsal).
†Marketed as an ophthalmic preparation but popular for intra-articular use at this dose.
‡Marketed as a topical preparation for wounds but used intra-articularly.
§Marketed for both intravenous and intra-articular use.

influences of exogenously administered hyaluronan are attributable to indirect effects on synovial and cartilage metabolism, because intra-articular half-life is only several hours in normal joints[122,123] and is further reduced in synovitis.[124] Favorable in vitro effects of hyaluronan are supported by observations of cartilage-sparing effects after intra-articular administration of hyaluronan in animal models of arthritis.[106,107,125-128]

Hyaluronan preparations of a molecular weight exceeding 1×10^6 d may provide superior clinical and chondroprotective effects, although this claim is controversial.[129] Certainly, many potentially beneficial effects observed in vitro depend on molecular weight,[99,103,108,118] but whether these effects occur in vivo is unclear. Cartilage-sparing properties of a high molecular weight hyaluronan were superior to those of a less polymerized preparation in rabbits in a limb immobilization model,[128] and better clinical results in horses were reported when using hyaluronan exceeding 1×10^6 d, rather than lower molecular weight formulations.[130,131] Conversely, others maintain clinical efficacy is related to the purity of the preparation, rather than molecular weight.[98,132]

Recently a form of hyaluronan intended for intravenous use has become available. The precise mechanisms of action are uncertain, but beneficial clinical and biochemical effects were observed in an equine lameness model,[133] suggesting that the product need not cross the blood-synovial barrier to exert its action(s). When used in horses with synovitis, intravenous hyaluronan appears to decrease lameness and synovial effusion. Recent studies have demonstrated that hyaluronan receptors exist on numerous extra-articular cell types, and if involved with joint inflammation, intravenous hyaluronan may exert a positive effect.[134,135]

In the horse, hyaluronan appears to be most effective in treating acute synovitis, and results when used in more advanced disease are frequently disappointing. The frequency of hyaluronan administration for the treatment of osteoarthritis in people (often weekly) is higher than that typically used in the horse. Optimal results in horses may require more frequent injections than routinely recommended, particularly in those with established joint lesions. Although often heralded as being virtually free from side effects, data from animal models suggest that despite its putative chondroprotective properties, continued joint deterioration in treated animals may occur when the primary cause of osteoarthritis remains uncorrected.[105-107]

Polysulfated Glycosaminoglycan

Polysulfated glycosaminoglycan (PSGAG) is a semi-synthetic preparation from bovine trachea, comprised principally of chondroitin sulfate, a glycosaminoglycan found in the aggregating proteoglycan of cartilage. The molecular weight of PSGAG varies between 2 and 16 kd; the mean molecular weight is 6 kd. PSGAG is purported to have chondroprotective and anti-inflammatory properties; however, the exact nature and mechanism(s) by which it exerts these effects are unknown. Thorough reviews of the biological properties and clinical use of PSGAG are available in the veterinary literature.[136,137]

PSGAG reduces the severity of clinical signs in people and horses with arthritis.[138-143] Clinical improvement is likely attributable to anti-inflammatory effects, including the inhibition of prostaglandin E_2 synthesis and inhibition of cytokine release.[117,144,145]

PSGAG is reported to have numerous favorable effects on joint metabolism, including stimulation of hyaluronan production by synoviocytes and synthesis of proteoglycans and collagen by chondrocytes.[146-149] The latter effects are thought to be more pronounced in arthritic cartilage.[149] Despite the existence of in vitro studies supporting anabolic properties of PSGAG, subsequent research has questioned modes of action, particularly PSGAG putative stimulation of cartilage proteoglycan synthesis. Dramatic stimulatory effects on chondrocyte biosynthesis were not clearly demonstrable in studies using equine cartilage explants, particularly those in which normal or mildly osteoarthritic cartilage was used.[150,151] An anabolic response may be more evident in chondrocytes from joints with significantly deranged cartilage metabolism,[149] but clinical benefit of such response is questionable. Moreover, PSGAG has limited effects on the early healing of cartilage lesions, and repair tissue formed in healing cartilage wounds under the influence of PSGAG may be inferior to that in untreated animals.[152,153]

Although the nature and magnitude of its anabolic effects are unclear, PSGAG is widely held to have anti-catabolic effects. PSGAG is capable of inhibiting the activity of a number of degradative enzymes known to be present in articular tissues, including elastase, some cathepsins, serine proteases, and neutral MMPs.[58,136,137,154-156] Mechanisms of enzymatic suppression are characterized incompletely, but they may include direct inhibition or abrogation of the synthesis or activity of other mediators that stimulate degradative enzyme release. For example, our recent data (J.P.C.) indicate that PSGAG inhibits inducible nitric oxide synthase expression and synthesis in cytokine-stimulated equine chondrocytes in culture. A variety of animal models of arthritis have provided support for a cartilage-sparing effect of PSGAG in vivo,[153,157-160] and in most cases beneficial effects were attributed mainly to the inhibition of degradative enzymes.[155,160]

PSGAGs can be administered intra-articularly or intramuscularly, but slight risk of infection (quantitatively exceeding that of corticosteroids) with intra-articular administration of PSGAGs has reduced enthusiasm for this route.[161] Most clinicians now combine an aminoglycoside (e.g., amikacin, 125 mg) with PSGAG as a preventative measure. Articular cartilage concentrations of PSGAG after intramuscular administration are capable of inhibiting some cartilage-degrading enzymes,[162] yet the duration of a persistent effective concentration is unclear. As for other anti-arthritis preparations, the frequency of administration of PSGAG usually is based on the therapeutic response and its duration. Typically, when a favorable therapeutic response occurs, it is rapid. For intramuscular use the manufacturer recommends that PSGAG be given at a dose of 500 mg every 4 days for 28 days. Clinical experience suggests that if improvement is not evident within 7 to 10 days using this schedule, a dramatic response from subsequent injections is unlikely.

Pentosan Polysulfate

Pentosan polysulfate is a compound prepared by sulfation of beechwood hemicellulose, consisting of xylopyranose chains, to which are attached methylated glucuronyl rings at regular intervals. Like PSGAG, pentosan polysulfate is a highly sulfated molecule, a property that likely contributes to its biological activity.

Pentosan polysulfate appears to have chondroprotective effects similar to PSGAG, including promotion of anabolic activity of chondrocytes and synoviocytes and inhibition of degradative enzymes.[163,164] Studies using animal models support these chondroprotective effects.[161,163,165,166] Pentosan polysulfate is not presently available in North America but is approved for use in Australia, and anecdotally, pentosan polysulfate is effective in reducing lameness in racehorses with chronic osteoarthritis.[163] Experienced clinicians report pentosan polysulfate provides symptomatic relief in some horses that have been unresponsive to PSGAG. The recommended dosage regimen is 2 to 3 mg/kg intramuscularly every 7 days for 28 days, and the series can be repeated every 3 months as required.[163] Further clinical experience with pentosan polysulfate is required to define specific indications.

Glucosamine and Chondroitin Sulfate

Glucosamine and chondroitin sulfate are compounds extracted from animal products that have been used to treat osteoarthritis in people in Europe for more than a decade. In North America an interest in using these products in people and animals has developed. These supplements reportedly possess anti-inflammatory and disease-modifying effects, and because of the relative safety, they could have great use in managing osteoarthritis even if they are only modestly effective. In vitro and appropriately designed and rigorous clinical trials are lacking, but available data suggest beneficial effects.

Glucosamine sulfate is a precursor of the disaccharide subunits of cartilage proteoglycans. Glucosamine salts appear to be well absorbed after oral administration.[167] In vitro studies indicate that glucosamine sulfate increases proteoglycan synthesis by chondrocytes and may have a number of anti-inflammatory activities.[168-171] Glucosamine was protective for proteoglycan loss and inhibited MMP synthesis and activity in equine cartilage explants.[172] Follow-up experiments using a variety of glucosamine derivatives and sulfated glucose controls demonstrated that cartilage-sparing properties of glucosamine are shared by the 3-sulfate and hydrochloride salts but that glucose-3-sulfate and *N*-acetylglucosamine are devoid of chondroprotective effects at equimolar doses.[173] Glucosamine also appears to reduce the suppressive effects of some cytokines on cartilage proteoglycan synthesis.[174] The hydrochloride salt of glucosamine is less expensive than the sulfated forms and constitutes the principal form of glucosamine marketed in North America.

Chondroitin sulfate consists of chains of sulfated galactosamine and glucuronic acid molecules and is the principal glycosaminoglycan of aggregating proteoglycan (aggrecan). Chondroitin sulfate is less sulfated but resembles PSGAG in structure and mechanisms of acton. Data from in vitro studies have demonstrated anti-inflammatory properties of the drug on leukocytes.[175] Experiments have provided evidence of chondroprotective effects of chondroitin sulfate, including stimulating the synthesis of proteoglycans and inhibiting the activity of certain matrix degrading enzymes, particularly when chondroitin sulfate is present in a polymerized or long-chain form.[176-178] Chondroitin sulfate also has protective effects for cartilage proteoglycan loss in animal models of joint inflammation.[179,180]

Controversy persists regarding the efficacy of enteral absorption of chondroitin sulfate in a biologically active (long-chain) form. In monogastric species, the oral bioavailability is less than 20%,[175] and although isotope recovery is possible in animals fed radiolabeled chondroitin sulfate,[181] absorbed radioactivity may reside in small-chain or monomeric forms of the sugar that lack the biological effects of polymers.[182] Gastrointestinal absorption of polymerized chondroitin sulfate in the horse is unknown.

Clinical trials with chondroitin sulfate and glucosamine, used alone or in combination in people, revealed symptomatic relief similar to that of ibuprofen.[183] Unfortunately, most studies lack appropriate controls and are complicated by the co-administration of other medications.[184,185] Recommendations from recent meta-analyses, a type of study using stringent criteria for inclusion and statistical analyses of other published studies, call for additional trials with larger cohorts of patients studied for longer periods of time.[186,187] Interestingly, in military personnel with spinal and knee osteoarthritis, symptomatic relief was reported using a variety of subjective measures, but running times were unaffected by treatment.[188]

To date, clinical research in horses is limited,[189] and reported results vary from beneficial effects to limited value. Which horses will respond to oral supplementation is not known. Formulation of glucosamine and chondroitin sulfate–containing supplements is not regulated by the Food and Drug Administration (United States), so purity and content are not always ensured. Products with certified content from reputable sources should be used.

Common Practices in Medical Management

There are two principal objectives of medical management of the equine athlete: symptomatic relief and arresting progression of joint degeneration. As for people with rheumatic disease, no equine compound exists for which incontrovertible in vivo evidence of disease modification exists. Efficacy of most drugs is based subjectively on clinical experience. To assess trends in current practices of medical management of osteoarthritis, we sent questionnaires to 20 members of the American Association of Equine Practitioners for whom the treatment of performance horse lameness was particularly common. The survey included questions regarding the use and perceived efficacy of NSAIDs, corticosteroids, hyaluronan, PSGAG, and nutriceuticals (glucosamine and chondroitin sulfate–containing products). Fourteen responses were returned, including from five veterinarians working exclusively with Thoroughbred racehorses, two exclusively with Standardbred racehorses, four with Western or show horses (hunting, jumping, dressage, other show) or both, and three who treated racing and show breeds. Collectively, respondents treated an estimated 17,000 horses with osteoarthritis annually and most estimated that osteoarthritis accounted for at least 60% of their lameness case load.

Non-Steroidal Anti-Inflammatory Drugs

All respondents used NSAIDs and phenylbutazone was the most frequently used. Cited advantages included cost, efficacy, and anti-inflammatory potency compared with other compounds; ability to administer the drug by a variety of routes; and relative lack and predictability of toxic effects. NSAIDs were considered adjunctive and were seldom used alone.

Corticosteroids

All respondents but one used intra-articular injections of corticosteroids regularly (29%) or frequently (71%). Common indications for corticosteroid use included recurrent osteoarthritis in horses that had previously responded well to intra-articular medication and subacute or chronic osteoarthritis that was unresponsive to other therapies. Acute signs of osteoarthritis were managed less commonly with intra-articularly administered corticosteroids. Some clinicians combined corticosteroids with local anesthetic solutions when performing diagnostic analgesia. Corticosteroids were seldom used alone and in 50% of horses were used with intra-articularly administered hyaluronan.

Among the available products, methylprednisolone acetate was the most popular; it was used (although not exclusively) by 12 respondents. Among these veterinarians, eight opined that methylprednisolone acetate was more effective or had a greater duration of activity than other corticosteroid preparations. The second most popular preparation was triamcinolone acetonide. Dose varied among veterinarians and was also dependent on the volume of the synovial cavity being treated. About 50% of clinicians used methylprednisolone acetate and triamcinolone acetonide at relatively low doses (e.g., 40 mg and 6 mg per articulation, respectively), and the rest used higher doses (e.g., 100 to 120 mg and 12 to 18 mg per articulation, respectively). Intra-articularly administered corticosteroids were more popular and tended to be used at higher doses by racetrack clinicians than by those treating other sport horses. Overall, intra-articularly administered corticosteroids were considered effective for managing horses with acute synovitis and somewhat less so in those with established osteoarthritis (Fig. 85-2, *A*). Notably, estimates of efficacy were similar, regardless of whether low or high doses were used.

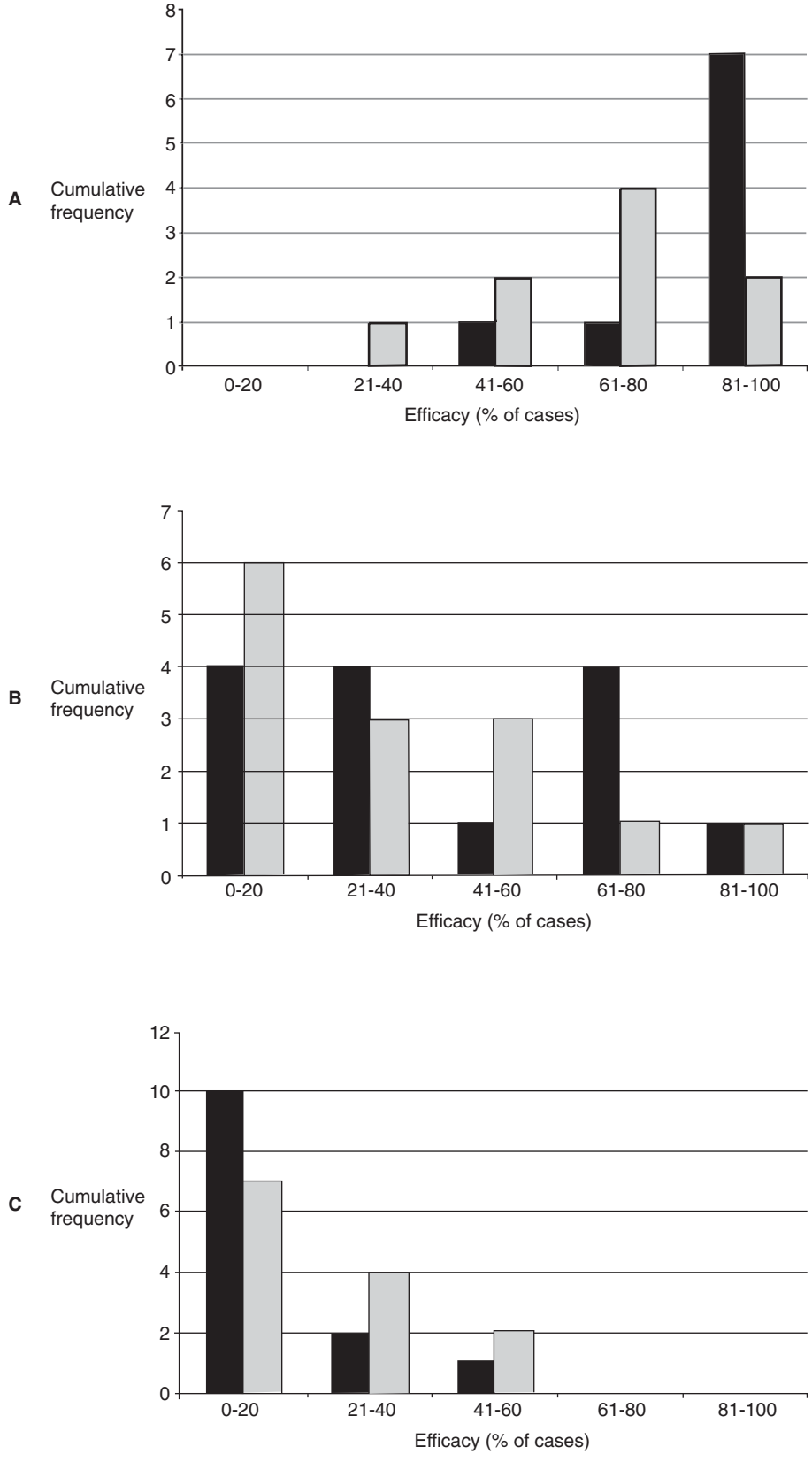

Fig. 85-2 Selected survey results to a joint disease medication survey of 14 members of the American Association of Equine Practitioners for whom the treatment of performance horse lameness was particularly common. The graphs represent the perceived efficacy of the symptomatic treatment of acute synovitis *(solid bars)* and established joint disease *(gray bars)* as follows: **A,** intra-articularly administered corticosteroids; **B,** intravenously administered hyaluronan; and **C,** orally administered glucosamine/chondroitin sulfate–containing nutriceutical products.

Hyaluronan

All respondents used intra-articularly administered hyaluronan, but for hyaluronan to be used initially or alone was uncommon, particularly in horses with established osteoarthritis. Twelve (86%) of 14 supplemented hyaluronan injections with other forms of treatment, most commonly concurrent administration of intra-articularly administered corticosteroids, particularly among those who treated Thoroughbred racehorses. Although combined use of corticosteroids and hyaluronan was also common among veterinarians treating show horses, two used hyaluronan alone 80% of the time.

Interestingly, seven clinicians chose to use hyaluronan preferentially in high-motion joints (e.g., middle carpal joint), whereas seven used the drug equally between high-motion and low-motion joints (e.g., centrodistal and tarsometatarsal joints). A variety of hyaluronan preparations were used and respondents were divided in their views on the relative efficacy of high and low molecular weight products. An unbiased evaluation of the therapeutic efficacy of intra-articularly administered hyaluronan was hampered by the co-administration of a corticosteroid preparation.

Most clinicians had experience treating horses with intravenous hyaluronan, but few, regardless of the type of horse treated, were impressed by its efficacy, particularly when used alone (Fig. 85-2, *B*). They opined that effects on osteoarthritis signs were not dramatic and duration was short.

Polysulfated Glycosaminoglycan

PSGAG use was common. Six of seven racehorse veterinarians used intra-articularly administered PSGAG at least occasionally, whereas a similar number of non-racehorse veterinarians avoided the practice. Intra-articularly administered PSGAG was used most commonly in horses with recurrent clinical signs or those with sub-acute or chronic conditions that were unresponsive to other therapy. Indications for intramuscular use varied widely. In addition to the indications listed for intra-articular administration, intramuscularly administered PSGAG was used to treat horses with acute or recurrent chronic osteoarthritis, or both types. PSGAG was also used as a preventative measure. PSGAGs were used with other treatments and estimates of efficacy were diverse, ranging from completely ineffective, to constituting a major element of therapy.

Glucosamine and Chondroitin Sulfate

Most respondents had experience with nutriceuticals, but unlike other compounds they were used most commonly as a preventative measure, often at the request of an owner or trainer. These products were considered substantially less effective than the other medications (Fig. 85-2, C). Most considered these products adjunctive at best, and no trend favored one product over another.

Overall, considerable variation existed regarding medical management of osteoarthritis, but certain themes emerged. Phenylbutazone remains the most popular NSAID, was effective, and often is used with intra-articular injections. Intra-articular injections of corticosteroid and hyaluronan, often in combination, are frequently used to manage acute and chronic osteoarthritis in all sport horses. Opinions were divided regarding the purported superiority of high molecular weight hyaluronan when injected intra-articularly, but the consensus was that intravenously administered hyaluronan was of limited value. PSGAG use remains popular, but considerable variation existed in the indications for use and perceived efficacy. Little support was given for the routine therapeutic use of nutriceuticals.

Intra-Articular Medications: Practical Considerations

Intra-articularly administered medications are important in managing osteoarthritis, yet no clear consensus is apparent regarding specific practices and precautions. Given the gravity of post-injection infection, in particular when resistant bacteria such as methicillin-resistant *Staphylococcus aureus* are involved, strict adherence to aseptic technique is mandatory. An appropriate standard includes thorough surgical preparation of the intended injection site and using sterile gloves and single-use syringes and needles. Multiple dose bottles or vials of any preparation intended for intra-articular use should be avoided. Although long accepted, the practice of clipping or shaving the injection site has been challenged recently by a study demonstrating that bacterial numbers were comparable between surgically prepared clipped and unclipped injection sites of the equine carpus and distal interphalangeal joints.[190]

No consensus exists on duration of rest after an intra-articular injection, largely from lack of objective information. Supported by various reports, recommendations vary from immediate resumption of exercise to 30 days of rest from training after any injection. Variation in recommendations is not unique to the equine practitioner. In a survey of the members of the American College of Rheumatology, recommendations for rest after intra-articular corticosteroid injections in people varied from 48 hours or less to 1 week or more.[191] Clearly the nature and severity of osteoarthritis should be considered. Because compelling evidence exists that corticosteroids have inhibitory effects on cartilage matrix metabolism, the dose of corticosteroid must be considered. Mechanical properties of cartilage in corticosteroid-treated joints are altered,[192] and although normal loading of cartilage has beneficial metabolic effects, heavy loading can compound the inhibitory effects of corticosteroid on matrix synthesis.[193] Unfortunately, most studies have used generous corticosteroid doses and normal cartilage that responds differently to osteoarthritic cartilage.[82-84,194] Nonetheless, the deleterious effects of exercise after intra-articular corticosteroid injections have been over-rated.[82,83] Moreover, industry pressures often militate against prolonged periods of rest after injections. After considering all factors, we recommend horses be given 2 to 3 days of box (stall) rest with hand walking, followed by a gradual return to light training, after intra-articular injections of corticosteroid. Similar recommendations are given for injections with hyaluronan and PSGAGs, even though issues such as inhibition of matrix synthesis are less important with these drugs.

Despite a near complete lack of knowledge of effects of mixing local anesthetic solutions with corticosteroids for intra-articular injections, rheumatologists commonly mix both in the same syringe to reduce pain, dilute corticosteroid suspensions, reduce post-injection flare, and help diagnose the source of pain.[191] Equine veterinarians often combine the two solutions to investigate and treat a source of pain simultaneously. Conversely, some clinicians avoid a second intra-articular injection if local anesthetic solution was injected recently. Local anesthetic agents cause a mild inflammatory response in joints,[195] but it is unclear whether inflammation or drug interactions influence the therapeutic response to concurrently or sequentially injected medications or substantially increase the risks of post-injection infection. We do not recommend routine use of combined injections.

Post-injection flare causes pronounced synovial effusion and often substantial lameness and usually occurs 4 to 24 hours after injection. Rapid response to lavage and anti-inflammatory therapy is usually seen. However, appropriate antimicrobial therapy should be instituted, and synovial fluid samples should be submitted for culture and susceptibility testing and cytological examination.

Medical Management: Conclusions

From objective and subjective information presented, clearly specific recommendations for all situations are difficult to make. Management choices are still made based on general principles, past experience, and economic considerations,

and the evolution of these regimens often is determined by therapeutic response. Given the complexity and variations of osteoarthritis and paucity of specific knowledge regarding the effects of available medications, veterinarians still must tailor treatments individually.

SURGICAL TREATMENT

Diagnostic and Surgical Arthroscopy

The application of arthroscopic techniques to the horse has revolutionized the treatment of traumatic joint injuries.[196,197] The advantages and disadvantages of arthroscopic surgery are discussed in Chapter 23, but importantly, arthroscopic surgery is the imaging modality of choice to evaluate articular cartilage (Fig. 85-3), although magnetic resonance imaging may supersede it in the future, particularly in joints with limited surgical access (see Chapter 21). Diagnostic arthroscopy is particularly useful in horses without radiographically visible lesions.[198]

Arthroscopic surgery is useful in surgical management of osteoarthritis. Since the advent of arthroscopic surgery, fractures of the proximal, dorsal aspect of the proximal phalanx are once again removed, with favorable results. The practice had previously been suspended when arthrotomy was performed, because low-grade lameness attributed to capsular fibrosis or calcification was often seen.[196,199] Intra-articular lavage is a benefit from arthroscopic surgery, because inflammatory debris and cartilage fragments are washed from the joint. Joint lavage is itself an important consideration in managing inflamed joints. In people with osteoarthritis, joint lavage alone is comparable to other forms of medical therapy. In Standardbreds in training, injection of small volumes of saline solution combined with 3 weeks' rest was more effective than rest alone in alleviating clinical signs of traumatic arthritis.[200-203]

Arthroscopic surgery is most useful in surgical management of osteoarthritis by eliminating the inciting cause and preventing the progression of osteoarthritis rather than treating established lesions. The most common indication for arthroscopic surgery is the removal of osteochondral fragments of traumatic or developmental origin. The ideal candidate is a horse that has not been injected previously with corticosteroids. Prognosis is favorable for horses after arthroscopic surgery to remove small osteochondral fragments, but before secondary changes of osteoarthritis have developed. However, osteochondral fragments often occur with, or result from, osteoarthritis (see Chapter 39) and this reduces the prognosis.[204,205] Presence of radiographic changes of osteoarthritis before surgery reduces prognosis in any horse with osteochondral fragments. In horses with advanced radiographic evidence of osteoarthritis, arthroscopic surgery may be useful to assess the magnitude of cartilage damage and to provide temporary symptomatic relief, but it is of dubious value in making a positive impact on long-term prognosis. In horses with established osteoarthritis, client education is important and horses may best be managed medically. Case selection is most important when considering arthroscopic surgery or arthrotomy for management of any articular problem (see Chapter 23).

Specific arthroscopic techniques are well established and detailed descriptions are available.[196] Controversy lingers regarding the vigor with which debridement of articular tissues should be performed. Currently, minimal effective debridement is advocated because articular cartilage has limited capacity for intrinsic repair.[197] Conversely, diseased bone should be completely removed.

Care after surgery involves the principles of providing an environment for resolution of inflammation, providing stability and restricting joint motion to facilitate optimal healing of articular tissues, and preventing post-operative contamination. Rest is important to reduce articular trauma and to minimize disruption of fragile repair tissues. NSAIDs are administered for 3 to 5 days to reduce inflammation from surgical trauma. Appropriate bandaging and rehabilitation are important (see Chapters 87 and 92). Although popular, the intra-articular use of hyaluronan and PSGAG after surgery had no beneficial effects on healing experimental cartilage defects.[206,207] Prognosis depends on several factors, but most importantly the magnitude and location of cartilage damage should be considered. For example, horses with carpal osteochondral fragments with 50% or more cartilage or subchondral bone loss have a significantly inferior prognosis than those with less severe lesions.[208]

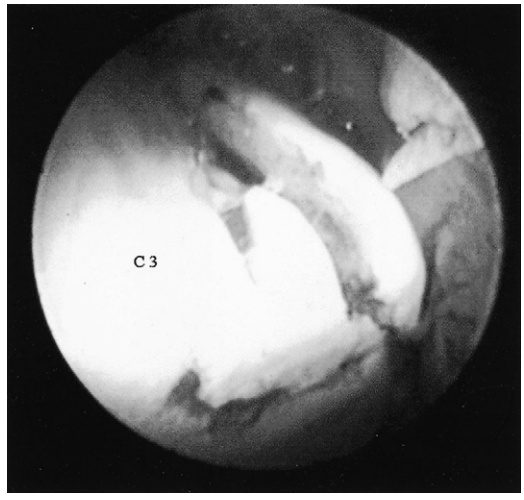

Fig. 85-3 Arthroscopic view (proximal is to the bottom) of the middle carpal joint in Standardbred racehorse with sizeable osteochondral fragments from the third carpal bone (C3). Beyond removal of osteochondral fractures, arthroscopic examination allowed determination of the magnitude of cartilage damage extending onto the weight-bearing surface of the third carpal bone and assessment of the degree of cartilage damage to the opposing surface of the radial carpal bone.

Internal Fixation of Intra-Articular Fractures

Common intra-articular fractures managed by lag screw techniques include third carpal bone slab fractures, lateral (and medial) third metacarpal/metatarsal bone condylar fractures, and sagittal fractures of the proximal phalanx. Internal fixation of displaced, intra-articular fractures involving sizable portions of the articular surfaces of these and other bones offers the greatest possibility for the horses to return to function. Repair of non-displaced fractures may improve healing time and substantially reduced risk of delayed displacement of fracture fragments. In addition, experimental evidence suggests that cartilage healing is superior with stable internal fixation of intra-articular fractures.[209]

Internal fixation can and should be combined with diagnostic arthroscopy. Arthroscopic surgery can be useful to assess cartilage damage, facilitate the identification and removal of small osteochondral fragments and debris in the fracture line, assess for comminution,[210] and evaluate reduction before implants are placed. The size and number of implants is dictated by fracture configuration and surgeon preference, and cortical bone screws are placed according to principles of the Association for the Study of Internal Fixation.[211,212] Appropriate positioning of implants to optimize reduction and congruity of articular components is facilitated by intra-operative radiography or fluoroscopy.

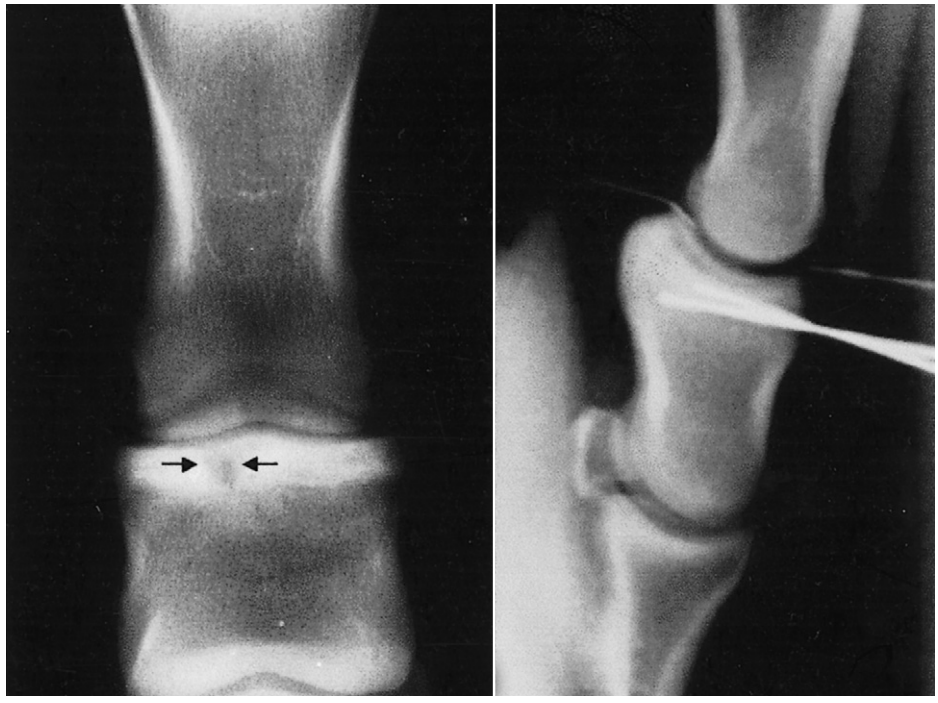

Fig. 85-4 **A,** Dorsopalmar and **B,** lateromedial radiographic views of the distal limb of a yearling with lameness referable to the proximal interphalangeal joint. **A,** There is an osseous cyst-like lesion (*arrows*) in the proximal aspect of the middle phalanx. **B,** The area was decompressed using transcortical drilling, and the procedure successfully abolished lameness.

Osseous Decompression

On occasion, joint pain may be referable to lesions of subchondral and epiphyseal bone that are not accessible arthroscopically. For example, subchondral cysts or osseous cyst-like lesions lacking a direct articular communication may occur under weight-bearing surfaces. Transcortical decompression has been successful in reducing lameness (Fig. 85-4). This empirical treatment is based on the principles of debridement of malacic bone and on the physiological premise that elevated metaphyseal pressures contribute to articular pain.

Synovectomy

Synovial resection, or synovectomy, involves removing inflamed, hypertrophic, or infected synovial membrane and can be accomplished surgically, with chemical agents, or using radioactive isotopes.[213] Synovial resection can be local (partial synovectomy) or generalized (subtotal or complete synovectomy).

In people with rheumatoid arthritis, early synovectomy provides temporary pain relief and reduces synovial inflammation. Symptomatic relief is typically more long-lasting for patients treated early in the course of the disease.[214,215] The rationale for the use of synovectomy in rheumatoid arthritis is intuitively clear, because the synovium has a well-established role in the disorder. In osteoarthritis the synovium plays a minor role in the disease process, and only mild response is observed in people with osteoarthritis treated by synovectomy.[216]

Specific indications and potential benefits of synovectomy in equine joint surgery have yet to be determined. Regeneration of the equine synovium after arthroscopically assisted synovectomy is slow compared with other species, but the procedure has negligible ill effects.[217-220] Arthroscopic synovectomy is considered beneficial in reducing bacterial load and attenuating profound synovitis in chronically infected equine joints.[221] Results of synovectomy in managing horses with chronic, unresponsive infectious arthritis of the tarsocrural joint are favorable (Fig. 85-5).

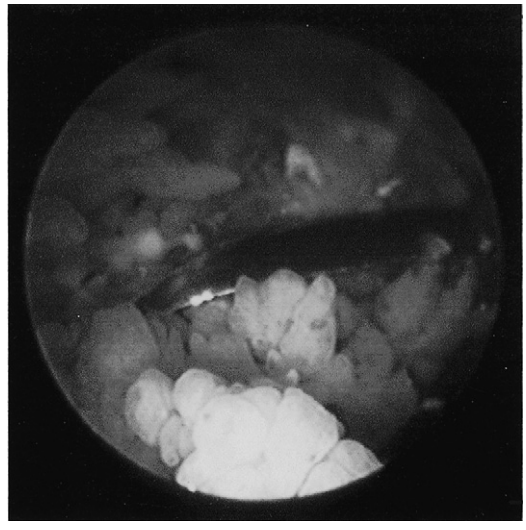

Fig. 85-5 Arthroscopic view of the tarsocrural joint of a racehorse with chronic active synovitis that was refractory to medical treatment. Greatly enlarged and club-like synovial fronds (under the stainless steel elevator) are evident. The gelding responded favorably to subtotal synovectomy.

To date, no compelling evidence supports the routine use of subtotal synovectomy in treating horses with osteoarthritis. Synovectomy was reported to be effective in reducing lameness from chronic proliferative synovitis of the fetlock and carpal joints of racehorses.[222]

Arthrodesis

In some horses, osteoarthritis is severe and cannot be managed medically or by conventional surgical techniques. Often concern

exists about laminitis of the contralateral limb and euthanasia may be considered if the horse has no residual value as a breeding prospect. In horses with chronic, severe, and refractory osteoarthritis of low-motion joints, such as the proximal interphalangeal and distal tarsal joints, arthrodesis can preserve athletic function.[223-227] Arthrodesis can be performed using internal fixation and external coaptation or by using chemical means.[228,229] Arthrodesis of high-motion joints, including the scapulohumeral, antebrachiocarpal or middle carpal, metacarpophalangeal, and distal interphalangeal joints, is performed as a salvage procedure.[230-233]

INVESTIGATIONAL TREATMENTS

Experimental treatments used in people may be applicable to horses. Active areas of research include cartilage resurfacing and limiting the effects of known biological mediators of osteoarthritis. Preliminary results using these approaches, alone or in combination, have been encouraging, though a number of biological and technical obstacles remain.

Joint Resurfacing

Cartilage loss is one hallmark of osteoarthritis. Cartilage has limited capacity for repair and considerable effort has been expended in attempts to augment intrinsic healing or to replace it. A number of approaches currently are being explored to provide a biochemically and biomechanically suitable repair tissue for equine osteochondral defects.

The healing of partial thickness cartilage defects is ineffectual, and the recruitment of a larger population of cells than that which exists in the adjacent matrix is required for optimal healing to occur. Attempts to improve the natural healing process typically involve debridement of the subchondral plate, or other methods to recruit pluripotent cells from the marrow cavity (stimulated endogenous repair), or the delivery of chondrogenic tissues, chondrocytes, or pluripotential cells to the defect (articular grafting). To date, the long-term efforts to modulate healing of cartilage defects have been disappointing, because formation of true hyaline cartilage has not occurred.[234-236] The biochemical composition of repair tissues frequently lacks appropriate macromolecules, specifically type II collagen, and a normal concentration of proteoglycans of suitable size and structure. Lacking the required composition, the biomechanical performance of the tissue is less than ideal and ultimately leads to its breakdown.

Stimulated Endogenous Repair

Stimulating endogenous repair in equine joints involves techniques to provide access of marrow elements to the cartilage defect (e.g., subchondral drilling), but results have not paralleled those in laboratory species. Problems of dedifferentiation of the repair tissue to a largely fibrous tissue composition have occurred. This fibrous tissue lacks the required functional characteristics.[237,238]

More recently, a technique involving the creation of microfractures in the subchondral plate to allow access of marrow pluripotential cells to the cartilage defect was developed. Known as *micropicking* or *breaching,* a stainless steel awl is used to create small crack-like defects in the articular end plate, usually under arthroscopic control.[239,240] Micropicking is a simple and atraumatic way to provide pluripotent cells, and proponents cite the lack of heat generated as one reason that results are superior to drilling techniques.[241] Another advantage of micropicking is that it preserves the subchondral plate. This is noteworthy because destruction of the subchondral plate typically compromises any resurfacing effort.[234] Experimental use of micropicking in normal horses resulted in more voluminous repair tissue than controls; however, little difference in repair tissue quality was apparent.[242] Although reports of using micropicking to treat traumatic injuries in human athletes are encouraging, the indications and long-term results of the procedure in the horse are unknown.

Articular Grafting

Initially, studies using onlay grafts of periosteum or perichondrium for resurfacing of cartilage defects in laboratory animals generated much excitement. Attempts to apply this approach in the horse were disappointing and research efforts have shifted focus to grafts of cultured chondrocytes or cartilage-progenitor cells contained in an appropriate substrate.[235,243,244] To date, uniformly successful results have not been achieved using these methods. The problems of graft incorporation, cell survival, and an inability to maintain a functional hyaline cartilage repair persist. Recent studies are beginning to shed more light on the specific idiosyncrasies of cell metabolism in cartilage defects, and soon improving repair tissue quality through more precise regulation of cell metabolism in the various strata of the healing wound may be possible.[245-247]

Biologically Based Therapies

Biological therapy is intended to modulate the extent of immunological or inflammatory events while maintaining appropriate tissue function and responses. The goal of this approach is to control the excessive activation of the various mediators leading to cartilage degradation. Inhibition can be accomplished directly or indirectly. Areas of active investigation include administering MMP blockers, drugs that directly inhibit inflammatory cytokines (e.g., interleukin-1), and providing natural inhibitors of proteinases or cytokines. For example, efforts at suppressing cartilage matrix degradation have been conducted using natural[248,249] or synthetic matrix MMP inhibitors.[248,250,251] Another strategy to prevent proteolytic cartilage degradation is to inhibit the synthesis or activity of mediators responsible for stimulating the synthesis enzymes. For example, matrix MMP release is stimulated by interleukin-1, a process that can be blocked by interleukin-1 receptor antagonist protein, a naturally occurring inhibitor of interleukin-1. Intra-articular administration of interleukin-1 receptor antagonist protein slows the progression of lesions in experimental osteoarthritis.[252,253] Similarly, antibodies and soluble receptors to tumor necrosis factor-α, another pro-inflammatory cytokine implicated in arthritis, reduced cartilage loss in collagen-induced arthritis in mice.[254,255] In addition to specific inhibitors, treatment with proteins that indirectly block the effects of inflammatory mediators may be of therapeutic value. An example includes administering interleukin-4, a blocking cytokine with metabolic effects opposite to those of recognized pro-inflammatory forms, such as interleukin-1 or tumor necrosis factor-α.[256] Another approach is to attempt to enhance matrix synthesis and repair. This method involves administering cytokines such as basic fibroblast growth factor or insulin-like growth factor, which have been shown to influence cartilage metabolism and healing favorably.[257,258]

Unfortunately, the actions of many of these therapeutic proteins are short-lived, which necessitates frequent administration. Gene therapy represents a potential means by which delivery of some of the mentioned therapeutic proteins may be possible.[259,260] Although technical aspects of the approach vary, the general principle involves introducing a gene that enables cells within the joint to synthesize one or more therapeutic product(s) (Fig. 85-6). Genes coding for anti-arthritic proteins are the most obvious choice, but bioactive ribonucleic acid (RNA) molecules or antisense RNA could also be valuable. The advantage of gene therapy is that, once the gene is present, theoretically the gene could be transcribed and translated many millions of times to produce a therapeutic level of its product. Candidate proteins for gene therapy include natural MMP inhibitors (e.g., tissue inhibitors of metalloproteinases), cytokines (e.g., insulin-like growth factor), and cytokine

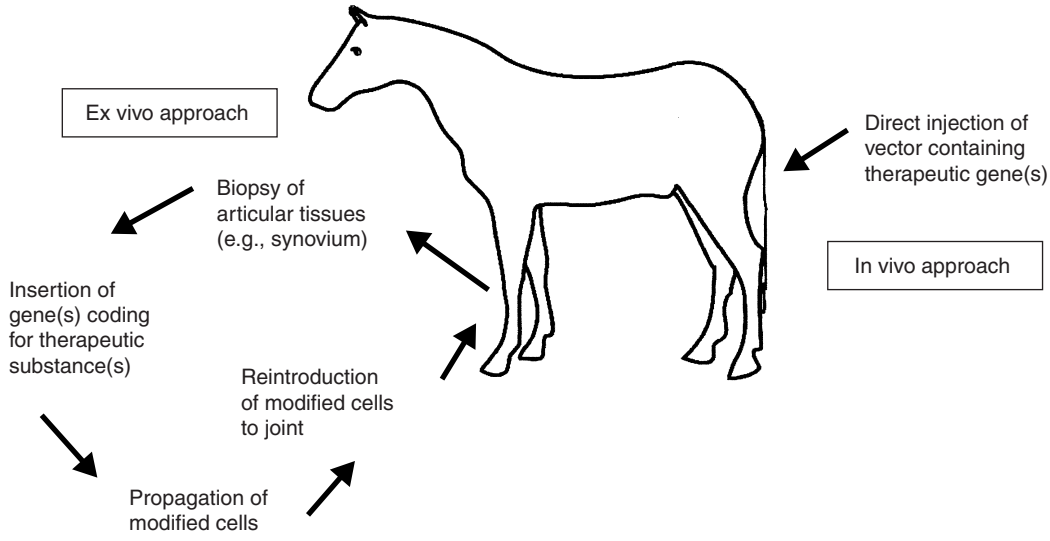

Fig. 85-6 General approaches for equine joint disease using local gene therapy. A vector containing one or more therapeutic genes can be injected directly into the affected joint (in vivo approach), or a biopsy containing patient cells may be transfected with a vector (frequently viral) containing the genes of interest, after which the cells are propagated in culture and re-introduced to the joint in large numbers (ex vivo approach).

inhibitors (e.g., interleukin-1 receptor antagonist protein). Inhibition of cartilage degradation has occupied much of the research efforts to date, many involving experiments designed to block the effects of interleukin-1 on cartilage degradation.[261,262] The interleukin-1 receptor antagonist gene was introduced successfully into normal equine joints, and preliminary results in an experimental joint disease model were favorable.[263,264] Promoting matrix synthesis is another potential application of gene therapy. For example, transfer of growth factor constructs (transforming growth factor-β1, insulin-like growth factor 1, and bone morphogenic protein 2) to chondrocytes was successful in increasing synthesis of collagen and proteoglycan.[265,266]

Durable resurfacing of cartilage defects ultimately may be accomplished with a combination of chondroprogenitor cell transplantation and gene therapy, with the introduction of genes coding for one or more selected growth factors. Theoretically, if the correct combination of proteins could be expressed in appropriate quantities, at the correct times during healing and incorporation of the transplanted cells, modulation of grafted or endogenous repair tissues to hyaline cartilage may well be possible.

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CHAPTER • 86

Epidural Analgesia and Hindlimb Lameness

Rose M. McMurphy

The benefits of providing analgesia for acute and chronic pain have been well established in many species. Inadequate treatment of pain in animals can result in inappetence and weight loss, increases in serum cortisol and catecholamines, tachycardia, hypertension, and compromise of the immune system.[1] Increases in serum cortisol and catecholamines can cause derangements in serum glucose, protein metabolism, and immune function. In addition, horses with a painful limb may remain recumbent for prolonged periods of time, with resultant decubital ulcer formation and secondary infection.

Designing an appropriate plan for analgesia in horses, particularly for those with severe, chronic pain can be challenging. Parenterally administered opioids and α_2-adrenergic agonists may be associated with side effects such as ataxia and excitement, and adverse effects on the gastrointestinal and cardiovascular systems. Non-steroidal anti-inflammatory drugs (NSAIDs) can cause gastrointestinal ulceration or renal disease, and they may be inadequate for horses with acute, intense pain. Epidural administration of drugs provides a more localized analgesia and fewer systemic effects than parenteral administration.

Epidural drug administration places a drug in close proximity to its site of action within the spinal cord or spinal nerves as they exit the spinal cord. A greater analgesic effect may be achieved with a smaller total dose of a drug. The duration of analgesia is usually longer than with parenteral administration.

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CHAPTER • 86

Epidural Analgesia and Hindlimb Lameness

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Designing an appropriate plan for analgesia in horses, particularly for those with severe, chronic pain can be challenging. Parenterally administered opioids and α_2-adrenergic agonists may be associated with side effects such as ataxia and excitement, and adverse effects on the gastrointestinal and cardiovascular systems. Non-steroidal anti-inflammatory drugs (NSAIDs) can cause gastrointestinal ulceration or renal disease, and they may be inadequate for horses with acute, intense pain. Epidural administration of drugs provides a more localized analgesia and fewer systemic effects than parenteral administration.

Epidural drug administration places a drug in close proximity to its site of action within the spinal cord or spinal nerves as they exit the spinal cord. A greater analgesic effect may be achieved with a smaller total dose of a drug. The duration of analgesia is usually longer than with parenteral administration.

Pain that is related to a disease process of the hindlimb is particularly amenable to treatment with epidural analgesia. The site of injection for epidural drug administration in horses is usually the first coccygeal (caudal) interspace. When deposited into the epidural space, the drug diffuses across the meninges, into the cerebrospinal fluid (CSF), and then into the spinal cord. The degree of cephalad diffusion of the drug within the CSF depends on several factors, including the volume of the drug injected, concentration of the drug, and lipid solubility.[2] Although studies in dogs and people have reported analgesic effects with epidural morphine or α_2-agonists that extend to the mid-thoracic area or farther cranially,[3,4] this generally does not occur in horses.[5-8] This limits the use of epidural analgesia for treatment of limb pain in horses to the hindlimb.

TECHNIQUE FOR EPIDURAL ADMINISTRATION

Caudal epidural drug administration can be done by single injection at the first coccygeal interspace or by placing an epidural catheter at this same site for repeated drug administration. The lumbosacral space is also a potential site of injection and may have a more rapid onset of hindlimb analgesia. However, positioning the tip of the needle within the epidural space rather than the subarachnoid space can be difficult. Subarachnoid administration of drugs in the horse is certainly acceptable, but the dose should be reduced by 40% to 50%.

The first coccygeal interspace (between the first and second coccygeal vertebrae) is identified while raising and lowering the tail and palpating for the articulation between these two vertebrae. This space is just caudal to the most angular portion of the bend of the tail, about 5 cm cranial to the first long tail hairs (Fig. 86-1). The site is prepared aseptically. Administration of 2 to 3 ml of 2% lidocaine subcutaneously using a 25-gauge needle helps decrease the response of the horse to placement of the needle for epidural injection. An 18- or 20-gauge, 6.35-cm spinal needle with stylet is recommended for epidural injection in horses, although many clinicians use a standard 18-gauge needle. The bevel of a spinal needle is not as sharp as a standard needle, and the bevel angle is less acute. This design difference makes it easier to identify penetration through the interarcuate ligament and the subsequent loss of resistance as the epidural space is entered. The needle is inserted at a 30° to 60° angle to horizontal, with the tip pointed cranioventrally, and is advanced until it contacts the floor of the vertebral canal (Fig. 86-2). The depth of insertion is 3 to 6 cm, depending on the size of

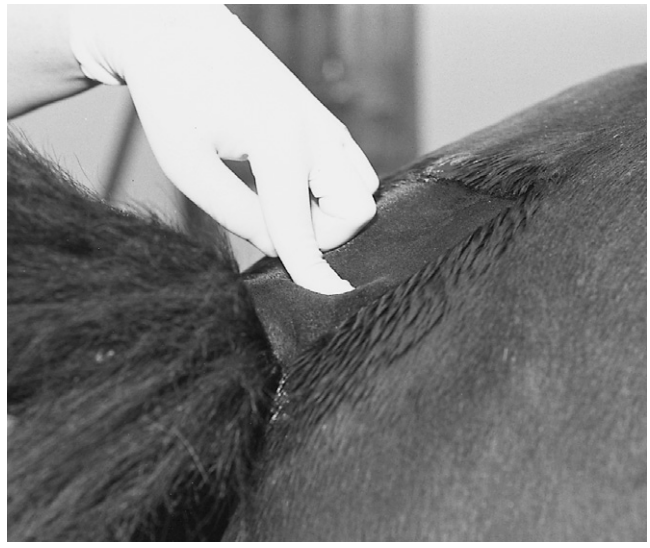
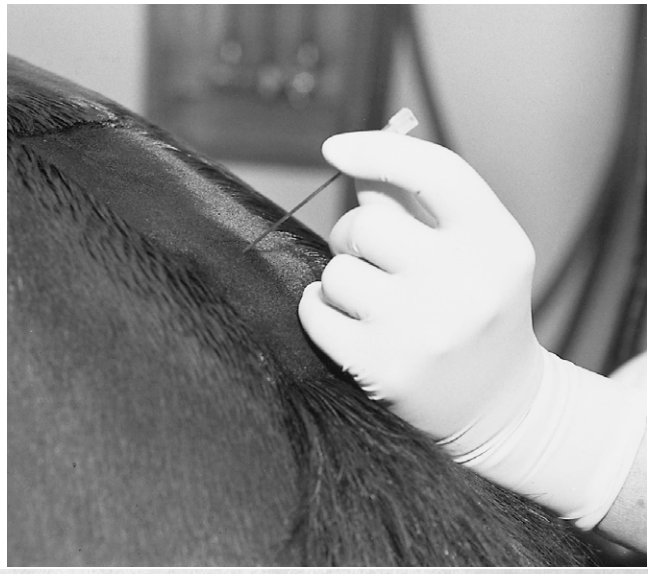
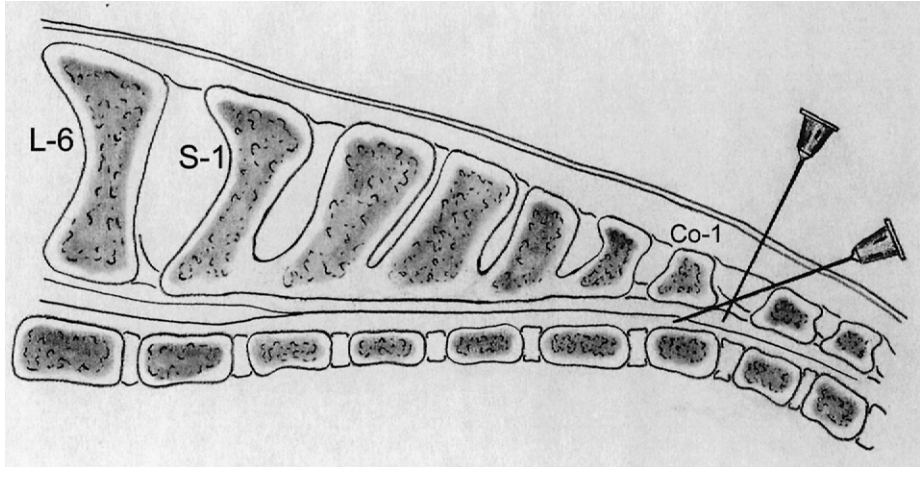


Fig. 86-1 The first coccygeal interspace between the first and second coccygeal vertebrae is identified while raising and lowering the tail and palpating for the articulation between these two vertebrae. This space is just caudal to the most angular portion of the bend of the tail.



A



B

Fig. 86-2 **A,** The needle is inserted at a 30° to 60° angle to horizontal and is advanced until it contacts the floor of the vertebral canal. **B,** Sagittal view of the sacral vertebrae and placement of the needle within the epidural space. *Co-1*, First coccygeal vertebra; *S-1*, first sacral vertebra; *L-6*, sixth lumbar vertebra.

the horse and the angle of the needle. The needle placement can be tested by attempting to inject 2 to 3 ml of air or solution in a 3-ml syringe. Resistance to injection should be little or absent. Appropriate epidural injection of local anesthetic solution and xylazine often is confirmed when anal tone decreases and the tail relaxes, but because the drugs most commonly used for epidural analgesia of the hindlimb (morphine, detomidine) have little or no effect on motor nerves, these responses will be absent.

EPIDURAL CATHETER PLACEMENT

The site for inserting an epidural catheter is the same as for a single epidural injection. Placement of the catheter requires the use of a needle with a curved point (Tuohy, Hulstead; Becton Dickinson, Franklin Lakes, NJ) that will direct the catheter cranially, along the floor of the vertebral canal (Fig. 86-3). The epidural catheter is made of polyamide (nylon) or teflon, with a closed or open end, and it can be purchased with a wire stylet if desired. I use an 18-gauge, 8.89-cm Tuohy needle and a 20-gauge, 100-cm radiopaque, polyamide catheter with a closed tip (bullet tip) without a stylet (Fig. 86-4). Epidural catheters have marks every centimeter and multiple marks at 10, 15, and 20 cm from the end. Before needle and catheter placement, slide the catheter inside the needle and note the distance from the tip of the needle to the hub on the catheter. The clinician then should determine which mark on the catheter will be at the hub of the needle once the catheter is advanced to the desired position.

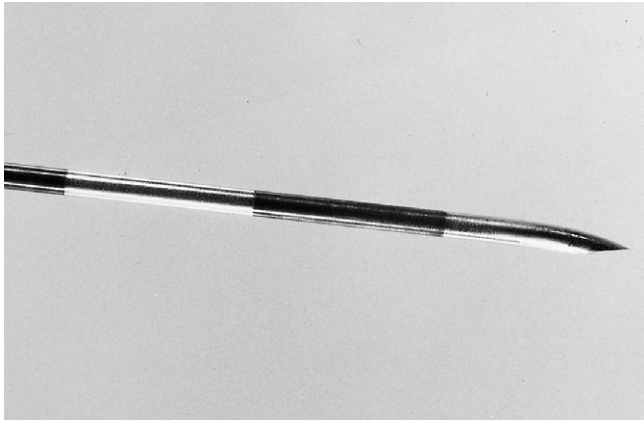


Fig. 86-3 Curved point of a Tuohy needle.

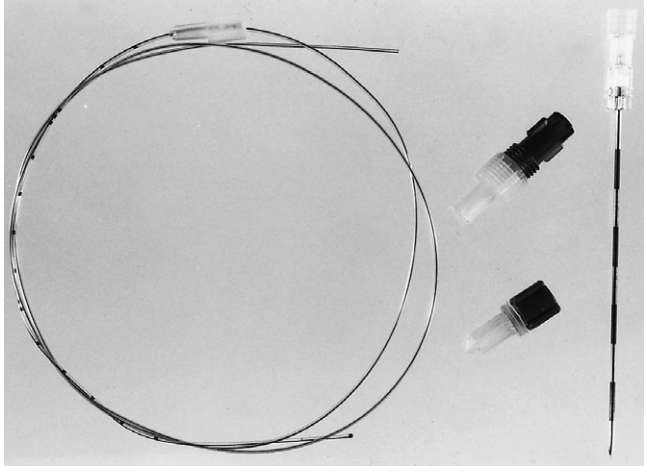


Fig. 86-4 Tuohy needle and epidural catheter.

As with the single epidural injections, strict attention should be paid to aseptic technique, and sterile gloves must be worn. Lidocaine is injected into the subcutaneous tissues at the desired site. A small incision is made through the skin with a No. 11 scalpel, because the Tuohy needle has a blunt tip. Once the needle is positioned, the bevel should be directed cranially (the notch on the hub of the needle should face cranially). The catheter is threaded through the needle and along the floor of the vertebral canal (Fig. 86-5). Provided the needle is positioned in the epidural space, the advancing catheter receives little resistance. Getting the catheter to advance initially into the canal can be difficult, however. If the tip of the needle is angled incorrectly, the catheter tends to bump up against the vertebrae and will not advance. If this occurs, the needle is withdrawn 1 to 2 mm and the catheter is rotated slightly as it

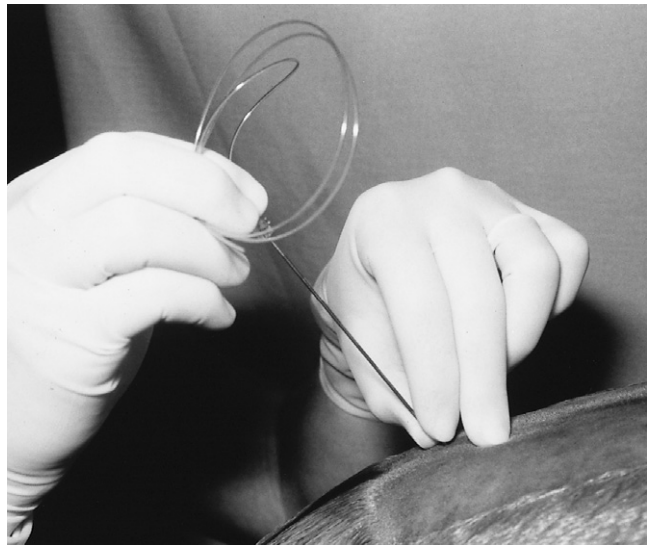


Fig. 86-5 The epidural catheter is threaded through the needle and along the floor of the vertebral canal. If the catheter will not advance, the needle is withdrawn 1 to 2 mm, and the tip of the needle is angled up slightly.

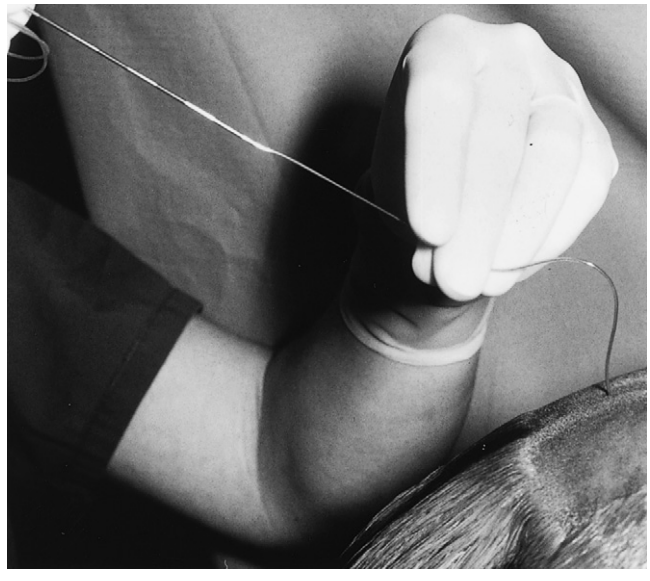


Fig. 86-6 Once the catheter is in place, the needle is carefully withdrawn over the catheter and removed.

is advanced. It is critical that once the catheter has advanced any distance outside of the needle that it is never withdrawn back into the needle. This may result in cutting or sheering off the catheter. The catheter is advanced 5 to 10 cm past the tip of the needle, and the tip is positioned in the mid-sacral region. Once the catheter is positioned, the needle is carefully withdrawn over the catheter and removed (Fig. 86-6). Most epidural catheters intended for human use are long to facilitate positioning of the injection port in a convenient place on the body. I usually cut the catheter with a scalpel (scissors tend to crush the catheter), leaving about 10 cm of the catheter to secure to the horse. The catheter/ syringe adapter is attached and an injection cap secured to the adapter. A portion of tape is secured to the catheter at the point of insertion and then sutured to the skin (Fig. 86-7). The syringe adapter also should be sutured to the skin, making sure tension will be minimal on the catheter during any movement of the horse. The catheter and injection cap then are covered with a sheer, adhesive dressing. Each injection into the catheter is made through the catheter cap after thoroughly cleaning it with alcohol and while wearing sterile gloves. It is critical that the catheter system remains sterile. The catheter site should be examined daily for signs of inflammation or infection, which would necessitate removing the catheter and submitting the tip for culture. With diligent aseptic technique and catheter care, we have kept catheters in place for 2 weeks in clinical patients, and some researchers have reported epidural catheters being maintained for 5 weeks in research horses.[9]

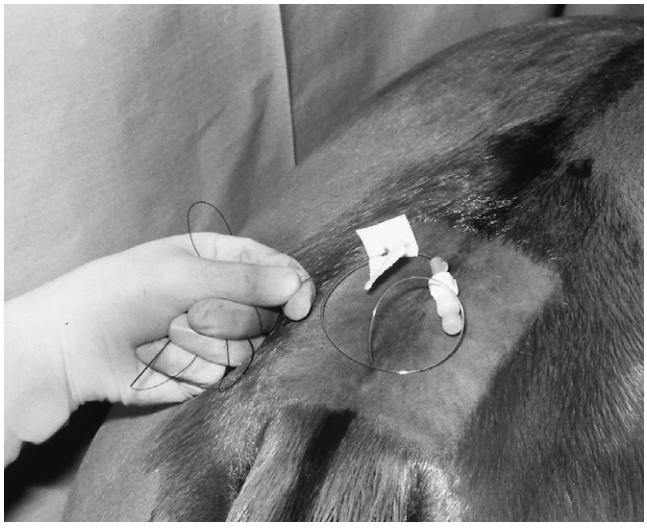


Fig. 86-7 The catheter/syringe adapter is attached and an injection cap is secured to the adapter. A portion of tape is secured to the catheter at the point of insertion and then sutured to the skin.

DRUG SELECTION

Epidural Opioids

The primary advantage of epidural administration of an opioid is the intense and prolonged segmental (localized) analgesia achieved without either sedation or possible excitement that may accompany the parenteral administration of an opioid. Unlike epidural local anesthetics or xylazine, epidural opioids do not affect neuromuscular function or the sympathetic nerves.

The analgesia obtained with epidural opioids is primarily from the effect of the opioid within the spinal cord rather than supraspinal. Opioids bind to presynaptic receptors of the afferent nerve terminals in the dorsal horn and inhibit the release of excitatory neurotransmitters, such as glutamate and substance P. Opioids also act postsynaptically to inhibit transmission of impulses in ascending tracts. Evidence also shows that opioids enhance the effects of descending inhibitory pathways on the processing of pain within the dorsal horn.[10]

The onset and duration of action of epidural opioids vary greatly between drugs. The time to onset of analgesia reflects the time required for a drug to diffuse from the epidural space, across the meninges, into the spinal fluid, and ultimately into the spinal cord. Physicochemical properties of the drug such as molecular weight, molecular shape, degree of ionization, and particularly lipid solubility influence the diffusion across the meninges.[3,11] The more lipid-soluble drugs, such as fentanyl and butorphanol, have a rapid onset but a shorter duration, whereas the more water-soluble drug, morphine, has a delayed onset and a long duration (Table 86-1).[3]

Table • **86-1**

Characteristics of the Analgesia and Side Effects after Epidural Administration of Various Drugs

DRUG	DOSE (mg/kg)	ANALGESIA		SEDATION	ATAXIA	COMMENTS
		ONSET (MIN)	DURATION			
Morphine	0.1*	150-180	12-18 hr	Minimal	No	Onset time for peak analgesia may be up to 6 hours.
Butorphanol	0.04	†	2.5-3 hr	No	No	Degree of analgesia may be inadequate.
Xylazine	—	—	—	—	Marked	Drug is not suitable for hindlimb analgesia.
Detomidine	0.05	10-25	2-3 hr	Moderate	Mild	Heart rate, blood pressure, and respiratory rate are decreased.
Ketamine	1-2	5-10	30-75 min	Mild	Mild	Ataxia occurs only at higher dose.
Detomidine and morphine	0.03 and 0.1-0.2	10-25	12-18 hr	Moderate	Mild	Heart rate, blood pressure, and respiratory rate are decreased.

*Although a dose of 0.2 mg/kg has been used and reported in horses, I no longer recommend this high dose.
†Onset time has not been documented.

The more water-soluble opioids also tend to have greater degree of craniad diffusion within the CSF.[2]

Morphine is the drug of choice (see Table 86-1).[5-7,12] Fentanyl has a faster onset of action but a considerably shorter duration of action (4 to 6 hours). Butorphanol provides inadequate analgesia. Oxymorphone is too expensive. I use preservative-free morphine (Abbot Laboratories, North Chicago, IL) that is intended for epidural use at a dose of 0.1 mg/kg. There are some parenteral preparations of morphine that have been used for epidural administration in horses in an attempt to lower the cost, but some of these may contain substances such as phenol or formaldehyde, which may be neurotoxic. The preparation of morphine (25 mg/ml) with sodium bisulfite as the preservative has been used in the horse epidurally, but the dose to be administered should be diluted in 20 ml of sterile saline solution. Epidural morphine has been used at a dose of up to 0.2 mg/kg combined with epidural detomidine.[13,14] The higher dose may potentially speed the onset of action and extend its duration, but it may increase the incident of side effects.

Potential side effects include respiratory depression, pruritus,[15] and urinary retention. These are rarely seen in horses and can be treated with naloxone (0.005 to 0.01 mg/kg). I have experience with a horse that developed substantial central nervous system effects after administration of morphine (0.2 mg/kg in 50 of ml saline solution), resulting in collapse, tachypnea, muscle rigidity, and hypoxemia. The horse was treated with naloxone (0.01 mg/kg), furosemide (2 mg/kg), diazepam (0.01 mg/kg), and a neuromuscular blocker to facilitate positive-pressure ventilation after intubation and anesthesia with isoflurane. The horse recovered.

Epidural α_2-Adrenergic Agonists

Epidural administration of an α_2-adrenergic agonist can induce profound analgesia mediated by α_2-adrenergic receptors in the spinal cord.[16] The exact mechanism for the analgesia is not known. However, the α_2-adrenergic agonists traditionally have been thought to induce analgesia by mimicking the action of norepinephrine released from descending noradrenergic inhibitory pathways.[17] Additional proposed mechanisms of action include modulation of pain via serotonergic and adenosinergic pathways, inhibition of the release of substance P, and effects on opiate receptors.[18,19]

Xylazine, an α_1- and α_2-receptor agonist, is commonly used to induce analgesia of the perineal area of horses.[20] However, xylazine is not suitable for severe hindlimb lameness because of its effects on somatic motor innervation.[8] Doses that exceed 0.25 mg/kg can cause substantial ataxia and have the potential to cause temporary hindlimb paralyis. Detomidine is a more selective α_2-adrenergic agonist, which primarily affects nerve fibers involved in pain transmission (C fibers and A delta fibers) and appears to have limited effect on somatic motor function at clinically used doses. Detomidine therefore is less likely to cause motor dysfunction and hindlimb paralyis, making it more appropriate for the treatment of horses with hindlimb pain. However, detomidine has a relatively short duration of action (see Table 86-1) and also has systemic effects, such as decreased blood pressure and heart and respiratory rates. Detomidine is best used with morphine and can provide additional analgesia. I use a combination of morphine (0.1 mg/kg) and detomidine (30 µg/kg).

CLINICAL APPLICATIONS

Epidural analgesia can make a profound difference in the comfort and convalescence of equine patients and is frequently used with other forms of analgesia such as NSAIDs. Epidural analgesia is often used during the first stages of recovery from various disease processes, when the pain may be more intense, or immediately before or after surgical treatment of pelvic limb injuries or disease. Procedures such as fracture repair and arthrodesis can be painful intra-operatively and post-operatively. Pre-operative administration of epidural opioids can decrease the concentration of inhalation anesthetic required, and some evidence shows that pre-operative administration of epidural opioids is effective pre-emptive analgesia. Evidence also shows that effective pre-emptive analgesia can make post-operative pain management more successful. Because the onset of action of epidural morphine is prolonged in horses, administration of the drug 1 to 3 hours before anesthesia and surgery may be necessary to achieve the greatest effect post-operatively. A clinical impression exists that horses treated with epidural morphine have longer recovery times. My impression is that recovery might be prolonged slightly, but not dangerously so, and that the horses are far more comfortable in the immediate post-operative period. Epidural administration of morphine provides far superior analgesia to that which we can achieve with NSAIDs and parenterally administered opioid agonist-antagonists, such as butorphanol, without substantial systemic effects.

Horses with trauma to the hindlimb that require long-term therapy may benefit from the placement of an epidural catheter. I have left catheters in place for 1 to 2 weeks in some of these patients and, as of yet, have seen no complications attributable to the catheter. In some horses, however, the analgesia seemed gradually to become less effective, despite increasing the dose and frequency of epidural morphine. The addition of 30 µg/kg of epidural detomidine every 6 to 8 hours was beneficial in these horses. Ultimately ketamine[9] or other α_2-agonists, such as medetomidine, may have a role in treating horses with pain that is refractory to epidural morphine.

CONTRAINDICATIONS AND COMPLICATIONS

Complications associated with epidural analgesia can be grouped according to those caused by the drugs administered, those caused by the actual insertion of the needle or the catheter, and those caused secondary to the maintenance of an epidural catheter. Complications reported with epidural morphine are rare in horses. Detomidine may cause cardiopulmonary depression, sedation, and ataxia. Careful assessment of the patient's cardiovascular status and appropriate dosing of the drug can minimize the risks associated with epidural detomidine.

Introduction of the needle or catheter into the epidural space potentially can cause trauma to the spinal nerves roots or the epidural venous sinus. Substantial bleeding in patients with normal clotting times and platelet numbers is unlikely. The possibility exists that if the tip of the needle is in the venous sinus, the drug administration will be intravenous as opposed to epidural and will not have the desired effect of prolonged, segmental analgesia. I have also, on one occasion, inadvertently catheterized the epidural venous sinus. If blood is obtained after aspiration of the catheter, the catheter (and needle if still in place) should be removed and a new catheter should be placed.

Potential complications secondary to the maintenance of an epidural catheter include localized soft tissue inflammation, and rarely, epidural abscess, vertebral osteomyelitis, and meningitis. The catheter site should be inspected daily for signs of infection or inflammation.

Possibly the most serious potential complication would be the inadvertent injection of an inappropriate substance or an overdose of the drug into the epidural catheter. The epidural catheter should be marked clearly as such, and all personnel working with that patient should be instructed in the care of the catheter and the amount and type of drugs that are administered through the catheter.

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CHAPTER • 87

Bandaging, Splinting, and Casting

Alan J. Ruggles and Sue J. Dyson

Indications for bandaging and cast application include protection of limbs during transport and performance, reduction of soft tissue swelling, protection of surgical wounds, management of skin defects and granulation tissue, protection of surgical implants, management of fractures, and first aid before transport of injured horses. This chapter discusses methods of bandage, splint, and cast application and the acute management of a horse with a suspected fracture or soft tissue injury.

STABLE AND TRAVELING BANDAGES

Bandages used routinely as stable bandages or for transport consist of a padded, quilted, fleece or interwoven sheet cotton as a protective layer, held in place by a flannel wrap or commercially available bandage material. Horses that continually wear stable bandages tend to develop ridges in the hair coat, and this should be noted at a prepurchase examination. Proper bandaging prevents limbs from filling in a stabled horse and prevents injury during transport. The coronary band region is particularly vulnerable during transport, loading, or unloading, and the bandage ideally should extend to cover this area. Alternatively, overreach (bell) boots should be used. Improper bandage application or management can cause the development of white hairs, transient edema, structural damage to soft tissue structures and, under severe circumstances, pressure necrosis. An excessively tight bandage can result in severe tissue necrosis within 24 hours, resulting in full-thickness skin loss and damage to the underlying soft tissues. The mid-metacarpal region seems to be particularly vulnerable. Alternatively, commercially available boots can be used for transport, the best of which extend from proximal to the dorsal aspect of the carpus, or the plantar aspect of the tarsus, to cover the coronary band. The use of exercise bandages and boots for protection and support is discussed in Chapter 38.

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BANDAGING WOUNDS

The principles of bandages used to protect wounds are to absorb exudate, reduce soft tissue swelling, and provide an environment conducive to wound healing. Each wound has its own characteristics that make a specific type of bandage, or even the absence of a bandage, ideal. All bandages prevent or reduce edema by providing pressure. A bandage can immobilize a region to a certain extent; the degree of immobilization depends on the type of material used and the manner of its application.

Surgical wounds are generally created under ideal conditions and proper apposition of the skin edges occurs during suturing. Protection is provided by a non-adherent, porous dressing (Telfa, Kendall Co., Mansfield, MA) over the wound. The dressing is held in place with a sterile gauze roll (Conform, Kendall Co.) and then a cotton combine roll 45 cm wide (for the lower limb only), or alternatively a soft conforming bandage (Soffban, Smith & Nephew, Hull, North Humberside, UK). The gauze is held in place with an elastic conforming bandage (Vet-Wrap, 3M Animal Care Products, St. Paul, MN; Elastikon, Johnson and Johnson, Arlington, TX). These dressings are changed every 2 to 3 days, or earlier as needed, until suture removal. If a full-limb bandage is placed to extend above the carpus or tarsus, an additional bandage 40 cm wide is applied above the first bandage. For bandaging hocks, one author (A.J.R.) prefers to use a 45-cm wide combine roll on the distal limb and then 0.5-kg roll of cotton wool, which is divided lengthwise over the hock to provide greater compression. A layer of gauze (Ken Vet Co., Ashland, OH) is used over the combine and cotton, and an elastic conforming bandage (Elastikon) is then applied. Finally, additional elastic tape is used to secure the top of the bandage to the skin. These types of bandages are used post-operatively for most horses with orthopedic procedures, such as arthroscopy or splint removal, and for most horses with limb wounds, assuming the limb is stable.

In a forelimb the area of the bandage over the accessory carpal bone is incised with a scalpel blade to prevent rub sores from developing. In a hindlimb the point of the hock can be covered by the bandage if bandaging is anticipated for less than 1 week. If prolonged bandaging is required, the point of the hock should not be covered, to prevent rub sores and potential development of white hairs.

Roll cotton (cotton wool) rather than combine roll often is used for the proximal portion of carpal bandages, because it stays in position best. Roll cotton is particularly useful for reducing soft tissue swelling after desmotomy of the accessory ligament of the superficial digital flexor tendon. A non-adherent layer and conforming gauze is covered by elastic adhesive bandaging tape before applying a full-limb padded bandage. This provides protection of the wound from the environment and prevents hematoma and seroma formation. Care must be taken to avoid excessive tension being applied to the elastic adhesive tape to prevent pressure necrosis.

Pressage Bandages

Pressage bandages (Pressage bandage, S.C. Meades, Cardiff, Wales) are commercially produced elasticized bandages designed specifically for the carpus and hock and available in three sizes. They provide an excellent method of securing a light bandage in place and providing pressure, with a minimal risk of pressure sores, and are used routinely by one author (S.J.D.). Two turns of an elastic conforming bandage over the proximal extent of the Pressage bandage and application of a stable bandage in the more distal part of the limb help to keep the Pressage bandage from slipping.

Ether Bandage

Ether bandages are used to cover surgical incisions, but they do not provide any compression to the incision. For the ether bandage to stay, the area to which it is applied must be clipped, or preferably, shaved. Cleansing of the skin with surgical scrub, followed by alcohol rinsing, provides the best environment on which to apply this bandage. Strips of adhesive elastic tape about 15 cm in length are cut and ether is applied using a syringe to the adhesive side of the bandage tape. The ether soaked bandage is then applied over gauze sponges and held in placed while the ether evaporates and the adhesive dries to the skin and elastic tape. Three to four strips of elastic tape typically are used. Ether bandages are useful to protect surgical wounds made for stifle arthroscopy, bone grafts from the tuber coxae, and plate fixation of the olecranon. Because of the explosive nature of ether, proper precautions should be followed in its use and storage. Commercially available Primapore dressings (Smith & Nephew) provide a simpler alternative.

Stent Bandages

Stent bandages are towels or gauze rolls that are sutured over wounds to protect the wound from the environment or to relieve tension at the sutured site. A sterile hand towel or gauze sponges, rolled like a cigar to the proper width of the wound, are sutured over the wound with No. 2 or larger suture. A cruciate pattern is usually used. Stent bandages are used after shoulder arthrotomies, after olecranon or femur fracture repair, to cover wounds over the point of the hock, and for other incision sites unsuitable for bandaging. If the stent is to be changed routinely, then loop sutures are placed on either side of the incision, and umbilical tape can be used as suture to hold the bandage in place between changes.

Wet-to-Dry Bandages

Wet-to-dry bandages are used to absorb exudates and to provide an environment conducive to wound healing, particularly in the proliferative phase of wound healing. After cleansing of the wound, sterile sponges moistened with saline solution are applied to the wound and held in place with normal bandaging techniques. The bandaged is changed daily, or as needed, and the process can be repeated as often as necessary until the granulation bed has formed.

With any wound there is a combination of dressings, bandaging, immobilization, and systemic therapy that is appropriate. The management changes as the wound heals and sometimes alternative strategies need to be adapted. Careful attention to the progress of the wound and understanding of wound healing principles optimize outcome.

Foot Poultice

If a subsolar abscess is suspected, applying a poultice may be necessary, along with foot soaks, to soften the sole to permit drainage and to draw the abscess. A commercial poultice such as Animalintex (3M Animal Care Products; Robinson Animal Healthcare, Chesterfield, UK) is the simplest to use. A half piece is usually adequate. It should be thoroughly soaked in hot water and partially squeezed out before being applied to the sole of the foot. The poultice is covered by a thick layer of cotton wool and then conforming bandage is wrapped around the foot to hold the poultice in position. To keep the moisture in, the foot can be placed in a used fluids bag, which is then covered by duct tape to provide a durable bandage.

Robert Jones Bandage

A Robert Jones bandage is used when additional support or compression is required. The bandage is particularly useful for immobilizing the limb of a horse with a suspected fracture for transport or for recovery from general anesthesia after fracture or wound repair; for example, after lag screw repair of a simple proximal phalangeal fracture or a condylar frac-

Fig. 87-1 A forelimb full-limb Robert Jones bandage. Note the cylindrical shape and proximal extent of the bandage.

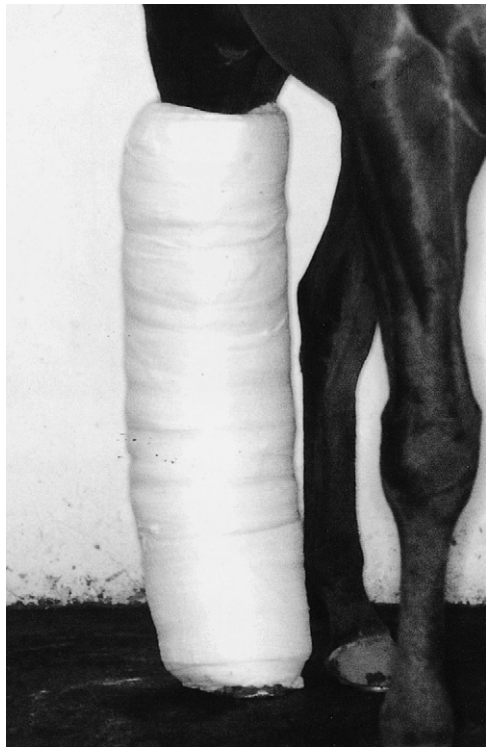

Fig. 87-2 A full-hindlimb Robert Jones bandage, before application of elasticized tape, extending to the proximal tibia.

ture of the third metacarpal bone. The Robert Jones bandage also can provide limb support if the suspensory apparatus is disrupted, can control severe post-traumatic limb edema, and can provide substantial pain relief to a horse with a severely injured superficial digital flexor tendon. This bandage has a multi-layered construction. Compared with a single-layered bandage, a Robert Jones bandage compresses air-filled cotton wool layers to increase rigidity and spread pressure evenly. A half-limb bandage requires 4 to 5 rolls of cotton wool, 8 to 10 conforming bandages, and 3 to 4 rolls of elastic adhesive tape. A full-limb bandage needs twice as much. For a half-limb bandage in a forelimb the cotton wool should be applied snuggly from the foot to the distal aspect of the carpus, incorporating the foot. The first layer using 1½ to 2 rolls of cotton wool should be 2 to 3 cm thick. This layer is then compressed using several conforming bandages at least 15 cm wide. Each bandage is applied using fairly firm, constant pressure to compress the cotton wool evenly. Filler layers of cotton wool usually are needed at the top and bottom of the bandage to create a uniform cylinder. A length of cotton wool is folded in half and placed around the top and bottom of the bandage as a filler layer. Another 2- to 3-cm layer of cotton wool is then applied and is compressed using a conforming bandage. At least 3 layers are constructed to create a thickness of 6 to 8 cm. The entire length of the bandage is now covered with elasticized tape, which should extend the entire length of the bandage and above and below it, to prevent dirt and bedding material from getting into the bandage. The end of one roll of elasticized bandage and the beginning of the next should overlap to prevent unraveling of the bandage. The end of the last roll is covered by zinc oxide tape. A full-limb bandage is applied similarly. In a forelimb the bandage should extend the entire length of the limb, incorporating the elbow (Fig. 87-1); in a hindlimb the bandage should extend to the proximal aspect of the tibia (Fig. 87-2).

SPLINTING

Splints should be used to support unstable fractures, when disruption of the suspensory apparatus is suspected, and when a full-limb Robert Jones bandage is used to provide additional support. Proper splinting helps protect bone from further trauma, prevents further soft tissue damage, and may also increase the horse's comfort. Wood (45 × 20 mm), or plastic guttering (diameter about 112 mm) cut in half to give a U shape, can be used. Polyvinyl chloride (PVC) piping is lightweight, inexpensive, and strong, but wood is generally more suitable for lateral splints. The length of the splint depends on the size of the horse and the position of the injury. It is critical that the splints are adequately padded, especially at the top and bottom, to avoid rub sores. A gutter splint can be padded throughout its entire length using cotton wool. Wooden splints should be padded at the top and bottom. The splint should be covered with elastic adhesive tape proximally and distally to prevent it from becoming damaged and developing rough, sharp edges. A splint generally is applied over the dorsum to immobilize the distal limb. If a transverse or oblique fracture is suspected, or support of the suspensory apparatus is lost, the limb should be splinted to align the dorsal cortices of the third metacarpal bone and phalanges to eliminate the bending forces of the metacarpophalangeal joint. An assistant is needed to hold the limb off the ground, supporting it under the antebrachium (Fig. 87-3). One layer of the Robert Jones bandage is applied and then the splint is strapped to the dorsum of the limb before applying the second layer of the Robert Jones bandage (Fig. 87-4). The splint should be well reinforced at the toe. If a sagittal plane fracture or subluxation or luxation of a joint is suspected, supporting the limb in its normal position is preferable. The splint then can be applied over the full thickness of the Robert Jones bandage. To immobilize the carpal region, or if a fracture in

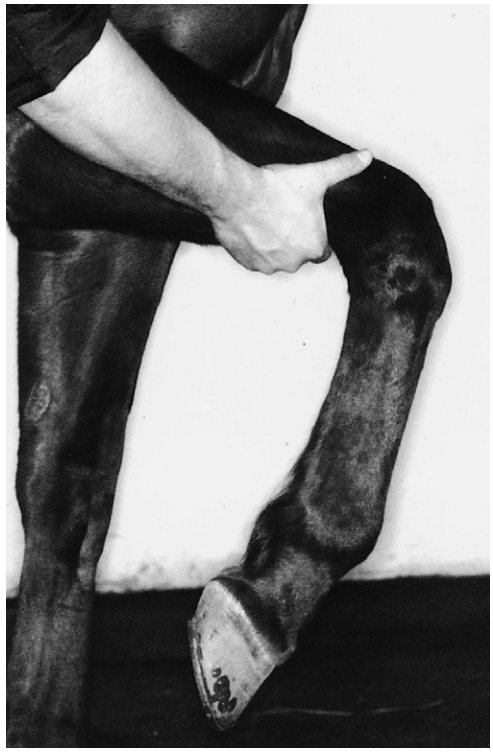

Fig. 87-3 An assistant holds a forelimb before application of a half-limb Robert Jones bandage, aligning the dorsal cortices of the distal limb bones.

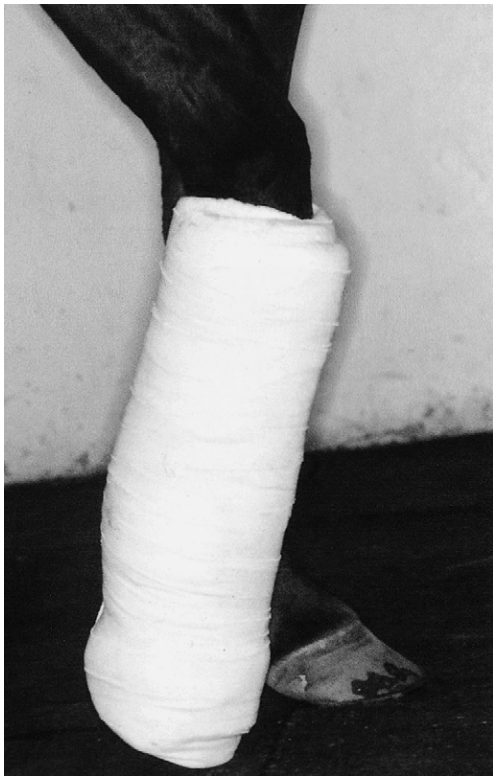

Fig. 87-4 A half-forelimb Robert Jones bandage incorporating a dorsal splint. The horse is standing happily, bearing weight on the toe.

the mid-metacarpal region or distal radius is suspected, caudal and lateral splints are used, extending to the elbow. With a fracture of the mid- or proximal radius, abduction of the limb is prevented by placing a lateral splint extending from the ground to mid-scapula level. The top of the splint must be well padded to prevent rub sores. With a fracture of the ulna and loss of triceps function, it is best to fix the carpus in extension by using a caudal splint extending from the ground to the proximal olecranon. Similar principles apply in a hindlimb. Not all horses tolerate immobilization of the hindlimb and the clinician must be prepared for the horse to react adversely when it first moves. For horses with fractures of the distal metatarsal region and distally, the limb is held above the hock by an assistant while the Robert Jones bandage and dorsal splint are applied. With a suspected fracture of the mid-metatarsal and proximal metatarsal regions, the Robert Jones bandage should be applied with the limb bearing weight, and caudal and lateral splints are applied. The caudal splint should extend up to the calcaneus, to fix the tarsus to the distal limb fracture. Wood splints are stronger than gutter splints. Splinting of the hock and distal tibial regions aims to counteract the medial forces of the lateral musculature of the femur and the destabilizing effect of flexion of the stifle through the reciprocal apparatus. To immobilize the hock, a lateral splint is contoured to the angle of the hock by heating PVC gutter pipe over a flame, or by using a 12-mm steel rod that can be shaped by hand but is strong enough to provide support. The splint should extend to the proximal tibia. An additional contoured splint can also be placed distal to the stifle and proximal to the fetlock on the plantar (caudal) side.

Several commercial splints are available that are particularly useful in an emergency situation, being easy to apply and rapidly providing pain relief by immobilization of the limb. These include the Kimzey Leg Saver Splint (Kimzey, Woodland, CA), the Monkey Splint (Kruuse, North Yorks, UK), and the Australian Equine Salvage Splint (Ballarat Veterinary Practice, Ballarat, Victoria, Australia). They are all designed to align the dorsal cortices of the distal limb bones and are appropriate for fractures distal to the distal third of the metacarpal or metatarsal regions or suspensory apparatus breakdown. The Kimzey splint also has an extension that reaches to the proximal antebrachium and is suitable for use in horses with fractures up to the carpus. The Kimzey splint generally is well tolerated. The Monkey and Australian splints have an extra heel and some horses walk awkwardly.

TRANSPORT OF AN INJURED HORSE

With appropriate immobilization of a limb, a horse with a suspected fracture or major soft tissue injury can be safely transported long distances to a clinic with suitable diagnostic and surgical facilities and a skilled surgeon. Ideally the horse should be transported in a low-loading vehicle. Alternatively the horse should be loaded via a loading ramp, or with the ramp of the vehicle placed on a slope, so that the incline up the ramp into the vehicle is shallow. Ideally a horse with a forelimb injury should travel facing backward so that major load is placed on the hindlimbs when the vehicle decelerates. A horse with a hindlimb injury should travel facing forward. A clinic expecting to receive such horses also should have a loading ramp to minimize the incline down which the horse has to walk when unloaded.

CAST BANDAGE

Cast bandages are a combination of a standard bandage, casting tape, and splint material (typically PVC). After place-

ment of a standard bandage, two layers of cast material are placed over the bandage in the area to be splinted. The splint is then applied and held in place while at least two additional layers of casting tape are applied. In 2 to 3 days the cast material is split to create a bivalve to allow access to the surgical site. The cast can then be reapplied to provide immobilization. Clinical indications for this technique include management of wounds over the carpus, including transverse lacerations or hygroma resection.

Cast Application

Indications for cast application include after fracture repair or suturing of lacerated tendons or for wound management. Commonly used materials include plaster of Paris, resin-augmented plaster, and fiberglass. Fiberglass casting tape consists of knitted fiberglass fabric impregnated with polyurethane resin and is used most commonly because of its greater strength, lighter weight, and quicker setting times, despite increased cost. Full- (up to elbow or stifle) and half-limb (up to carpus or tarsus) casts can be applied. Full-limb casts are not well tolerated by all horses, and the horse's temperament should be assessed carefully, particularly before applying a full-hindlimb cast. Casts are changed as needed because of rub sores, suture removal, breaking or buckling of the cast, or usually within 4 weeks after placement. In some circumstances a cast can be left on up to 6 to 8 weeks; however, the risks of cast rubs increase the longer the cast is left on the horse.

Casts can be applied in a conscious, sedated horse but are commonly applied with the horse under general anesthesia, after internal fixation or wound repair. The limb should be clean and dry before cast application. Sterile dressings are applied over the surgical site, and non-binding materials such as cotton cast padding should be used to secure the wound dressings. A double layer of sterile stockinette is applied over the limb and extends above the proximal extent of the cast. Four to 5 cm of orthopedic felt is placed at the proximal extent of the cast. Wider felt is used in full-limb casts. Additionally, orthopedic felt with the centers cut out can be placed over bony prominences, such as the styloid processes or accessory carpal bone, to avoid cast rubs. The felt is secured with adhesive tape. Wire can be placed through holes placed in the hoof wall near the toe and secured to a twitch handle to provide a means for an assistant to hold the limb and maintain the proper angle of the digit. In general, the hoof is placed at or near a weight-bearing position. Alternatively, the hoof can be extended to a lesser extent, by manual pressure at the toe. Cast padding is applied and overlapped about 50%. The use of 3M Custom Support Foam (3M Healthcare, St. Paul, MN), which has a polyurethane resin–impregnated cast padding, is helpful in reducing cast rubs. It is important not to use too much foam, because the cast will compress it and the cast will become loose, predisposing the horse to rub sores. Casting tape (3M Scotchcast, 3M Healthcare; 10 to 12 cm wide) is applied, beginning on the orthopedic felt at the top of the cast. The material is spiraled down the limb, taking care not to apply it too tightly, while avoiding wrinkles. Because the limb is not a perfect cylinder, wrinkles tend to develop in areas where contouring is necessary. Tension across the width of the casting tape eliminates potential folds or wrinkles. The next roll of casting tape begins where the first leaves off. At least five rolls of casting tape are used on the limb and sometimes one or two more additional rolls. When finished, more casting tape should be at the fetlock and distally, because this is the most common site of breakage. If the distal limb is extended using wires through the hoof wall, the toe region is not cast until the rest of the limb is finished and the twitch handle is no longer necessary. The wire and twitch handle are removed and the foot is covered with one to two rolls of casting tape. If a heel wedge is required, folded casting tape or a wood block can be incorporated in the casting tape applied to the hoof. Polymethylmethacrylate (Technovit, Jorgenson Laboratories, Loveland, CO) is applied to the bottom of the cast, especially if the cast is to remain more than 2 weeks, to prevent wearing through of the casting tape.

For application of a cast in a standing horse the same protocol is followed, except a 2.5- to 5-cm block is placed under the horse's hoof, so that the heel is left hanging slightly over the edge of the block, and the cast is applied. After the limb is cast, the limb is lifted and casting tape and polymethylmethacrylate are applied to the hoof. When standing casts are applied for first aid treatment of unstable fractures, a dorsal splint can be incorporated to maintain the bony column in a straight line. Foot casts are sometimes useful in managing heel bulb lacerations.

Potential cast complications include rub sores and cast breakage. Rub sores can occur over any bony prominence or an area where the cast folds or fingerprints are present. Common sites for cast rubs include the proximal dorsal metacarpal and metatarsal regions, proximal sesamoid bones, and heel bulbs in half-limb casts and the elbow, distal radius, accessory carpal bone, stifle, and point of the hock in full-limb casts. Increased lameness, fever, drainage, heat, foul odor, and proximal swelling are signs of cast rubs. Rub sores are common complications of cast application and usually are managed easily by cast change or removal. Failure to recognize rub sores can lead to serious problems, such as large wound defects, infections of synovial structures, or laminitis in the supporting limb. Proper application and careful monitoring of cast is essential to prevent serious rub sores.

Transfixation Pin Casts

The use of transfixation pin casts is discussed in Chapter 88.

CHAPTER • 88

External Skeletal Fixation

David M. Nunamaker

HISTORY AND DEVELOPMENT

Using external skeletal fixation to treat fractures in the horse has, until recently, received little enthusiasm from equine surgeons. Although external skeletal fixation of fractures works well in small animals and has enjoyed periods of enthusiastic use, these same devices do not withstand the loads of weight bearing in the adult horse and therefore are not versatile enough to meet the needs of the equine surgeon. Using transfixation pins in plaster, or more recently transfixation pins in fiberglass casts, has allowed salvage of some horses with difficult fractures that could not have been saved using internal fixation.

The incorporation of a walking bar cast, with transfixation pins placed in the bone above the fracture, was described in 1999 to manage fractures in horses and ponies. An overall success rate of 57% was reported for a variety of fractures in 35 horses and 21 ponies. The authors suggested that using transfixation pins incorporated into the cast material may help prevent fracture through these pin sites. Major complications of the technique included infection in nine animals, fracture through the bone or pin sites in six animals, and loss of circulation to the distal phalanx in two animals. Complications in the remaining seven non-survivors were not reported.[1] Using in vitro tests, McClure et al.[2,3] suggested that the walking bar was not necessary when using fiberglass casts and that divergent transfixation pins may be helpful in preventing fracture through the pin tract sites.

Although transfixation pins have been used successfully in fiberglass casts, this technique represents a compromise compared with classical external skeletal fixation. In horses managed with transfixation pin casts, wounds, skin, and pin sites must be covered in the cast, and at the time of every cast change, fractures are remobilized, allowing fracture collapse or shortening of the limb segment. In addition, pin loosening and local infection at pin sites are common sequelae using this form of transfixation pinning, often necessitating pin replacement through new pinholes.

The development of an external skeletal fixation device has lagged behind other internal fixation devices for use in the horse because of perceived or real problems with device design, strength, and versatility. Our early experience with the AO Tubular System (Synthes, Paoli, PA) or Hoffmann Device (Howmedica, Inc., Rutherford, NJ) in the 1970s showed the inability of these human-oriented systems to withstand the rigors of full weight bearing in the horse. Design and development of an equine external skeletal fixation device was undertaken in 1981 in the Richard S. Reynolds, Jr. Comparative Orthopedic Research Laboratory at New Bolton Center, University of Pennsylvania. A grant from the Swiss AO/ASIF North American group was instrumental in early design and development. The original device incorporated three threaded transfixation pins in the intact bone above the fracture. The pins were connected to sidebars that extended distally to a footplate that allowed the animal to bear full weight on the limb but protected the immobilized fracture below the last transfixation pin. The hoof was attached to the footplate using conventional hoof nails, assuring immobility of the fracture with continued full weight-bearing function of the limb (Fig. 88-1). The results using the external skeletal fixation device in the initial 15 horses were reported in 1986.[4] Eight horses were admitted with open fractures that were not amenable to other acceptable forms of treatment. Primary fracture healing was achieved in seven horses and an additional three horses progressed to frame removal during the course of treatment. Only 4 of 15 horses survived long term. Fatal complications included unrelenting infection in four horses. Two horses fractured the third metacarpal bone (McIII) through the pin sites while wearing the device, and two horses fractured McIII through the pin holes during anesthetic recovery to remove the external skeletal fixation device. We changed the transfixation pin diameter and the ground contact area of the footplate to address these early bone failure problems. In addition, removal of the frame while the horse was standing prevented any further failures through the pin holes in the bone, associated with recovery from anesthesia. In 1991, we reported our continuing experiences with the external skeletal fixation device, which included using the device in another 20 clinical patients. The results of 11 horses with fractures of the proximal phalanx in which the external skeletal fixation device was used were reviewed.[5] Five (45.5%) of 11 horses survived. We felt overall that improvement in survival continued from our original reports, attributed in part to improvements in the design of the external skeletal fixation device. Overall, using the original and modified device in 26 horses, we found the survival rate for the first 13 horses was 15%, whereas the survival rate for the second 13 horses was 53% (overall survival rate 34%).

We continued our work to attempt to reduce fractures through the pin sites (approximately 15%), a complication that occurred with the earlier frame design. In 1994 we described a newly developed concept of loading the external skeletal fixation pin in shear, instead of bending[6] (Fig. 88-2). The concept involved using large-diameter sleeves over the transfixation pins, which were bi-axially loaded in tension and shear. The device used only two pins and incorporated the pins in a stronger, lighter frame (Fig. 88-3). Compared with the old configuration that allowed for pin bending, the loads to bone failure with the new design were nearly an order of magnitude higher. Because bone failure occurs at a finite strain level, it appeared that the larger loads to failure were indicative of lower strain levels in the bone at the working stress level required by the horse fully weight bearing in the frame. Although research and development of this device continues, the current external skeletal fixation device is available commercially (Ron Nash Engineering, Magnolia, AR).

Regardless of the type of external skeletal fixation used, concerns regarding the pin holes remain. Experimental studies in other species suggest that pin holes are remodeled and stress concentrations reduced in 8 to 12 weeks. The holes themselves may never fill in completely. Holes in the bone change sectional properties and bone strength may not equal

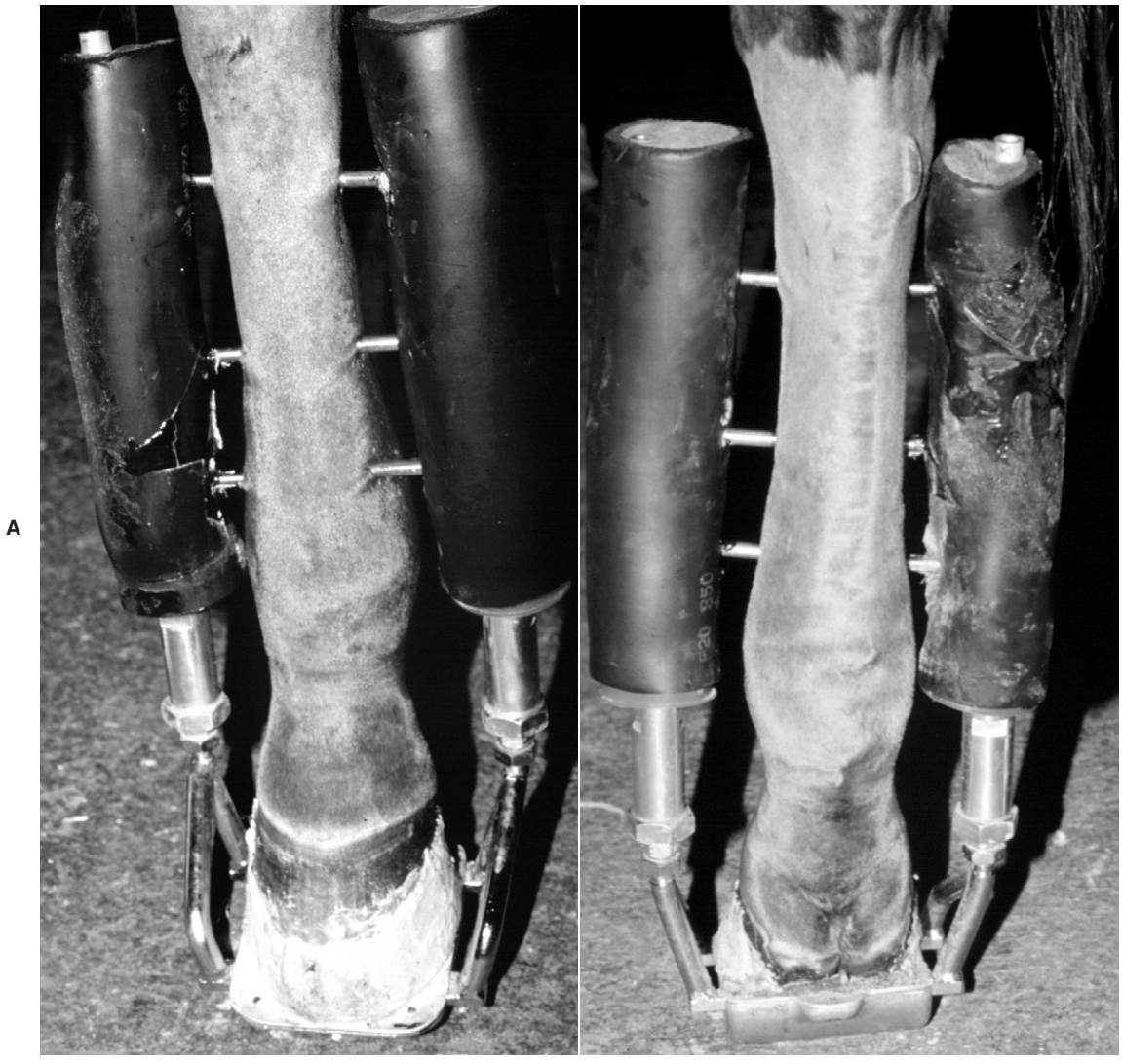

Fig. 88-1 **A,** A dorsal view of the three-pin external fixator shows the relationship of the pins traversing the intact third metacarpal bone and supporting the distal fracture of proximal phalanx. **B,** A plantar view shows the back of the limb with the same fixator in place. The base of the device is attached to the hoof using the glue-on system.

that of bone without holes. Treatment with large diameter external skeletal fixation pins should be reserved for horses that are not expected to return to athletic pursuits and where other methods of fixation are unlikely to preserve the horse's life. Most horses in which external skeletal fixation is contemplated presently have life-threatening injuries and are not likely to return to athletic competition. Treatment of these fractures aims to allow the patient to become pasture sound or to be used for breeding. It is noteworthy that several horses treated with the external skeletal fixation device have returned to light riding or carriage driving, even with fusion of the joint involved in the original fracture.

PRE-OPERATIVE PLANNING, INDICATIONS, TIPS, AND LIMITATIONS

The external skeletal fixation device was designed to treat horses with catastrophic injuries of the distal limb. Indications include comminuted fractures of the proximal phalanx when no strut (intact piece of bone from the proximal to the distal

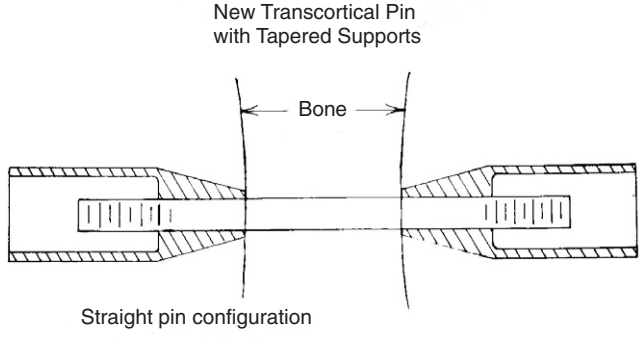

Fig. 88-2 This drawing shows the concept of sleeves that slide over the transfixation pins to give protection from bending in the external skeletal fixation device. The large diameter of the sleeves and the proximity to the bone eliminate bending as a loading mode and ensure a more uniform loading on the pin in shear. This protects the outer bone cortex from the loads of a bending pin and allows the device to be loaded to a greater extent before bone or pin failure.

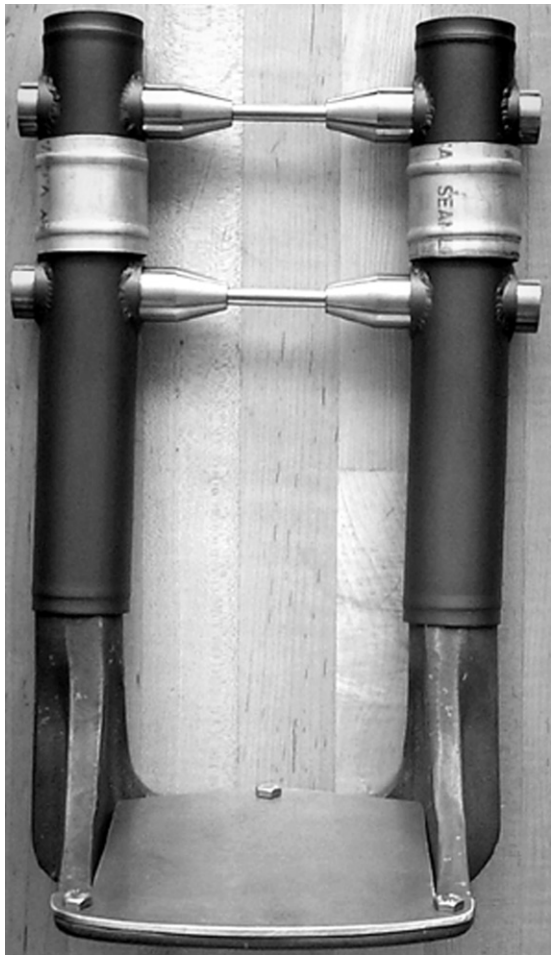

Fig. 88-3 The present external skeletal fixation device design is shown with two transfixation pins and tapered sleeves. The device is assembled easily over the tapered sleeves using the footplate attached to the base.

articular surface) is available for use with internal fixation (see Chapter 36), comminuted fractures of the middle phalanx, fracture dislocations, or traumatic disruptions of the suspensory apparatus with massive soft tissue injury (see Chapter 37). Expanded use of the external skeletal fixation device could be considered in horses with severe laminitis. In its present form the external skeletal fixation device is designed for use when McIII or the third metatarsal bone (MtIII) is intact. The only other requirement is that the blood supply to the distal limb must be intact. In addition to applying the external skeletal fixation device, it is sometimes helpful to insert bone screws, used in lag fashion through stab incisions, to hold large fragments together. This can be done under radiographic guidance and these fixations do not need to resist weight-bearing forces, because the external skeletal fixation device supports the entire weight of the animal. It is necessary that the hoof fits on the base plate of the device and, if large, the hoof needs to be trimmed. This must be accomplished to allow the aluminum base plate to attach to the magnesium-alloy base using the three cap screws. A compression bandage (elastic bandage) is wrapped around the fractured bone before and during surgery to bring the bone fragments closer together and reduce swelling, which helps with reduction of badly comminuted fractures.

Abbreviated instructions for application of the external skeletal fixation device are included to show the simplicity of the system. The procedure is completed with the horse under general anesthesia. The only surgical requirement for application of the external skeletal fixation device is to drill two holes in McIII, although additional internal fixation can be used when appropriate. The footplate is attached using nails or a glue-on system. The limb is prepared for aseptic surgery and, using the drill guide supplied, two 7.94-mm holes are drilled, one at the junction of the proximal and middle thirds and the other at the junction of the middle and distal thirds of McIII from the lateral aspect. Holes are placed in the center of the bone and marrow cavity and perpendicular to the long axis of the bone. This process is facilitated by using a dual drill guide. Next the 7.94-mm pins are inserted using the threaded cap to protect the threads. The sleeves over the pins are assembled on the medial and lateral sides and secured with nuts. The sleeve-skin junction is wrapped with a sterile bandage and the rest of the assembly is done using non-sterile technique. The side tubes are then assembled using ring connectors over the tapered sleeves on both sides of the limb. The base is slid into the distal ends of the tubes, and the base is fastened to the footplate using cap screws supplied. The reinforcing rods are then inserted into tubes on the medial and lateral sides and electrical tape is placed around all the tubular junctions to prevent leakage. The fracture is reduced and stabilized by placing traction on the limb in a position that allows the tubes to be filled. The polymer (Conathane UC41, Cytec Industries, Inc., Olean, NY) is then mixed (2:1 by volume) and poured into the sidebars. The tubing is cooled, using cold saline solution if necessary, to prevent heating of the pins. Last, the now wet sterile wrap around the tapered sleeves is removed, and the external skeletal fixation device is covered with a bandage. The horse is then recovered from general anesthesia.

POST-OPERATIVE CARE

After anesthetic recovery the horse is immediately fully weight bearing and comfortable on the limb. In fact, this is a major advantage of this type of fracture management. Skin care around the pin sites is important during the initial post-operative period and is done easily using antiseptic-soaked sponges. The external skeletal fixation device is checked for tightness using an appropriate deep socket wrench every few days. Pin loosening does not occur, because one has the ability to tighten the pins with the fastener nuts already in place, which is easily done and does not require sedation of the patient. Reducing pin loosening helps to diminish pin tract infections and allows the horse to remain fully weight bearing. Local antiseptic application at the pin sites may decrease severity of pin hole infections. Some patients may be discharged from the hospital with the external skeletal fixation device in place. If the owner or trainer can provide pin care, discharging the horse as soon as possible decreases the cost of hospitalization. The horse is returned to the hospital for removal of the external skeletal fixation device, which is usually after 8 to 10 weeks. Although the external skeletal fixation device can be left on longer, substantial osteoporosis of McIII or MtIII below the lower pin may be detrimental to the end result. Radiographically the fracture site may not appear healed (based on the observation of mature callus) at this time, because osteoid will have formed, but calcification of this soft callus will not have occurred without the stimulus of weight bearing. The external skeletal fixation device is removed in the standing, sedated horse, and a fiberglass cast is applied. Serial radiographic examinations are then performed until radiographic evidence of fracture healing is visible. Premature removal of the external skeletal fixation device may result in collapse of the fracture. Mild collapse of the fracture may occur if the external skeletal fixation device is left in place for only 6 to 8 weeks. However, by this time skin and soft tissues will allow for some fracture collapse without

penetration of bone fragments, and the horse can be managed in a fiberglass cast without complications. Spontaneous fusion of the proximal interphalangeal joint will occur in horses with comminuted proximal phalanx fractures. If fusion of the metacarpophalangeal or metatarsophalangeal joint is necessary, additional internal fixation is needed because spontaneous fusion does not usually occur.

REMOVING THE EXTERNAL SKELETAL FIXATION DEVICE

With the horse sedated, the external skeletal fixation device is removed by using the special pin removal device supplied with the instrumentation. If the footplate has been glued on using the Sigafoos glue on system (Sound Horse Technologies, Unionville, PA), the hoof and footplate are separated using a sharp knife to cut the hard glue. If the footplate was nailed on, the cap screws, bolt, and nut should be removed from the alloy base using a wrench. A hacksaw is then used to cut the lateral sidebar tube just below the lower tapered sleeve. The nuts are removed from the threaded pins on the tapered sleeves. The pin extractor is placed over the distal tapered sleeve and the pin is engaged and removed by turning the extractor with an end wrench. This can be repeated in the proximal tapered sleeve if necessary. Once the pins are removed, the frame can be lifted away and the base plate can be removed like a shoe, if the base plate was nailed in place.

A standing cast is recommended to preserve the structure and function of the extremity for additional time, until healing is sufficient for unrestricted weight bearing. Radiographic control is helpful in making those decisions.

In one horse managed using the original three-pin external skeletal fixation device for a severely comminuted, open proximal phalanx fracture, inadequate reduction of the metacarpophalangeal joint at the time of application of the fixator was noticed. However, rather simple fixation was used to fuse the metacarpophalangeal joint without major collapse of the proximal phalanx. This animal survived and was used successfully as a broodmare. The current external skeletal fixation device is shown in Fig. 88-4.

Using external skeletal fixation for treatment of horses with severe, comminuted fractures or other injuries in the distal extremity is a reasonable choice, but device design and development are still being refined. Transfixation-pin casts are a suitable alternative to rigid external skeletal fixation devices, but soft tissue and pin care cannot be performed.

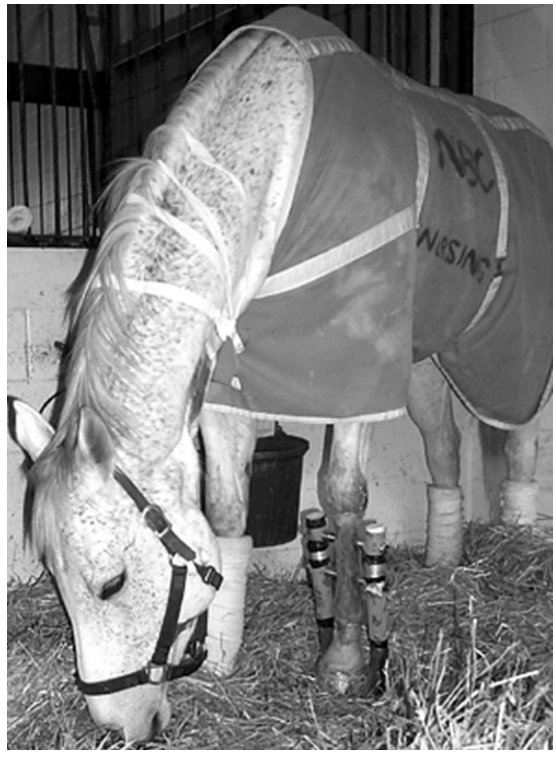

Fig. 88-4 An external skeletal fixation device has been used to manage a comminuted fracture of the middle phalanx.

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CHAPTER • 89

Counterirritation

David Ellis and Stephen P. Dey III

BRIEF HISTORY AND OUTLINE OF TECHNIQUES

Counterirritation techniques have long been a part of veterinary therapy, particularly in horses. Major veterinary texts, such as those by Percivall[1], Wooldridge,[2] and even Stashak[3] as late as 1987, devoted considerable attention to indications and techniques. To some this now seems misguided and even cruel, but one only has to read a few of the older texts to admire the authors' inquiring minds and their thorough observations on matters medical and particularly orthopedic. They were not far from the truth, but they did not have the benefit of modern anesthetics or imaging equipment to learn and apply more direct and rational therapies. Techniques currently used include hot or cold firing and blisters, but use of those therapies is much reduced because of changes in the horse ownership and veterinary attitudes. This chapter considers both British (D.E.) and American (S.P.D.) perspectives.

BLISTERING

Limited availability of ingredients and ignorance of preparation and safe use has restricted blistering of horses in the United Kingdom. Although veterinary surgeons or equine wholesalers are the source, the horse owner is usually the user. More severe blisters, such as those containing cantharides or croton oil, are used less commonly than the working blisters that are iodine or mercuric iodide based and mainly used on splints, sore shins, or curbs. The technique involves rubbing or brushing the liquid preparation onto the clipped skin once daily until a scale forms. The animal is kept in light exercise, such as walking, trotting, or hack cantering, and this phase usually is undertaken after the initial rest and anti-inflammatory treatment has removed heat, swelling, and pain, 7 to 10 days after initial signs. When the heat and soreness from counterirritation has settled, which takes 2 to 3 weeks, the horse resumes work.

Irritation from working blisters is minimal, and protective measures such as anti-inflammatory treatment, keeping the horse cross-tied, or fitting a bib or cradle are not necessary.

Strong blister was made mostly from cantharides (Spanish fly) or croton oil, but nowadays these ingredients are difficult to obtain and, because they are not licensed drugs in the United Kingdom, are probably illegal for a veterinary surgeon to import without permit. As a consequence the strong blister that is used most commonly is made as a cream using red mercuric iodide. The skin is clipped and the blister rubbed into the area for 5 minutes. Excess is removed and petroleum jelly is applied to flexor surfaces distal to the blistered areas. The horse is kept shod for blistering, in case it paws the ground, and a neck cradle is applied until the initial inflammatory phase has subsided. It is essential that the horse continues walking exercise, usually 30 minutes twice daily. A substantial risk of laminitis or lymphangitis exists if blistered horses do not have this regular exercise. When blisters form and burst, they are left alone and sprayed with antibiotic powder. Rarely is the reaction severe enough to warrant anti-inflammatory treatment. After 10 days the blistered areas of the legs are smeared with petroleum jelly, which is left on overnight and then washed off with warm water and soft soap. This softens and lifts the scabs free, without causing bleeding or further irritation. Any blistered areas that are still moist are dressed with antibiotic wound powder and walking is continued. Scabs that persist after 7 to 10 days are removed by repeating this procedure. After 6 weeks the horse can be turned out to pasture or start light ridden exercise. The program for a return to training depends on the nature and progress of the original injury.

Areas treated most commonly with strong blister are the flexor tendon region of forelimbs and the fetlock joints. Indications are tendonitis, tenosynovitis, suspensory desmitis, and chronic effusion of fetlock joints.

In North America, curbs frequently are treated by blistering and both hindlimbs are usually treated. The area is clipped and rubbed aggressively for 3 minutes with medical-grade turpentine and then with kosher salt for 3 minutes, alternating for a total of 15 minutes. This is repeated once daily for 3 days. The horse is exercised immediately after each treatment for at least 20 minutes. The skin excoriates and remains thickened and inflamed for some time, but therapeutic effects are almost immediate.

Blistering also is used to manage suspensory desmitis, tendonitis, sesamoiditis, and other fetlock problems. The severity of the injury determines which blister is used and the degree of exercise modification. If suspensory desmitis or tendonitis is being treated, the metacarpal region usually is wrapped (bandaged) with a gauze layer, a leg quilt, and a bandage to control swelling. Moving areas such as the carpus or tarsus are not wrapped.

The blistering process is allowed to continue for 5 days and then a firing paint is applied to the area for another 7 days, with the bandages reset every day if the blistering process involves the distal area. After the firing paint application period ends, usually an iodine glycerine–based product is applied every day to soften the blister scabs and help lift the outer skin layers that have separated. Once a horse begins to be painted with the firing paint, it can be exercised on a walker or in a small paddock. This reduces inflammation and begins to re-establish normal venous and lymphatic flow.

Internal blisters have more widespread use in North America. A 2% iodine mixture, in almond oil or a lighter base, can be injected into tissues. One author (S.P.D.) treats muscle soreness by injection of 1 ml of internal blister per site using an 18-gauge needle of variable length, depending on the depth of the injection. The mechanism of action is unknown, but trainers usually report that the gait improves. Internal blisters also are used to treat horses with suspensory desmitis, curbs, and splints. Varied results may reflect the inconsistency of the available products.

ACTUAL CAUTERY OR FIRING

Cautery or firing is performed less frequently in the United Kingdom since the Royal College of Veterinary Surgeons deplored the practice in 1991. Cautery or firing is used on soft

and hard tissues, and some clinicians and clients are still enthusiastic about its value. Pin or needle firing is the most common technique.

Cold firing is used mainly for persistent sore shins or splints, and the thermal injury is made with liquid nitrogen (see Chapter 90). The procedure is done soon after the initial inflammatory phase has subsided and is preferred for horses with intractable injuries that have not responded to anti-inflammatory measures or blisters. The area is clipped and sedation and local anesthesia are administered. An ordinary firing iron can be cooled by leaving it in the liquid nitrogen, or a special "gun" point freezes a small area of skin. Spots of lubricant gel on the areas to be fired improve the contact. The pattern should not allow the frozen spots to be closer than 3 cm apart. No systemic or local treatment is needed and the horse is kept in walking exercise for 6 weeks. Ultimately the skin develops persistent white point scars. Results have been good enough to ensure its continuing use, but the rationale is difficult to ascertain. Cold firing probably causes local analgesia of some duration within periosteal nerves and may necrose small areas of the periosteum and underlying cortical bone.

Acid firing enjoyed a vogue 10 to 25 years ago but has deservedly fallen from use. Concentrated nitric acid was soaked into a small cork, which was then pressed onto the clipped and desensitized skin. The inflammatory reaction was substantial but, like line firing, appeared only to affect the skin and not the underlying tendon.

Line or bar firing was used for tendonitis or curbs. For the lines to be made evenly, the area was clipped the day before firing and covered with a mustard plaster overnight, which caused subcutaneous edema to smooth out the grooves between the structures before firing. Curved, blade-shaped irons were used, and management was the same as for pin firing. One author (S.P.D.) has minimal experience with this technique but agrees with Silver et al.[4] that it would not be "a useful therapeutic exercise."

Horses are still pin fired regularly in the United Kingdom. The procedure is used for superficial digital flexor tendonitis, suspensory desmitis, and sesamoiditis and has value for horses with persistently painful splints. Pin firing is no longer used for bone spavin or carpitis. The horse is fired after the initial inflammatory response has subsided and when the area is no longer warm or painful, 4 to 6 weeks after the injury. The area is clipped and anesthetized before being dressed with surgical spirit. Firing is started 10 to 15 minutes later and patterned no closer than 3 cm apart over the structure being treated. It is important to use a fine point so that minimal burning of the adjacent skin occurs. The point penetrates the underlying tendon or periosteum. An iodine-based ointment is then applied, and petroleum jelly is smeared distally, particularly in the flexural regions of joints. The horse is cross-tied, or fitted with a cradle, and walked in hand twice daily for 30 minutes. As mentioned before for blistered horses, walking is essential. It is wise to keep front shoes on and only rarely is analgesia indicated. The area is powdered with antibiotic daily and after 10 days is smeared with petroleum jelly, which is left on overnight and washed off the following morning, as previously described. Walking exercise continues for at least 6 weeks and then the horse can start to spend increasing periods of time turned out in a paddock. The horse should not return to training following a soft tissue injury for about 1 year after the injury. Those horses that have had bony tissue fired, such as with splints, can return gradually to training after the 6 weeks of walking.

In North America, pin firing is used mostly to treat splints; curbs; tendons and ligaments; and fetlocks and surrounding soft tissue injuries. A similar pattern is used for pin firing curbs and splints (S.P.D.). Marks are made down the center of the lesion, 1 cm apart. Alternating rows, medially and laterally, 0.5 cm between the original marks are then made. When firing tendons or ligaments, a similar pattern is used, but the point never goes through the deep layer of epidermis. However, areas of maximum damage are re-fired to ensure deeper penetration to focus the healing process. The horse may require sedation and analgesia for 24 hours after treatment to control pain. The area is painted with firing paint for 10 days, covering it with gauze, a quilt, and bandage. Another 30 days of rest are required before re-evaluating the horse. The total convalescent period depends on the initial injury. In sport horses, use of cryotherapy to treat splints and some soft tissue injuries is more common. Although cryotherapy is less invasive, the procedure is often less effective (see Chapter 90).

Current thoughts on thermal injury to tissues still emphasise the gulf between veterinary science and veterinary practice. Although much excellent science has illuminated the injury and healing of strained tendons, such injuries are still the most significant injuries a flat racehorse can sustain and are a long-term threat to the horse's continuing career. Horses that race over fences (steeplechasers) or hurdles seem to manage better than those that race at a faster pace on the flat. The expectation that counterirritation would produce a fiber pattern with crimp and better-quality collagen may have been ill-founded, but the results that veterinarians have experienced over the years need to be explained. Perhaps the mechanical side effect of pin fire scars, evening out the differing strength and elasticity along the length of the injured tendon and in the contralateral limb, has kept pin firing in equine orthopedics. Beyond enforced rest and low-grade exercise, seeing benefit accruing from blisters used in horses with tendon strain is difficult.

Aggressive anti-inflammatory treatment in the early post-injury phase is important, but one author (D.E.) is uncertain if subcutaneous corticosteroids are essential to a good long-term result.

New treatment regimens such as intralesional glycosaminoglycans or β-aminoproprionitrile fumarate have disappointed, and surgical splitting or proximal accessory desmotomy have not always justified the expense in the United Kingdom. Prolonged rest and controlled exercise regimens seem as successful as any treatments.

The choice of treatment still emerges from consensus between the experiences of the veterinary surgeon, the trainer, and the owner. All equine veterinary surgeons share the fervent hope that successful and rational therapy for strained flexor tendons will emerge soon.

Restrictions on using firing by the Royal College of Veterinary Surgeons were initiated by a question in the Houses of Parliament about the firing of horses. The Horserace Betting Levy Board then funded a research program by the Bristol group, led by Professor Ian Silver. This work was published in a supplement to the *Equine Veterinary Journal*.[4] Extensive discussion followed, but in 1991 the Royal College of Veterinary Surgeons tried to outlaw the technique of firing tendons, by declaring that they deplored it and would bring disciplinary action against any member performing it. This was not well received by a substantial number of respected equine veterinary surgeons and the policy was one that had been poorly thought through, did not recognize hard tissue firing, and proved to be unenforceable. A compromise was later reached, whereby veterinary surgeons had to show that other treatments had failed and to justify their choice of firing a particular horse. The technique continues to be used, but since the introduction of ultrasonography and the retirement of veterinarians who preferred it, firing has diminished considerably. One author (D.E.) believes that firing would have gradually disappeared without legislation.

Techniques currently used in equine orthopedics have become more conservative since the introduction of ultrasonography and the development of finer sensitivity among horse owners. The regular monitoring of these horses has

enabled more rational advice to be given as to healing of the lesions and programming of exercise. Thus the implementation of direct therapy has taken second place among certain groups of clinicians. They remain cynical about therapy while maintaining client confidence through regular observation of the patient. Splitting or needle decompression of a tendon core lesion soon after injury is a rational first step, followed by regular monitoring and controlled, graded exercise throughout a long convalescence. The old adage of the longer the rest, the better still seems valid.

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CHAPTER • 90

Cryotherapy

Kjerstin M. Jacobs and Thomas P.S. Oliver

Cryotherapy has been used widely in veterinary medicine since the 1970s, primarily for tumor ablation. Percutaneous cryotherapy, called *freeze firing* or *freezing,* is a useful palliative technique for various musculoskeletal disorders in the horse, but little research has been published on specific techniques or results.[1-4] Most of the information in this chapter comes from our clinical experience using cryotherapy to manage selected lameness conditions. Cryotherapy generally is used for pain management and our recommendations are made assuming the horse will continue or resume athletic performance.

MECHANISMS OF CRYONECROSIS

Freezing mammalian tissue results in direct and indirect cell destruction. Direct cell injury occurs by formation of intracellular and extracellular ice crystals, which destroy cell walls and cause intracellular dehydration, respectively. Intracellular dehydration causes severe electrolyte concentration and pH shifts, which damage lipoprotein membranes and organelles. Loss of cellular homeostasis results in cell death. Indirect cell injury occurs by damage to the endothelium of arterioles and venules, causing increased vascular permeability, edema, and hemoconcentration. Local tissue damage occurs from thrombosis and infarction of small vessels. Two rapid-freeze, slow-thaw cycles are used. Rapid freezing maximizes intracellular crystal formation and crystal size. Slow thawing causes additional cell damage by a process of re-crystallization, during which time crystals increase in size before melting. Pre-cooled tissue freezes faster than normal tissue; therefore a second freeze-thaw cycle optimizes the processes of tissue destruction.

Because cryotherapy causes tissue destruction in situ, fibrous structures such as epineurium remain intact. This allows for regeneration of large myelinated nerves. Experimental percutaneous cryotherapy of equine palmar and plantar digital nerves resulted in neuropraxia (temporary ablation of nerve function, but the axons remaining intact) or axonotmesis (Wallerian degeneration of axonal tissue, with the fibrous supportive tissue remaining intact). All nerves regenerated.[5] The degree of nerve damage is temperature dependent, with lower temperatures resulting in longer duration of analgesia.[6]

Presumably, percutaneous cryotherapy causes destruction of local type C nerve fibers. Type C fibers are small, unmyelinated, nociceptive nerve fibers that contribute to chronic pain. The inflammatory response to cryotherapy appears to cause thickening and fibrosis of certain soft tissues, such as the suspensory ligament and subcutaneous tissues, and although this effect has not been studied, the clinical result is a stronger ligament and one less likely to be re-injured in the same location. Cryogens must be used judiciously on the distal limb to avoid cryonecrosis of tendon, ligament, joint tissues, or cortical bone.

BASIC TECHNIQUE

Cryotherapy instruments and cryogens are described elsewhere, and only basic principles are discussed here.[7-9] Instruments used to apply cryogens vary from simple cryoprobes to cryounits with continuous closed-system flow of cryogen liquid. We use liquid nitrogen that is stored in a commercial 10- to 20-L tank. For all techniques we use individual brass probes, 1.5 cm in diameter (Fig. 90-1), pre-cooled in liquid nitrogen and positioned on the lesions for a double freeze-thaw cycle consisting of freezing (60 seconds), thawing (60 seconds), and freezing (60 seconds). Local edema formation is minimal and has no effect on the second freezing cycle. Earlier work suggested that a thaw duration of 15 seconds was optimal, but our modified cycle appears effective.[4] We prefer to use solid metal probes, because consistent freezing to a specific depth is easy to control (Fig. 90-2). The cooling ability of solid metal probes has been questioned, but in one study solid metal probes were most effective in freezing to specific depths.[7]

Cryotherapy is well tolerated by most horses, but sedation and a twitch are used. Local analgesia (a ring block proximal to the freeze site) may be necessary for highly strung or fractious horses. The area over the lesion is clipped with a No. 40 blade and cleaned of gross contamination with iodophor or chlorhexidine solution. The limb is dried and a 0.5-cm thick

enabled more rational advice to be given as to healing of the lesions and programming of exercise. Thus the implementation of direct therapy has taken second place among certain groups of clinicians. They remain cynical about therapy while maintaining client confidence through regular observation of the patient. Splitting or needle decompression of a tendon core lesion soon after injury is a rational first step, followed by regular monitoring and controlled, graded exercise throughout a long convalescence. The old adage of the longer the rest, the better still seems valid.

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CHAPTER • 90

Cryotherapy

Kjerstin M. Jacobs and Thomas P.S. Oliver

Cryotherapy has been used widely in veterinary medicine since the 1970s, primarily for tumor ablation. Percutaneous cryotherapy, called *freeze firing* or *freezing,* is a useful palliative technique for various musculoskeletal disorders in the horse, but little research has been published on specific techniques or results.[1-4] Most of the information in this chapter comes from our clinical experience using cryotherapy to manage selected lameness conditions. Cryotherapy generally is used for pain management and our recommendations are made assuming the horse will continue or resume athletic performance.

MECHANISMS OF CRYONECROSIS

Freezing mammalian tissue results in direct and indirect cell destruction. Direct cell injury occurs by formation of intracellular and extracellular ice crystals, which destroy cell walls and cause intracellular dehydration, respectively. Intracellular dehydration causes severe electrolyte concentration and pH shifts, which damage lipoprotein membranes and organelles. Loss of cellular homeostasis results in cell death. Indirect cell injury occurs by damage to the endothelium of arterioles and venules, causing increased vascular permeability, edema, and hemoconcentration. Local tissue damage occurs from thrombosis and infarction of small vessels. Two rapid-freeze, slow-thaw cycles are used. Rapid freezing maximizes intracellular crystal formation and crystal size. Slow thawing causes additional cell damage by a process of re-crystallization, during which time crystals increase in size before melting. Pre-cooled tissue freezes faster than normal tissue; therefore a second freeze-thaw cycle optimizes the processes of tissue destruction.

Because cryotherapy causes tissue destruction in situ, fibrous structures such as epineurium remain intact. This allows for regeneration of large myelinated nerves. Experimental percutaneous cryotherapy of equine palmar and plantar digital nerves resulted in neuropraxia (temporary ablation of nerve function, but the axons remaining intact) or axonotmesis (Wallerian degeneration of axonal tissue, with the fibrous supportive tissue remaining intact). All nerves regenerated.[5] The degree of nerve damage is temperature dependent, with lower temperatures resulting in longer duration of analgesia.[6]

Presumably, percutaneous cryotherapy causes destruction of local type C nerve fibers. Type C fibers are small, unmyelinated, nociceptive nerve fibers that contribute to chronic pain. The inflammatory response to cryotherapy appears to cause thickening and fibrosis of certain soft tissues, such as the suspensory ligament and subcutaneous tissues, and although this effect has not been studied, the clinical result is a stronger ligament and one less likely to be re-injured in the same location. Cryogens must be used judiciously on the distal limb to avoid cryonecrosis of tendon, ligament, joint tissues, or cortical bone.

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Cryotherapy is well tolerated by most horses, but sedation and a twitch are used. Local analgesia (a ring block proximal to the freeze site) may be necessary for highly strung or fractious horses. The area over the lesion is clipped with a No. 40 blade and cleaned of gross contamination with iodophor or chlorhexidine solution. The limb is dried and a 0.5-cm thick

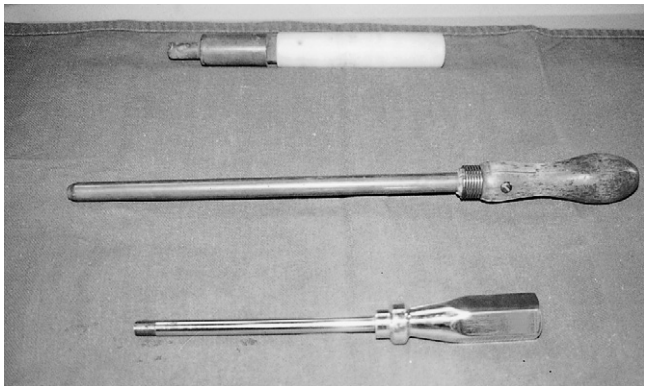

Fig. 90-1 Solid brass cryoprobes commonly used for cryotherapy in the horse.

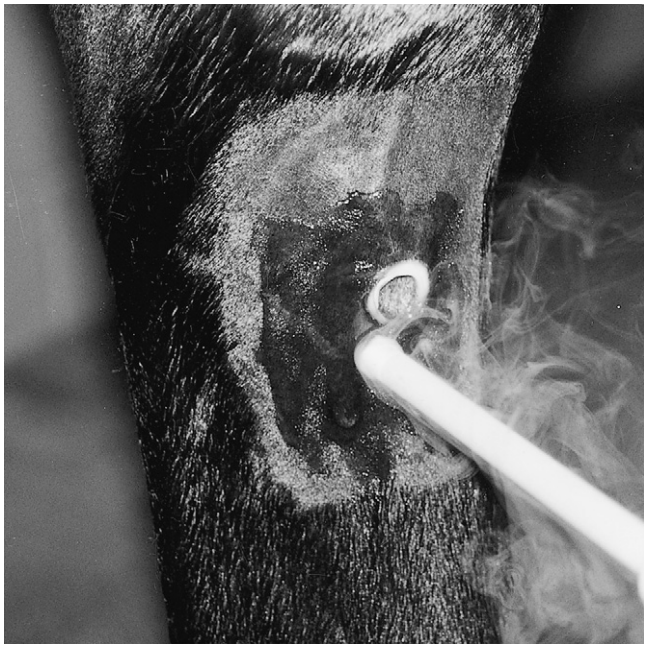

Fig. 90-2 Cryotherapy is being used to manage pain associated with exostosis of the fourth metacarpal bone in a Standardbred racehorse. A double freeze-thaw cycle has been used in one site, and the probe is being applied at a distal site.

layer of water-soluble gel is applied generously over the site. This layer provides a template to ensure accurate positioning for the second freeze cycle.

The metal probe tip is applied to the skin for 60 seconds, then removed to allow thawing, and re-applied (see Fig. 90-2). No benefit accrues from a third freeze-thaw cycle, and in fact a third cycle may lead to underlying bone necrosis. Freeze sites should be located 1 cm apart in staggered, parallel rows that cover the lesion site and extend 1.5 cm beyond the margins of the lesion. If freezing sites are closer than 1 cm, the areas of skin necrosis will coalesce and later slough.

After completing the procedure, the leg is cleaned of lubricant and the sites are covered with an anti-bacterial spray. The leg should be left unbandaged, but it is kept dry, and antibacterial spray is applied daily until skin healing is complete. Local soft tissue swelling and edema develops and persists for 7 to 10 days and then gradually resolves. The administration of anti-inflammatory agents should be avoided, because this medication limits the intended effect of cryotherapy. Percutaneous cryotherapy destroys melanocytes in skin cells, so hair regrowth will be white. This negative cosmetic effect should be considered when discussing complications of cryotherapy.

TREATMENT OF SPECIFIC LESIONS

Diagnosis always should be confirmed using diagnostic analgesia, because results obviously are improved if the painful area is identified correctly. Most horses return to full function and race performance within several days to weeks, but some (e.g., those treated for severe suspensory desmitis) require up to 120 days of rest after treatment. When managing horses with suspensory desmitis, rest is paramount but is a source of frustration for trainers and owners, who often want to rush a horse back to the races.

Splint Exostoses (Splints)

Exostoses of the second, third, and fourth metacarpal and metatarsal bones should not be treated until acute inflammation has subsided, because cryotherapy may exacerbate periosteal reaction. Horses are given 2 weeks of rest during which a poultice is applied for 5 days, followed by the application of a cedar oil blister for 5 to 7 days. Scurf from the blister is removed by sweating before cryotherapy is performed. Horses with chronic splints can be managed with cryotherapy without this 2-week preparation period. After cryotherapy in any horse, stall rest is recommended for 3 to 5 days. The horse then may be jogged or galloped lightly and brought back into full training within 7 to 10 days after treatment. Prognosis is good for return to racing. The procedure may be repeated at the same site, or at additional sites, if pain recurs or new sites develop.

Second and Fourth Metacarpal or Metatarsal Fractures

Ostectomy of distal fragments of the second or fourth metacarpal or metatarsal bones is ideal, but in some horses surgery is not an option and cryotherapy can provide analgesia. Acute inflammation must be resolved before treatment. A single freeze site is placed directly over the fracture site, and four more sites are frozen in a circular pattern around the fracture site. Prognosis and rest depend mostly on extent of associated suspensory desmitis, if present, because horses with simple splint bone fractures can return to work immediately. Horses with mild suspensory desmitis have a good prognosis for return to racing and usually receive 60 days of rest after cryotherapy. Horses with severe suspensory desmitis require a minimum of 120 days of rest and have a guarded prognosis.

Periostitis of the Third Metacarpal Bone (Bucked Shins)

Cryotherapy can be used to manage pain associated with bucked shins once acute inflammation has subsided. A dorsal cortical fracture of the third metacarpal bone must be ruled out, because cryotherapy in horses with fracture is contraindicated. Once swelling and palpable pain resolve, cryotherapy is performed using three vertical rows of eight to nine freeze sites per row. After cryotherapy, 2 weeks of stall rest with hand walking followed by 4 weeks of turnout exercise is given. Prognosis is excellent and few horses require additional treatments.

Suspensory Desmitis

Ultrasonography must be performed first to determine the severity and extent of desmitis. Before cryotherapy, acute inflammation is managed using rest, anti-inflammatory medication, and local therapy, such as cold-water hosing and poultice application. Cryotherapy is then performed using a vertically

oriented row of freeze sites, beginning 3 cm proximal and extending 3 cm distal to the lesion. It is crucial that each freeze site is 1 cm apart and is located directly over the body of the suspensory ligament and not over the deep and superficial digital flexor tendons. Cryotherapy is performed over each affected branch. If suspensory body desmitis is present, the procedure is performed on the medial and lateral aspects. Horses are given a minimum of 2 weeks of stall rest with hand walking, followed by 2 weeks of turnout or swimming physiotherapy. The suspensory ligament and overlying tissue become fibrotic and rigid after treatment and, although tissue may appear strong to laypersons and the horse may be sound at the walk and trot, it is essential to recommend a conservative return to training and racing. Prognosis always is guarded, but most Standardbred racehorses return to racing. Suspensory desmitis is often a compensatory condition caused by contralateral limb lameness, or lameness in a diagonal or ipsilateral limb, and management of the primary problem is critical in decreasing recurrence. Cryotherapy is seldom effective for long-term management of suspensory desmitis if primary lameness continues. After developing suspensory desmitis, horses may drop in race class and value, but cryotherapy has been a useful adjunct in managing pain and allowing continued racing.

Distal Hock Joint Pain (Jack Spavin or Cunean Tendonitis)

Cryotherapy is useful in managing horses with distal hock joint pain. A double horizontal row of freeze sites over the proximal and distal borders of the cunean tendon is used. The horse is given stall rest with hand walking for 2 weeks. Prognosis is excellent for return to racing in the Standardbred racehorse.

Osteoarthritis of the Proximal Interphalangeal Joint

Chronic pain from osteoarthritis of the proximal interphalangeal joint may be alleviated partially using cryotherapy and is most useful in those horses in which palmar digital neurectomy fails to abolish all pain originating from the proximal interphalangeal joint, especially in those with severe bone lysis and proliferation and in which arthrodesis is not an option. Pain relief sometimes can be achieved by placing cryotherapy sites 1 cm apart, encircling the proximal interphalangeal joint proximal to the lesion, and directly over the sites of bony proliferation. It is important to avoid the superficial digital flexor and deep digital flexor tendons. After treatment, horses are given stall rest with hand walking for a minimum of 3 weeks. Prognosis is guarded.

Curb

Curb, a collection of injuries located along the distal, plantar aspect of the tarsus, can be managed successfully using cryotherapy (see Chapter 79). Acute inflammation must be resolved, a process that may take as long as 7 to 10 days. The number of cryotherapy sites depends on the size of the curb, but 8 to 12 sites are usually required. Horses are given stall rest with hand walking for 2 to 3 weeks, but further rest depends on the structure involved and severity of initial lameness. Prognosis is usually good, but some horses require additional treatment within 6 to 12 months.

Cryoneurectomy

Cryotherapy can be useful to provide analgesia to the palmar or plantar aspects of the digits, but the degree of cryoanalgesia depends on the technique used. Percutaneous cryoneurectomy has limited clinical use, because only temporary pain relief (3 to 4 weeks) is achieved.[5] Cryoneurectomy was more effective in producing neuroanalgesia than was simple transection, but the former procedure requires a surgical approach.[10] The nerves are exposed and transected, and the proximal ends are frozen.

A modified approach can be used.[5] The palmar or plantar digital nerves are exposed and a double freeze-thaw technique is used to freeze the nerve within the perineurium, but the nerve is not transected. Analgesia is longer than with percutaneous cryotherapy, but having to expose the nerves limits the practical application of this technique.

Tendonitis

We feel management of superficial digital flexor or deep digital flexor tendonitis with cryotherapy is contraindicated. After cryotherapy, fibrous tissue formation along the tendon fibers and possible scarring and adhesions of the tendon sheath can occur and limit prognosis. Although we are aware of horses that have raced after cryotherapy for tendonitis, we suggest that other management options be explored (see Chapters 70 and 71).

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CHAPTER • 91

Radiation Therapy

Alain P. Théon

Radiation therapy is the use of ionizing radiation to treat patients with malignant tumors and, occasionally, selected benign diseases. The analgesic action of ionizing radiation for chronic orthopedic conditions has long been recognized and was first reported soon after the discovery of x-rays.[1] The effectiveness of radiotherapy has been demonstrated in people suffering from painful, degenerative joint diseases that were refractory to first-line conservative treatment; for example, non-steroidal anti-inflammatory drugs and physiotherapy.[2] In horses radiation therapy was found to be effective for painful degenerative and inflammatory musculoskeletal conditions early in the development of veterinary radiation therapy.[3] However, limited access to treatment facilities for horses, fear of the long-term hazards of ionizing radiation, and the availability of potent anti-inflammatory drugs have decreased the impact of radiation therapy in the overall management of equine lameness. Although most clinical studies on using radiation therapy for equine chronic orthopedic conditions were reported in the 1960s and the 1970s, current interest is renewed because of a better understanding of the mechanisms of action of radiation and identification of specific indications for treatment. Two studies are currently under way at the University of California, Davis, Veterinary Medical Teaching Hospital for treatment of chronic synovitis and prevention of excessive bone proliferation. In addition the emergence of the specialty of veterinary radiation oncology will provide the expertise and personnel for expanding the applications of radiation therapy for treatment of non-cancerous conditions at veterinary schools and private referral practices.

RADIOBIOLOGICAL ASPECTS

Radiotherapy of non-malignant conditions requires low doses of radiation, usually one fifth to one tenth the dose used in cancer treatment. The radiobiological mechanisms involved in the therapeutic effects of low-dose radiation used for benign conditions are different from those mediated by high radiation doses used to treat cancer. The therapeutic effects of radiotherapy for cancer result from cell death and inhibition of tumor cell proliferation. The therapeutic effects of low-dose irradiation in treating painful musculoskeletal disorders result from several distinct mechanisms.

Early data indicating an increase in dermal blood flow and cutaneous temperature after treatment[4] led to the misconception that radiation therapy produces a deep counterirritation that promotes increased blood flow and cell recruitment to the treated area to expedite healing.[5] This theory, however, is not supported by any current data. The previously observed effects merely reflect inaccurate dosimetry that resulted in high radiation doses to the skin. In addition, evidence shows that counterirritation during the chronic stage of inflammation prolongs healing time and results in further damage.[6]

Modern theories on mechanisms of low- to medium-dose radiation for treatment of non-cancerous conditions are based on new experimental data. The mechanisms are grouped into anti-inflammatory effects, analgesic effects, and anti-proliferative effects.

Anti-Inflammatory Effects

Whereas good evidence exists that high radiation doses induce strong inflammation in normal tissues, equally good evidence exists that low radiation doses have the opposite effect in inflamed tissues. Low-dose radiation therapy has a pronounced anti-inflammatory effect on acute and chronic inflammatory processes in joints and peri-articular tissues. In experimental animal models of osteoarthritis, low-dose irradiation reduced bone loss, synovial proliferation, cartilage degradation, joint swelling, and pain.[7]

The anti-inflammatory effects of irradiation are not caused by cytotoxic effects, because the low doses used are not lethal to cells, and activated inflammatory cells, including lymphocytes,[8] monocytes, and macrophages,[9] have lost the clonogenic potential and are therefore radioresistant.

Clinically observed anti-inflammatory effects of low-dose irradiation appear to result from functional alteration in cells involved in the inflammatory response. Adhesion of white blood cells to activated endothelial cells and induction of nitric oxide synthetase in activated macrophages are reduced. Radiation decreases leukocyte adhesion, thereby reducing recruitment of granulocytes to inflamed tissue, which results in decreased proteolytic enzyme release and reduced tissue necrosis. Radiation-induced inhibition of nitric oxide production results in reduced inflammatory reaction.[9,10] Good evidence also exists that radiation-induced reduction of monocyte and macrophage cytokines prevents excessive fibrosis.[11]This may explain subjective reduction in joint capsule fibrosis and the functional improvement after treatment of osteoarthritis.[12]

Analgesic Effects

The analgesic effects of ionizing radiation on degenerative and inflammatory disorders are manifested by early effects of short duration, followed by long-term, delayed effects. The delayed effects, characterized by long-lasting pain relief that develops several weeks after treatment, result from the anti-inflammatory effects of radiation. Early effects, characterized by fast pain mitigation, result from radiation-induced modulation of the afferent nociceptive pathways. Analgesia involves non–opioid- and opioid-mediated mechanisms. Although the opioid-mediated analgesia is poorly understood,[13] the non-opioid mechanisms (which include modification of pain transmission and perception) appear to be mediated in part by nitric oxide.[14-16] Radiation-induced decrease in local nitric oxide production results in desensitization of the nociceptors[17] and prevention of neuropathic pain development.[18]

Anti-Proliferative Effects

Ionizing radiation inhibits cell proliferation and temporarily reduces the production of new cells. This mechanism is assumed for prevention of new bone growth and treatment of chronic synovitis. Low-dose irradiation does not interfere with normal bone healing, because native osteoblasts are not

irreversibly inactivated by radiation. However, irradiation can inhibit excessive new bone formation from existing bone and within muscle after trauma or surgery. Radiation target cells are pluripotent mesenchymal stem cells that are stimulated to proliferate and differentiate into osteoblasts after trauma (e.g., injury or surgery) as a result of the healing process.[19]

Surgical trauma initiates a sequence of events during which cell proliferation takes place within a specific time. Radiation done during this period induces a delay in cell production that results in a full and permanent therapeutic effect. In people the ability of irradiation treatment to interfere with the formation of heterotopic bone is limited to 4 hours before surgery to 48 hours after surgery.[20] In horses the timing of radiotherapy to prevent new bone growth is also important and irradiation usually is done immediately after surgery. In ponies with experimental osteochondral defects in the antebrachiocarpal and middle carpal joints, radiation therapy was not effective in preventing periostitis and peri-articular osteophytes when given 6 weeks after surgical trauma.[21] For mature ossification, irradiation alone has no value beyond pain control.

In treating chronic synovitis the anti-proliferative effects of irradiation only delay progression of the disease process, because no critical period exists during which cell proliferation is required for the expression of damage. The goal of radiation-induced synovectomy is to ablate inflamed, proliferative synovium, with the expectation that after treatment the regenerated synovium will be free of disease.

Treatment Side Effects

Much of the concern surrounding using irradiation to manage benign conditions is the presumed risk of radiation-induced malignancy. The low doses used, however, are below the threshold associated with an identifiable risk of malignant transformation. In horses, development of in field sarcoma secondary to radiation therapy to manage benign conditions has not been reported. Reported skin and bone damage likely reflect inadequate treatment techniques according to modern standards.

Skin Damage

Skin damage including epilation, dry desquamation, and regrowth of depigmented hair was often seen when low-energy orthovoltage radiation or insufficiently filtered radioisotope sources were used. The use of modern irradiation techniques and high-energy megavoltage radiation has eliminated the risk of skin overdose and skin reactions are no longer observed.

Osteopenia

Similar to findings in people, experimental[21,22] and clinical (University of California, Davis, Veterinary Medical Teaching Hospital, unpublished data) studies in mature horses have shown that low doses of radiation (<10 Gy) do not affect bone density measured radiographically and do not increase risk of fracture. Although irradiation inhibits excessive bone remodeling and periosteal reactions after surgery, it does not appear to interfere with bone healing. In immature horses, the use of radiation therapy to treat periostitis and arthritis is discouraged because radiation interferes with bone growth.[23]

IRRADIATION TECHNIQUES

The administration of ionizing radiation is analogous to the prescription of medications based on pharmacological principles. Radiation doses are expressed in gray (Gy), the Système International unit, which has replaced the roentgen (R) and the rad (radiation absorbed dose; 1 Gy = 100 rad).

Before treatment, areas to be irradiated must be identified radiographically to ensure delivery of the radiation dose to the appropriate target volume. Radiotherapy techniques vary considerably based on tissue involved, location, and equipment available. Two methods of irradiation available for treatment of benign lesions in horses are teletherapy (external beam therapy) and brachytherapy.

External Beam Irradiation

Teletherapy involves the external administration of radiation therapy (at a distance of 50 to 100 cm from the area to be treated) by machines that emit x-rays or γ-rays, which can be collimated and directed precisely at the lesion. No difference exists in the mechanism of action between x-rays or γ-rays. In the past, treatments were done with low-energy orthovoltage x-ray units[24-26] producing low-energy x-rays (60 to 90 keV), and the radiation dose was given in a series of three to five exposures. Dosimetry was inaccurate because of the poor penetration of radiation and movement of the standing horse that was physically restrained during treatment. Currently, teletherapy is done exclusively with linear accelerators that produce high-energy x-rays or telecobalt units that produce high-energy γ-rays.[27] The advantages of high-energy (>1000 keV) radiation include skin sparing, deep penetration into tissue, and precise dosimetry. A distinct disadvantage of this technique is the need for general anesthesia to ensure accurate positioning and immobilization of the horse during treatment. Delivering a single radiation dose ranging from 8 to 12 Gy takes 6 to 8 minutes, so complete immobilization is crucial. Accurate radiographic definition of the area to be treated is essential for efficacy and sparing of the surrounding normal tissues.

This treatment method requires high investment in equipment and involves the registration of the radiation therapy unit with government agencies. As a result, such treatments for horses are usually provided at veterinary schools.

Brachytherapy

Brachytherapy is a treatment in which radioactive sources are applied directly to the area to be treated. The short distance between the radiation source and the lesion allows delivery of the radiation dose with minimal exposure to surrounding, uninvolved normal tissues. The disadvantage is radiation exposure to the radiotherapist during positioning and removal of the sources and to personnel responsible for the care of horses during treatment.

Treatment involves using sealed radioactive sources implanted directly into tissues (interstitial brachytherapy) or arranged into an applicator positioned on the skin above the area to be treated (surface brachytherapy). An alternative approach consists of injecting unsealed radioisotopes into a joint space (intracavitary brachytherapy) to deliver the radiation dose directly to the synovium.

This treatment method requires minimum facility and equipment investment, but obtaining a license for laboratory and industrial use of radioactive materials is necessary. This type of license is issued only to an individual that can document extensive training and experience in the safe use of radioisotopes for medical and veterinary purposes.

Interstitial Brachytherapy

Radon gas (^{222}Rn) in beads and radioactive gold (^{198}Au) pellets that emit γ-rays have been used for interstitial brachytherapy. Radioactive sources are inserted permanently and slowly deliver the radiation dose to the surrounding tissue, until complete decay occurs. To minimize personnel exposure, the implantation procedure is done in two steps: first, the insertion of unloaded needles in tissues according to specific radiation planning rules; and second, the loading of the needles with radioactive sources using a special implantation instrument. Achieving consistently satisfactory implants requires a good deal of practice. Treatment is expensive, because radioactive

sources can be used only once. This technique is now used rarely and is not allowed in most areas of the United States (the reader may inquire at the local department of social and health services for state regulations, or with the appropriate government agency in other countries). The major drawback is potential radiation hazard, because the implant is still radioactive when the horse is discharged from the hospital.

Surface Brachytherapy

Surface brachytherapy (plesiotherapy) involves using a surface applicator containing an array of linear radioactive sources. The most common brachytherapy technique involves inserting radioactive sources in the form of rigid needles applied on the skin overlying the lesion. Radioactive cobalt (^{60}Co) and cesium (^{137}Cs) emit a useful beam of γ-rays and are the most common radioisotopes used. Rigid (cast)[28] or flexible (pack)[29] applicators must be light, immobile, well fitted, and comfortable because they are left in position for an extended period to deliver the radiation dose. Dose distribution in tissues depends on arrangement and number of radioactive sources. Treatment doses range from 5 to 15 Gy, given at a low-dose rate over 48 to 72 hours (Table 91-1). Horses must be confined to an isolation facility during treatment to manage the radiation safety hazard posed by large ambient exposure rates around the applicator. Once the dose is delivered and the pack is removed and stored in a shielded container, a radiation hazard no longer exists. The horse then can be discharged safely from the hospital.

Table • 91-1

Radiation Therapy for Painful Degenerative and Inflammatory Musculoskeletal Conditions in Horses

CONDITION	TREATMENT TECHNIQUE	RADIATION DOSE	NO. OF CASES	RETURN TO RACING (%)	REFERENCES
Osteoarthritis					
Carpus	Plesiotherapy with γ-radiation (^{222}Rn, ^{60}Co)	5-10 Gy	39	80	12, 24, 36, 37
	Teletherapy with orthovoltage x-rays (HVL*: 0.5-1.3 mm Cu)	5-15 Gy given in 3 to 7 dose fractions	38	71	24-26
PIP joint	Plesiotherapy with γ-radiation (^{60}Co)	8 Gy	1	100	24
	Teletherapy with orthovoltage x-rays (HVL: 0.5 mm Cu)	4-10 Gy given in 3 to 7 dose fractions	21	62	26
MCP joint	Plesiotherapy with γ-radiation (^{222}Rn, ^{60}Co)	5-9 Gy	19	74	12, 24, 37
	Teletherapy with orthovoltage x-rays (HVL: 1.3 mm Cu)	12-15 Gy given in 3 dose fractions	5	40	24
Sesamoiditis	Plesiotherapy with γ-radiation (^{222}Rn, ^{60}Co)	5-9 Gy	10	80	12, 24, 36, 37
	Teletherapy with orthovoltage x-rays (HVL: 1.3 mm Cu)	10-12 Gy given in 3 dose fractions	4	75	24
Navicular osteitis	Plesiotherapy with γ-radiation (^{222}Rn)	4.5-9 Gy	4	50	36
	Teletherapy with orthovoltage x-rays (HVL: 1.3 mm Cu)	12-15 Gy given in 3 dose fractions	2	100	24
Villonodular synovitis	Post-operative teletherapy with orthovoltage x-rays	12 Gy in 4 dose fractions	14	90	33
Tendonitis/Desmitis					
Suspensory ligament	Plesiotherapy with γ-radiation (^{222}Rn)	4.5-7 Gy	4	50	36
Flexor tendon	Plesiotherapy with γ-radiation (^{222}Rn, ^{60}Co, ^{182}Ta)	7-15 Gy	4	25	28, 37
	Teletherapy with orthovoltage x-rays (HVL: 0.5-1.3 mm Cu)	4.5-15 Gy given in 3 to 7 dose fractions	5	80	24, 26
New Bone Growth					
Splints	Plesiotherapy with γ-radiation (^{222}Rn)	4.5-9 Gy	4	50	36
	Post-operative plesiotherapy with γ-radiation (^{60}Co)	5 Gy	5	100	12
McIII periostitis	Plesiotherapy with γ-radiation (^{222}Rn)	9 Gy	2	100	35
	Teletherapy with orthovoltage x-rays (HVL: 0.5 mm Cu)	4-10 given in 3 to 7 dose fractions	8	37	26

* *HVL,* Half-value layer; *PIP,* proximal interphalangeal; *MCP,* metacarpophalangeal; *McIII,* third metacarpal bone.

Surface application has a distinct disadvantage over a therapeutic x-ray machine, because movement of the pack above the skin affects the radiation dose distribution on the area to be irradiated. The potential for source damage, release of radioactive material, and contamination of the facility for several years are other disadvantages.

Intracavitary Brachytherapy

Intracavitary brachytherapy is a new approach developed to concentrate the radiation dose in the target tissue and is used primarily to treat synovitis. Radiation is delivered by intra-articular administration of unsealed β-emitting radioisotopes. The short emission range of the β particles (electrons) provides a high radiation dose to the synovium in contact with the radioisotope. Diseased synovium is ablated, but the effects on the peri-articular tissues and underlying cartilage are minimal.

Several formulations have been developed to prevent leakage of radioisotope from the site of injection. Radiocolloids labeled with radioactive gold (^{198}Au), yttrium (^{90}Y), phosphorus (^{32}P), and rhenium (^{186}Re) and particulate radiopharmaceuticals labeled with radioactive dysprosium (^{165}Dy), holmium (^{166}Ho), and samarium (^{153}Sm) have been used successfully in people. The radioisotope used is based on the expected thickness of the synovium.[30] Hydroxyapatite microspheres labeled with ^{153}Sm have been shown to be safe and effective for intra-articular administration in horses. The estimated treatment doses ranged from 7 Gy to 34 Gy.[31]

CLINICAL APPLICATIONS

Irradiation has been shown to be efficacious for clinical treatment of painful degenerative and inflammatory musculoskeletal conditions in horses. The treatment goal is long-term pain relief and return to racing or performance. Although modern standards of clinical research cannot be applied to these clinical studies, the general message is a remarkable consistency in the response pattern.

Although radiation therapy should not be used indiscriminately, it should not be viewed as a last resort. Radiation therapy is effective in managing refractory, chronic stages of various degenerative disorders and should be used for conditions that do not respond within a few months to conservative treatments, including administration of non-steroidal anti-inflammatory drugs, local injections (corticosteroids or other anti-inflammatory products), physical therapy, and physiotherapy. Strenuous exercise should be avoided, and the horse should be given state-of-the-art physiotherapy after radiation therapy is used. General recommendations include stall rest with hand walking for 30 to 90 days and a gradual return to regular training over 60 to 90 days.

A summary of clinical results of radiotherapy for painful refractory orthopedic conditions is found in Table 91-1. Overall success rate was 60% to 70%, although data are not contemporary and techniques are different from those practiced today. Onset and duration of analgesia were often difficult to measure. Low success rates were found in horses with chronic conditions or in those in which many unsuccessful treatments had previously been performed.

Chronic Synovitis

When synovitis plays a major role in the arthritic process, radiation therapy may be advantageous. Treatment is directed at reducing synovial inflammation and proliferation to prevent progressive joint deterioration and control of joint effusion, fibrosis, and pain. Inflamed synovium induces damage to the articular surfaces by causing effusion, up-regulation of endothelial adhesion molecules, liberation of inflammatory mediators and destructive enzymes, and alterations in the synovial fluid constituents.[32]

Chemical, surgical, or radiation-induced synovectomy can be performed. Similar to surgical synovectomy, radiation synovectomy destroys diseased synovium, and the intent is to allow regeneration of normal synovium and amelioration of clinical signs. In people the duration of response after radiation synovectomy is related inversely to the amount of cartilage and bone destruction. These findings emphasize the importance of screening horses for radiological evidence of cartilage and bone destruction before considering radiation synovectomy. It is imperative to discriminate pain arising from inflamed synovium from that originating from cartilage or bone damage, so that horses with pain from the latter, which is likely to be unaffected by radiation synovectomy, do not undergo unnecessary treatment.

Irradiation is recommended in horses with chronic osteoarthritis, after osteochondral fragment removal if synovium is proliferative, in horses with idiopathic synovitis, and potentially as an ancillary treatment for infectious arthritis. The role of radiation therapy in treating infectious arthritis is limited to preventing chronic synovitis and treating reactive arthritis. Radiation therapy should not be used until bacteria are eliminated, because the anti-inflammatory effects of radiation can exacerbate infection. In addition, radiation therapy should be limited strictly to adult horses, because the anti-proliferative effects of radiation damage growing bones and joints in young horses. External beam irradiation can be used as adjuvant to incomplete surgical synovectomy.[33,34] Radiation doses of 7 to 10 Gy do not cause delayed wound healing or wound complications when the incision is excluded from the field of radiation. Intra-articular administration of ^{153}Sm is used as a single modality when thickness of the inflamed synovium does not exceed 1 mm.[30] Treated horses may experience transient pain (lameness), pitting edema, and effusion. In people, concomitant intra-articular injection of triamcinolone acetonide has been shown to prevent transient local reaction and to reduce pain.

Degenerative Bone and Joint Disorders

Radiation therapy is used to treat various types of equine lameness caused by inflammatory and degenerative changes affecting bones, joints, and tendons. The most common indications for radiation therapy include chronic traumatic arthritis and osteoarthritis of the carpal and fetlock joints, sesamoiditis, active carpitis with swelling and mild mineralization, and chronic carpitis. Radiographic examination should be performed before radiation therapy is performed. This helps ensure that the cause of lameness is not an osteochondral fragment or another condition that is best handled surgically. The anti-inflammatory and analgesic effects of radiation may stabilize the injury process in subacute and chronic painful disorders, when peri-articular new bone proliferation is minimal and damage to cartilage is not obvious. Joints with end-stage osteoarthritis are unlikely to benefit from irradiation.[25] Because radiation has no inhibitory effect on the degenerative processes in osteoarthritis, the treatment goal is limited to pain relief.

Radiation therapy can be used for tendonitis, tenosynovitis, and desmitis. Long-term results in horses with acute and subacute conditions are better than those with chronic conditions. In horses with chronic osteoarthritis, in which substantial peri-articular radiographic changes have occurred, radiation therapy is used to control inflammation and pain, and to prevent excessive fibrosis and calcium deposition, which can result in dysfunction, reduced joint motion, and deformity. Horses with severe osteoarthritis, which show extensive new bone, large amounts of mineralization of soft tissues, and evidence of cartilage destruction, are not treated routinely because they rarely improve enough to perform satisfactorily.

Treatment results have not been uniform because of many variables, including severity and location of the lesion, degeneration of the articular cartilage and pre-existing arthritis,

radiation dose and technique used, and duration of rest after treatment. In addition, questionable causes and pathogenesis make degenerative disorders prone to polypragmasy. Finally, the effects of enforced rest periods in the overall management are not known, which makes definitive assessment of the value of radiation therapy impossible.

The efficacy of plesiotherapy using ^{137}Cs packs was evaluated in 147 horses (46% Thoroughbreds, 27% Quarter Horses, and 12% Standardbreds) treated at the University of California, Davis, Veterinary Medical Teaching Hospital. The treatment dose was 12 Gy given over 24 to 32 hours. Treatment responses were judged to be good, meaning that the horse returned to racing and was sound, or poor, meaning that the horse showed no response or the response was temporary. The most common use was to treat osteoarthritis (characterized by bone remodeling, with lysis or proliferation, and narrowed joint spaces) affecting the carpal, metacarpophalangeal, and proximal interphalangeal joints. Treatment was most effective for carpitis (84% good response, $n = 51$), osteoarthritis of the proximal interphalangeal joint (77% good response, $n = 22$), and sesamoiditis (80% good response, $n = 12$), and least effective for osteoarthritis of the metacarpophalangeal joint (60% good response, $n = 25$) and splints (55% good response, $n = 20$). Overall results were less favorable for treatment of desmitis and tendonitis (64% good response, $n = 17$); however, horses treated in this study had a long history of lameness. Prognosis for horses with mild lameness caused by curb appeared favorable, because five of seven horses had a good response. Compared with previous reports (see Table 91-1), the better responses observed in this study may reflect the use of a higher radiation dose. The results should be interpreted cautiously, because this was not a controlled study and objective criteria of response were not available.

Preventing New Bone Growth

Post-traumatic exostosis occurs after repair of bone fractures (third carpal bone and third metacarpal or metatarsal bone condylar fractures) and after certain surgical procedures, such as arthrodesis and distal splint bone removal. The incidence of radiographic findings is higher than that of clinical signs and functional impairment. Risk factors that predispose horses to develop exostoses are not known. A history of previous new bone growth after surgery is the most substantial predisposing condition. Although a report described regression of excessive callus on a splint bone,[35] horses with painful mature exostosis or peri-articular and soft tissue calcification usually do not benefit from radiotherapy except for pain relief. These patients require excision of calcified tissue followed by post-operative irradiation.

Radiation therapy is a powerful prophylactic treatment to prevent abnormal ossification in stress-related bone and periosteal injury in racehorses. Treatments should be done with high-energy radiation, such as that produced by linear accelerators, or telecobalt units. Doses of 8 to 10 Gy are given in a single treatment. The effective therapeutic window reaches from a still undefined pre-operative time to about 2 days after surgery or traumatic soft tissue injury. In an ongoing, prospective study conducted at the University of California, Davis, Veterinary Medical Teaching Hospital, prophylactic irradiation was given to horses at risk of developing abnormal mineralization after surgery or horses with a previous history of peri-articular calcification and exostosis. A single dose of 10 Gy (1000 rad) was given using a linear accelerator (Clinac 4, Varian, Lake Oswego, OR) immediately after surgical curettage of the excessive bony proliferation. While they were under general anesthesia, horses were moved from the operating room to the radiation therapy suite. Close proximity of the two rooms at the hospital minimizes the potential for surgical and anesthetic complications during transportation. Interim results in 15 Thoroughbreds with a minimum of 12 months follow-up after treatment indicate that irradiation is effective in long-term prevention of abnormal mineralization, even after unsuccessful previous treatments including surgery, medical treatment, laser therapy, acoustic shock wave therapy, and ultrasound hyperthermia. Horses entered in this study had exostosis after trauma or fracture of splint bones, osteoarthritis of the proximal interphalangeal joint, or osteophytes associated with mild osteoarthritis.

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CHAPTER • 92

Rest and Rehabilitation

Barrie Grant

The number of publications about the effects of exercise on bone, cartilage, and tendons has increased substantially in the past decade. The results of these investigations have allowed not only the research community, but also practicing veterinarians, to gain more insight into the effects of mild, moderate, and strenuous exercise on the skeletal system, especially regarding the pathogenesis of fractures and osteoarthritis. Unfortunately, results of controlled studies of the effects of deconditioning on bone, cartilage, and tendons have not been disseminated widely. Presently each clinician recommends a rest and rehabilitation program based many times on intuition, anecdotal experience, and tradition. These recommendations have withstood the test of time, however, and can serve as a guide.

In the last decade the number of alternative therapies and therapists has also increased. Although some benefits to this approach are possible, many of the modalities have not been tested in a standard scientific method, and popularity often is based on the principle that alternative approaches are less expensive, with faster results.

PRINCIPLES OF REST AND REHABILITATION PROGRAMS

The following are some basic principles that should be considered when formulating a specific plan for a specific injury.

1. Is the diagnosis complete? An incomplete diagnosis and thus treatment lead to poor results. Arthroscopic removal of a carpal fracture in the apparently lame limb will have poor results if an osteochondral fracture is in the contralateral fetlock joint. Hindlimb suspensory desmitis is unlikely to resolve satisfactorily if mild ataxia is concurrent. An expedient, complete, and accurate diagnosis affords the best chance for long-term success.
2. Horses can tolerate extensive periods of stall confinement without becoming deranged psychopaths. They do need to be in clean airy stalls, so they can see other horses, and energy intake should be reduced. Clinicians should avoid walking horses every 4 or 5 days to see how they are doing, because horses tend to find any excuse suddenly to jump or rear and possibly cause re-injury.
3. The heart and muscle do not undergo any significant deconditioning for at least 4 weeks after a standard anaerobic training program. Therefore many horses with minor injuries, or even a suspicion of a minor ligament or tendon problem, possibly can heal with 4 weeks of rest, with little loss in performance. The cardiac parameters actually improve 5 days after the last extensive training effort. Little will be lost and much could be gained if experienced jumping and dressage horses with lameness problems could be walked under tack for a week before major events.
4. An earlier return to training, in the form of walking under saddle, swimming, or controlled exercise on a treadmill, can reduce the potential chance for further injury than if a previously stall-confined horse is turned out in a paddock and allowed to run, buck,

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4. An earlier return to training, in the form of walking under saddle, swimming, or controlled exercise on a treadmill, can reduce the potential chance for further injury than if a previously stall-confined horse is turned out in a paddock and allowed to run, buck,

slide, and turn quickly. If paddock turnout is used, then administering sedation and gradually increasing the size of the paddock are recommended. Leaving a horse in a paddock continuously also is best once the horse has been introduced to the freedom. If a horse is stabled each evening, then it tends to buck and play again each morning when released.

REST

The basic principle of rest is to reduce the force and strain on injured tissue and allow the normal reparative processes to proceed without further insult. Muscle tissue can heal in a much shorter period than tendons or ligaments, but the repair of cartilage is accomplished by the metaplasia of replacement tissue, which takes 4 to 6 months. The quality and quantity of rest depends on the severity of the condition, the disposition of the horse, the owner, and the trainer, and their expectations.

Complete Immobilization

Although complete immobilization can be accomplished with internal fixation, external coaptation with using a cast also can reduce the motion of fracture fragments and excessive strain placed on damaged ligaments or tendons. The long-term use of cast immobilization can result in osteoarthrosis (fracture disease) and osteopenia. Normal joints can tolerate at least 4 weeks of cast immobilization without significant irreversible change to the articular cartilage.[1] Well-designed splints and braces offer an alternative for conditions that do not require complete rest or as intermediary support in the transition from a cast to no external support.

Stall Rest

Stall rest is the most common form of rest recommended and is highly effective as long as weekly episodes of outside activity do not interrupt it. The effects of controlled exercise on articular cartilage remain controversial. French et al.[2] concluded that early post-operative exercise is not detrimental to repair of experimentally created osteochondral defects of the carpus. Other investigators suggested deleterious effects of early exercise on the healing of carpal osteochondral defects.[3] However, exercise may be beneficial in maintaining normal concentrations of glycosaminoglycan in insulted articular cartilage.[4] The administration of polysulfated glycosaminoglycans may have a protective effect against the deleterious results of early post-operative exercise on experimentally induced osteochondral defects.[3] Only a cost-benefit investigation with a significant number of horses will show if the expense of the medication is justified, with an earlier return to competition compared with horses restricted to stall rest.

Owners are always concerned about the effect that enforced stall rest can have on the mental status of horses, but provided energy intake is restricted and horses have visual contact with other horses, most adapt satisfactorily. Many horses lose muscle mass and tone during inactivity, which are rapidly restored, and horses that become overweight are less likely to return to athletic competition than leaner cohorts. It is important to stress good husbandry practices, because soiled wet bedding or infrequent hoof care can lead to chronic hoof problems. I have seen many horses with joint disease develop subsolar hematomas on the opposite limb predisposing to or because of the joint disease. These hematomas can become subsolar abscesses if the husbandry is inadequate.

Experience has shown that 60 days is the minimum amount of time necessary for the repair of soft tissues following orthopedic surgery,[2] but convincing eager and aggressive owners and trainers that this time is a positive economic decision is often difficult.

Reduction of Excessive Movement

Recent improvement in slings has helped horses with severe orthopedic and neurological problems to reduce the risk of further injury from difficulties in lying down and getting up and the risk of contralateral laminitis. We prefer the Liftex sling (Liftex, Inc., Warminster, PA) to the more complicated Anderson sling (CDA Products, Potter Valley, CA) for everyday use, although we recognize that the Anderson sling has some features that are helpful in lifting severely injured or neurologically compromised patients. Not all horses accept a sling, especially those under a year of age, and further injury can possibly occur if a horse becomes violent. Giving the horse a small amount of acepromazine reduces anxiety before a sling is applied. Having a short introductory period for the first session and rewarding acceptable behavior with food is helpful. Slings are not meant to provide vertical stability to patients who cannot stand by themselves. They are best used to provide a method for injured horses to reduce weight bearing for short periods. Experienced horses take full weight off the legs and sit as a person might in a hammock for short periods, two to three times an hour.

Use of cross-ties or overhead chains can also prevent excessive movement and force on injured areas from horses lying down and struggling to rise. Young horses do not accept this form of restraint well, which may increase the chance of injury because they fight the system. It is important to allow horses three to four sessions a day with the head down to prevent aspiration pneumonia that can occur with improper drainage of pulmonary secretions.

Paddock Rest

After stall confinement, tradition has stressed a period of paddock rest before a horse is returned to active training. When and how much paddock rest is based on each individual horse. The type of injury, response to treatment, personality of the horse and owners, and the season of the year are major considerations. In my experience horses can over-exercise and re-injure healing tissue or incur new injuries when turned out. The use of small paddocks (6 × 6 m) to inhibit running is a suitable compromise.

The initial introduction of the horse to the paddock is the time when most mishaps can occur. Sedation, protective boots (tendons and bell or overreach boots), and avoiding muddy, snowy, frozen conditions and unfamiliar horses greatly reduce injuries. Ideally the horse should be allowed to remain in the paddock at all times, instead of being turned out each morning after a night of stall confinement. It is necessary to continue to monitor the general health care of the patient, including anthelmintic prophylaxis and immunization against respiratory pathogens that are spread easily with horses communicating over adjacent fences. Foot care is still a concern. Many horses may require shoeing to prevent abnormal hoof wear, which could result in an uneven gait when they return to active training.

Ridden Walking

After stall confinement and hand walking, I prefer to ride the horse at walk, beginning with 10 minutes a day and progressing to 30 minutes a day after 30 days, rather than use paddock exercise. This increase of activity is felt to stimulate the maturation of repair tissue in cartilage and tendons.

EXERCISE

Passive Exercise

Passive exercise can begin immediately after joint surgery to reduce the amount of post-operative immobility. Theoretically, passive exercise improves distribution of nutrients to chondrocytes and reduces the number of synovial adhesions to any osteochondral defects on the edge of the joint. The type of

reparative tissue that develops under synovial adhesions is fibrous and not the hyaline-like material that fills a full-thickness defect when no synovial tissue has adhered to the defect.[5] We routinely begin passive joint movement on the first post-operative day. The joint is flexed just to the position that produces discomfort 15 to 20 times twice a day, for 30 days. Passive exercise is not indicated in joints with any detectable instability or in which a problem with primary healing of the joint capsule exists.

Hand Walking

Hand walking is the mildest form of active exercise and often is recommended along with stall rest. Although recommending hand walking early in the post-operative period is easy to appease owners, trainers, and the caretakers, it is also essential to realize that early disruption of a primary clot in an osteochondral defect can result in poor healing of the defect.[5] The chances of excessive and unpredictable behavior that can result in removal of the clot are increased with removing active horses from their stalls for short periods. The benefits of complete stall rest for the first 30 days far outweigh any benefit that 5 to 10 minutes of hand walking may produce. Hand walking for 5 to 10 minutes a day beginning at day 30, and gradually increasing to 30 minutes at post-operative day 60, is common practice. Hand walking seems to be the most objectionable form of exercise to owners and caretakers, because horses act up or the potential for them to do so is anticipated and dreaded. The new form of caged horse walkers appear to be much safer and, although horses are constantly turning, this appears to be a suitable form of exercise to replace hand walking. Alternatively, ridden walking exercise can be used.

Aquatreds and Swimming

Using swimming pools and submerged treadmills (aquatreds) is still popular and at least in specific locations has withstood the test of time. Minimal controlled data are available to document the benefits of these modalities for treating athletic injuries. That the cardiopulmonary system responds significantly to swimming has been documented, but only anecdotal information exists for aquatreds. Both modalities assist in increasing joint mobility and producing some physical activity in a controlled environment. The force to the axial skeleton is reduced and this benefits horses with a non-displaced fracture or a joint with advanced osteoarthritis. However, the reduction of the weight-bearing force in rehabilitation may not be desirable, because osteoporosis develops when training is curtailed for more than 30 days.[6] Returning the skeletal system to its previous competitive state requires a longer period of time than for muscles and the cardiopulmonary system. Reconditioning the muscles by swimming or aquatreds, before reconditioning the skeletal system, increases the possibility of having horses that are too eager and able to produce more force than the bones, joints, tendons, and ligaments can tolerate. The recognition that stress fractures of the humerus are more common in horses returned to training after only 30 to 60 days of rest bears testimony to the inability of the skeletal system to adapt to the stress of training as quickly as the muscle does.[7]

High-Speed Treadmills

The increase in the number of privately owned treadmills could increase the quality of healing of athletic injury. High-speed treadmills have a number of benefits for reconditioning equine athletes. The speed, distance, slope, heart rate, and climatic conditions can be controlled, monitored, and indeed with some systems, even programmed. Work effort can be increased with a weighted saddle and raising the slope of the treadmill, without having to increase the speed. The force on the tendons can be increased slowly by increasing the slope of the treadmill slowly.

Using treadmills for exercising horses is now accepted widely because the injury rate has been low if good judgment is used, especially when introducing a new horse. It is important to realize that training on a treadmill is not the same as racing or training on a racing surface. Horses require twice as much work on the treadmill at maximum oxygen consumption (V_{O_2}) to produce the same muscle changes as galloping on a training track with a rider.[8] Horses also require speeds of at least 14 m/sec to produce the same changes on implanted strain gauges in the third metacarpal bones as breezing on a racetrack.[9] Some treadmills cannot achieve this speed and lay operators often are reluctant to exercise horses at 14 m/sec, because they feel the horses may become injured.

We have found little problem with long-term training on a treadmill. Horses usually trot 1000 m at 4 m/sec and then gallop for 2000 m at 10 m/sec. We have not recognized any foot problems that may result from excessive time on the treadmill,[10] although the training plates are hot to the touch after a training session. A constant fine mist applied to the treadmill belt is supposed to reduce the heat. Recently it has been shown that prolonged treadmill training can result in carpal articular cartilage changes.[11]

Active Training

The importance of periodic monitoring for any subtle clinical signs as a horse progresses through the final stages of training before competition cannot be overstressed. The trainers with the most success in returning horses with athletic injuries to a successful career are those who are patient and careful observers. Horses often show signs of increased inflammation and gait abnormalities as the training intensity increases from tack walking and jogging to galloping. Reducing the amount and quality of the exercise for 30 days may be all that is necessary to reduce the inflammation, without medication.

Hill Training

It is possible to produce the same training effect by exercising horses on moderate inclines (e.g., ½ mile at 13% incline) at a 50% reduction in the speed of horses trained on a racetrack.[12] Trotting downhill is not necessary for most conditioning programs other than endurance or cross country eventing. Trotting downhill may lead to an increase in the incidence of proximal suspensory problems and also increases the forces on the stifle joint. I used interval training in fifteen 2-year-old Thoroughbreds being trained for racing, using a program consisting of three intervals with the first at an extended trot, the second at a canter, and the third at a gallop, and found the horses had minimal unsoundness and were competitive. Walking the horses downhill allowed time to recover and kept the horses relaxed and manageable.

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SECTION • 2
Complementary (Nontraditional) Therapy

CHAPTER • 93

Acupuncture

EQUINE ACUPUNCTURE FOR LAMENESS DIAGNOSIS AND TREATMENT

• Allen M. Schoen

Recently interest in veterinary acupuncture for lameness diagnosis and treatment has increased greatly in the public and veterinary medical communities. With this increased awareness has been an increase in veterinary acupuncture research and thus a better understanding of the physiological basis of acupuncture and its clinical applications. Acupuncture may be used as an adjunct diagnostic and therapeutic tool to our traditional lameness examination and to the treatment of lameness, but it is not meant to be a substitute.

With the increased interest in equine acupuncture, there has also been additional controversy and use and misuse of this modality. It is important to understand the scientific basis, traditional Chinese medical theories, and clinical indications and limitations for acupuncture to become part of an integrated approach to equine lameness in a professional manner. Currently, equine acupuncture is practiced by veterinarians trained in the Western medical perspective of scientific acupuncture and the traditional Chinese and Japanese medical theories. The approach may vary slightly based on the practitioner's perspective.

The history of equine acupuncture dates back to 2000-3000 BCE during the Shang and Chow dynasties in China. Around 650 BCE Bai-le wrote, *Bai-le's Canon of Veterinary Medicine*, one of the first veterinary textbooks, which described acupuncture and moxibustion in equine medicine. The first atlas of equine acupuncture points and channels, *Ma Jing Kongxiue Tu*, was written during the Sui period, between 581-618 CE.[1] Equine back pain was first addressed in the ancient veterinary textbook *Yuan-Heng Liao Ma Ji* (*Yuan-Heng's Therapeutic Treatise of Horses*).[2] Periodically, anecdotal reports of using equine acupuncture would come to the Western world. The first substantial introduction occurred in the 1970s, when the International Veterinary Acupuncture Society was established and developed training programs in veterinary acupuncture in the United States. In 1996 the American Veterinary Medical Association stated, "Veterinary acupuncture and acutherapy are considered valid modalities, but the potential for abuse exists. These techniques should be regarded as surgical and/or medical procedures under state practice acts. It is recommended that extensive continuing education programs be undertaken before a veterinarian is considered competent to practice acupuncture."[3] Postgraduate education in veterinary acupuncture throughout the world—including Australia, Europe, Scandinavia, North and South America, and other areas—is offered by the International Veterinary Acupuncture Society, based in Fort Collins, Colorado.[4] The Chi Institute of Traditional Chinese Medicine in Reddick, Florida[5]; Colorado State University College of Veterinary Medicine continuing education program in Fort Collins, Colorado[6]; Tufts University School of Veterinary Medicine in North Grafton, Massachussetts[7]; and the Veterinary Institute for Therapeutic Alternatives in Sherman, Connecticut,[8] also offer postgraduate programs in the United States.

SCIENTIFIC BASIS

Acupuncture may be defined in a Western medical perspective as the stimulation of specific predetermined points on the body to achieve a therapeutic or homeostatic effect. Recent research has provided evidence for the anatomical classification of acupoints. Acupuncture points are areas on the skin of decreased electrical resistance or increased electrical conductivity. Acupuncture points correspond to four known neural structures. Type I acupoints, which make up 67% of all acupoints, are considered motor points. The motor point is the point in a muscle which, when electrical stimulation is applied, will produce a maximal contraction with minimal intensity of stimulation. Motor points are located near the point where the nerve enters the muscle. Type II acupoints are located on the superficial nerves in the sagittal plane on the midline dorsally and ventrally. Type III acupoints are located at high-density foci of superficial nerves and nerve plexi. For instance, acupoint GB-34 is located at the point where the common fibular (peroneal) nerve divides into the deep and superficial branches. Type IV acupoints are located at the muscle-tendon junctions, where the Golgi tendon organ is located.[9] Recently, histological studies have revealed that small microtubules, consisting of free nerve endings, arterioles, and venules, penetrate through the fascia at acupuncture points (Fig. 93-1).[10]

Acupuncture has many varied physiological effects on all systems throughout the body. No one mechanism can explain all the physiological effects observed. The traditional Chinese medical theories have explained these effects for four thousand years, based on empirical observations and descriptions of naturally occurring phenomena. The Western medical theories include the gate and multiple gate theories, autonomic theories, humeral mechanisms, and bioelectric theories.[10] Detailed discussions of the neurophysiological basis of acupuncture are reviewed in a number of texts.[9-12] Essentially, acupuncture stimulates various sensory receptors (pain, thermal, pressure, and touch), which stimulate sensory afferent nerves, which transmit the signal through the central nervous system to the hypothalamic-pituitary system (Fig. 93-2). The acupoints correlate with cutaneous areas containing higher concentrations of free sensory nerve endings, mast cells, lymphatics, capillaries, and venules. Various neurotransmitters and neurohormones are then released and have subsequent effects throughout the body. Recent research in rabbits using electroacupuncture-induced neural activation, detected by the use of manganese-enhanced functional magnetic resonance imaging, demonstrated a corresponding cerebral link between peripheral acupoints and central neural pathways. Research like this is aiding in further

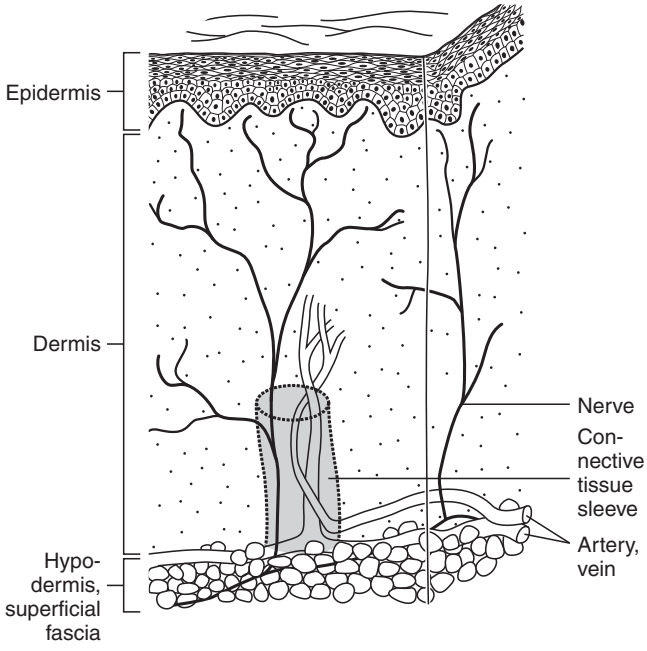

Fig. 93-1 Schematic drawing of the skin, showing a neurovascular bundle wrapped by a sleeve of loose connective tissue deep to an acupoint. (From Schoen AM, editor: *Veterinary acupuncture: ancient art to modern medicine,* ed 2, St Louis, 2001, Mosby.)

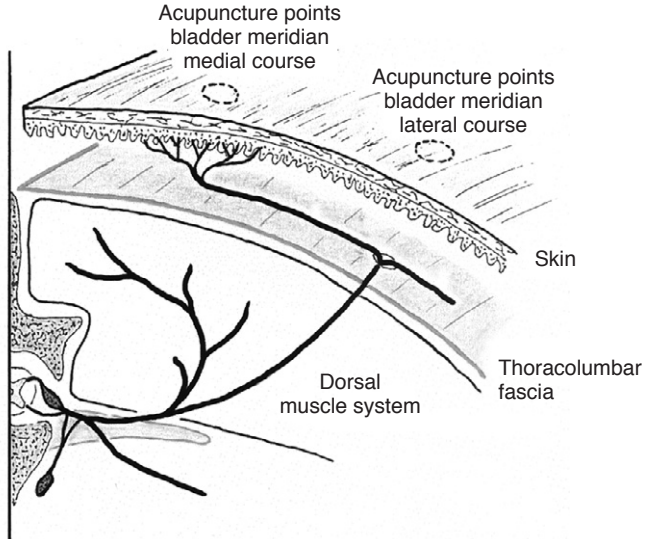

Fig. 93-2 Cutaneous nerve entering the dermis at an acupuncture point along the bladder meridian. (From Schoen AM, editor: *Veterinary acupuncture: ancient art to modern medicine,* ed 2, St Louis, 2001, Mosby.)

understanding the effects of local peripheral stimulation on specific areas in the brain.[13] Electroacupuncture has also been found to stimulate endogenous opioids, vasoactive peptides, adrenocorticotrophic hormone, cortisol, and catecholamine in cerebrospinal fluid and peripheral plasma in ponies.[14] Bossut et al.[15] documented changes in plasma cortisol and β-endorphin in horses subjected to electroacupuncture for cutaneous analgesia. Changes in serum proteins and blood gases also have been demonstrated in donkeys following acupuncture.[16,17] Xie et al.[18] documented that electroacupuncture could relieve experimental pain in the horse via the release of β-endorphin. They found that acupuncture stimulation using local acupuncture points with high frequency (80 to 120 Hz) is more effective than using distal points with low frequency (20 Hz). They found that acupoints close to the painful areas require high-frequency electroacupuncture stimulation, whereas the acupoints far from the painful areas may be stimulated with low-frequency electroacupuncture. Electroacupuncture generally is considered to have stronger effects than other types of acupuncture methods.[18] The degree of stimulation appears to depend on the location of the specific acupoints.[19,20]

Through understanding the neurophysiology and neuroanatomy of acupuncture, one can appreciate that acupuncture may stimulate nerves, increase local microcirculation to joints and muscles, relieve muscle spasms, and cause a release of various neurotransmitters. Clinically, one may see the benefits of these effects in treating equine lameness caused by soft tissue injury, muscle spasms, nerve trauma, and other causes.

TRADITIONAL CHINESE MEDICAL THEORIES

Acupuncture has been used in China to treat equine lameness for a few thousand years. Chinese acupuncture was based on the empirical use of acupoints for certain conditions. Historically, acupuncture meridians were not acknowledged in animals. Specific empirical points based on experience were identified and named for location or function. Equine acupuncture meridians, or pathways, have been transposed onto horses from human acupuncture maps only in the past thirty years.

Traditional Chinese medicine acupuncture diagnosis and treatment are based on the pattern differentiations that are developed from two main theories of traditional Chinese medicine: the Five Element theory and the Eight Principles, which require a great deal of study.[21] The traditional Chinese medicine diagnosis is defined as a specific pattern based on imbalances in the five elements (also called five phases), imbalances and disorders in specific meridians or organs, or specific patterns of disharmony. The diagnosis is based on a physical examination of the horse, including evaluation of the tongue and pulse. Tongue inspection includes evaluation of the color of the tongue, the moisture, and the color and thickness of any tongue coating. Palpation of the jugular pulse would be described as weak, strong, wiry, and so on. Tongue and pulse examination and an overall examination would lead to a traditional Chinese medicine diagnosis and treatment of appropriate acupuncture points. Xie[22] describes a traditional Chinese medicine five-step method for diagnosis and treatment. This includes the following:

1. Collection of empirical data from the history, tongue, and pulse diagnosis
2. A Western medical diagnosis
3. A traditional Chinese medicine pattern differentiation
4. A treatment strategy based on traditional Chinese medicine
5. A selection of appropriate acupuncture points, Chinese herbs, or both

In contrast, the Japanese technique, frequently termed *meridian therapy,* is based more on palpation of the acupuncture points and the reaction. The anatomical location of equine acupoints is based on the traditional Chinese acupuncture atlases or a transpositional atlas based on transcribing human acupuncture points onto the equine anatomy. The location of the points is similar in each method, though anatomical variation exists, as discussed by Fleming[23] (Fig. 93-3).

TECHNIQUES AND INSTRUMENTATION

Numerous techniques exist to stimulate acupuncture points. The following modes of stimulation are commonly used in equine acupuncture: dry needle stimulation, electroacupuncture, aquapuncture, moxibustion, laser stimulation, gold

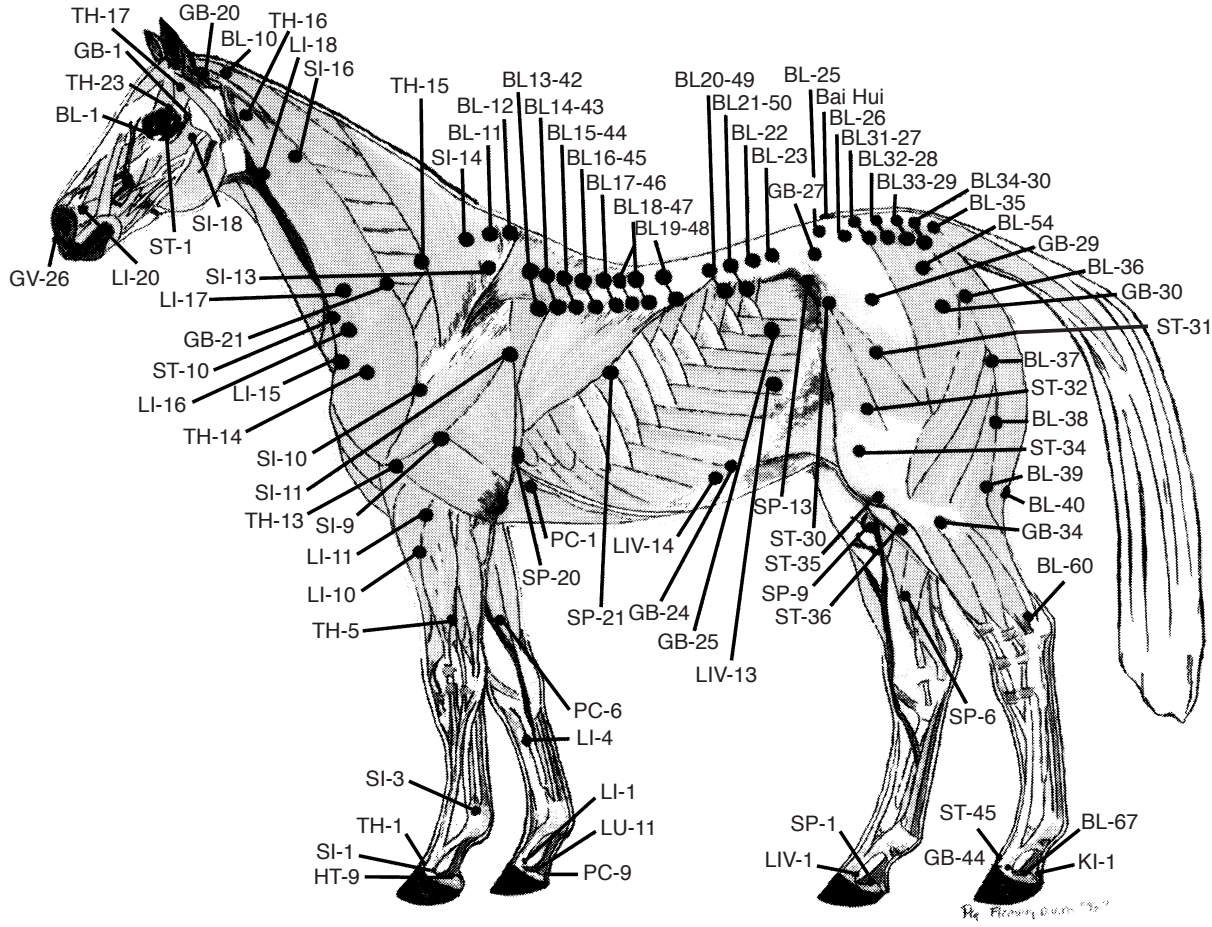

Fig. 93-3 Acupoints of the horse: lateral view in relation to muscles. (Courtesy Peg Fleming. From Schoen AM, editor: *Veterinary acupuncture: ancient art to modern medicine,* ed 2, St Louis, 2001, Mosby.)

implants, and acupressure. Each method has its indications and limitations. Dry needle and aquapuncture are used most routinely in the West.

Aquapuncture is the injection of various solutions into the acupuncture points. The hypodermic needle is stimulating the acupuncture point as a traditional acupuncture needle would. In addition, the solution is stimulating the pressure receptors for a period of time, and the solution is having its specific effect as well. Solutions injected include saline, vitamins such as B_{12}, non-steroidal analgesics, local anesthetics such as lidocaine, and occasionally homeopathic and herbal solutions, depending on the veterinarian's preference. Electroacupuncture, using a portable electroacupuncture unit, normally is used for nerve stimulation to treat severe chronic cervical, thoracolumbar, or lumbosacral problems or an affected joint, tendon, or ligament. These electroacupuncture units have electrodes attached to the acupuncture needles. Positive results with electroacupuncture have been documented for treatment of lameness in horses. Electroacupuncture decreased the lameness score in horses ($P < 0.001$).[24] Though poorly documented, electroacupuncture without needles is sometimes applied as well. Moxibustion is a form of thermostimulation using a Chinese herb, *Artemesia vulgaris.* Low-level or cold laser therapy is also used to stimulate acupuncture points. Gold implants have been used to treat navicular disease and chronic back problems and provide long-term stimulation of acupoints. This approach is an adaptation of the Chinese technique of sutures embedded into acupoints. Acupressure often is taught to the client to administer in between acupuncture treatments to potentiate the effect. Detailed descriptions of these techniques are described by Altman.[25]

From a Western medical perspective, acupuncture point selection is based on locating points on the body where stimulation produces a beneficial change in the central nervous system by modulating ongoing physiological activity, releasing local muscular spasms, and increasing local microcirculation. From a traditional Chinese medicine perspective, acupoints are selected based on a pattern differentiation developed from the Five Element theory or Eight Principles. The choice of acupoints based on the Eight Principles depends more on tongue and pulse diagnosis and a comprehensive history. The Japanese approach to point selection follows a diagnostic acupuncture point examination, palpating diagnostic acupoints, including association (*Shu*) points on the dorsal thoracolumbar region, alarm (*Mu*) points on the ventral abdomen, and other diagnostic points. Many clinicians choose their points based on a combination of Chinese, Japanese, and Western diagnostic techniques, including tongue and pulse diagnosis, diagnostic acupuncture point examination, sometimes including *Ting* points (points around the coronary band), and a comprehensive Western medical examination and history.[26,27] The number of acupuncture points treated may vary from one to more than 20, depending on the condition treated and the approach and experience of the practitioner. The depth of needle insertion varies from 1 mm to 12 cm, depending on the location of the point, such as the coronary band versus dorsal lumbar musculature. The number of treatments required depends on the condition treated and the chronicity of the problem, usually

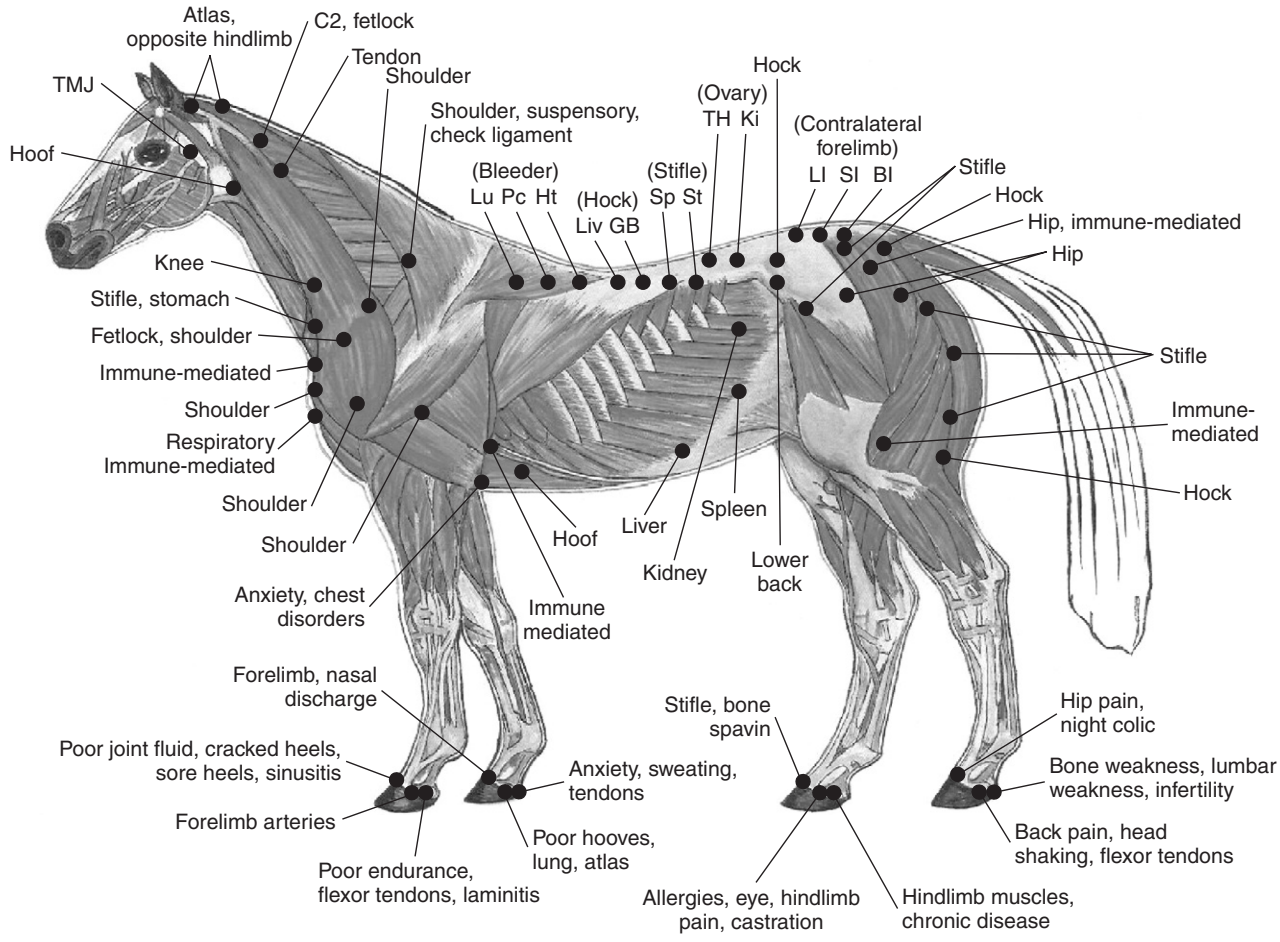

Fig. 93-4 Diagnostic acupuncture palpation points. (Courtesy Peg Fleming. From Schoen AM, editor: *Veterinary acupuncture: ancient art to modern medicine,* ed 2, St Louis, 2001, Mosby.)

ranging from one to eight treatments. The length of treatment varies from 5 to 30 minutes.

Manual therapies, such as chiropractic or osteopathy, are often applied along with acupuncture. They appear to work synergistically, acupuncture relieving muscle spasms and increasing circulation, chiropractic helping to correct skeletal fixations (see Chapter 94), and osteopathy affecting the fascia and skeletal structures (see Chapter 98). The combination of these therapies often leads to a more rapid response and greater efficacy, with fewer treatments required.

CLINICAL APPLICATIONS IN LAMENESS EXAMINATION AND TREATMENT

Acupuncture Diagnostic Examination

Acupuncture and manual therapies may be used diagnostically to aid in evaluating various lameness and performance problems. Acupuncture is an excellent diagnostic aid as an adjunct to our conventional lameness examination. Many of the diagnostic acupoints are located lateral to the dorsal midline, between the longissimus and iliocostalis muscles, along the acupuncture meridian known as the bladder meridian.[26] In addition, some diagnostic points are actually trigger points, knots or tight bands in a muscle. For instance, a triceps trigger point is often sensitive to palpation when a lower forelimb lameness is present and correlates with the acupoint Small Intestine 9. A triceps trigger point may not indicate exactly where the lameness is or what the cause is, but it does indicate that something is reactive in that region (Fig. 93-4).

Diagnostic acupoints also are located around the coronary band on the forelimb and hindlimb, known as *Ting* points (Fig. 93-5).[15] Each diagnostic acupuncture point may have four or five meanings, depending on which other points show up as reactive on the examination. For instance, one point, Large Intestine 16 (located in a depression on the cranial border of the scapula, at the intersection of the cranial margin of the scapular muscle and the caudal margin of the brachiocephalicus muscle, cranioventral to the first thoracic vertebrae) may be reactive in horses with forelimb lameness, cervical conditions, or a contralateral hindlimb lameness.[26] Sensitivity on acupoints along the bladder meridian, lateral to the dorsal midline, along the back may indicate that a hindlimb lameness is related to the stifle or hock, or that a primary back problem is related to the saddle fit, rider, or a conformational problem. Sensitivity at acupoints suggesting a problem in the hock region also may be reactive with nearby injuries, such as a proximal suspensory problem. In addition, reactivity may indicate internal organ problems via a somatovisceral reflex. The combination of reactive points often aids the clinician in localizing the cause of the problem and determining a diagnosis. Often a horse may have a localized lower limb lameness along with a back problem. Sometimes acupoint diagnosis assists the veterinarian in figuring out which may have come first, the lower limb lameness or the back problem, based on the degree of sensitivity of the various points. Acupuncture diagnosis can be an excellent adjunct to conventional lameness examination, flexion tests, diagnostic

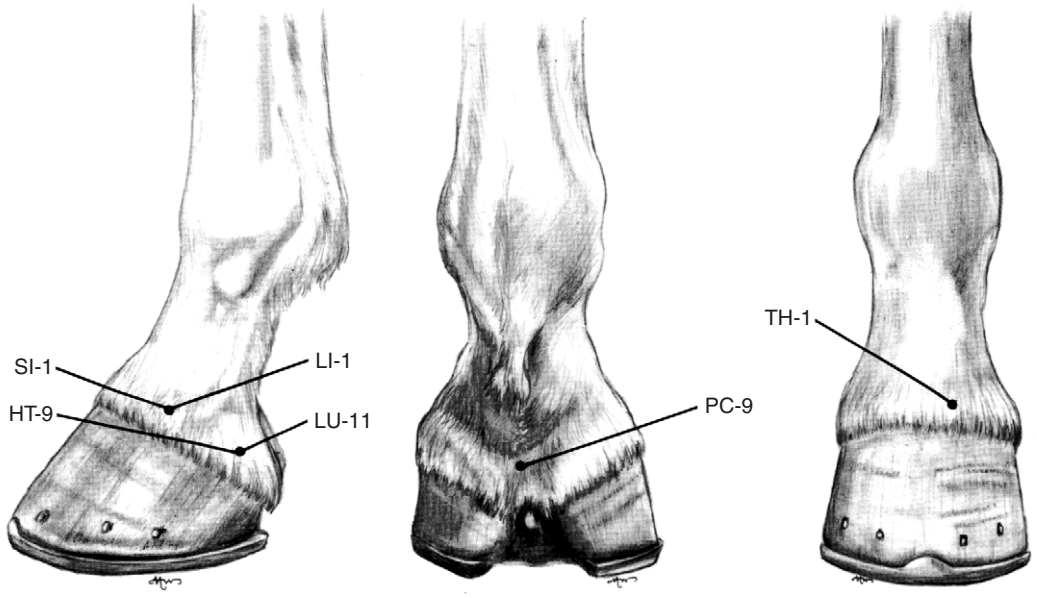

Fig. 93-5 *Ting* points of the equine front foot. In a lateral position the points are SI-1 and HT-9; in a medial position the points are LI-1 and LU-11. (From Schoen AM, editor: *Veterinary acupuncture: ancient art to modern medicine,* ed 2, St Louis, 2001, Mosby.)

analgesia, radiography, ultrasonography, and other diagnostic approaches. Not uncommonly we use all of our diagnostic capabilities, including nuclear scintigraphy and magnetic resonance imaging, and still do not arrive at a diagnosis. Acupuncture is often an excellent adjunct technique that may assist in elucidating the problem. In people, musculoskeletal pain often is accompanied by muscle shortening in peripheral and paraspinal muscles from spasms and contractures, and secondary trigger points and autonomic manifestations of neuropathies are also present in chronic musculoskeletal pain.[28] Patterns of trigger points distant to the primary problem, compensating for the primary musculoskeletal problem, also have been found.[29] These patterns are also evident in equine acupuncture lameness examinations. For instance, the horse may have a primary hock or stifle problem and then develops compensatory patterns of trigger points in the back and neck and contralateral forelimb. These patterns show up as standard patterns of trigger points that may assist the veterinarian in the primary diagnosis.

An integrative approach to diagnosis is incorporated along with the acupuncture diagnostic palpation examination and includes evaluation of conformation, saddle fit, shoeing, rider and training programs, and the conventional lameness examination. Chiropractic evaluation, including static and motion palpation, also are included, which allows for a more comprehensive evaluation of all potential causes of lameness.

Acupuncture Therapy for Lameness

Acupuncture has been reported to be effective for treating various musculosketetal pain-producing conditions and has been found to be beneficial in treating cervical, thoracolumbar and lumbosacral hyperpathia,[30,31] and chronic back pain, chronic lameness, osteoarthritis, and colic. Hyperpathia, increased pain sensation detected as muscles spasms and increased sensitivity of acupuncture points, is often secondary to numerous causes, including work-related muscle soreness and injuries, metabolic diseases, infectious diseases, and underlying soft tissue and orthopedic problems. Wherever possible, primary underlying causes of lameness should be explored with appropriate diagnostic procedures. Conventional and complementary therapies should be considered based on what is most appropriate for the particular condition.

Back Pain

Chronic back pain can be a major cause of poor performance and may be caused by soft tissue damage or lesions to the thoracolumbar vertebrae. Numerous studies have documented the benefits of acupuncture for equine back pain.[32-42]

Xie et al.[24] documented the success of electroacupuncture for treating chronic back pain in performance horses in a controlled clinical trial. They found that three electroacupuncture sessions were needed for clinical improvement. Conventional medical approaches, including the administration of muscle relaxants and analgesics, usually offer only temporary and minimal improvement, decreasing the clinical signs but not addressing the specific problems. Acupuncture is able to address specific muscle spasms and trigger points. Recent research suggests that electroacupuncture may be stronger and more effective than dry needle techniques or aquapuncture.[24] Clinically, aquapuncture and dry needle techniques appear to be effective.

Pain Associated with Lower Limb Lameness

Acupuncture has been reported to be beneficial in horses with various lower limb lameness[43-65] and has been beneficial in treating lameness related to the shoulder, elbow, carpus, tarsus, fetlock, stifle, hip, and numerous soft tissue injuries. In one experiment evaluating acupuncture in the treatment of lower limb lameness, Xie et al.[24] found that electroacupuncture can partially relieve pain caused by the mechanical pressure induced by tightening a screw against the sole. They found that electroacupuncture significantly reduced the degree of lameness ($P < 0.001$). Electroacupuncture simultaneously increased plasma β-endorphin concentration, which suggests that endorphin release may be one of the pathways in which acupuncture relieves experimental pain. Electroacupuncture did not alter adrenocorticotrophic hormone concentrations, which indicates that the hormone may not be involved in this type of analgesia. This indicates that the mechanism of acupuncture analgesia is not merely a nerve block, such as with local anesthetic solution, but also has central acting effects. This correlates well with other clinical studies demonstrating the benefits of acupuncture in relieving equine lameness caused by joint contusion, muscular atrophy, rheumatic pain, and laminitis. Using a similar experimental design in a controlled clinical trial,

Hackett et al.[65] found that acupuncture alleviated equine pain, based on heart rate measurements as an indicator of pain response. In this study acupuncture was found to be more beneficial than non-steroidal anti-inflammatory medications.

The experience of numerous clinicians suggests that acupuncture, combining local and distant points and using various techniques, can be beneficial as a primary or secondary modality. In clinical practice, acupuncture is beneficial in resolving chronic back pain. Establishing a primary cause of back pain is critical and includes evaluating the feet, saddle fit, rider, training, and conformation. If pain primarily is caused by conformation problems, long-term resolution may include periodic electroacupuncture, traditional acupuncture, or gold bead implantation.

Acupuncture and chiropractic are used successfully in treating various equine musculoskeletal conditions as primary treatments or as adjuncts to conventional veterinary therapeutic techniques. For instance, a horse may have primary distal hock joint pain and may be treated with an intra-articular injection. However, the injection may not completely resolve hock lameness. The horse may still "not be right" or "be off." Often secondary compensation and subsequent patterns of trigger points, muscular spasms in the longissimus, and vertebral fixations in the lumbar and cervical regions remain unresolved. Acupuncture and chiropractic therapy may then be used to treat the sequelae of the primary hock problem successfully. Hence the clinician then may resolve 100% of the lameness and increase client satisfaction.

Acupoint selection for lameness may include specific local points or *ah shi* (tender points) around a specific joint or region, points related to the secondary compensation for the primary lameness, and points based on traditional Chinese medicine or Japanese meridian therapy. Acupuncture may be used as primary therapy for lower limb lameness if conventional medical approaches have not demonstrated substantial improvement, or as an adjunct to conventional therapy. Horses with limb lameness that may benefit from acupuncture include those with laminitis; navicular disease; carpal, metacarpal, tarsal, fetlock, and pastern problems; soft tissue injuries; and some idiopathic problems. Horses with acute and chronic laminitis have responded favorably to acupuncture using dry needles, hemoacupuncture, or electroacupuncture. In the treatment of laminitis, local points around the coronary band are used along with distant points based on traditional Chinese medicine therapy.

SUMMARY

Acupuncture can be beneficial in diagnosing and treating various lameness conditions, including lower limb and back problems. A thorough conventional diagnostic examination should be conducted along with an acupuncture diagnostic examination. All therapeutic options appropriate for a specific lameness condition should be considered. The advantages and disadvantages of each therapy should be discussed. Acupuncture can be considered as a primary therapy, or as an adjunct treatment, depending on the condition. No one form of medicine has all the answers. Clinicians should consider the most appropriate conventional and complementary diagnostic and therapeutic modalities for the particular horse and its owner to develop a comprehensive, integrative approach to equine lameness. Acupuncture is one of these complementary approaches that may be beneficial for the patient.

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UNDERSTANDING THE USE OF ACUPUNCTURE IN TREATING EQUINE LAMENESS AND MUSCULOSKELETAL PAIN

• William H. McCormick

Mastery of the Oriental examination is the key to treating equine musculoskeletal pain with acupuncture. The basis of the traditional Chinese medicine examination is pattern differentiation. This principle, as stated in Chinese, is "*bien jheng lun jhi*," or in translation, "treatment is based on the pattern."[1] The *si jhun*, or four examinations of looking, asking, palpating, and listening/smelling, outline the procedure for determining patterns. A patient's pattern is defined by the sum total of the signs, symptoms, tongue and pulse, as determined by the *si jhun*.[2]

How does the Oriental pattern-based therapy differ from the Western disease-based approach? The Western clinician arrives at a diagnosis by using the available technologies, such as diagnostic analgesia, radiography, and nuclear scintigraphy. The strict traditional Chinese medicine practitioner must rely on the traditional patterns demonstrated by the patient using the *si jhun*, without technical support. Hypothetically, a horse with navicular disease might exhibit the following patterns: *blood stasis* or fixed pain; *Qi stagnation* or moving, referred muscle pain; *liver depression*, or anger expressed in stereotypical behavior; and *heart Qi vacuity*, or anxiety. The total of the patterns describes the presentation of the disease in traditional Chinese medicine. Any disease may have a number of different patterns. Therapy is defined by the pattern, because each pattern must be addressed in the subsequent therapeutic principles used in treatment. For example, the pattern of *blood stasis* (fixed pain) requires a traditional Chinese medicine therapy that will *quicken the blood* and *resolve stasis*. The stated functions of each acupoint or herbal medicinal are used to determine which acupoints to select. The proper use of the descriptive methodology of traditional Chinese medicine forces a clinician to depict each horse in terms of patterns that then will be used to direct therapy.

One also must evaluate acupuncture by the resultant effect on the described pattern. Any other therapy, be it Western or Oriental, can also be evaluated in terms of its effect on the pattern. For example, consider a horse with navicular disease that is not yet lame, but the rider describes a change in performance caused by pain, including stiffness in the corners and a sore back. In such a horse the traditional Chinese medicine pattern may add significant information, whereas radiography, scintigraphy, and magnetic resonance imaging are technological overkill, expensive, and perhaps misleading. Treating the horse as presented with available modalities, such as shoeing, drugs, acupuncture, and herbs, and subsequently evaluating the change in pattern and performance may be more efficient. Because patterns change over time, frequent follow-up examinations and good record keeping are required. Sophisticated imaging techniques can and should be used to establish an anatomical diagnosis. The therapeutic advantage of the integrated Western and Oriental approach is most applicable to a horse with subclinical lameness as opposed to overt lameness.

Channel diagnosis can add information about any horse's response to musculoskeletal pathological conditions. This technique involves palpation of acupoints to determine the status of *Qi* flow. The information attained can be useful in a Western context. Acupoint examination should include the following grading system for severity of reaction and a recording of those reactions over time. Grade I is normal, supple and non-painful response to deep palpation. Grade II is a slight muscle twinge that is fatigable. Grade III is consistent pain or avoidance response to deep palpation. Palpation of an acupoint with grade IV sensitivity elicits a sharp avoidance response, accompanied by a kick or a bite (Figs. 93-6 to 93-8). Grade III and IV sensitivity are of sufficient severity to warrant therapy. Each of the following acupoints corresponds to a traditional Chinese

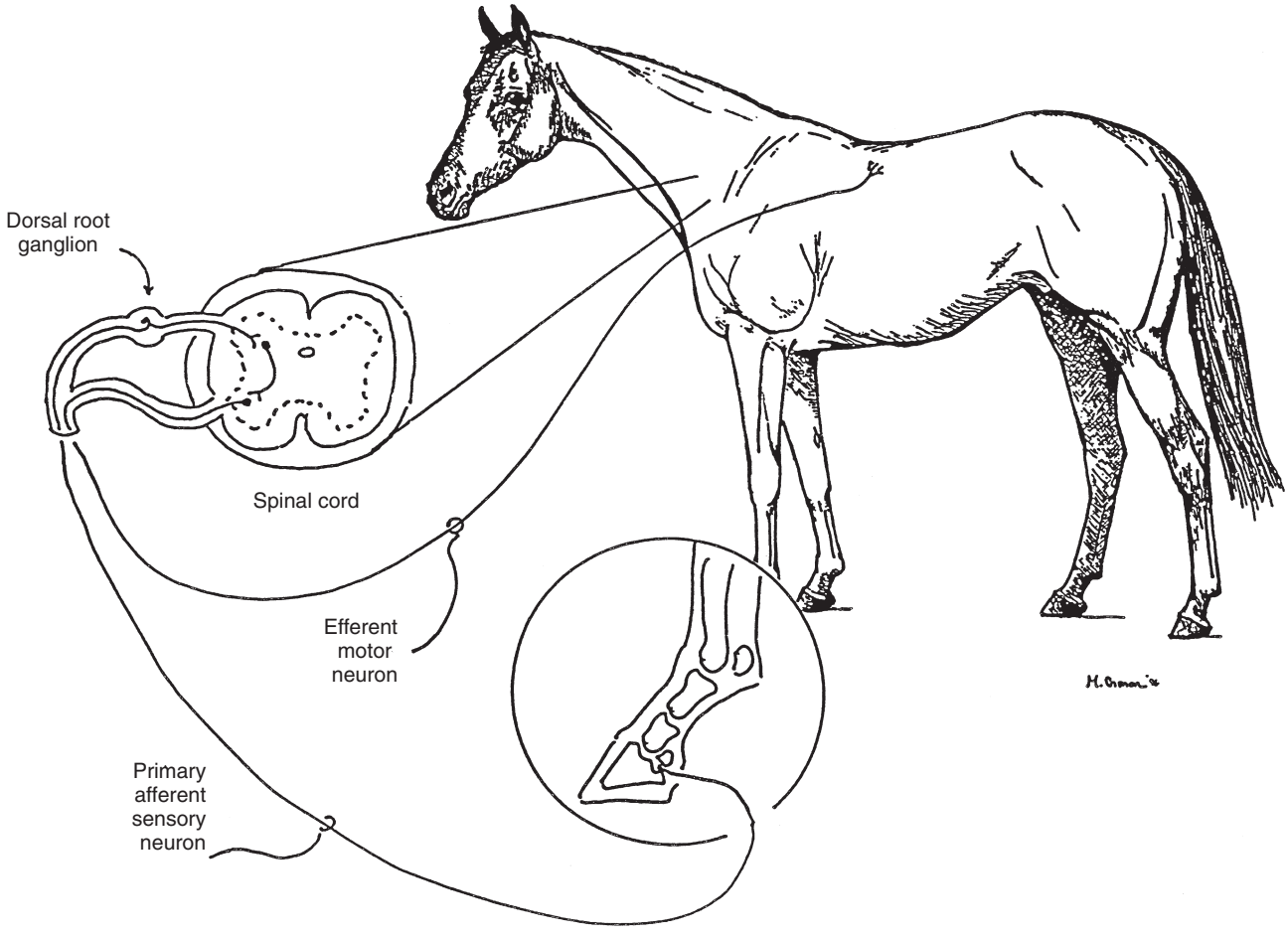

Fig. 93-6 The mediation of channel pain through the spinal reflex. (Courtesy William E. Jones.)

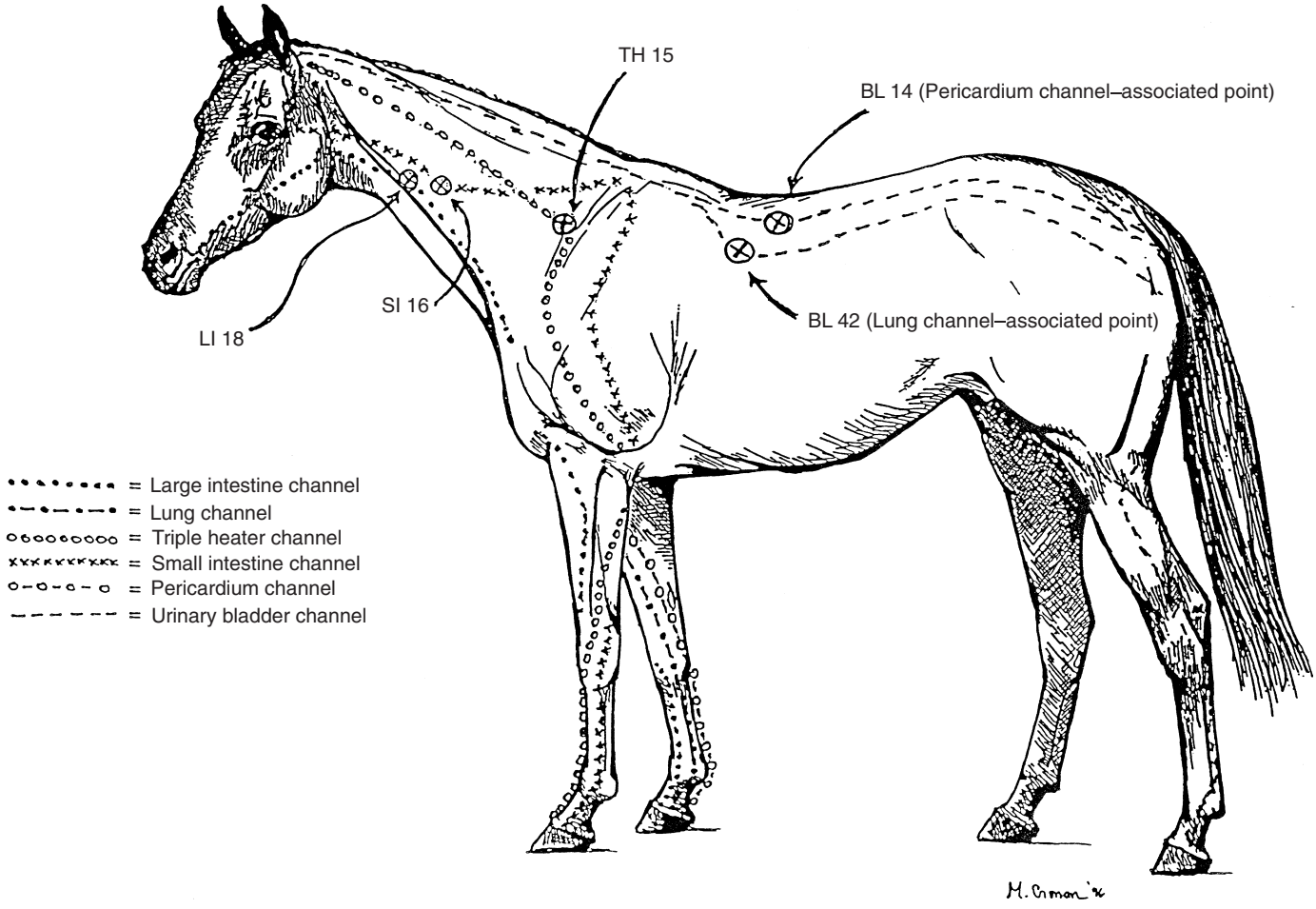

Fig. 93-7 The principal acupoints used in the diagnosis of channel pain referable to the equine limb. (Courtesy William E. Jones.)

medicine channel, which in turn is named after an internal organ. The 12 channels govern the flow of *Qi*, or life forces, for the whole body.

1. Large Intestine (LI) 18: An acupoint at the level of the caudal aspect of Viborg's triangle, on the ventral aspect of the sternocleidomastoideus muscle, at the junction of the third and the fourth cervical vertebrae. The distal course of this channel passes over the *dorsomedial aspect of the distal extremity of the forelimb.*[3,4]
2. Small Intestine (SI) 16: An acupoint located caudal to the angle of the mandible, in a depression on the dorsal border of the sternocleidomastoid muscle and dorsal to the transverse processes of the third and fourth cervical vertebrae, or 8 to 10 cm caudal to Large Intestine 18. *Distally the small intestine channel courses over the dorsolateral aspect of the forelimb.*[3,4]
3. Triple Heater (TH) 15: An acupoint located on the cranial border of the scapula, in a depression about one third of the distance from the cranial angle of the scapula to the shoulder joint. Triple Heater 15 is defined by the ventral borders of the cervical serratus and the cervical trapezius muscles. *The course of this channel crosses the dorsal midline of the distal extremity of the forelimb.*[3,5]
4. Urinary Bladder (UB) 42: An acupoint located in a depression over the seventh intercostal space, just caudal to the scapular cartilage, in a muscular groove between the longissimus thoracis and the iliocostalis thoracis muscles, about 20 cm from the dorsal midline. Urinary bladder 42 is the *shu* or associated point for the lung channel, which runs over the *medial aspect of the distal forelimb.*[3,5]
5. Urinary Bladder (UB) 14: An acupoint about 12 cm lateral to the midline and parallel to the caudal border of the dorsal spinous process of the ninth thoracic vertebra. In the ninth intercostal space, this acupoint is situated about 10 cm caudal and dorsal to Urinary Bladder 42. Urinary Bladder 14 is the *shu* point of the pericardium channel in the International Veterinary Acupuncture Society transpositional system. *The pericardium channel courses over the palmar midline of the distal digit.*[3,6]
6. Urinary Bladder (BL) 18, *Gan Shu*: An acupoint located about 12 cm lateral to the caudal border of the dorsal spinous processes of the thirteenth and fourteenth thoracic vertebrae, at the thirteenth and fourteenth intercostal spaces, in the muscular groove between the iliocostalis and the latissimus dorsi muscles. Urinary Bladder 18 is associated with the liver channel, the distal course of which passes over the *dorsomedial aspect of the distal hindlimb.*[3,7]
7. Urinary Bladder (BL) 19, *Dan Shu*: An acupoint located about 12 cm lateral to the caudal border of the dorsal spinous processes of the fifteenth and sixteenth thoracic vertebrae, at the fifteenth and sixteenth intercostal spaces, in the muscular groove

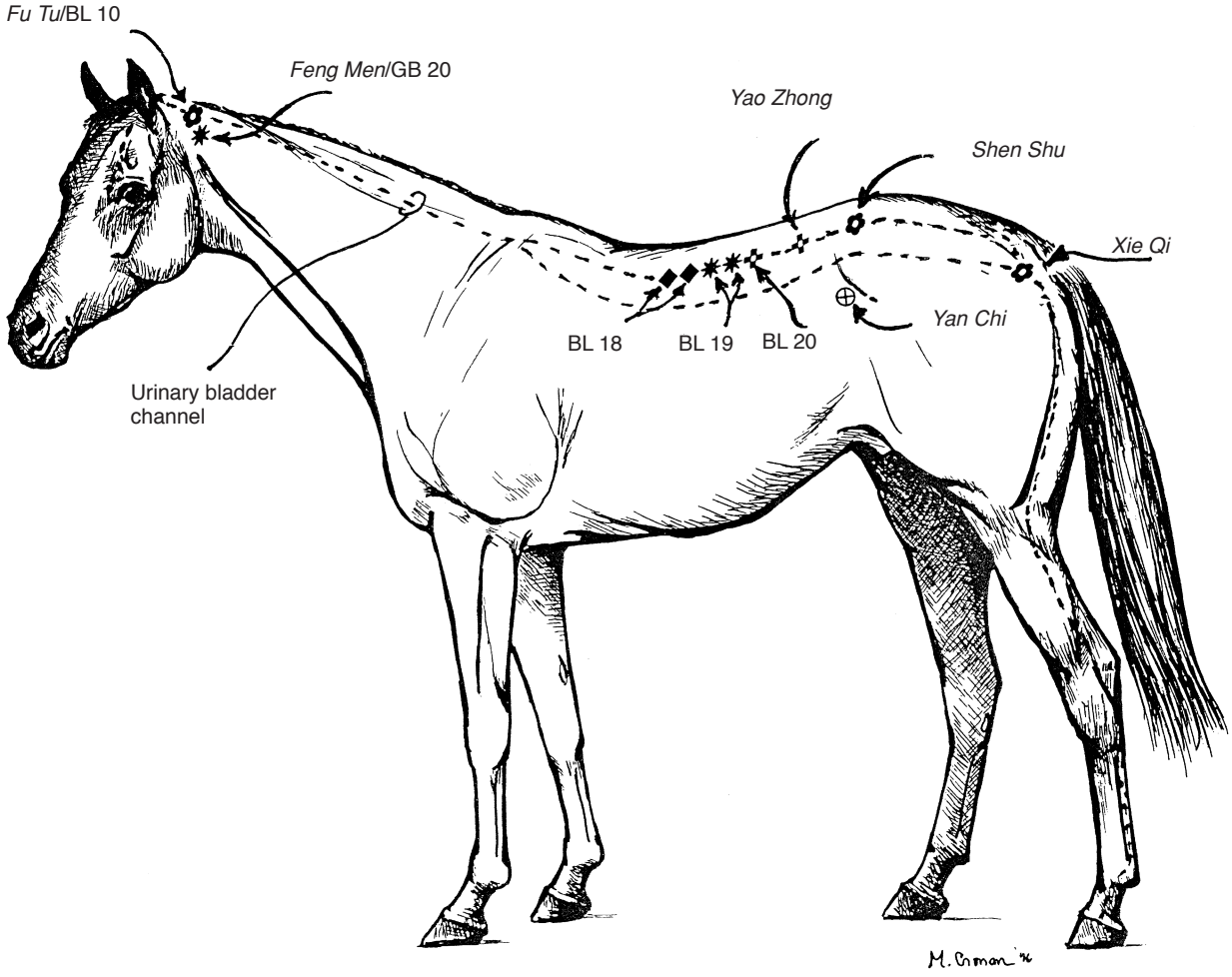

Fig. 93-8 The principal acupoints used in the diagnosis of channel pain referable to the equine hindlimb. (Courtesy William E. Jones.)

between the iliocostalis and latissimus dorsi muscles. Urinary Bladder 19 is associated with the gall bladder channel, which courses over the *dorsolateral aspect of the distal hindlimb.*[3,7]

8. Urinary Bladder (BL) 20, *Pi Shu*: An acupoint located about 12 cm lateral to the caudal border of the spinous process of the seventeenth thoracic vertebra, at the last intercostal space, in the muscular groove between the iliocostalis and latissimus dorsi muscles. Urinary Bladder 20 is associated with the spleen channel, which crosses the *medial aspect of the distal hindlimb.*[3,7]
9. *Yao Zhong*: An acupoint about 12 cm lateral to the dorsal midline, between the third and the fourth lumbar vertebrae, midway between the caudal border of the last rib and *Shen Shu*, a traditional veterinary acupoint. *Yao Zhong* is associated with the stomach channel, which courses over the *dorsal aspect of the distal hindlimb.*[5,8]
10. *Shen Shu*: An acupoint about 6 cm lateral to the lumbosacral space. *Shen Shu* is the traditional veterinary acupoint associated with the kidney channel, which courses over the *plantar medial aspect of the tarsus and the plantar midline of the distal hindlimb.*[5,8]
11. *Xie Qi*: An acupoint in the muscular groove between the biceps femoris and the semimembranosus muscles and at a level horizontal to the anus. *Xie Qi* is used as the associated point of the urinary bladder channel, which courses over the *plantarolateral aspect of the distal hindlimb.*[5,8]
12. *Feng Men* (GB 20): An acupoint in a depression cranial to the wing of the atlas, 6 cm ventral to the dorsal midline and 3 cm caudal to the ear base. *Feng Men* reflects the *dorsolateral aspect of the distal hindlimb.*[5,7]
13. *Fu-Tu* (BL 10): An acupoint in a depression cranial to the wing of the atlas, 6 cm caudal to the ear base and 4.5 cm ventral to the dorsal midline. *Fu-Tu* reflects the *plantarolateral aspect of the distal hindlimb.*[5,7]
14. *Yan Chi* (SP 14): An acupoint in a depression 4.5 cm ventral to the wing of the ilium. *Yan Chi* reflects the *medial aspect of the distal hindlimb.*[9,10]

The usefulness of recognizing reactive acupoints depends on the meaning attributed to those acupoints. I previously have published a series of articles on using reactive acupoints in pain referable to the shoulder joint,[11] metacarpophalangeal joint,[12] distal interphalangeal joint,[13] and the joints of the lower hindlimb.[14] These articles were based on 712 lame and sore horses. Over 14 years, which succeeded 13 years of strictly Western lameness practice, many thousands of examinations were conducted to establish a protocol and subsequently to confirm the following principles for evaluating reactive acupoints:

1. Palpable reactive acupoints in musculoskeletal disease depend on neural control through the spinal reflex (see Fig. 93-6).

2. Reactive acupoints that reflect the lower limb can be differentiated from somatovisceral pain, local muscular injury, and spinal pain by intra-articular medication. That is, if a pattern of reactive acupoints is present and intra-articular medication abolishes the pattern, then the treated joints alone are responsible for the changed reactive acupoints. Intra-articularly administered mepivacaine hydrochloride changes acupoint reactivity within 10 minutes. Intra-articularly administered triamcinolone acetonide, methylprednisolone acetate, or hyaluronan changes the acupoints within 24 hours.
3. In musculoskeletal extremity pain (*Bi* syndrome), acupuncture channel reactivity reflects only synovial inflammation in the distal interphalangeal, proximal interphalangeal, metacarophalangeal, and carpal joints and the distal interphalangeal, proximal interphalangeal, metatarsophalangeal, and distal tarsal joints and stifle in the hindlimb. Non-synovial pain, such as subchondral bone or extra-articular pain, are not reflected directly by reactive acupoints, except as a secondary event, such as metacarpophalangeal inflammation in tendon disease, or distal interphalangeal joint inflammation in laminitis.
4. Potentially any one joint can produce any pattern of reactive acupoints, but some joints have patterns that occur statistically more frequently than others, such as Urinary Bladder 14 in distal interphalangeal joint inflammation,[13] *Yao Jhong* in metacarpophalangeal joint inflammation,[12] or *Yan Chi* in stifle inflammation.[9,15]
5. Most complex diseases are represented by a number of different patterns occurring simultaneously. The projection of reactive acupoints by inflamed synovial structures is a clinical finding that can be confirmed easily by practical Western diagnostic tests. If a careful evaluation of the acupoints has been performed before joint injection, then the effect on the acupuncture pattern is evaluated easily.

In a sore horse that is not overtly lame and also has a multipattern presentation, intra-articular medication allows for the removal of patterns one at a time. A horse without overt lameness that has reactive acupoints reflecting forelimbs and hindlimbs could be treated only for distal tarsal inflammation. If the primary source of inflammation is the tarsus, then the whole acupuncture presentation will change. But if the metatarsophalangeal joint, stifle, or a forelimb joint is concomitantly involved, then only the pattern reflecting the distal tarsus will change. The other patterns will remain the same. Non-articular or non-traumatic pain, such as painful hind splints, suspensory ligament enthesopathy, or joint infection, do not directly cause a reactive acupoint pattern. Routinely re-examining horses that have received joint therapy or acupuncture the day after therapy and in 1 week is good practice to assess the rider's or trainer's evaluation and any change of pattern. If the pattern is balanced and the horse is doing well, then one's efforts should be directed toward maintaining the acupuncture balance. If a few remaining points show low reactivity, they usually can be treated with acupuncture. However, if lameness or sharply reactive acupoints remain, then the diagnosis must be re-evaluated. Other medications that effect, or are purported to effect, articular inflammation also can be assessed. Shoeing changes, track surfaces, and training or management regimens directly affect acupuncture balance.

No direct relationship exists between lameness and the severity and distribution of reactive acupoints. For example, a very sore racehorse can have severely unbalanced acupoints without appreciable lameness, whereas a horse with an acute radial fracture may have none. This is because acupoint reactivity in joint pain is a function of highly innervated, intra-articular synovial structures, which are activated by the mediators of traumatic inflammation.[16] The distribution, incidence, and severity of acupoint activity depend on the spatial distribution of nociceptor sites within a joint, and the quantity and characteristics of inflammatory mediators released within a joint. Lameness is a function of a much broader range of pain perception.

Joints that project reactive acupoints vary according to the occupation of the horse. Thoroughbred racehorses have a greater incidence of synovial inflammation in the metacarpophalangeal joint compared with the distal interphalangeal joint, whereas the converse is true for show hunters. Both occupations have a high incidence of distal tarsal inflammation. Steeplechase horses and show jumpers have a high incidence of metatarsophalangeal joint inflammation. Multiple joint involvement is not unusual. Determining multiple joint inflammation is difficult if lameness is not severe enough to warrant diagnostic analgesia, but careful observation of horses after joint therapy should reveal demonstrative changes in acupoint reactivity.

Two papers published in English define the limits of acupuncture. Martin and Klide's work on back pain showed the efficacy of modulation of muscle tension on the performance of equine athletes.[17] Steiss showed that acupuncture in foot lameness was no better than that expected in untreated controls.[18] Martin and Klide were treating the traditional Chinese medicine pattern of *Qi* stagnation, or abnormal muscle tension, as opposed to the Steiss' fixed-pain pattern of blood stasis. The strength of acupuncture is its ability to restore normal physiological activity in the face of functional abnormality. However, structural disease associated with foot pain cannot be reversed as easily as can abnormal muscle tension. Acupuncture can be used effectively as an adjunct therapy in structural disease, if muscle hyperactivity is part of the pattern. Careful examination of the channels should allow one to recognize patterns with a good therapeutic potential.

The basis of acupuncture diagnosis and therapy in musculoskeletal disease depends on pattern differentiation. The patterns most easily determined by the Western clinician involve the precise palpation of key acupoints in channel diagnosis. Acupoint reactivity of musculoskeletal dysfunction is a reflection of the body's response to intra-articular trauma. Because only highly innervated synovial structures of a small number of joints of the extremities are reflected by reactive acupoints, the primary disease may be reflected only indirectly or not at all. However, abnormal patterns are common and are worth investigating, regardless of the type of therapy contemplated. At the least, the abnormal pattern is a warning. The ease of elimination of a pattern gives an idea of the persistence and severity of the underlying traumatic pathological condition.

The pattern projected by synovial structures is a protective mechanism, the presence of which has the teleological function of limiting joint excursion. Therefore effective treatment must go beyond the elimination of acupoint imbalance and address all the underlying problems as well. Because disease in traditional Chinese medicine is described in terms of all of the patterns that are present, treatment requires evaluation and elimination of multipattern presentations. In my experience, the channel approach to sore but non-lame horses is effective and useful. With the exception of lameness caused by muscle tension, acupuncture is generally not sufficient by itself to manage frank lameness.

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CHAPTER • 94

Chiropractic Evaluation and Management of Musculoskeletal Disorders

Kevin K. Haussler

MANUAL THERAPY AND CHIROPRACTIC

Manual therapy involves the application of the hands directly to the body, with the goal of treating soft tissue injuries or articular dysfunction. Chiropractic, osteopathy, massage therapy, therapeutic touch, and certain physical therapy techniques are considered forms of manual therapy. Chiropractic is a health profession concerned with the diagnosis, treatment, and prevention of disorders of the musculoskeletal system and the effects of these disorders on the nervous system and general health.[1] The word *chiropractic* is derived from the Greek words *cheir,* meaning "hand," and *praktike,* meaning "business" or "to practice." The goal of chiropractic is to optimize health through the inherent healing ability of the body (i.e., homeostasis) as affected by and integrated through the nervous system.[2] The practice of chiropractic focuses on the relationship between structure (primarily the spinal column) and function (as coordinated by the nervous system) and how that relationship affects the preservation and restoration of health. Chiropractic uses controlled forces (i.e., adjustments), which are applied to specific joints or anatomical regions, to induce therapeutic responses through induced changes in joint structures, muscle function, and neurological reflexes. Human research has demonstrated reductions in pain and muscle hypertonicity and increased joint range of motion after chiropractic treatment.[2,3]

Joint mobilization and manipulation are two types of induced articular movements used in musculoskeletal rehabilitation to restore joint function. Mobilization is characterized as repetitive joint movements induced within the normal physiological range of joint motion (Fig. 94-1). Joint manipulation (e.g., chiropractic adjustment) occurs within the paraphysiological zone, which lies outside of the active (i.e., patient induced) and passive ranges of joint motion. In people, joint mobilization and manipulation induce different physiological responses. Manipulation in people has been shown to relieve adjacent spontaneous myoelectrical activity immediately, whereas mobilization has not.[2]

PRACTITIONER QUALIFICATIONS

Equine practitioners have seen a recent proliferation in the use of chiropractic techniques on horses, in one form or another. Veterinarians currently do not receive any formal education in chiropractic principles or techniques; therefore many equine clinicians do not have a basic understanding of chiropractic principles or clinical applications. Conversely, chiropractors (doctors of chiropractic) do not have any formal training in comparative anatomy, physiology, pathology, or clinical equine experience. Veterinary medicine, for the most part, has been forced to acknowledge the use of chiropractic

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CHAPTER • 94

Chiropractic Evaluation and Management of Musculoskeletal Disorders

Kevin K. Haussler

MANUAL THERAPY AND CHIROPRACTIC

Manual therapy involves the application of the hands directly to the body, with the goal of treating soft tissue injuries or articular dysfunction. Chiropractic, osteopathy, massage therapy, therapeutic touch, and certain physical therapy techniques are considered forms of manual therapy. Chiropractic is a health profession concerned with the diagnosis, treatment, and prevention of disorders of the musculoskeletal system and the effects of these disorders on the nervous system and general health.[1] The word *chiropractic* is derived from the Greek words *cheir,* meaning "hand," and *praktike,* meaning "business" or "to practice." The goal of chiropractic is to optimize health through the inherent healing ability of the body (i.e., homeostasis) as affected by and integrated through the nervous system.[2] The practice of chiropractic focuses on the relationship between structure (primarily the spinal column) and function (as coordinated by the nervous system) and how that relationship affects the preservation and restoration of health. Chiropractic uses controlled forces (i.e., adjustments), which are applied to specific joints or anatomical regions, to induce therapeutic responses through induced changes in joint structures, muscle function, and neurological reflexes. Human research has demonstrated reductions in pain and muscle hypertonicity and increased joint range of motion after chiropractic treatment.[2,3]

Joint mobilization and manipulation are two types of induced articular movements used in musculoskeletal rehabilitation to restore joint function. Mobilization is characterized as repetitive joint movements induced within the normal physiological range of joint motion (Fig. 94-1). Joint manipulation (e.g., chiropractic adjustment) occurs within the paraphysiological zone, which lies outside of the active (i.e., patient induced) and passive ranges of joint motion. In people, joint mobilization and manipulation induce different physiological responses. Manipulation in people has been shown to relieve adjacent spontaneous myoelectrical activity immediately, whereas mobilization has not.[2]

PRACTITIONER QUALIFICATIONS

Equine practitioners have seen a recent proliferation in the use of chiropractic techniques on horses, in one form or another. Veterinarians currently do not receive any formal education in chiropractic principles or techniques; therefore many equine clinicians do not have a basic understanding of chiropractic principles or clinical applications. Conversely, chiropractors (doctors of chiropractic) do not have any formal training in comparative anatomy, physiology, pathology, or clinical equine experience. Veterinary medicine, for the most part, has been forced to acknowledge the use of chiropractic

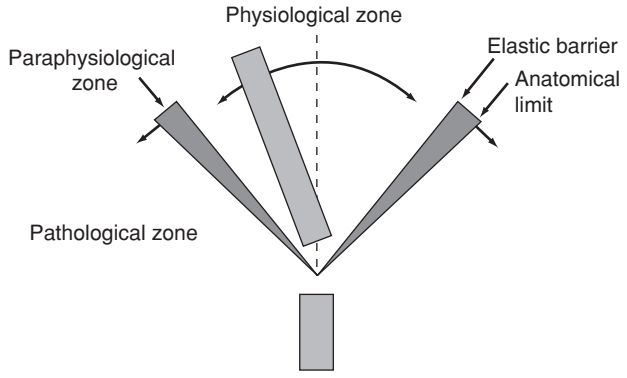

Fig. 94-1 Schematic representation of the three articular zones of joint motion: physiological, paraphysiological, and pathological. The elastic barrier and the anatomical limit mark the transitional boundaries between the three articular zones.

and other non-traditional modalities by horse owners that have sought practitioners who use these techniques and have experienced their perceived therapeutic effects.[4] However, limited research has been done to evaluate the clinical effectiveness of chiropractic techniques in horses. If veterinarians have not taken the time or effort to learn more about these non-traditional techniques, objectively evaluating the use of chiropractic, discussing the indications or contraindications for a specific treatment modality, or applying these techniques clinically is difficult. Therefore owners often seek advice about alternative therapies or treatment from someone who is not their regular veterinarian and often without his or her knowledge. To complicate matters, many laypersons claiming to be equine chiropractors are not professionally trained or licensed in chiropractic or veterinary medicine. These lay practitioners often have a limited knowledge of equine musculoskeletal anatomy, physiology, biomechanics, or pathology. Because of the potential misapplication, chiropractic evaluation and treatment should be provided only by licensed professionals (i.e., veterinarians or chiropractors working under the direct supervision of a veterinarian) who have pursued additional post-graduate training in animal chiropractic principles and techniques. The primary organization currently involved in training and certifying veterinarians and chiropractors in animal chiropractic is the American Veterinary Chiropractic Association. A similar post-graduate education program designed to qualify veterinarians and chiropractors as animal chiropractic practitioners exists at the RMIT University in Melbourne, Australia. Attendees can earn a master of chiropractic science (animal chiropractic) degree after successfully completing 3 years of part-time study. A recently offered veterinary manual therapy course at Colorado State University is available to veterinarians only and includes instruction in chiropractic, osteopathy, therapeutic massage, and other forms of manual therapy. Several other veterinary colleges are expected to develop post-graduate programs in the near future that include chiropractic and other forms of complementary medicine modalities.

Most state chiropractic and veterinary medical boards do not allow chiropractors to treat animals unless they are working under the direct supervision of a veterinarian. This requires that the veterinarian and chiropractor must work together in evaluating and treating the horse and provide appropriate follow-up care as indicated. It is strongly recommended that owners and referring veterinarians seek out licensed professionals (veterinarians or chiropractors) who have had specialized training and experience in chiropractic evaluation and treatment of horses. Veterinarians who have not pursued formal post-graduate training are not qualified to provide chiropractic care and risk producing more harm than potential benefit. It is a good idea to ask equine chiropractors about their professional and post-graduate training or certification, horse experience, and the types of techniques that they use (i.e., hands only versus more aggressive techniques or the use of additional instruments). Chiropractic requires a working knowledge and understanding of vertebral anatomy, physiology, biomechanics, pathology, and rehabilitation. Combining the knowledge and expertise of the veterinary and chiropractic professions provides practitioners with new insights and methods for diagnosing and managing horses with select vertebral or musculoskeletal disorders. A similar multidisciplinary approach has developed in human medicine to address chronic pain syndromes and vertebral column disorders in the last decade.

HISTORY OF EQUINE CHIROPRACTIC

Chiropractors often have been asked to treat the animals of clients who have experienced the benefits of chiropractic care for their own back or neck problems. Horse owners often want the opportunity to have the same type of care for their horses, without the potential adverse effects of medications or surgery. The recent increased awareness of the prevalence and management options to address back problems, with which traditional veterinary medicine has had difficulty in dealing, has also stimulated horse owners' interest in complementary forms of treatment.[5] Any vertebral column disorder can have serious effects on a horse's ability to perform. Back problems can be classified into three basic types of injuries involving the muscles, tendons and ligaments (soft tissue injuries), bones and joints (osseous injuries), or nervous system (neurological disorders). However, several concurrent injuries have been reported in 17% of horses with back pain.[6] Diagnosis of the underlying vertebral pathological conditions in horses with back pain is important for the appropriate treatment and management of these disorders (see Chapter 54).

Many horses in which chiropractic may be useful often have a history of a traumatic event or an injury related to overexertion.[7] Trauma may occur as a single event (i.e., macrotrauma), such as a trailer accident, flipping over backward, or substantial falls over jumps. Severe musculoskeletal injuries may improve gradually, but they never resolve totally or subsequently debilitating arthritis or soft tissue fibrosis may develop. Chronic, overuse injuries (i.e., microtrauma) usually are associated with poor saddle fit, improper riding techniques, inadequate shoeing, or faulty conformation. Long periods of confinement, inconsistent training programs, or cumulative stresses and strains related to prolonged, high-level athletic activities also may predispose horses to musculoskeletal injuries and reduced performance. Older horses, similar to elderly people, are susceptible to loss of vertebral column flexibility, joint degeneration, and loss of muscle strength. Aged horses also have increased healing times and increased chances of having chronic conditions or abnormal musculoskeletal compensations from prior injuries. Chiropractic techniques have helped identify and treat some of these previously undiagnosed or poorly managed problems in horses. Most veterinarians use chiropractic techniques to complement their conventional veterinary practice.

COMPLEMENTARY APPROACHES

Prevalence of back problems in horses varies greatly (from 0.9% to 94%), depending on the specialization or type of practice surveyed: general practice (0.9%); Thoroughbred

racehorse practice (2%); veterinary school referrals (5%); mixed equine practice including dressage, show jumpers, eventing (13%); spinal research clinic (47%); or equine chiropractic clinic (94%).[8] Clinicians often have difficulties when dealing with horses with no obvious localized pain or vague, unspecified lameness. Neck or back problems and limb injuries often are interrelated. Distal limb injuries can cause an alteration in carriage of the affected limb and altered gait, which subsequently can overwork or injure proximal limb musculature and the paraspinal musculature. Similarly, vertebral column injuries can produce gait abnormalities, increased concussive forces, and distal limb lameness. The diagnostic dilemma facing clinicians is to decide whether the limb or the vertebral column is the primary or initial cause of the horse's clinical problem. Unless the primary cause of the neck or back pain is identified and treated, most horses will have recurrent back pain when returned to work after a period of rest or trial of anti-inflammatory medications. Non-specific back pain most likely is related to a functional impairment and not a structural disorder. Therefore many back problems may be related to muscle or joint dysfunction, with secondary soft tissue irritation and pain generation.[9]

Chiropractic provides expertise in evaluating vertebral column disorders and can provide an additional means of diagnosis and early treatment options in certain types of gait abnormalities or performance problems. Prepurchase examinations using chiropractic examination techniques also can help identify horses that have chronic underlying neck or back problems.[7] Chiropractic addresses subclinical conditions or abnormal biomechanics, which may progress to future debilitating musculoskeletal injuries. Chiropractors also are trained in using physiotherapy modalities, strength training exercises, massage, stretching techniques, and other forms of musculoskeletal and nerve rehabilitation. Equine chiropractic is a complementary modality that can be used in veterinary medicine for the diagnosis, treatment, and potential prevention of select musculoskeletal disorders in horses. However, limited published information exists that investigates the effectiveness and safety of chiropractic procedures in veterinary medicine.

PATHOPHYSIOLOGY AND MECHANISMS OF ACTION

The vertebral motion segment is the functional unit of the vertebral column, which includes two adjacent vertebrae and the associated soft tissues that bind them together. The basic elements of joint dysfunction include altered articular neurophysiology, biochemical alterations, pathological conditions of the joint capsule, and articular degeneration.[2,3] Vertebral segment dysfunction (i.e., chiropractically defined subluxation) is a vertebral lesion characterized by the following:

1. Asymmetrical or loss of normal joint motion (Fig. 94-2)
2. Diminished pain thresholds to pressure in the adjacent paraspinal tissues or osseous structures
3. Abnormal paraspinal muscle tension
4. Visual or palpatory signs of active inflammation or chronic tissue texture abnormalities (i.e., edema, fibrosis, hyperemia, or altered temperature)[2]

Multiple theories have been proposed and tested over the years to explain the causes of vertebral segment dysfunction in people and its effects on the neuromusculoskeletal system.[2,3] The chiropractically defined vertebral subluxation complex is a theoretical model that incorporates the complex mechanical and biochemical interactions of injured nervous, muscular, articular, ligamentous, vascular, and connective tissues.[10] The theory of a "bone out of place" is outdated and not supported by current human spinal research.

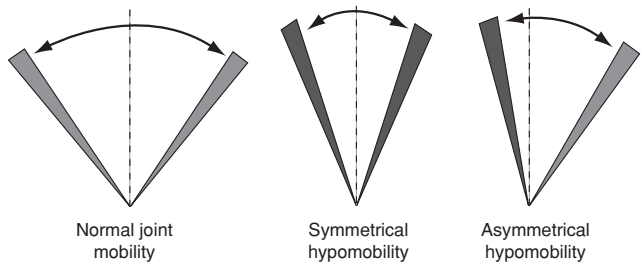

Fig. 94-2 Diagram of patterns of normal and altered joint range of motion. Symmetrical joint hypomobility is characterized by generalized stiffness. Asymmetrical joint hypomobility is characterized by reduced joint mobility in one or more directions (e.g., left lateral bending).

The goal of chiropractic treatment is to reduce pain and muscle hypertonicity, restore joint motion, and stimulate neurological reflexes. The exact mechanisms by which chiropractic techniques produce therapeutic effects are not certain. Chiropractic treatment may reduce musculoskeletal pain by stimulating nociceptive reflexes and release of neuropeptides (i.e., endorphins and enkephalins).[2,11,12] Concurrent muscle spasms restrict joint motion and may contribute to the further development of joint stiffness. In people, palpatory changes in osseous symmetry after manipulation often are associated with soft tissue alterations and not actual reduction of an articular misalignment.[9] Chiropractic care can improve restricted joint motion and may reduce the associated harmful effects of joint immobilization.[2,3] In response to chronic pain or stiffness, new movement patterns are learned by the nervous system and adopted in an attempt to reduce pain or discomfort. Long after the initial injury has healed, adaptive or secondary movement patterns may continue to persist that predispose additional joints or muscles to injury.[9] Chiropractic treatment is thought to affect mechanoreceptors (i.e., Golgi tendon organ and muscle spindles) to induce reflex inhibition of pain and reflex muscle relaxation and to correct abnormal movement patterns.[10,12] Additional modalities used to address altered movement patterns in people and horses include stretching or relaxing hypertonic muscles, strengthening weak muscles, and re-education of movement patterns.[9]

Successful chiropractic treatment requires specific techniques and psychomotor skills.[3] A thorough knowledge of vertebral anatomy and joint biomechanics is required for proper chiropractic evaluation and treatment. Joint manipulation often induces a palpable release or movement of the restricted articulations. An audible cracking or popping also may be heard during chiropractic treatment as the applied force overcomes the elastic barrier of joint resistance.[13,14] The rapid articular separation produces a cavitation of the synovial fluid.[15] Radiographic studies of synovial articulations after manipulation in people have shown a radiolucent cavity within the joint space (i.e., vacuum phenomenon) that contains 80% carbon dioxide and lasts for 15 to 20 minutes. A second attempt to recavitate the joint will be unsuccessful and potentially painful until the intra-articular gas has been reabsorbed (i.e., refractory period).

CLINICAL EVALUATION

Chiropractic, like any medical evaluation, begins with a thorough history, discussion of the chief complaint, and observation of the patient from a distance for conformation, posture, and signs of lameness. Chiropractic evaluation and treatment is not a substitute for a thorough lameness examination and diagnostic workup, because many horses have musculoskeletal

conditions that are identified readily and managed with traditional approaches. In veterinary medicine, many structural abnormalities of the vertebral column are becoming easier to diagnose with newer imaging modalities (e.g., computed tomography, scintigraphy, and ultrasonography). Currently most clinicians are not well educated or experienced in procedures required to perform a thorough functional evaluation of the equine vertebral column. Therefore horses with conditions not diagnosed readily using traditional modalities, or with suspected concurrent neck or back problems, may require referral for chiropractic evaluation.

Horses with conditions that may be responsive to chiropractic care have a variety of non-specific or vague problems (Box 94-1). The focus of the chiropractic examination is placed on evaluating static and dynamic characteristics of the musculoskeletal system. Initially the horse's general attitude and behavior are monitored for signs of pain or discomfort. Vertebral column conformation then is evaluated for proper alignment and symmetry, with special attention to the top line, shape and height of the withers, and osseous pelvic symmetry. A short-coupled horse is thought to have a higher incidence of osseous disorders, whereas a long-backed horses is more prone to soft tissue injuries.[16] Conformation is a structural relationship of body segments, whereas postural analysis deals more with functional relationships. The horse is made to stand on a hard, level surface and is evaluated for a preferred or shifting stance, head and neck carriage, vertebral curvatures, and muscular symmetry. Chiropractic gait analysis focuses on evaluating regional vertebral mobility and pelvic motion symmetry, in addition to the typical assessment of forelimb and hindlimb lameness. Gait analysis may help to rule out distal limb disorders and to rule in vertebral dysfunction, although limb lameness has been reported in about 85% of horses with back problems.[17] Motion asymmetries, restricted vertebral or pelvic mobility, not tracking straight, or lack of propulsion are a few characteristics that are evaluated. Tape on the tubera coxae or vertebral column midline may help to see subtle motion asymmetries. Normal vertebral column motion consists of small cumulative amounts of segmental motion, which produce an overall smooth curve or movement of the vertebral column (Fig. 94-3). Evaluation of the response to placing a saddle on the horse and being ridden is important for a complete assessment of horses with back problems. Inspection of the tack for proper use and fit are always suggested on initial examination. Saddles and restraint devices should be evaluated for proper fit, padding, and positioning on the horse.

A thorough physical examination is used to eliminate other more common causes of lameness or neurological disorders. Chiropractic evaluation focuses on evaluating and localizing segmental vertebral dysfunction, which is characterized by localized pain, muscle hypertonicity, and reduced joint motion.[10] Palpation is used to localize and identify soft tissue and osseous structures for changes in texture, tissue mobility, or resistance to pressure.[16,18] Soft tissue layers are evaluated from superficial to deep in two ways: by increasing digital pressure and by shifting attention with discrete palpatory movements. Shapes of structures, transitions between structures, and attachment sites also may be palpated.[9] Soft tissue texture and mobility can be compared between the skin, subcutaneous tissue, thoracolumbar fascia, and muscle. Patient response to palpation is important especially in evaluating tenderness or hypersensitivity. Osseous palpation involves evaluating osseous structures for pain, morphology, asymmetries, and alignment. Many horses with dental problems or malocclusion have localized pain during palpation of the temporomandibular joints and hypertonicity of the adjacent muscles of mastication. Osseous asymmetry of the space between the ramus of the mandible and the lateral wing of the atlas (first cervical vertebra) can be identified in horses with upper cervical congenital malformations, poll trauma caused by pulling back or falling over, or in some horses that head toss. The apices of the thoracic and lumbar spinous processes are readily palpable in most horses, unless they are grossly overweight. The dorsal apices of individual spinous processes are palpated with firm manual pressure, while monitoring for a localized pain response or muscle hypertonicity, indicative of local injury or impinged spinous processes. Palpable deviations of individual spinous processes are common, but usually

Box • 94-1

Potential Clinical Indications for Chiropractic Evaluation and Treatment of Horses

Poor performance
Back or neck pain
Reduced neck or back flexibility
Not able to raise or lower head and neck
Localized muscle tightness
Vague lameness
Uneven or asymmetrical gait
Recent change in spinal conformation
Difficult or improper saddle fit
Discomfort with saddle placement
Resentment of tightening of the cinch or girth
Stiff and slow to warm up
Bucking or pinning ears when ridden
Lame only when ridden
Constantly on one rein or line
Difficulty with a lead or gait transition
Refusing jumps
Resisting collection
Difficulty with turning in one direction
Consistent stumbling or toe dragging
Muscle mass asymmetry
Pelvic asymmetry
Not standing squarely on all limbs
Difficulty standing for the farrier
Holding tail to one side
Resentment of being groomed
Behavior or avoidance problem

Modified from Willoughby SL: *Equine chiropractic care,* Port Byron, Ill, 1991, Options for Animals Foundation.

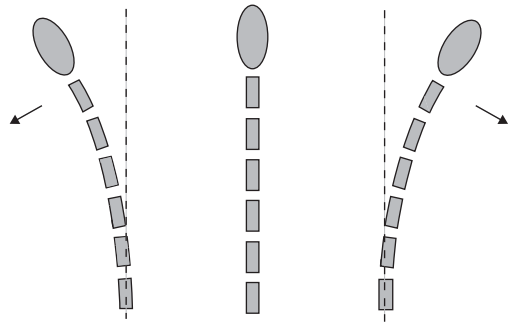

Fig. 94-3 Diagram of normal segmental vertebral contributions to overall vertebral column mobility during active left and right lateral bending. Note the smooth vertebral curve produced by cumulative segmental joint motion.

they are not associated with spinous process fracture or vertebral malposition (i.e., bone out of place), as is commonly thought. Overlapping or malaligned dorsal spinous processes are often caused by spinous process impingement, developmental asymmetries in the neural arch, or isolated dorsal spinous process deviation of unknown cause.[5,19] During induced kyphosis, the abaxial borders of each individual thoracolumbar spinous process and the overlying supraspinous ligament are palpated for pain, thickening, or deviation from midline. The tubera sacrale are palpated for height asymmetries and evaluated for a localized pain response to manual pressure applied dorsally or during abaxial compression. The apices of the sacral spinous processes (of the second to fifth sacral vertebrae) are palpated for pain or deviation from midline.

A complete musculoskeletal examination includes assessment of active and passive ranges of joint motion for all axial and appendicular articulations. Active joint range of motion is evaluated during induced vertebral movements (carrot stretches) and gait analysis. Assessment of passive range of motion requires muscular relaxation as the articulations are moved passively throughout the individual joint ranges of motion (Fig. 94-4). Abnormal segmental vertebral motion is detected when joint motion is asymmetrical or restricted bilaterally (see Fig. 94-2). Causes of segmental vertebral motion restrictions include capsular fibrosis, effusion, or inflammation. Regional causes of vertebral movement restrictions may include peri-articular soft tissue adhesions, musculotendonous contractures or, more commonly, protective muscle spasms. Combining the evaluation of joint range of motion and the presence or absence of pain at the extremes of joint motion, diagnostic interpretations can be implied.[20] Normal joint motion is painless, suggesting that articular structures are intact and functional. Normal joint mobility that has a painful end range of movement suggests that a minor sprain of the associated articular tissues is present. Painless joint hypomobility suggests that a contracture or adhesion is present. Painful hypomobility suggests an acute strain with secondary muscle guarding. Painless hypermobility of an articulation may indicate a complete rupture, whereas painful hypermobility suggests a partial tear of the evaluated structure.

Motion palpation is used to evaluate each vertebral segment for loss of normal joint motion and overall resistance to induced motion (Figs. 94-4 and 94-5). Vertebral segments with altered motion palpation findings can occur with or without localized muscle hypertonicity and pain. Using palpation to evaluate the musculoskeletal system requires an understanding of how joint motion is assessed.[9] Moving an articulation from a neutral position first involves evaluating joint motion that has minimal and uniform resistance. As the articulation is moved toward the end range of motion, a gradual increase in the resistance to movement occurs (i.e., elastic barrier) (see Fig. 94-1). End range of motion starts when any change in resistance to passive joint movement is palpable. The elastic barrier is evaluated by bringing the articulation to tension and applying gentle, rhythmic oscillations to qualify the resistance to movement. The normal joint end feel is initially soft and resilient and gradually becomes more restrictive as maximal joint range of motion is reached. This elastic barrier marks the end of physiological joint movement. A pathological or restrictive end range of motion is palpable earlier in passive joint movement and has an abrupt, restrictive end feel compared with normal joint end feel. The goal of palpating joint movement is to evaluate the initiation of motion resistance, the quality of joint motion and end feel, and the overall joint range of motion. Joint movement beyond its normal anatomical limits is characterized by ligamentous or articular capsule disruption and joint subluxation.

Individual vertebral segments are evaluated for altered motion palpation findings in flexion and extension (Figs. 94-6 and 94-7); right and left lateral flexion (see Figs. 94-4 and 94-5); and right and left rotation. In a relaxed horse the articulations of the individual second to sixth cervical vertebrae are assessed for the presence or loss of the normal elastic barrier during combined lateral flexion and rotation. The articulation between the fourth and fifth cervical vertebrae seems to be commonly affected in most performance horses, presumably because of locally altered biomechanical influences. The individual spinous processes of the third to twelfth thoracic vertebrae are deviated manually from midline, while monitoring for signs of reduced vertebral motion, localized pain, and induced muscle hypertonicity. Horses with poorly fitting saddles (i.e., tree too narrow) resent motion palpation of the affected vertebrae. The remaining thoracolumbar region is assessed in lateral bending and flexion and extension for similar signs of

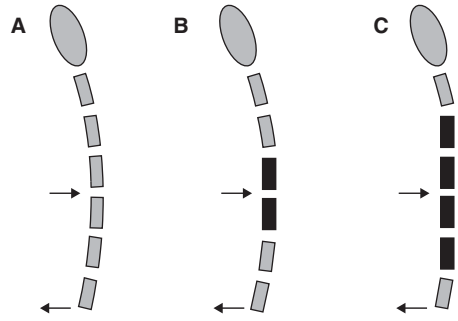

Fig. 94-4 Diagram of segmental vertebral motion during manually induced left lateral bending of the vertebral column. *Arrows* indicate direction of applied forces to vertebral column. **A,** Normal segmental motion of the vertebral column. **B,** Locally restricted vertebral motion involving two vertebrae. **C,** Regionally restricted vertebral motion involving several vertebrae.

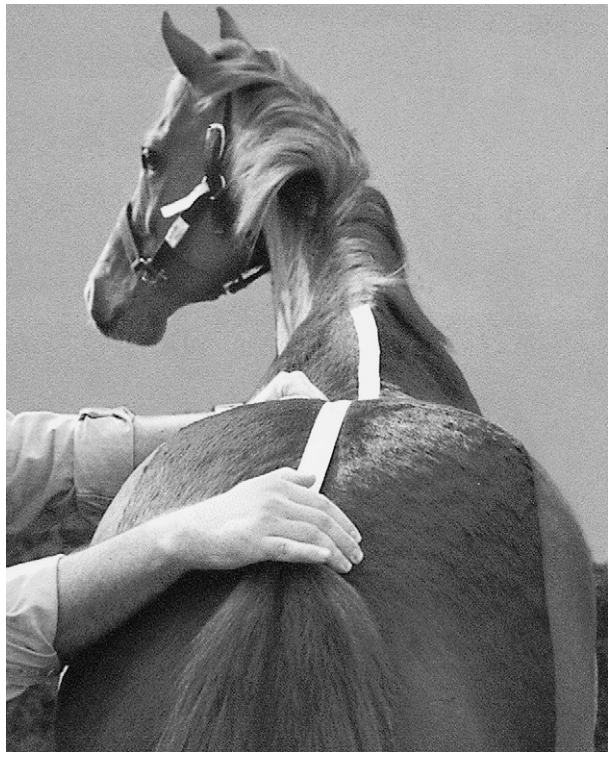

Fig. 94-5 Demonstration of motion palpation with induced left lateral flexion of the thoracolumbar vertebral column.

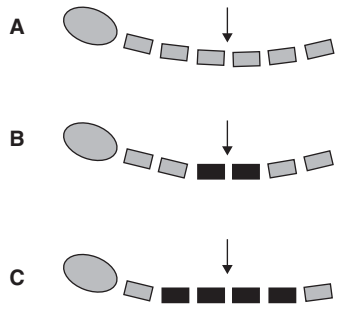

Fig. 94-6 Diagram of segmental vertebral motion during manually induced extension of the vertebral column. *Arrows* indicate direction of applied forces to vertebral column. **A,** Normal segmental motion of the vertebral column. **B,** Locally restricted vertebral motion involving two vertebrae. **C,** Regionally restricted vertebral motion involving several vertebrae.

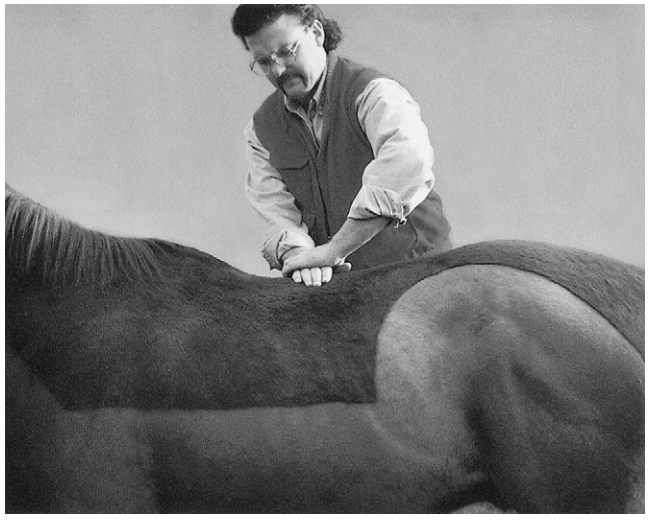

Fig. 94-7 Demonstration of motion palpation with induced extension of the thoracolumbar vertebral column.

joint dysfunction. While the clinician stands next to the horse, segmental vertebral motion in lateral bending is assessed with one hand lying over the intervertebral articulation to be evaluated. The other hand is placed at the tail head and is used to induce rhythmic oscillations to the caudal vertebral column. Normal lateral bending is maximal at the mid-thoracic vertebral region and gradually diminishes toward the lumbosacral junction. Conversely, flexion and extension are minimal in the thoracic region and gradually increase until the lumbosacral junction, the site of maximal flexion and extension. Segmental vertebral motion in flexion and extension requires the clinician to be on an elevated surface to induce ventrally directed rhythmic oscillations to the intervertebral articulations of the thirteenth to sixteenth thoracic vertebrae. The sacroiliac joints are evaluated for motion restriction or pain during induced joint motion, with an applied force directed ventrally over the tuber coxae, or during abaxial compression of the tubera sacrale (see Chapter 53). The caudal vertebrae are assessed by manipulation of individual vertebrae or by applying axial traction. The range of motion of the individual forelimb and hindlimb articulations also are evaluated in flexion and extension, internal and external rotation, abduction and adduction, and circumduction for signs of reduced joint motion, localized pain, and induced muscle hypertonicity. Palpation of the entire musculoskeletal system, including joint motion assessment of the axial and appendicular skeleton, can be accomplished within 15 to 20 minutes.

A neurological examination is indicated to evaluate horses with back problems to rule out traumatic, infectious, and toxic causes (see Chapter 11). Postural reactions also help to assess the proprioceptive status, which may be compromised in horses with certain vertebral column disorders. Rectal palpation is a commonly forgotten diagnostic test in horses with back problems. Osseous palpation rectally is useful for evaluating fractures, pelvic canal symmetry, and lumbosacral or sacroiliac joint degenerative joint disease. Externally induced pelvic motion during rectal palpation helps to assess lumbosacral joint motion internally. Palpation of the iliopsoas muscles for pain, swelling, or asymmetry is also important in evaluating horses with back pain. An orthopedic examination commonly is indicated to rule out or identify concurrent limb problems. Hematological evaluation, diagnostic analgesia, muscle biopsies, or cerebral spinal fluid analysis may be required in certain horses before chiropractic assessment and treatment. Imaging modalities that may contribute to a definitive diagnosis in horses with neck or back problems include radiography, myelography, ultrasonography, scintigraphy, computed tomography, and thermography. A thorough diagnostic workup and a definitive diagnosis, when available, are important for tailoring the appropriate chiropractic treatment and rehabilitation program. Horses with developmental osseous abnormalities, cervical vertebral fractures, thoracolumbar impinged dorsal spinous processes, equine protozoal myelitis, and sacroiliac joint luxation have been referred to me for chiropractic consultation. It was critical that these horses were properly diagnosed and that inappropriate chiropractic treatment was not applied, especially as the sole or primary therapeutic modality.

INDICATIONS FOR CHIROPRACTIC CARE

Chiropractic provides additional diagnostic and therapeutic approaches that are not currently available in veterinary medicine. The principal indications for equine chiropractic evaluation are acute or chronic neck or back pain, localized or regional joint stiffness, poor performance, and an altered gait that is not associated with overt lameness (see Box 94-1). Musculoskeletal conditions that are chronic or recurring, are not diagnosed readily, or do not respond to conventional veterinary care also may be indications for chiropractic consultation. A thorough diagnostic workup is required to identify soft tissue and osseous pathological conditions, neurological disorders, or other lameness conditions that may not be responsive to chiropractic care. Horses with a localized limb lameness or diagnosed neurological disease are better treated with conventional veterinary medicine. However, if a residual lameness continues or a secondary vertebral column disorder (e.g., stiffness or asymmetry) is identified, then concurrent chiropractic care is indicated. Horses that have concurrent hock pain (e.g., osteoarthritis) and a stiff, painful thoracolumbar or lumbosacral vertebral region are best managed by addressing all areas of musculoskeletal dysfunction. A multidisciplinary approach entails concurrent medical treatment of the hock osteoarthritis and chiropractic evaluation and treatment of the back problem. Most horses respond favorably to concurrent management, and owners appreciate a complete and thorough medical evaluation and treatment. Similarly, horses with chronic forelimb lameness often have compensatory pain and stiffness in the withers region that is readily addressed with chiropractic or physical therapy techniques.

The primary clinical signs that equine chiropractors assess are areas of localized musculoskeletal pain, muscle hypertonicity, and restricted joint motion. This triad of clinical signs can be found in a variety of distal limb disorders but it is most

evident in neck or back problems. In general, localized pain, reduced vertebral segment motion, and local muscle spasms in the vertebral column are indications of a primary spinal disorder. In contrast, regional or diffuse pain, generalized stiffness, and widespread muscle hypertonicity are indications of a chronic or secondary spinal disorder, and further diagnostics should be done to identify the primary cause of lameness or poor performance. Chiropractic care may provide symptomatic relief in horses with early vertebral osteoarthritis if related to joint hypomobility and subsequent immobilization degeneration. Research suggests that spinal manipulation also may affect certain visceral disorders (e.g., cardiovascular, respiratory, and gastrointestinal) through somatovisceral reflexes in animals and people.[2,3] However, consistent and predictable long-term changes in visceral disorders rarely occur with chiropractic treatment in people or horses.

Specially trained veterinarians or chiropractors, with advanced training and experience in animal chiropractic techniques, should be able to evaluate vertebral column disorders and determine if the condition will respond to chiropractic care, if further diagnostic evaluation is required, or if the patient would be better managed with traditional veterinary care. Unfortunately, equine chiropractors often are asked to treat animals as a last resort, when all else has failed, or the disease has progressed to an irreversible condition. Chiropractic care has helped some of these horses with chronic conditions when other types of conventional treatment have failed. However, chiropractic is usually much more effective in the early stages of clinical disease versus end-stage disease, where reparative processes have been exhausted. Chiropractic care and other holistic modalities often fail to produce their fully desired therapeutic effects when used as a last resort.

CONTRAINDICATIONS

Chiropractic is not a cure-all for all back problems and is not suggested for treatment of horses with fractures, infections, neoplasia, metabolic disorders, or non–mechanically related joint disorders. Serious diseases requiring immediate medical or surgical care need to be treated by conventional veterinary medicine before any chiropractic treatment is initiated. However, chiropractic care may contribute to the rehabilitation of most horses post-operatively, or those with medical conditions, by helping to restore normal musculoskeletal function. Chiropractic care usually is contraindicated in horses with acute stages of soft tissue injury. However, as the soft tissue injury heals, chiropractic has the potential to help restore normal joint motion, thus limiting the risk for future re-injury.[9] Acute episodes of osteoarthritis, impinged dorsal spinous processes, and severe articular changes, such as joint subluxation or luxation, are often contraindications for chiropractic. All horses with neurological diseases should be evaluated fully to assess the potential risks or benefits of chiropractic treatment. Cervical myelopathy occurs because of structural and functional disorders in the cervical vertebrae. Static compression caused by malformation and dynamic lesions caused by vertebral segment hypermobility are contraindications for cervical manipulation. However, adjacent hypomobile vertebrae may require chiropractic treatment to help restore joint motion and reduce biomechanical stresses in the affected vertebrae. Chiropractic care cannot reverse severe degenerative processes or overt pathological conditions.

CHIROPRACTIC TECHNIQUES

Chiropractic provides important therapeutic approaches that are not currently available in veterinary medicine. Most of the current knowledge about equine chiropractic has been borrowed from human chiropractic techniques, theories, and research and adapted to animals. Therapeutic trials of chiropractic manipulations often are used because knowledge is limited about the effects of chiropractic care on animals. Chiropractic addresses mechanically related disorders of the musculoskeletal and nervous systems and provides a conservative means of treatment and prevention for horses with back problems. Chiropractic treatment uses an applied, controlled force to a specific anatomical region or osseous structure to produce a desired therapeutic response. Chiropractic manipulations are applied to areas of vertebral segment dysfunction. The condition of the horse is monitored closely as the neuromusculoskeletal system responds to the applied treatment. The applied treatment influences joint, muscle, and nerve function via mechanical and biological mechanisms.[2] The therapeutic dosage of the applied chiropractic manipulation is modified by the number of vertebrae treated, the amount of force applied, and the frequency of treatment. The goal of chiropractic care is to restore normal joint motion, stimulate neurological reflexes, and reduce pain and muscle hypertonicity. Comparisons of sensitivity to palpation, muscle tone, and joint motion are made before and after treatment to evaluate the response to chiropractic treatment.

Clinicians and clients often ask how can a 500-kg horse be treated with chiropractic techniques? The answer is one vertebral segment at a time. Recent equine chiropractic research has demonstrated that forces applied to instrumented vertebral segments do induce substantial vertebral motion, usually beyond the normal range of segmental motion that occurs during locomotion.[21] Segmental vertebral motion characteristics induced during chiropractic treatment in horses are similar to those reported in people.[22] In a relaxed horse, the mass (i.e., vertebral segment) that is affected by the rapidly applied force is proportionately smaller than the mass of the clinician applying the treatment. However, if the horse does not relax the paraspinal musculature, then the mass that is affected increases dramatically from the mass of a few vertebral segments to the mass of the entire vertebral region, or potentially the entire horse. Effective joint mobilization or manipulation cannot be applied to a nervous, tense horse without risk of injury to the horse or the clinician. Chiropractic treatments in horses usually are done without any sedation or other medications, but they may occasionally be done with the horse under general anesthesia, or coupled with intra-articular injections, if indicated.[23] Typical indications for manipulation in people under anesthesia includes chronic myositis or fibrosis, or acute musculoskeletal pain, where reflex muscle spasms prevent a thorough assessment or impede manipulative treatment. Untrained professionals who do not have thorough understanding of joint physiology, vertebral anatomy, or chiropractic principles resort to overly aggressive and forceful means of applying an external force (e.g., mallets and 2 × 4s). Small, rapidly applied manual forces are easier to control and have a lower risk of soft tissue or bone injury than more often forceful types of manipulation. A good rule of thumb is that if the procedure does not look like something that the practitioner would be willing to have done to himself or herself, then maybe the procedure should not be done to a horse.

Horses are usually held by a trained handler on a loose lead during chiropractic treatment. The cervical vertebrae, sacrum, and extremities are evaluated and manipulated as needed from ground level. However, the thoracolumbar vertebrae and pelvis often require an elevated surface on which to stand for effective manipulation and proper positioning of the clinician (see Fig. 94-7). Equine chiropractic is physically demanding and requires significant mental concentration. The clinician and the horse must be relaxed and focused on each other. Environmental distractions are counterproductive to effective chiropractic care.

Muscle relaxation allows evaluation of the elastic barrier of the joint. Motion palpation is used to evaluate joint motion restrictions so that the manipulative thrust can be applied correctly. Stabilization of adjacent joints or vertebral segments is required for proper joint manipulation.

Typically, an immediate reduction in pain and an increase in segmental vertebral motion is noted. Most horses also have increased muscle relaxation, but other therapies (i.e., acupuncture or stretching) often are used with chiropractic treatment to completely resolve any remaining muscle hypertonicity. In general, conditions with an acute onset respond rapidly, whereas chronic conditions usually require longer treatment or rehabilitation. Horses with acute pain or vertebral column trauma may require initial anti-inflammatory medication, physiotherapy modalities (e.g., ice), or rest before chiropractic treatment. If stiffness, local muscle hypertonicity, or pain remains, then two or three chiropractic treatments may be indicated. Horses with chronic neck or back stiffness may require monthly evaluation and treatment for several months' duration. Owners often request chiropractic evaluation at the beginning of the performance season, a few days after athletic competition, or as a general assessment of the overall musculoskeletal system. Similarly, horses may benefit from chiropractic treatment several days before an event. Because of ethical considerations or possible masking of musculoskeletal dysfunction, it is not recommended that chiropractic treatment be performed immediately before or during any competition.

Post-treatment recommendations for actively training horses usually include stall rest or pasture turnout for one day, which provides an opportunity for the musculoskeletal system to respond to the applied treatment without immediate re-exposure to potential inciting factors of the vertebral segment dysfunction. The horse is asked to return to normal work the next day, unless other musculoskeletal injuries are present, for which appropriate supportive care is recommended. If stiffness or soreness is noted after chiropractic treatment, then an additional day of rest is suggested.

COMPLICATIONS OR ADVERSE EFFECTS

Potential adverse effects from properly applied chiropractic treatments include a transient stiffness or worsening of the condition after treatment (i.e., aggravated complaint, worsening of pre-existing state, regional soreness, or lameness).[3] Adverse reactions from properly applied vertebral manipulation are typically uncommon, but they may occur immediately after treatment or insidiously within 6 to 12 hours. The undesired effects usually last less than 1 to 2 days and resolve without concurrent medical intervention. If increased or acute musculoskeletal dysfunction or lameness is noted after chiropractic treatment, then a thorough re-examination and appropriate medical treatment or physiotherapy should be pursued. If the condition does not improve with conservative care, referral for more extensive diagnostic or therapeutic modalities is recommended. Potential harmful side effects from improperly applied manipulation from untrained individuals may include permanent articular damage or loss of function (i.e., torn ligaments, injured muscles, luxated joints, fractures, or possible paralysis if a severe underlying pathological condition is present).

ADJUNCT RECOMMENDATIONS AND PROGNOSIS

Chiropractic care often is supplemented with massage, physiotherapy modalities, and stretching or strengthening exercises to help soft tissue rehabilitation and to help restore normal vertebral joint motion (see Chapter 96). These concurrent therapies also help to encourage owner participation in the healing process and provide close monitoring of the patient's progress. Other recommendations may include changes in training schedules or activities, corrective shoeing, or tack changes. Horses with poorly fitting saddles often have localized pain and muscle hypertonicity in the caudal withers region. Saddle refitting, coupled with chiropractic treatment of the painful withers region, leads to rapid recovery and management of a common problem for many horse owners. A horse with a 6-month history of consistently resenting saddle placement and bucking the owner off was treated chiropractically and the next week won 11 ribbons at the local country fair. Many horses with repetitive-use disorders may benefit from cross-training activities. Clinicians also have reported synergistic therapeutic effects with the combined use of chiropractic, acupuncture, and other holistic modalities in equine patients. In general, horses with conditions with an acute onset respond rapidly and have a good prognosis for return to function. Horses with chronic injuries may gain only short-term improvement of restricted motion, pain, or muscle hypertonicity. This corresponds to current research on joint immobilization and spinal learning.[9] Horses with chronic conditions usually require a series of treatments to affect a more lasting improvement. Musculoskeletal health depends on movement and use. Scientific evidence suggests that long-term rest or inactivity is contraindicated for back problems in people.

SUMMARY

A thorough knowledge of equine vertebral anatomy, biomechanics, and pathology is required to understand the principles and theories behind chiropractic and to apply its techniques properly. Anecdotal evidence and clinical experience suggest that chiropractic is an effective adjunct modality for the diagnosis and conservative treatment of select musculoskeletal-related disorders in horses. Chiropractic provides additional diagnostic and therapeutic means that may help equine clinicians to identify and treat select musculoskeletal disorders. Chiropractic provides specialized evaluation and treatment of joint dysfunction and conservative treatment of neuromusculoskeletal disorders that currently lack treatments in traditional veterinary medicine. However, limited research is currently available on the efficacy of equine chiropractic. Direct measures of chiropractic techniques in horses demonstrate substantial induced vertebral motion, usually beyond the normal range of segmental motion that occurs during locomotion.[21] In 1996 the Committee on Alternative and Complementary Therapies of the American Veterinary Medical Association suggested that the research community should be encouraged to prioritize avenues of research and to allocate research funds to projects that will provide further scientific evaluation of these modalities.[24] Because of its potential misuse, animal chiropractic evaluation and treatment should be provided only by licensed and specially trained clinicians or chiropractors. Future studies should evaluate the use of chiropractic techniques in horses and evaluate the long-term functional effects.

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CHAPTER • 95

Therapeutic Use of Ultrasound, Lasers, and Electromagnetics

Michael A. Ball

The use of therapeutic ultrasound, lasers, electromagnets, and magnets may be important for lameness treatment, but the body of equine-specific research about correct usage is small. These treatment modalities are widely promoted to horse owners without veterinary involvement. No statutory control is exercised over the manufacturing and sale of this equipment, which may not meet the minimum technical requirements to be useful. Certain types of equipment may cause harm if used incorrectly.

Evidence exists that therapeutic ultrasound, lasers, and magnets do have value to manage some equine injuries, and there are vague therapeutic protocols derived from limited objective research to allow for safe clinical experimentation.

THERAPEUTIC ULTRASOUND

Objective study of therapeutic ultrasound in people or horses is limited and conflicting. Comparison of studies is difficult because of the use of differing treatment protocols. A placebo effect does appear to occur in people. Nonetheless, I believe that therapeutic ultrasound can be beneficial, but more information is needed about appropriate machine settings, particularly because ultrasound does have the potential to damage tissues.

Equipment

Therapeutic ultrasound machines are available as portable units powered from wall outlets (110 V or 220 V) or cordless and battery operated. Transducer size varies from 5 to 10 cm in diameter surface, appropriate for treatment of distal extremities or large muscle masses, respectively. The sound waves emitted from regular transducers are relatively unfocused and have the potential to penetrate all tissue directly underneath, with the waves being most intense at the skin level and attenuating as they pass through soft tissue. Newer transducers allow for the focusing of ultrasound waves on the target tissue, thereby decreasing exposure to surrounding tissue. Time, frequency, power as watt per square centimeter (W/cm^2) or overall watts, or duty cycle can be controlled. *Duty cycle* refers to the ultrasound emissions being continuous

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CHAPTER • 95

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or pulsed. To avoid potential equipment damage the clinician should never turn on ultrasound emission without the transducer being properly coated with contact jelly and in contact with the tissue being treated.

Mechanism of Action

The exact mechanisms by which sound waves are potentially beneficial to damaged tissue are unknown. The main effect is the production of heat, because sound energy is converted to thermal energy as it interacts with tissue. Mechanical effects are also caused by the high-frequency vibrational energy, such as cellular friction and cavitation, which are proposed to have a micromassage effect at the cellular level. Factors influencing the production of thermal energy within the tissues and the mechanical bioeffects include the output power and frequency, the profile of the ultrasound beam, and temporal aspects of the sound emission. Some evidence indicates that the biomechanical effects are responsible for some of the positive alterations in cellular physiology within damaged tissue, such as increases in cellular protein synthesis and effects on fibroblasts. Possibly, pulsed ultrasound emissions could decrease the overall heat production within the tissue, decreasing the potential for thermal injury at the cellular level, while maintaining the positive bioeffects of isonation (the exposure of tissue to ultrasound energy).

In vitro studies have demonstrated alterations in blood flow and angiogenesis and increases in collagen and protein synthesis and in osteogenesis. The effects depend on the power and frequency of the ultrasound applied.[1,2] In vivo studies have demonstrated improved healing of pressure sores and surgical wounds in people.[3,4] Clinical trials have shown conflicting results for therapeutic ultrasound in treating tendon injuries.[5-7] Therapeutic ultrasound had no effect on regeneration of experimentally injured muscle fibers[8] and failed to alleviate chronic heel pain in people.[9]

The effect of therapeutic ultrasound on surgically split superficial digital flexor tendon (SDFT) was assessed in horses with the contralateral limb acting as control. The treated limb received 1.5 W/cm^2 with continuous duty cycle for 10 minutes daily, 6 times weekly for 2 weeks. At 4 and 6 weeks the control tendons showed perivascular inflammatory reactions and degeneration of tendon tissue, and the treated limbs showed good healing characterized by vascularization and fibroblastic proliferation.[10]

More is not always better. Excessive exposure to ultrasound may cause thermal injury. Certain therapeutic benefits observed using one treatment dosage are not observed when a higher dosage is used in the same situation. The interval between injury and treatment is important: the clinician should avoid treatment during the acute phase of inflammation. Cold hydrotherapy and anti-inflammatory medications should be used for acute tendon and ligament injuries for 3 to 5 days before using therapeutic ultrasound.

Clinical Application

Clipping the hair over the treated area aids transmission of ultrasound waves. A liberal amount of contact gel should be applied to both the treated area and the head of the transducer to minimize the risk of thermal damage. The transducer should be in constant motion when emitting and in contact with the body. I usually move the transducer slowly up and down the leg when treating tendons or in a spiral circle pattern if treating muscles.

To treat tendons or ligaments, I use 3 MHz with 1.5 W/cm^2 power and a continuous duty cycle for 10 minutes of therapy once daily for 14 days.[10] The treatment is started 3 to 5 days after the initial injury, when the acute inflammatory phase is subsiding. For muscle tissue I use a lower frequency (1 MHz) to provide deeper penetration.

No good clinical trials are available that document the efficacy of therapeutic ultrasound, but subjectively I believe that some horses do benefit when this treatment is used with other standard therapies. More information is needed about the ideal time and duration of treatment and the power and frequency of the ultrasound for different injuries.

THERAPEUTIC LASER THERAPY

Laser Physics

The term *laser* stands for *l*ight *a*mplification by *s*timulated *e*mission of *r*adiation. A laser emits monochromatic, parallel, coherent light, creating a uniform, focused beam of photon energy. The physical characteristics of laser light are determined by the wavelength and power. Power is the energy contained in the photon beam in watts, which is equal to joules per second. The power output of lasers used in medical physical therapy is in the milliwatt range. The wavelength is the relationship of the frequency of the emitted laser light to the speed of light; the wavelength of laser light used in low-intensity laser medicine is between 300 and 10,600 nm. The intensity is the relationship of power to the surface area of the tissue irradiated and is indicated by watts per square centimeter. The dose summates the total of power, time, and area of tissue irradiated and is indicated by joules per square centimeter (J/cm^2).

Medical Laser Equipment

A variety of laser equipment is marketed for equine use, some of which comes with detailed treatment protocols, offering settings for a variety of musculoskeletal problems. However, these protocols are not likely supported by scientific study because of the dearth of literature related to laser use in the horse. It is essential that the power and wavelength output are adequate to penetrate the horse's skin. Many machines currently available are inadequate. Even deep therapeutic lasers with wavelengths of 632 to 950 nm penetrate only 0.5 mm beyond human skin.[11-14]

Mechanism of Action

The mechanisms behind the biostimulatory or biomodulatory effects of low-intensity laser irradiation are not understood fully, but they are thought to include alterations in mitochondrial enzymes, cytochromes within the electron transport chain, and cellular calcium channels.[15-17] The net effect of these cellular alterations includes the triggering of mitosis and cellular proliferation.[18] Laser light of the correct wavelength and intensity may have a variety of biomodulatory effects, including alterations of circulation, production of pharmacologically active compounds, and analgesia; excessively high doses inhibit cellular metabolism.[19,20] Any effect depends on power, wavelength, dosage, and intensity.

Clinical Application

Low-intensity laser irradiation may influence wound healing. Application of an 860-nm diode laser at a dose of 2 J/cm^2 increased cell proliferation and enzyme activity of human fibroblasts. Experimentally created teat wounds in cattle were treated with a 632-nm continuous wave helium-neon laser, with an output of 8.5 mW and a dosage of 3.64 J/cm^2. Healing was improved by minimizing inflammation and edema formation, improvement of skin regeneration, and enhancement of collagen synthesis.[21] To my knowledge, only one experimental study has evaluated the effect of laser therapy on wound healing in the horse, which showed no benefit in treatment with an 830-nm laser at a dose 2 J/cm^2.[22]

I have obtained beneficial results treating skin wounds and after excision of exuberant granulation tissue, using a 69-diode laser cluster probe containing 34,660-nm and 35,950-nm diodes, with a total power of 865 mW for an

approximate dosage of 25 J/cm^2 (continuous wave) daily for 7 to 10 days. The cluster probes are advantageous because they allow treatment of a larger unit of area per unit of time, thereby decreasing the overall treatment time.

Several uncontrolled clinical studies have evaluated laser treatment of superficial digital flexor tendonitis in the horse.[23,24] Whether current equipment has adequate penetration is debatable,[12,13] although using detection equipment, I estimated that a 200-mW diode laser at 810 nm penetrated up to 2 cm in equine neck muscle.

Low-level laser therapy may provide analgesia.[25-27] An 830-nm diode laser used at an intensity of 3 W/cm^2 and a dosage of 24 J/cm^2 reduced post-operative abdominal incisional pain in people.[25] A large meta-analysis assessed the efficacy of analgesia for patients with osteoarthritis or rheumatoid arthritis of the knee, hand, or fingers.[27] Patients were treated three times weekly for 3 to 6 weeks. An analgesic effect was achieved at 632 nm or 830 nm, although the lower wavelength appeared superior. Lower doses (≤3 J/cm^2) tended to be more effective than higher doses.

No studies have evaluated analgesia in the horse, except relating to acupuncture. I have used a 200-mW single-diode laser operating continuously at 810 nm at a dose of 3 J/cm^2 as an adjunct therapy for horses with centrodistal and tarsometatarsal osteoarthritis. The treatment targeted the joint spaces, moving circumferentially around the joints, especially treating the area of the cunean tendon. The treatment was performed three times weekly and was subjectively beneficial in some horses. Several horses with pronounced pain on palpation of the cunean tendon were treated only with laser therapy and had reduced pain and lameness after six treatments. Further controlled clinical trials are essential in the horse to determine whether beneficial therapeutic effects can be achieved and the optimum treatment protocols.

I believe that therapeutic laser can be a useful adjunct therapy but not a replacement for any traditional therapies for musculoskeletal lameness. I do feel that the benefits of therapeutic laser on wound healing are profound and recommend its use as part of primary wound care where applicable.

MAGNETIC AND ELECTROMAGNETIC THERAPY

Considerable controversy surrounds the use of magnetic and electromagnetic therapy, with conflicting published results. A large amount of anecdotal information exists, with a strong marketing push by equipment manufacturers to the lay horseman, with a general lack of technical information, understanding, and direction for use.

Basic Physics

An electromagnetic field is created by passing an electric current through a coil of insulated wire. The strength of the magnetic field measured in gauss is proportional to the magnitude of the current. The nature of the Gaussian field can be altered by pulsing the electric current and altering the waveform of the signal. With a single coil the magnetic field emanates perpendicular to the coil and then bends. Use of two coils is necessary for a magnetic field to pass through tissue staying perpendicular to a particular plane. To treat a horse's third metacarpal bone coils are placed lateral and medial to the bone. The distance between the coils cannot be greater than the diameter of the coils, otherwise the uniform nature of the magnetic field breaks down. If a single coil is used, the magnetic field only penetrates tissue to a depth of one half the diameter of the coil.

Magnetic Equipment

Magnetic/electromagnetic devices available range from small, solid magnetics (stationary magnets) imbedded in bell (overreach) boots and a variety of other boots and bandages, to full-size blankets containing large coils and transformers, allowing for adjustable field strength and characteristics or pulse therapy (electromagnet). The U.S. Food and Drug Administration regulates the use of electromagnetic therapy in people, but no quality control is exercised over the products available for use on the horse.

Mechanism of Action

Since the discovery of the piezoelectrical properties of bone, a large body of research has supported the effects of an electromagnetic field on bone healing.[28-31] Electroporation is a universal, non-thermal, bio-electrochemical phenomenon relating to the rate of two-way transmigration of chemical ions through cell membranes, defining the metabolic rate and ultimately the energy level of the cell. Pulsed electromagnetic field therapy may stimulate electroporation and provide pain relief.[32] Pulsed electromagnetic field therapy enhanced nerve regeneration in experimental rat models[33-36] and was beneficial in treating rotator cuff tendonitis, using a magnetic field generated by a single coil, peaking at about 27 G (specific waveform varied over 4.23 ms) pulsed at 73 Hz. The treatments were applied for 1 hour, 5 to 9 hours per day.[37]

Studies of electromagnetic therapy in the horse are limited. Some benefit in fracture healing was observed using two coils coupled to a signal generator creating a 50-ms, 2-G field pulsed at 2 Hz, applied for 2 hours per day.[38] Horses with bucked shins also improved. However, pulsed electromagnetic field therapy delayed repair tissue maturation and collagen-type transformation in experimentally created defects in the SDFT in horses.[39] In contrast, electromagnetic stimulation increased tissue maturation, tensile strength, and collagen content in experimentally damaged rabbit collateral ligaments[40] and increased breaking strength in experimentally damaged rat tendons.[41] Variations in temporal relationship of therapy to injury and overall dosage protocol may mean the difference between no effect, positive effect, and negative effect.

Despite the widespread use of electromagnetic treatment in the horse, I am skeptical that many commercially available magnetic products generate a magnetic field strong enough to have any bioeffects. The effects of magnetic treatment on bone healing are well documented, but for other conditions determining the degree of therapeutic benefit, if any, is difficult because many horses receive a combination of treatments. I do not use any form of magnetic therapy as a primary treatment for any musculoskeletal disorders. I believe more research is required to demonstrate efficacy. If I am approached by a client who is using magnetic therapy, I encourage him or her to allow me to make a diagnosis and perform more standard treatments. Many clients use magnetic body blankets as an adjunct treatment for back muscle soreness, but results are anecdotal.

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CHAPTER • 96

Massage, Stretching, Homeopathy, and Herbs

Joyce C. Harman

The addition of massage, stretching, herbal treatment, and homeopathy to a conventional treatment protocol can help return horses with sports injuries to full performance more quickly. Performance problems may be alleviated despite the absence of obvious painful foci.

RECOGNIZING MUSCLE PAIN, TENSION, AND LOSS OF JOINT MOTION

The clinical examination should include assessment of muscle tension and joint mobility. Muscle pain can result in subtle or overt lameness and a variety of behavioral problems. A horse with a recent muscle injury often becomes sorer toward the end of a ride, whereas a horse with an old muscle injury starts out stiff and sore, takes a long time to warm up, and then improves. The clinician should observe the natural stance of the horse at rest, because many horses with pain develop a compensatory positioning of the limbs. Symmetry should be assessed from all angles, including from above, by standing on a stool behind the horse. Many horses with muscle pain move freely on the lunge, but they may become stiff or sore when ridden, resulting in stiffness or resistance when asked to perform certain movements, such as circling left or right, rather than overt lameness.

The clinician should gently palpate the muscles of the neck, back, and thorax, looking for tension, sensitivity, flinching, or discomfort. A healthy muscle feels soft and springy. Muscle under tension feels tight and hard and has little spring. Muscle spasms and fasciculation can be seen both locally and at a distance from the palpation site of tense muscle. Muscle in spasm is more prone to injury and takes longer to warm up because blood flow is restricted. A horse with muscle pain may splint its back or hold it rigidly in place to avoid movement. The clinician should then probe more deeply into the muscle. If the horse is accustomed to a light touch, any spasm, hardening, or flinching with deeper palpation confirms and defines sites of pain and hard knots in the muscle. If deep palpation is started too quickly, the horse may flinch from the touch rather than from pain.

The clinician should assess joint mobility by using the stretches described subsequently. Healthy joints move freely and have spring to them, whereas problem joints often feel stiff and have reduced motion.

MASSAGE

Massage techniques can be used to treat horses with specific muscle injuries, to prevent injuries, for pre-event preparation, and for post-event recovery.[1] Damaged, contracted muscle has less blood flow through the capillaries than healthy muscle. One of the primary benefits of massage is restoration of even blood flow to the muscle, a result that can be confirmed thermographically. Mechanical breaking of scar tissue is often reported, but evidence that physical change occurs in any tissue is lacking. Massage currently is used routinely on many human athletic teams to prevent and treat muscle injuries and might be useful if used more routinely in equine athletes.

Much subtle lameness can be attributed to muscle damage that responds well to massage.[2] Frequently injured muscles include the adductor, gracilis, psoas, semimembranosus, semitendinosus, gluteals, quadriceps, longissimus dorsi, rhomboideus, trapezius, and ventral strap muscles of the neck. With recent injury massage is used to enhance the clearing of lymphatic fluid and blood from the damaged area. Massage is used in horses with older, chronic injuries to restore circulation and flexibility.

Massage therapy consists of manipulation of the muscle using a variety of techniques. Massage can be specific to treat a small area, known as *trigger point therapy* (also known as *myotherapy* or *neuromuscular therapy*), where concentrated finger pressure is applied to trigger points (painful irritated areas in muscles) to break cycles of spasm and pain. Trigger points commonly occur in the gluteal muscles, the longissimus dorsi muscles over the last few ribs, and the lumbar muscles. However, trigger points can occur in any muscle. Swedish massage is a system of long strokes, kneading, and friction techniques on the more superficial layers of the muscles, combined with active and passive movements of the joints. Generally this form of massage feels good and relaxes the horse, but it is less effective for treating a problem. Several forms of deep tissue massage are also used and release the chronic patterns of tension in the deeper layers of muscle tissue, through slow strokes and deep finger pressure on the contracted areas, following or going across the grain of muscles, tendons, and fascia.

Myofascial release is a particularly useful form of massage for horses that releases tension in the fascial covering of the muscle. Long, stretching strokes are used to release muscular tension. In many horses the fascia feels tighter than the underlying muscle. *Shiatsu* and acupressure are Oriental-based systems of finger pressure that treat special points along acupuncture meridians (Chapter 93). Acupressure can be a useful form of massage, although its effects generally are longer lasting and require less repetition.

Massage usually is performed by laypersons, many of whom have little training other than a 1-week course. Currently the best way to find a qualified person is to locate someone who has completed a full 500-hour massage course for people and who has then taken one of the more extensive equine courses. Clinicians are advised to locate a few top-quality massage therapists for client referrals, so that effective therapy is performed and the clinician stays involved with the horse. Riders also should be advised of the benefits to muscle of slow warm-up before exercise and a cool-down period after exercise to facilitate clearance of lactic acid.

STRETCHING

Stretching is one of the simplest, most effective ways to enhance performance, aid in rehabilitation after an injury, maintain or restore full joint mobility, and assess flexibility.

Stretching can be performed by an owner regularly or by a clinician as part of an examination and treatment. In my experience, horses that are stretched regularly (daily or at least 2 to 4 times a week) are generally more flexible and stay sounder than horses that are not stretched.

Stretching must be done correctly and carefully, keeping the horse comfortable and relaxed, to avoid injury. The use of force can damage muscles and joints. Any joint can be stretched safely through its full range of motion if the handler holds the leg in position and waits for the horse to release and relax the muscles; then the stretch can be taken further. A horse is always stronger than its handler, and if the horse pulls back, it tightens the muscle the handler is trying to loosen. An analogy is persons bending down to touch their toes. Without some warming up and gentle stretching, many persons feel pain and tension at the back of the knees, but with time and progressive further stretching, reaching further without pain is possible.

Upper Body

Carrot stretches, where the horse is asked to reach to the tuber coxae and down between the forelimbs, are one of the most effective stretches to increase flexibility. A carrot or other treat is used to entice the horse to stretch. A horse with normal flexibility can reach each tuber coxae and come close to its stifle, as well as reach down well between its forelimbs. This stretch restores flexibility to the neck, withers, and thoracic area, with some muscle stretching even as far as the lumbar area. Many horses have restricted motion in the neck after trauma such as pulling back when tied and falling. Some horses may need chiropractic care to restore complete motion (see Chapter 94).

Motion through the thoracic vertebrae and ribs is critical to allow a horse to perform lateral movements demanded by many sports, including cutting, reining, barrel racing, dressage, and jumping. Subtle lameness and particularly stiffness in one direction often results from lack of motion through the rib cage. The stretch to diagnose and treat restricted motion through the thorax is the belly lift or sideways belly lift. Regular use of these stretches can improve performance substantially. To perform the belly lift, a handler places finger tips on the midline of the sternum behind the elbows and presses upward. The fingers can be moved along the midline to the center of the abdomen to raise different parts of the back. The handler starts with a light touch and increases pressure until the back rises. For some horses a blunt plastic tool such as a needle cap or plastic writing pen may be needed to raise the back. To perform the sideways belly lift, the handler stands on one side of the horse and reaches across the midline to the girth area behind the elbows on the opposite side and then pulls the rib cage diagonally upward toward the withers on the side on which the handler is standing (toward the handler's head).

When belly lifts are comfortable for the horse, the abdominal muscles should contract, the longissimus muscles should fill out and relax, the withers should rise, and the head and neck should stretch out and down. With sideways belly lifts the rib cage should move easily laterally, the same amount from both sides. The withers (trapezius and rhomboideus) on the side at which the handler is standing should fill with relaxed muscle, while the opposite side should form an even concave bend. The head and neck should stretch down and forward, curved slightly away from the handler.

When pain and tension exist, parts of the longissimus group of muscles contract, as can be seen by depressed areas in the muscle as the back is raised. The withers may or may not rise, depending on the location of the pain, and the head and neck usually rise up. Many horses are incapable of rising or moving the back laterally without significant pain, and they kick or bite; thus caution must be used. The pain preventing a comfortable belly lift may be from muscle tension in the psoas, lumbar area, intercostal muscles, pectorals, or withers. Pain also results from the loss of motion or arthritis that may be present throughout the thoracic vertebrae and may require chiropractic treatment (see Chapter 94).

The withers stretch can relieve tension and pain through the cranial thorax and partly under the shoulder blades. The handler places his or her hands over the withers and then leans back, gently pulling on the withers, starting at the cranial edge and moving toward the caudal part of the withers. When the stretch is ended, the handler should release the withers slowly. If little pain is observed, and the horse is enjoying the stretch, the horse will pull against the handler and relax into the stretch. If pain is substantial, muscle fasciculations and spasms may be felt under the handler's hands, and the horse does not pull into the stretch. This is especially useful with horses wearing tight blankets and those that appear to be lame while wearing blankets.

The tail stretch relieves tension in the tail and in the dorsal and ventral muscles surrounding the sacral area (sacrocaudalis dorsalis and ventralis). Many horses have substantial tension in the tail and sacral area, which can contribute to muscle tension in the hindlimbs, especially the hamstring area. The tail stretch is performed in several stages, with the handler first taking the tail and rotating it in circles, holding it at the base and the tip of the bone. After some relaxation is achieved, the tail may be brought gently up over the back, waiting for the muscles to relax as the stretch goes further. Then the tail can be pulled slowly caudally away from the body. When this part of the stretch is complete, the handler releases the tail slowly; otherwise, tension increases as the tail snaps back.

Lower Limbs

Stretches for the joints and muscles of the limbs include leg circles, psoas stretch, and hamstring stretch. These stretches are particularly for the large joints, hip and shoulder, and associated large muscle groups. Subtle lameness sometimes can be attributed to restriction of motion of the hips and shoulders and pain in the large muscle groups.

Leg circles are performed by holding the foot in the position for cleaning and then, as if a pencil were attached to the toe of the foot, a circle is drawn in the ground five or six times in clockwise and counterclockwise directions, with the entire leg moving. A horse with a full range of motion produces an even circle in all directions. A horse with restricted motion may give an uneven circle, square, or oblong pattern, with intermittent signs of discomfort. Using circles as a regular stretch improves range of motion, stride length, and ease of shoeing.

The psoas and hamstring stretches are perhaps the most important stretches for a performance horse. The psoas muscle is a large hip flexor muscle that inserts on the ventral side of the ribs and is often tight, causing many horses to have difficulty stretching the hindlimb caudally. The pull of a tight psoas muscle often restricts the flexibility of the rib cage in the belly lifts. These stretches are performed by holding a hindlimb cranial or caudal to the vertical, at the limit of the range of motion, and then waiting for the horse to release the leg a bit. As the muscles relax, the stretch can go further, until the leg reaches the back of the carpus (hamstring stretch) or can touch the ground behind the vertical. If force is used, the horse pulls back and tightens the muscle. If the horse is comfortable with the stretch, it releases the leg quickly, but if uncomfortable, the horse may only release small amounts at a time.

HOMEOPATHIC TREATMENT OF INJURIES

Homeopathy is a branch of complementary medicine that is perhaps least well understood, but it does have a place in

the management of lameness. Homeopathic remedies can be used with any conventional treatments, but faster results are achieved by using the remedies alone, together with supportive care such as cold hosing, bandages, or controlled exercise. The principles of homeopathy are discussed elsewhere.[3]

Homeopathic remedies usually are supplied in small tablets or sand-size granules listed with a potency of 30X, 30C, or 200C. The standard dose is six to eight tablets, or one-half teaspoon of granules for an adult horse and less for a pony or foal. The remedies can be given once or twice daily for the 200C formulation, or 2 to 3 times daily for the 30X or 30C potency, and can be fed with small quantities of food or placed directly in the mouth. Homeopathic remedies are prepared by diluting the original substance and therefore do not yield positive results in a drug test during competition in the strengths just described. Several remedies are particularly useful for treating lameness. I have used *Arnica montana* successfully to decrease the pain, swelling, stiffness, and healing time in many traumatic injuries. *Arnica* can be given 1 to 3 times daily, with the more frequent dosing used for more severe injuries. *Arnica* can be administered at any point in the healing process to improve healing, but it is most effective if used for horses with acute injuries.

Ruta graveolins is a remedy that has particular affinity for injuries to the periosteum, including splints, ringbone, strained joints, and blows to the bone. In my experience, the injuries listed previously heal more quickly and with less exostosis than would be expected with conventional treatments. For acute injuries, *Arnica* is used for the first few days or a week until the main swelling and pain is reduced, and then *R. graveolins* is used for 3 to 5 days. Horses with chronic ringbone respond well to *R. graveolins* administered once or twice a week as needed to keep the horse comfortable and in many horses, sound. *R. graveolins* also is used for horses with acute tendonitis or desmitis and is given 2 to 3 times a week for several weeks and then is followed by *Rhus toxicodendron*. *R. toxicodendron* is indicated when an injury to muscle, tendon, ligament, or joint has healed to the point where the horse is stiff when starting out but warms up and moves much better. In the later stages of a tendon injury, *Rhus toxicodendron* is given for 2 to 5 days in a row and then 1 to 3 times weekly for a few weeks. *R. toxicodendron* also is used commonly for arthritic conditions, administered once or twice weekly.

HERBAL THERAPY FOR LAMENESS

Herbs have been used for centuries to treat various injuries, and in China martial arts practitioners also used herbal preparations to strengthen tendons and ligaments. Herbs can be used internally (as powders or teas) and externally as ointments, liniments, or poultices.[4] Herbs contain active ingredients and some, such as yucca and white willow bark, yield test positive results in blood and urine testing for drugs, mainly when the herbs are ingested rather than topically applied.

A. montana can used as a poultice, ointment, body wash, or liniment as a topical treatment for bruising or muscle, tendon, and ligament injuries and overworked and tired muscles. *Arnica* should never be used on broken skin because it irritates the skin. *Arnica* has mild anti-inflammatory activity.

Comfrey has anti-inflammatory properties and is best used as an external poultice. The herb is easy to grow, and a poultice can be made simply by crushing the leaves slightly and bandaging them onto the injured area. Some horses have sensitive skin and should have the poultice applied for only part of the day.

Internally administered dried herbs or liquid extracts of devil's claw, meadowsweet, white willow bark, and yucca are known for anti-inflammatory action. In general, herbs such as these take several days of feeding or longer for the effects to be seen, so they are usually more effective when given to horses with chronic rather than acute problems. Products containing herbs with anti-inflammatory effects can be used in acute situations; however, homeopathic remedies may be more effective.

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CHAPTER • 97

Physical Therapy and Rehabilitation

Michael A. Ball

Physical therapy and rehabilitation techniques have been well defined in human medicine for many decades and play an important role in recovery from a variety of injuries. Programs use trained therapists and an array of therapeutic equipment to help persons regain strength, stamina, balance, and coordination, and to assist healing of specific injuries while attempting to minimize the risk of re-injury. The field of physical therapy and rehabilitation focusing on horses has grown tremendously in the past 10 years, but it is still in the embryonic stages of development. Much is still to be learned about the best ways to rehabilitate various injuries in the horse and how to evaluate and treat the variety of compensatory problems that can develop. I believe this area will continue to evolve, with the growth of specialty centers providing physical therapy and rehabilitation services for horses, working with clinicians.

Equine physical therapy and rehabilitation techniques have been reviewed recently.[1] In Canada, the United Kingdom, and

the management of lameness. Homeopathic remedies can be used with any conventional treatments, but faster results are achieved by using the remedies alone, together with supportive care such as cold hosing, bandages, or controlled exercise. The principles of homeopathy are discussed elsewhere.[3]

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Equine physical therapy and rehabilitation techniques have been reviewed recently.[1] In Canada, the United Kingdom, and

several other countries some physical therapists are certified, having completed an extensive training protocol, and work collaboratively with veterinarians. No training or certification process is available in the United States, and worldwide many completely unqualified persons practice under a number of titles. An accurate diagnosis of lameness is essential before any physical therapy begins, because inappropriate treatment could lead to serious complications.

MASSAGE

Massage may be beneficial in treating a variety of muscle problems to promote relaxation and improve venous return[1] (see Chapter 96). I have used massage for horses with focal muscle spasms or knots, particularly on the longissimus muscles of the back, where tightness and spasm is often secondary to poor saddle fit or padding. The primary problem also must be identified and treated. I have found light massage therapy to be helpful in horses in the later stages of convalescence from an episode of exertional rhabdomyolysis when the serum creatine kinase concentrations are nearly normal. In addition to hand massage a number of massage aids are available, such as handheld units with knob-like protrusions and electric vibrating devices.

MUSCLE STIMULATION

Selective muscle groups can be stimulated to work using an electric muscle stimulator or transcutaneous electrical nerve stimulation (TENS). A variety of devices are available that trigger the contraction of a muscle body through the application of an interrupted direct current across the skin, in the vicinity of a major motor nerve. Some units have handheld electrodes, whereas others have connectors for contacts on stick-on electrodes or attachment to acupuncture needles. Intensity and duration of the current can be controlled. The current is typically pulsed at a rate of 15 to 30 contractions per minute, allowing for complete relaxation and cycling of the muscle group before the next contraction. The current jumps into the nearby motor nerve, causing its depolarization and subsequent contraction of the muscle. I have found this modality to be particularly useful in horses recovering from neurogenic atrophy. TENS provides some analgesia, and care must be taken to avoid over-exercising an injury when pain has been masked.[1]

CONTROLLED EXERCISE

Advanced versions of the traditional hot walker enclose a horse in a system of moving partitions or gates, allowing for forced or controlled exercise. The horse's head is free, allowing for a natural gait. The speed can be controlled accurately from a walk to a fast trot (or faster). If weakness in a limb is apparent, the muscles of the affected limb can be loaded by adding extra weight (2 kg) to the limb by using a weighted boot or applying a heavier shoe.[1] By selectively loading muscle groups, an isolated increase in work can increase strength and power development relative to the non-loaded muscle groups. This technique also can be used with the horse exercising under saddle or in long lines, but care should be taken not to over-work the horse. The clinician should ensure that the injury is at the appropriate stage of healing for exercise to be beneficial and should enter this stage of rehabilitation gradually and with careful monitoring.

Treadmill exercise can be useful, but it does not induce a natural gait because of the effects of the moving traction belt under the horse's feet. The treadmill forces the hindlimbs into protraction farther behind the horse and the forelimbs into retraction farther underneath the horse, leading to an imbalance of working muscle groups. Treadmill exercise quickly builds loin muscle, but because of the protracting action of the forelimbs the horse tends to over-develop muscles over the shoulder. Many horses with chronic lameness develop an asymmetrical or unbalanced gait during compensation for the lameness and often retain some degree of abnormal gait (a memory effect) after the lameness has been resolved. The straight-moving traction belt of a treadmill makes maintaining an asymmetrical stride length difficult for a horse, thus forcing it back into balance. Forced exercise on a treadmill for about 5 minutes daily develops a normal pre-injury gait pattern after 5 days.[1]

SWIMMING

Swimming is anaerobic exercise and helps to retain cardiovascular fitness. Heart rate increases up to 200 beats/min in 30 seconds.[1] The horse is not a natural swimmer and often cannot breath efficiently when forced to swim. If an individual horse does not take readily to swimming and resists the process, the horse may do itself more harm than good. Some horses develop back soreness during swimming, so the exercise should be used with care.

A treadmill in water reduces load on the muscles because of buoyancy of the water and may decrease the gait alterations observed with a treadmill alone, allowing for a better balance of muscle group working while maintaining a forced symmetrical gait. Moving against water increases the work while reducing the excessive lordosis or kyphosis of swimming, which predisposes the horse to back pain, and increasing the cardiovascular benefits at low speeds.

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CHAPTER • 98

Osteopathic Treatment of the Axial Skeleton of the Horse

Chris Colles and Anthony G. Pusey

Osteopathy originated in the United States with a Kansas physician, Andrew Taylor Still. He envisaged a system of healing that placed emphasis on the structural integrity of the body as being vital to the well-being of the organism. As knowledge of physiology and anatomy have advanced, a concept has evolved of the nervous system as an information network constantly changing and adapting in response to sensory information from the body and the environment. This knowledge allows a move away from the ideas of pathological dysfunction as the only cause of illness to a concept of physiological (or somatic) dysfunction.[1] To use a computer analogy, the body may suffer from software failures (physiological malfunction) and hardware failure (pathological malfunction). To extend the analogy, osteopathy may be thought of as a form of reprogramming.

Chiropractic treatment evolved from osteopathy and is similar, but subtly different. The differences are largely philosophical; both forms of treatment rely on manipulation. Chiropractors in animal practice tend to look primarily for positional changes associated with skeletal dysfunction. Treatment is directed principally at restoring joint mobility, with manipulation directed to the local site of dysfunction.

The osteopathic approach considers local tissue dysfunction as the direct result of trauma or the breakdown of compensatory mechanisms consequent to past trauma. The diagnostic approach is based on identifying patterns of dysfunction, with minimal reliance on appositional factors and emphasis on the interactions of the entire spine. Treatment is aimed not only at restoring local function, but also at identifying and removing factors that predispose to acute relapse. Treatment therefore frequently involves tissues distant to the perceived area of local dysfunction.

NEUROPHYSIOLOGICAL BASIS OF OSTEOPATHY

The concept of somatic dysfunction is fundamental to osteopathy. In other words, somewhere in the course of entering information from the environment and body, processing the information in the central nervous system, and then generating a motor response, something has gone wrong. Clinically this manifests as a horse with clinical signs such as stiffness, loss of performance, poor coordination, or gait abnormality, where no pathological process can be identified. Osteopathic treatment is directed at changing the signals to the neural network to modify the way sensory information is processed and thus to correct the motor response generated in the central nervous system.

In the normal animal the body constantly makes small adjustments to movement to interact with the environment. The basic motor pattern is one of mutual inhibition of flexor and extensor neurons,[2] providing a balance between agonist and antagonist muscles. To refine the movements, proprioceptive information is sent constantly from muscle spindles and Golgi tendon organs back to the spinal cord. As the nerves enter the spinal cord, they send off sprays of ascending and descending collateral fibers, which synapse on interneurons with connections within the spinal cord segment, with other spinal segments up and down the cord, and with tract cells up to the brainstem. The result is a network of neurons processing information from the body and environment that generate flexible patterns of motor activity (sometimes referred to as *central pattern generators*). Thus a pattern of nerve activity is generated where the primary function is to move a limb, but simultaneously vital secondary functions are engendered—for example, changing the tone in the central musculature to counteract the changing load on the axial skeleton. With this skeletal activity, autonomic changes such as alterations in blood flow are incorporated to provide the necessary humeral environment. The number and variety of interconnections, determined by cellular and synaptic connections of the neurons, together with the balance of excitation and inhibition from many different tissues, allow the central pattern generators to generate many different routine motor patterns. These patterns develop over time by a process of repetition and learning. Thus the inconsistent swing of the learner golfer is trained to become a confident, automatic swing after many hours of repetitive practice. This involves a process of chemical changes, new synaptic connections and fast tracking, resulting in a consistent and reproducible pattern of motor activity.

The ability to generate rapid, appropriate patterns of motor responses to environmental demands depends on sensory information. This information comes from an appreciation of the position of the body (proprioception) and conditions prevailing in the outside world (exteroception). Both inputs significantly modify the central pattern generators.

The greatest concentration of proprioceptors is found in the upper cervical spine,[3] a vulnerable area when a horse falls. Joint stiffness and muscle pain in this area after an injury alter proprioceptive feedback from this level. This affects the ability of motor pattern generators throughout the spinal cord to modify activity in response to alterations in body position and may produce changes ranging from subtle gait disorders to ataxia. Clinically this may not result in obvious clinical signs, but it does affect the horse's potential and leaves the horse more susceptible to acute injury.

Environmental information is provided by exteroceptors that continually sample external conditions by precise localizing receptors of the A-afferent system (e.g., touch receptors) or by the more primitive, poorly localized pain receptors (nociceptors) of the B-afferent system. In a clinical setting, the effects of the B-afferent system are commonly encountered. A painful stimulus is conveyed to the spinal cord and passes on to the brain to register pain and also stimulates motor neurons in the ventral horn, resulting in paravertebral and peripheral muscle spasm,[4] and stimulates sympathetic activity in the lateral horn causing reduced blood flow to the skin.[5] To avoid constant discomfort, a control mechanism exists at the level of the spinal cord that is mediated by the A-afferent input of

discriminatory touch and proprioception and by descending inhibitory activity (creating a gate for painful stimuli). This can be demonstrated by rubbing an injured area to ease pain. The balance between A-afferent stimulation and B-afferent input determines the activity of the interneurons and the sensory, motor, and autonomic responses.

This balance can be affected in a number of ways:

1. A-afferent activity from joint receptors is reduced if movement is restricted. Thus inhibitory effects on the interneurons are reduced, and B-afferent activity may even increase and register as pain, without direct stimulation of pain receptors. Physical treatments to increase mobility restore the A-afferent input and reinstate the gating effect.
2. B-afferent activity, if intense or prolonged, causes changes in the chemistry and structure of the interneurons,[6] which can be traced 3 to 7 days after only 45 minutes of moderate nociceptive stimulation.[7]

These changes reduce the threshold for interneuron firing in a process called *facilitation*. They become supersensitive to afferent input and to internal network activity within the spinal cord. This mechanism underlies hyperesthesia. Facilitation is interesting for two reasons. First, the peripheral endings of sensory nerves feeding into the sensitized interneuron pools produce pro-inflammatory neuropeptides,[8] which further stimulate the nerve endings. This increases central stimulation, thus creating a spinal cord response out of proportion to the peripheral stimulation. This may explain horses with recurrent joint and soft tissue swellings. They may respond to corticosteroid injections that reduce inflammation at the local site, but if sensitization of the central interneuron pool remains, the inflammation returns.

Second, even when afferent activity has been blocked by sectioning the dorsal (sensory) root, a sensation of pain and the autonomic and motor responses may still be present, driven by the output of the sensitized interneurons. This means that pain may persist long after the original injury has resolved. Even when pain is no longer felt, the neuronal pool remains sensitive, and nociceptive inputs produce a pain response inappropriate to the magnitude of the stimulus. The interneurons respond to inputs on a segmental level, but also to input from distant segments, given the span of nociceptive fibers over several segments.

These mechanisms may explain some of the non-specific lameness and back pain that tend to be recurrent. A diagnosis based on pathological condition of tissue (demonstrated by x-ray, scintigraphic examinations, or nerve blocking) is not possible, because the abnormality results from physiological changes in the motor patterns generated by the facilitated neuronal segment. Gait disturbance may be observed and muscle spasm, joint stiffness, and tissue texture changes may be appreciated by palpation and passive motion testing. One diagnostic tool that does reflect this concept of somatic dysfunction is thermography. In the normal horse a reproducible thermographic pattern is observed, reflecting cutaneous blood flow (see Chapter 25).[9] Where facilitated segments exist, sympathetic activity in the lateral horn reduces surface blood flow, giving distinctive segmental cooling. In addition to local segmental changes, the pattern may be disrupted throughout the length of the spine, giving a visual record of changes in autonomic nerve output.[10]

EFFECTS OF OSTEOPATHIC TREATMENT

Osteopathic treatment aims to reverse changes in the areas of somatic dysfunction. Most techniques are directed toward restoring movement to stiff joints to optimize maintenance of postural position. This causes relaxation of the local musculature and improved mobility, which is associated with a resultant increase of mechanoreceptor activity stimulating the A-afferent system, thereby inhibiting the interneuron pool and blocking the B-afferent (pain) signals. The treatment may take a number of forms, including soft tissue techniques, articulatory techniques, and mobilization and functional techniques.

Soft tissue and articulatory techniques stretch the skin, fascia, and muscles to improve pliability of and nutrition to the peri-articular tissues and also allow the joints to move in a full range. This increased movement increases A-afferent input from the muscles and joint structures, inhibiting (or gating) incoming pain signals. These techniques include massage and repetitive flexion and extension of restricted joints.

Mobilization techniques involve taking a joint with poor mobility to the point of maximum resistance (the restrictive barrier) and pushing through this barrier with a short-amplitude, high-velocity thrust. This causes relaxation of the muscles and improved mobility resulting from a sudden increase of mechanoreceptor activity stimulating the A-afferent system, thereby inhibiting the interneuron pool and blocking the B-afferent (pain) signals.

A useful approach in horses with long-standing, complex injuries is the use of functional techniques, using the concepts of ease and bind. A normal joint reaches a point (usually at the middle of its range of movement) where tension on the capsular ligaments and the overlying muscles is minimal. This is the point of ease, where a joint tends to rest naturally. Any movement away from the point increases tension or bind. This information is used by the central nervous system to monitor joint position and to generate an appropriate pattern of motor activity. Where the normal relationships between the joint structures have been disturbed, this point of ease is offset and afferent information from that joint is changed. New reference points become imposed on the established networks and the joint is less able to perform appropriately or to coordinate movement with other joints.

This new abnormal point may be detected clinically by testing each range of movement (flexion/extension, side bending, rotation, translocation, and traction/compression). With the joint held in the position of ease, tension is minimal and therefore afferent input into the spinal cord is minimal. This appears to reduce conflicting information entering the network and allows the old pattern to reassert itself. The old pattern is preferred by the system, because over time neuronal connections have been created that fire more readily to generate this original learned response. This form of treatment relies on the osteopath establishing and maintaining one or, more commonly, several joints at the point of ease, until the original central pattern generators can reassert influence over the newly acquired patterns. This is a learned skill on the part of the osteopath and requires considerable tactile sensitivity and skill to adjust the tissues as the neurological patterns are modified. Among injured horses is a clinically challenging subgroup of 4- to 6-year-old horses that develop clinical signs as work becomes more demanding, rather than as a response to direct trauma. In these horses, it is tempting to conjecture that some form of perinatal injury may have occurred, which has prevented the normal patterns from developing. These horses frequently undergo more treatment and require more careful rehabilitation than other horses.

Details of osteopathic treatment techniques are beyond the scope of this chapter, but several excellent textbooks[9] are available on the subject.

DIAGNOSIS OF SUITABLE CASES FOR OSTEOPATHIC TREATMENT

Diagnosis should involve positive selection of horses with a somatic dysfunction and exclusion of horses with a pathological condition. Initially a conventional veterinary lameness

examination should be performed. If the examination indicates a change in quality of movement rather than lameness, or that lameness is centered in the axial skeleton and no pathological process can be demonstrated readily (e.g., by x-ray or scintigraphic evaluation), then the likelihood of a somatic dysfunction as the cause of lameness should be considered. Somatic dysfunction alters the way a horse moves and so may contribute to pathological change (e.g., synovitis in an overloaded joint). Once a primary pathological condition has been excluded, the process of making an osteopathic diagnosis can proceed. This diagnosis is based on somatic dysfunction, that is, how the component parts of the horse work together as a whole and how different areas of dysfunction or stiffness may contribute toward the problem.

An accurate case history is essential and should include the type of work the horse is expected to do and the level it is expected to attain. The history also highlights past injuries and illnesses and may reveal idiosyncrasies in behavior or movement that, while noticed by the owner, have not interfered with performance sufficiently to be a cause for concern. When considering the history, these subtle disturbances in motor function may, in retrospect, have been early signs of the coming crisis.

Examination begins by assessing muscle development and distribution of weight through all limbs in the standing horse. The horse should stand naturally with all cannon bones vertical and not shift weight constantly between limbs. Muscle development should be symmetrical, and the areas of muscle should be consistently developed throughout the horse. Areas of poor development over the top of the back, neck, or quarters should be noted. Similarly, uneven development or hypertrophy may be substantial. In particular, hypertrophy of the pectoral muscles may indicate forward propulsion of the horse unduly depends on the forelimbs because of poor hindlimb function. Hypertrophy of the muscle over the atlas and axis frequently accompanies restricted joint movement at this level. Foot placement, shape, and shoe wear are also important for locating areas of dysfunction. When assessing feet, however, it is necessary to differentiate between change caused by altered limb movement and problems caused by poor farriery.

Movement at a walk is more revealing than at the trot, when the horse tends to fix the neck and back to guard against pain. Observation gives an impression of how the horse moves as a whole and indicates which areas are functioning poorly. Balance and fluidity of movement from head to tail are as important as stride length, foot placement, and frank lameness. How the horse copes with long and short turns indicates problems with lateral flexibility. In particular, when the horse is turned short, the head and neck should flex evenly in the direction of the turn and the hindlimbs should be crossed as the quarters move around. All limbs should be moved with easy flowing strides, showing consistent stride length and straight limb flight. The feet should be placed easily, and in particular the hind feet should not dig into the ground when placed. Limb movement that appears heavy, stiff, inconsistent, or mechanical should alert the observer to potential problems. Excessive head and neck movement in time with the limbs is also abnormal. Substantial lateral movement of the pelvis, often associated with patches of worn hair under the back of the saddle, indicates reduced movement in the lumbosacral spine. The pelvis and tail being carried to one side also indicates uneven tone in the spinal musculature. Uneven height between the two tubera coxae also should alert the clinician to potential problems.

The transition from walk to trot can be revealing, and any tendency to raise the head or skip in the transition indicates problems, probably in the region of the cervicothoracic junction. Assessing individual joint movements is useful, but it requires considerable skill and powers of observation. Running the hands down the paravertebral muscles from the occiput to the tail may reveal areas of muscle hypertrophy or spasm, and most importantly tissue texture can be felt. Moving the joints through the relevant ranges of motion identifies those that are stiff.

Diagnosis requires consideration of past injuries, the way the horse moves overall, and identification of dysfunction in specific areas. Initially, the clinician may only be able to form a working hypothesis, which will be refined once treatment is begun. Where problems have been present for some time, the horse will have developed altered patterns of posture and movement to work round poorly functioning areas. These alterations may occur at several levels because of the interconnections of the neural networks. Ascertaining what is the site of the primary injury and what are secondary changes may be difficult.

INFRARED THERMOGRAPHIC IMAGING

Thermographic examination in the horse has proved to be an invaluable tool to confirm somatic dysfunction.[11] It is important to realize that areas of cooling are more important in diagnosing sympathetic dystonia than are increased temperatures. Thermography is prone to artifactual results and interpretation can be reliable only if image acquisition is carried out in a temperature-controlled environment (between 18° and 22° C), using an absolutely consistent technique.

The horse must be clean and dry, because moisture absorbs infrared radiation and causes false readings. Hair length should be consistent, because hair has an insulating effect, and horses that have clipped coats or rubs from rugs present difficulties in interpretation. Different coat colors may be of different hair length or density, which causes artifacts. When moulting, areas of different colored hair may moult at different times, again giving rise to artifactual results. Before thermography, horses should be acclimatized to the room temperature for as long as it takes for the surface temperature to remain stable. The time required depends on the difference in ambient temperature of the environments inside and outside the thermography room. The room should be free of drafts and direct sunlight.

In a normal horse a reproducible thermographic pattern is observed, reflecting cutaneous blood flow.[10] Where facilitated segments exist, sympathetic activity in the lateral horn reduces surface blood flow, giving distinctive segmental cooling. In addition to local changes, the pattern may be disrupted throughout the length of the spine, giving a visual record of changes in autonomic nerve output. Certain specific injuries do give areas of increased blood flow and rise in surface temperature. These injuries are usually around the head, or in the lower limbs, where little or no overlying muscle is present.

In the normal horse the surface temperature should be within 1° C over the neck and body of the horse and in the limbs proximal to the carpus and tarsus. The head and lower limbs are generally cooler. A useful guide when setting the thermographic factors is to assume the eye will be the hottest normal area and to set up the camera with this as the maximum temperature. Changes of 5° to 6° C are severe, so a temperature range of about 6° C is ideal. If the horse has parts of the coat clipped, then changes in the level of the window may be required to accommodate the clipped areas of the body. Particularly important is the dorsal view of the back, where a relatively warm dorsal midline stripe is seen, with the muscle on either side symmetrical and between 0.5° to 1° C cooler. When interpreting thermographs of the neck and back, changes of less than 1° C from the normal may be discounted. Changes in excess of 1° C usually indicate sympathetic dystonia, the importance of which must be interpreted in the light of clinical information regarding clinical signs seen and the use to which the horse is put. It is likely that horses frequently suffer low-grade

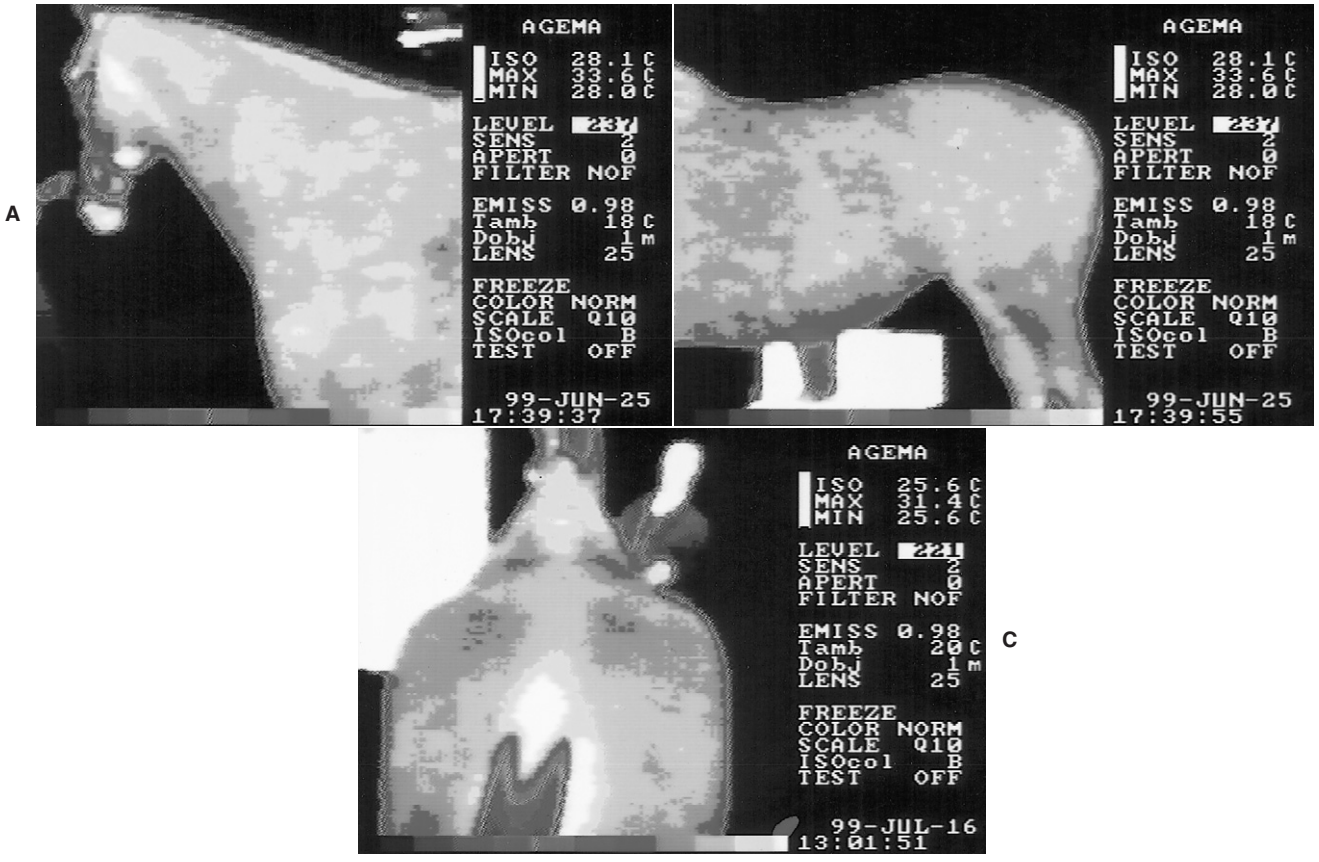

Fig. 98-1 Thermographs show the appearance of normal horses. The color scale represents 5.6° C, using 10 colors; that is, each color represents about a 0.5° C change in temperature. The color bar under the image shows the colors used; those to the right-hand side are the hottest and those to the left are the coldest. White is above the top of the scale and black below the scale. **A,** Lateral view of the head, neck, and shoulder. **B,** Lateral view of the thorax, abdomen, and hindquarters. **C,** Oblique dorsal view from behind the horse looking toward its head and neck. Temperature variation is 1° C over the neck, trunk, and hindquarters. A warm midline dorsal stripe along the back extends from the withers to the base of the tail, with symmetrical muscle temperature on either side.

back pain or stiffness, which may not be of particular significance if they live relatively sedentary or gymnastically undemanding existences (e.g., a racehorse or hunter performs satisfactorily with minor changes in the upper neck or thoracolumbar spine, which would cause unacceptable stiffness or gait modification in an event or dressage horse).

Fig. 98-1, *A* to C (Color Plate 5), show the thermographic appearance of a completely normal horse. One should bear in mind that few horses meet this degree of perfection, but they may still perform adequately at the required use. The most common area of the spine to show joint stiffness is the upper neck, with changes to the occipito-atlantal and atlanto-axial joints. This group of injuries results in a cold band running obliquely down the neck from about the level of the occiput (Fig. 98-2; Color Plate 6). This band becomes narrower with time, and although a substantial temperature change is visible in the acute stage, over an initial period (probably about 2 years) the temperature change becomes more noticeable. Stiffness or injury farther down the neck is accompanied by cooling of the surface temperature overlying the affected area (Fig. 98-3; Color Plate 7). Because the nerve supply to the muscles of the shoulder arises from the lower neck, injuries to the spine in between the shoulders are difficult to see thermographically, but a cold band running vertically down the back of the shoulder region (Fig. 98-4, *A*; Color Plate 8, *A*), usually accompanied by cooling of the entire thoracic area and hindquarters, is an indicator of injury to the cervico-thoracic area. Cooling of the hindquarters arising caudal to a diagonal band from the level of about the twelfth thoracic vertebra generally indicates injury at this level of the thoracic spine (Fig. 98-4, *B*; Color Plate 8, *B*). Two other changes are of particular clinical significance. Any break in the normal dorsal stripe running down the midline of the back, or significant cooling on either side of this stripe, indicates a change in sympathetic activity in that region (Fig. 98-5; Color Plate 9). Second, cooling of the distal limb may be substantial. If the ambient temperature drops below about 15° C, normal physiological changes reduce blood flow in the distal limb to conserve body temperature. This causes cooling of the distal limb (distal to the carpus or tarsus, unilaterally or bilaterally), but such limbs should still show a surface temperature 5° to 6° C cooler than the trunk temperature. If severe injury has occurred at the base of the neck, or in the lumbar region, then the temperature of the distal limb may drop to equal the ambient temperature. More detailed analysis of the thermographic changes seen is beyond the scope of this chapter.

TREATMENT

Because of the vast number of interneural connections and the ramification of effects on the spinal cord, the entire spine must be considered as one interrelated structure, and treatment may have to be provided at several levels. Sedating the

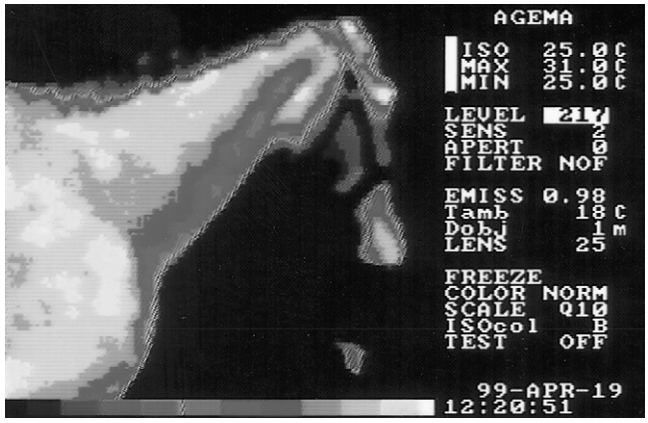

Fig. 98-2 Lateral view of the neck of horse with reduced mobility in the occipito-atlantal and atlanto-axial joints. A cool line runs obliquely from the region of the atlanto-axial joint caudally to the base of the neck and is 1.5° C cooler than the surrounding muscle, indicating an area of sympathetic dystonia.

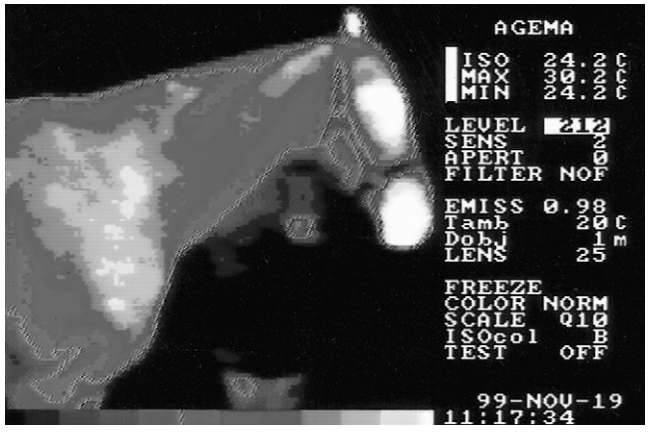

Fig. 98-3 Lateral view of the neck of horse with reduced mobility in the neck. Note the significant cooling from the occiput back to the level of the sixth and seventh cervical vertebrae, indicating a problem involving all joints in the neck.

horse is useful, because treatment requires identification of specific areas of stiffness or reduced movement. If the horse is sedated adequately, then the absence of conscious movement allows the osteopath to feel underlying deficiencies more easily. Sedation is particularly helpful when using functional techniques, which monitor subtle changes in joint position. Osteopaths in the United Kingdom undergo a 4-year university course, which includes considerable theory and practical training. The techniques used require considerable practical experience to be successful, so this chapter can only begin to explain the outlines of treatment.

After the osteopath performs the static and moving examinations described earlier, he or she starts a physical examination, feeling the tone and physical character of the muscles and the extent of movement of the joints, usually starting at the head and working back down the neck and to the tail. It is necessary to try to determine which joints show restricted movement and to decide which areas show abnormalities caused by primary injury and which changes are secondary to the principal affected areas. Experience allows the osteopath to determine which area must be treated first. The areas showing somatic dysfunction tend to be interrelated and must be treated in a specific order if treatment is to be successful. Starting treatment on an area showing restricted movement secondary to another abnormal area is doomed to failure.

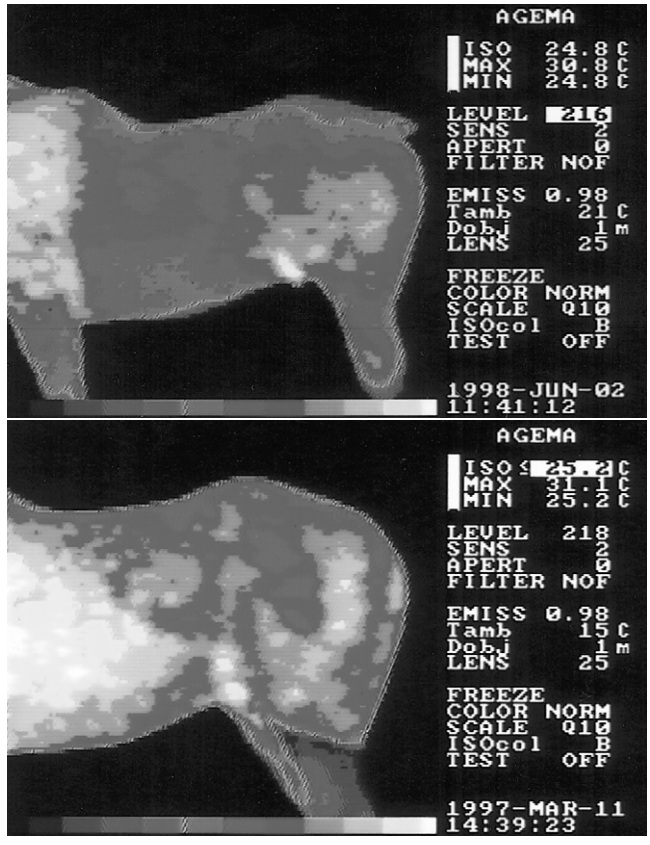

Fig. 98-4 Lateral views of the thoracolumbar area of two horses, both showing significant cooling in the musculature of the dorsal spine from the saddle region caudally. **A,** The vertical cranial boundary to the zone of cooling indicates that the injured area is in the region of the cervicothoracic junction, but the shoulder muscles overlying this area receive innervation from the lower neck, partly masking the muscles supplied by the upper thoracic area. **B,** The typical appearance of cooling in the muscle resulting from an injury to the region of the twelfth thoracic vertebra, with the cranial border of the region running obliquely down and back. This horse has been clipped, but long hair left on the hind limb shows as an area 5° C cooler than the surface temperature of the thorax of the horse.

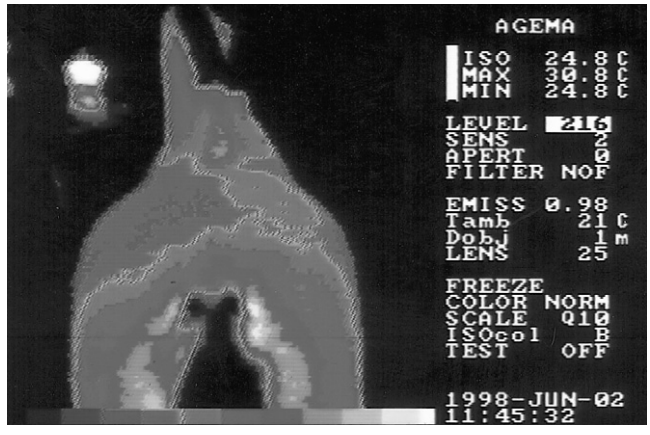

Fig. 98-5 Dorsal view of horse showing abnormal heat patterns of the thoracolumbar spine. Note the complete loss of the normal central stripe, with significant cooling indicating the presence of reduced mobility of the entire thoracolumbar spine and pelvis. The asymmetrical nature of the heat pattern indicates the horse is likely to move with an asymmetrical gait, resulting from increased muscle tone on the left-hand side of the body.

Treatment of a key area, however, often results in dramatic relaxation throughout the spine. The basic techniques of treatment have already been outlined and rely on stimulating the activity of the A-afferent system, thereby inhibiting the interneuron pool and blocking the B-afferent (pain) signals. The results are effective in most horses, and once the neural activity has been returned to normal, the horse tends to maintain this state.

In a small number of horses the problem may be so ingrained and widespread that general anesthesia may be needed to facilitate assessment and treatment. The plane of anesthesia is light, the horses are in good health, and so the risks of the procedure are minimal. Assessment of individual joint complexes can be made throughout the spine with the horse under general anesthesia. It is remarkable how often gross dysfunction of these complexes can be detected with the horse under general anesthesia, when they previously had been concealed effectively by the capacity of the body for setting up compensatory mechanisms. Treatment is still based on the principles outlined so far, but the horse is positioned on its back, allowing movements such as rotation of the thoracolumbar spine, which cannot be achieved in a standing horse. Using all limbs simultaneously is also possible, allowing functional techniques (outlined previously) to produce a massive A-afferent input to the interneuron pool. This is done by moving each limb to its position of ease, and then holding the position until relaxation is felt. By moving the limbs to maintain the positions of ease, the sympathetic dystonia can be unwound gradually. This technique, used with the horse under general anesthesia, allows a potent but gentle treatment to be carried out. In a standing, conscious horse techniques are necessarily limited and are most effective with relatively short-term injuries.

RESULTS OF TREATMENT

Pusey et al.[12] reported on a trial carried out over four years, treating horses with chronic dysfunction using osteopathic techniques. Eighty percent of treated horses returned to the previous use, performing at the same or a better level than before treatment, at least 12 months after the last treatment. As might be expected, the authors concluded that results were substantially better among the later horses because of increasing experience of treatment techniques.

Another study was made of horses that had been treated under general anesthesia, having failed to respond satisfactorily to treatment under sedation. Follow-up information was obtained at least 1 year after the treatment was finished. Thirty-five horses were treated. Twenty-four (71%) of these had maintained improvement and returned to the previous level of work, 11 at the previous level and 13 at a higher level than previously. Eight (24%) horses did not respond to treatment, and two horses deteriorated. One horse was lost to follow-up. The authors concluded that given that these horses had long-term intractable problems, this was a justifiable and useful treatment technique.

We hope this brief overview shows the potential for osteopathy in a complementary role, alongside veterinary science, in treating neuromusculoskeletal problems in the horse.

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CHAPTER • 99

Extracorporeal Shock Wave Therapy

Darryl Bonder and Joseph K. Boening

Extracorporeal shock wave treatment developed from lithotripsy, a technique in which shock waves are used to treat renal and ureteral stones. Extracorporeal shock wave treatment has been used to manage selected orthopedic conditions in people for some years and more recently has been used in horses. Extracorporeal shock waves are acoustic waves generated outside the body, characterized by transient high peak pressures, followed by negative pressure and then return to zero pressure. Peak pressures achieved by currently available equipment range from 10 to 100 MPa, with a rapid rise time (30 to 120 ns) and short pulse duration (5 µs).[1,2] Other variables include energy level, defined as energy flow density (mJ/mm^2) or as kilovoltage, pulse frequency, and depth of penetration.

Shock waves can be defined as focused or radial. Focused shock waves can be generated electrohydraulically, piezoelectrically, or electromagnetically. Radial shock waves are generated pneumatically. Each mechanism creates a characteristic waveform and energy density. Focused shock waves converge on a small target point and do not expose surrounding tissues to the same peak pressures, and the target point is guided by ultrasonography or radiography. Radial shock waves have lower peak pressures and reduced depth of penetration and expose all tissues to similar pressures.

When a shock wave meets an interface of different impedance, compression and shear loads develop.[1] Cavitation occurs, with development of gas bubbles, resulting from the rapid interaction between compression and shear. The collapse of the gas bubbles leads to development of fast flows, or jet streams, that are postulated to have an effect on bone. However, the exact mechanisms of action of shock waves on bone or soft tissues are not known and the effects are influenced by the pressure, energy flow, and pulse frequency. The optimal combinations of energy level, pulse frequency, and number of pulses, and the number of treatments and interval between treatments to exert therapeutic effects are currently unknown. Work in laboratory animals has demonstrated dose-dependent effects, including microfracture of cortical bone, medullary hemorrhage, subperiosteal hemorrhage, and stimulation of osteogenesis.[3-6] Relatively low energy levels may have no osteogenic effect. Dose-dependent effects also have been demonstrated in tendons, with high pressures (>0.28 mJ/mm^2) having the potential to cause hematoma formation and tendon cell damage.[7] Shock wave treatment may increase regional blood flow, have direct cellular effects, and activate osteogenic factors.[4] That shock waves can provide analgesia is well recognized, but the duration of this effect and the mechanism of action currently are defined poorly. Analgesic properties are of concern to racing officials, because horses with fractures or other injuries might suffer severe injury if treated before racing.

Extracorporeal shock wave treatment is used in people to treat plantar fasciitis or calcaneal spurs (heel spurs), epicondylopathic humeri radialis (tennis elbow), tendonitis calcarea of the shoulder, and selected non-union fractures.[8,9] Clinical trials using focused and radial shock wave treatment for tennis elbow and heel spur have shown similar results, with improvement in clinical signs in approximately 80% of patients.

Limited experimental work has been done in the horse and much of the currently available information concerning efficacy of extracorporeal shock wave treatment is anecdotal. In an experimental study, subperiosteal (cortical) and endosteal hemorrhage was induced in third metacarpal and metatarsal bones treated using 1000 pulses from an electrohydraulic generator at an energy level of 0.89 to 1.80 mJ/mm^2.[10] No observable lesions occurred in the overlying soft tissues and no evidence of bone microfracture was found. Comparing the results of currently published clinical trials using extracorporeal shock wave treatment is difficult, because diagnostic criteria, duration, severity of injury, and degree of lameness vary. Treatment protocols are not identical, with variable peak pressures, energy levels, pulse frequency, depth of penetration, and number of treatments. Some trials have used focused shock waves, whereas others have used radial. Focused shock wave machines are considerably more expensive than radial shock wave machines, but the latter are more mobile. Greater depth of penetration, variable energy levels, and the ability to treat the desired tissue specifically are potential advantages of the focused machines.

Numerous conditions have been treated in the horse, including the bucked-shin complex, tibial stress fractures, proximal sesamoid bone fractures, incomplete proximal phalangeal fractures, subchondral bone pain, insertional desmopathies (notably proximal suspensory desmitis, suspensory branch insertions, and avulsion fractures at the proximal attachment of the suspensory ligament), impinging dorsal spinous processes, osteoarthritis of the distal hock joints, navicular disease, and superficial digital flexor tendonitis.

The treatment itself is painful and requires sedation of the horse or, for some procedures, perineural analgesia or general anesthesia.[11] Detomidine or a combination of detomidine and butorphanol is usually effective. The area to be treated is finely clipped and thoroughly cleaned, and coupling gel is applied to ensure good transmission of the energy waves. Usually up to 2000 impulses are delivered to the target area at a frequency of about 10 per second. Energy levels may be increased during treatment. During treatment a local analgesic effect may be induced. Horses usually tolerate the procedure well. One to three treatments are administered at 1- to 2-week intervals. Horses are restricted to box rest and controlled exercise between treatments. Precise exercise levels and posttreatment convalescent time is guided by the nature of the original injury and serial clinical and radiographic or ultrasonographic re-examinations.

Generally no adverse effects from treatment occur. Small areas of hair loss may develop at the treatment site. Because of the analgesic effect, the potential exists for premature resumption of work, so control of the exercise regimen is important. Rough guidelines for return to work after treatment of a bone injury (D.B.) include 1 to 2 weeks of walking under tack for 20 minutes daily; 1 to 2 weeks of walking under

tack for 10 minutes, jogging for 10 minutes (1 mile), and walking for 10 minutes; 1 to 2 weeks of jogging under tack for 15 minutes (1.5 miles) plus walking; and gradual resumption of full work. A typical program following proximal suspensory desmitis (PSD) includes 6 weeks of hand walking for 20 minutes; 6 weeks of walking under tack for 20 minutes; 2 weeks of walking under tack for 15 minutes and 5 minutes of trotting; and 2 weeks with increased walking and trotting.

The most widespread use of extracorporeal shock wave treatment has been for PSD. Data from clinical trials in Germany using radial and focused shock wave treatments showed similar results.[12,13] Using a focused machine with energy flow density of 0.07 to 0.17 mJ/mm^2 and 2000 impulses, 83% of horses with forelimb or hindlimb PSD were improved 6 months after treatment. Radial shock wave treatment with maximum energy flow density of 0.16 mJ/mm^2 resulted in 71% of treated horses in full work 6 months after treatment, compared with only 50% of untreated control horses.[13] In a similar study in the United States, 71% of horses with chronic PSD were treated successfully.[14]

Further clinical trials with large numbers of horses with accurate diagnoses, well-documented lesions, and accurate follow-up data are required to validate the success of these methods of treatment for different clinical conditions, to compare success rates with other treatment modalities, and to determine optimum treatment parameters and exercise programs during convalescence. Further information is required about the usefulness of focused or radial shock wave treatment for specific conditions. Extracorporeal shock wave treatment seems to have a role in chronic pain management and may speed recovery in some injuries, permitting a more rapid return to full athletic function, and may stimulate repair in some chronic injuries that are refractory to conventional management.

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SECTION • 1
Poor Performance

CHAPTER • 100

Poor Performance and Lameness

Sue J. Dyson

Horses may be presented to a veterinarian because of a change in performance, or behavior or failure to live up to the expected level of performance, rather than because the rider or trainer has recognized overt lameness. Many of these horses do have musculoskeletal problems. The type of complaint often reflects the discipline in which the horse is used. For example, an event horse may be assessed because it has started stopping at drop fences, whereas a dressage horse may be evaluated because it takes uneven steps behind in passage or piaffe.

CHALLENGE OF ASSESSMENT

This type of horse presents the veterinarian with a diagnostic challenge, which may involve assessment not only of the horse, but also of the horse and rider together and the rider's ability. Such an assessment requires knowledge of the sport in question and an ability to recognize when the problem is a lack of athletic ability or a mental problem, rather than a pain-related musculoskeletal disorder, or some other pathological condition, such as an upper airway disorder. The assessment also requires good knowledge and appreciation of how horses normally move, the variation between different breeds and types, and an ability to recognize subtle changes in gait, such as slightly reduced lift to the stride or stiffness in movement of the back. The assessment requires basic knowledge of equine locomotion and how gait may be modified by pain and also requires the ability to recognize when a rider may actually be creating the problem. Training methods and style of riding may contribute to the development of lameness.

This section sets out to discuss some of the different problems that may be encountered and the types of conditions that may be the cause of poor performance and to suggest some methods of approach to diagnosis. These may vary to some extent depending on whether the clinician is dealing with a competent professional rider or trainer, or an inexperienced or even experienced amateur, who lacks ability.

HISTORY

It is essential to obtain a comprehensive history to determine whether the horse has genuinely performed better previously at this level of competition, or moved better, and precisely what changes have been observed. It is important to listen carefully to the owner to understand properly the perceived problem, however small it might be. This is particularly important if the clinician does not have prior knowledge of the horse.

Determine the answer to the following questions:

1. What is the age and training history of the horse?
2. Does the horse have the musculoskeletal coordination and strength to do what is being asked?
3. Has the horse recently moved up a level of competition: Does the horse have the confidence or athletic ability to cope?
4. What is the current work program? How does this vary from day to day? Is the horse allowed turnout?
5. Has a recent change in the work pattern or intensity or some other change in management occurred?
6. Has the bit been changed?
7. Did the horse sustain a fall or any other trauma before the onset of the problem?
8. Has a recent change in the rider or trainer occurred?
9. Has an alternative rider been tried?
10. Has the horse ever suffered exertional rhabdomyolysis?
11. Under what circumstances is the problem apparent?

Viewing videos of the horse when it was performing normally sometimes can be helpful to compare with the findings of your own clinical evaluation. If the horse has recently changed ownership, comparing video recordings of the horse ridden by the previous rider and by the current rider can be useful to determine whether a change in riding style or training techniques may be responsible for making a previously subclinical problem become symptomatic, or to determine if the problem could be related directly to the manner in which the horse is being worked.

The types of clinical problems that may be encountered include the following:

- Not jumping as well: knocking down rails. This may be pain related or caused by the rider presenting the horse poorly to the fence.
- Not jumping as well: stopping. This may be a horse or rider confidence problem or pain related.
- Not making distances in combination fences. This may be because the horse is landing too steeply, saving one or both front feet and therefore having too much ground to make up, or through lack of push from behind, reflecting a hindlimb lameness. Alternatively, if the horse is presented poorly to the first element of the fence, the subsequent elements become increasingly difficult.
- Reluctance to land leading on a specific leg. This can reflect a forelimb or less commonly a hindlimb lameness. Peak ground reaction forces at landing are significantly greater in the trailing forelimb than the leading forelimb[1]; therefore a tendency to land with the right forelimb leading is most likely to reflect pain in the right forelimb.
- Napping (resistance) on the approach to a fence off a turn. Nappiness may be pain related or behavioral.
- Not jumping straight (e.g., jumping to the right), with a tendency for the hindlimbs to drift toward the direction in which the horse is jumping (to the right). This usually reflects pain or weakness in the ipsilateral hindlimb of the side to which the horse is jumping. Less commonly the problem may be caused by reluctance to land on the contralateral forelimb (e.g., the left).

- Loss of hindlimb power. This may be caused by back pain or hindlimb lameness.
- Change in the shape the horse makes over a fence; for example, loss of bascule (jumping with a rounded arc over a fence). This may reflect back pain or forelimb or hindlimb lameness. It may also reflect the way in which the horse is presented to the fence.
- Rushing fences. This can be the way an excitable horse always jumps, but if the horse used to jump normally, rushing the fence usually reflects a painful problem. However, if the horse has been stopping for whatever reason and has been chased to the fences, it will inevitably rush.
- Loss of action. This can reflect the way in which the horse has been ridden and trained. Some loose, free-moving young dressage horses become much more restricted in their stride when ridden exercise is commenced. An apprehensive rider may restrict a potentially exuberant horse. A bored horse, particularly with a Warmblood mentality, may just switch off and refuse to go forward freely and loosely. Loss of action also may reflect forelimb or hindlimb lameness or back pain.
- Stiffness. Stiffness should be evaluated carefully to differentiate between loss of action and back stiffness, or restriction of gait caused by a bilateral forelimb or hindlimb lameness.
- Inability to perform medium or extended trot. Unless a horse is properly balanced with adequate hindlimb impulsion, it cannot perform medium or extended trot. Horses vary considerably in the ability to collect and extend. In general the Thoroughbred breed has much less natural ability than many of the Warmblood breeds. Some horses have to learn how to perform a medium and an extended trot and must first develop sufficient muscular coordination and power before they are able to do so. However, if a horse was previously able to work in medium and extended paces and now cannot do so, or the rhythm becomes irregular, this can reflect forelimb or hindlimb lameness or back pain.
- Inadequate hindlimb impulsion in trot, with a tendency to break to canter if asked to work harder. This usually reflects hindlimb lameness.
- Difficulty in performing specific dressage movements; for example, right half pass.
- Tendency to become disunited behind in canter. This usually reflects hindlimb lameness. However, it is important to recognize that young horses can find maintaining true canter difficult, sometimes just in one direction, or sometimes in both directions. This often can be overcome by training and development of muscular strength and coordination. Some trained horses that canter true when ridden may become disunited when cantering on the lunge. Hindlimb lameness may predispose to a horse becoming disunited. If the problem occurs to a similar extent on both reins, it often reflects a bilateral problem, but if it occurs on only one rein, the problem is more likely to be unilateral.

  Although the canter stride is initiated by the trailing hindlimb, which bears weight alone, the stance time, peak vertical ground reaction forces, and range of motion of the proximal limb joints are higher in the leading hindlimb.[2,3] Therefore if the horse consistently becomes disunited on the left rein, this is likely to reflect discomfort in the leading left hindlimb.
- Late flying changes, or difficulty in changing from right to left, or from left to right. This usually results from hindlimb lameness.
- Unlevelness in certain movements. Mild irregularities in rhythm may be detectable only in certain movements; for example, left half pass and right shoulder in. Such irregularities can reflect forelimb or hindlimb lameness or may be induced by the rider overrestricting with the hand and not creating sufficient hindlimb impulsion. Dressage riders often refer to bridle lameness, implying that lameness is not true because it cannot be detected when the horse is trotted in hand or lunged. This usually is a misnomer, because most bridle- or rein-lame horses have a genuine lameness, which may be apparent only when the horse is ridden.
- Crookedness, or reluctance to take the bit evenly on the rider's left and right hands. This can result from a training problem or be caused by lameness or unwillingness to accept the bit because of oral pain.
- Taking unequal length strides behind in walk, despite appearing sound in all other gaits. This can be seen in some otherwise normal horses that show no response to analgesics. Alternatively, irregular strides may reflect hindlimb lameness.
- Lameness apparent only when the horse is working to a contact, on the bit, and not when worked on a long rein. Problems in these horses can be difficult to solve. Some reflect a forelimb lameness, upper forelimb muscular pain, or caudal cervical pain.
- Inability to engage the hindlimbs; on the forehand. This usually reflects hindlimb lameness or back pain.
- Hanging, or on one line. This often reflects hindlimb lameness or forelimb lameness in a Standardbred.
- Loss of power cross country. This may be caused by subclinical exertional rhabdomyolysis, discomfort associated with superficial digital flexor tendonitis, or other causes of lameness. Alternatively, the horse may have acute onset back muscle pain.
- Reluctance to jump drop fences. This can be a rider or horse confidence problem, but it also may be caused by forelimb lameness or back pain.
- Cold-backed behavior when tacked up or first mounted. This is manifest by the horse tensing, roaching the back, sometimes freezing and refusing to walk forward, and then sometimes exploding into a series of violent bucks. Often this abnormal behavior stops within a few minutes, and the horse then works completely normally.

  Cold-backed behavior can be unrelated to pain and may be a problem initiated by pain, which then becomes a behavioral response. Such behavior may reflect fear, especially if a rider has fallen off several times, and less commonly is associated with chronic pain because of a rib fracture, fractured sternum, or nerve-related pain.

  The behavior may be precipitated by rapidly tightening the girth, especially if the girth has an elastic inset.

  Cold-backed behavior is generally manageable by an experienced rider, but such behavior is potentially dangerous to a nervous or inexperienced rider. Affected horses should be tacked up slowly and the girth tightened progressively, walking the horse forward each time. The horse should be lunged and made to go forward at the trot and the canter, before being mounted. The rider should be legged up onto the horse and should not attempt to mount from the ground.

  Horses with cold-backed behavior may improve with careful management but should never be trusted completely.
- Bucking. Bucking behavior can be similar to cold-backed behavior, or it may occur only after the horse has been ridden for a period. Although the horse may appear to buck with a flexible back, primary back pain is sometimes the underlying cause. Back pain secondary to hindlimb lameness may also result in bucking. Bucking can be a behavioral problem unrelated to pain.

- Rearing. Rearing is often part of nappy or resistant behavior: the horse tests the rider. Relatively rarely is rearing associated with a pain-related problem.
- Tendency for the saddle to slip to one side. This may be because of the rider's inability to sit straight, a poorly fitting saddle, or unilateral hindlimb lameness.
- Reluctance to work on the bit. Difficulties in working on the bit may be one of the first signs noted by a rider when the horse develops a minor musculoskeletal problem involving the back or limbs, or a mouth problem.
- Unwillingness to work. This may be behavioral or pain related. Warmblood breeds in particular are strong-willed horses, which easily recognize when their rider is not competent enough, and rapidly a vicious circle can ensue. However, reluctance to work equally may reflect lameness or back pain.
- Nappy (resistant) or evasive behavior. This may reflect pain or the dominance of the horse over the rider. Although some horses have a compliant temperament and never take advantage of an incompetent handler or rider, other horses rapidly recognize lack of ability, or apprehension of the rider, and develop resistant or awkward behavior.
- Progressive agitation with work, with or without loss of action. This may reflect the horse's temperament or pain caused by exertional rhabdomyolysis, for example.
- Episodic complete loss of rhythm and lack of synchronization of movement of all limbs. This may reflect a multilimb lameness, but it may also be an evasion or reflect tension. If the horse puts its head up and hollows its back, maintaining a regular rhythm is difficult. Working the horse in draw reins, a change in work environment, and the use of sedatives or analgesic drugs can help to differentiate a pain-related problem from an evasion.

Investigation of this type of horse is time consuming and usually requires repeated clinical examinations, together with ancillary diagnostic techniques. Ideally the horse should be in full work at the time of the investigation. The horse must be assessed in its entirety, including full visual appraisal and palpation at rest, and evaluation of the horse moving in hand and on the lunge on soft and hard surfaces, and the response to flexion tests.

CLINICAL ASSESSMENT

It is generally essential to see the horse ridden by the regular rider, performing the movements that are causing difficulty. If the veterinarian does not feel competent to judge the rider and the influence the rider may be having on the problem, the assistance of a professional rider or trainer may be necessary. However, it is important to recognize that not all so-called experts are truly experts, and the advice of a misguided professional may only serve to muddy the waters.

It is important to appreciate the profound influence that back pain arising from the thoracolumbar or sacroiliac regions can have over the horse's entire manner of moving. Back pain may not only induce back stiffness, but also may result in the horse holding itself tensely and not accepting the bit properly, with a restriction in stride length and reduced lift to the stride in all limbs.

Clinical assessment of back pain is not easy. A normal horse should be able to flex and extend the thoracolumbar spine repeatedly in the sagittal plane and flex from side to side with ease, without inducing tension in the epaxial muscles. Holding the back stiffly, sinking on the hindquarters to avoid extension, and evidence of muscle fasciculation or spasm may indicate pain. Alteration of facial expression and a tendency to bite or kick out may also reflect pain. However, some nervous, thin-skinned horses guard the back and do not flex normally unless they are relaxed completely. Some stoical cob-type horses show little response at all. Some horses respond to digital palpation, whereas in others firmer pressure must be applied using, for example, the closed tips of a pair of artery forceps to induce flexion and extension of the back.

In a normal horse at the trot and canter appreciable up-and-down movement of the back occurs, along with swing from side to side, with an easy swinging movement of the tail. The degree of movement is to some extent determined by the horse's natural athleticism. The degree of movement often is reduced with back pain. The degree of restriction of movement reflects the temperament of the horse and hence its reaction to pain and the severity of the pain. Clinical signs of back pain may be subtle unless the horse is assessed while it is ridden. Restricted back mobility may be more obvious when the rider sits continuously in the saddle, in sitting trot and canter, compared with when the rider sits light, or rises up and down in the trot. Movement of the tail may be restricted. The restricted movement of the back may be much more obvious to a rider than to an observer and may induce back pain in the rider. Further investigation of thoracolumbar and sacroiliac pain is discussed elsewhere (see Chapters 52 through 54).

It is important to recognize that many horses with hindlimb lameness show signs only when the horse is ridden. The modification of stride variables because of hindlimb lameness is much less than for forelimb lameness. The diagonal on which the rider sits in rising trot often substantially influences the lameness. Lameness is usually most obvious when the rider sits on the diagonal of the lame limb and may be unapparent on the other diagonal. The horse may deliberately try to make the rider sit on the non-lame diagonal. Low-grade irregularities in gait may be most apparent when the horse changes direction when performing small figures of eight or when decelerating from canter to trot or from trot to walk. Subtle delayed release of the patella may be apparent only as a horse decelerates.

Recurrent low-grade equine rhabdomyolysis may occur almost every time an affected horse is ridden, without any of the classical clinical signs of tying up. Measurement of serum concentrations of creatine kinase and aspartate aminotransferase reveal elevation of both. Some of these horses have increased uptake of radiopharmaceutical in the affected muscles in bone-phase images if examined using nuclear scintigraphy.

It is often necessary to compare the horse's performance when ridden by a different rider, bearing in mind that riders with varying abilities can make horses appear different, and riders of differing weights and ability to follow the rhythm can make a great difference in the horse's gait. A good rider can make a slightly nappy horse go forward freely, with a totally rhythmical stride, whereas a less competent rider may be unable to ride the horse through, and the horse may take uneven steps behind because it is not going forward enough. A heavy rider or a rider who is unable to ride properly in balance may induce hindlimb lameness. The same horse ridden by a lighter rider may be completely sound. A horse that evades the bit and runs along with its head in the air may take irregular steps in front and behind and appear stiff in the back, especially if a rider attempts to make the horse work on the bit. The same horse, when encouraged to submit and work on the bit when ridden by an expert with draw reins, may appear completely different, relaxing the back, stepping under more from behind and becoming much more rhythmical. It is often necessary diplomatically to separate the owner from the horse to assess how the horse responds to a skilled rider over several days.

It is also important to recognize that inappropriate work patterns on the flat or over fences actually can induce muscu-

loskeletal pain, which may progress to chronic lameness. For example, repeatedly placing the horse too close to a jump (putting the horse in deep), which also alters the bascule over the fence and the way in which the horse lands, may predispose the horse to patellar ligament soreness. This may alter the horse's way of moving and predispose to the development of pain elsewhere. In the early stages the problems potentially may be managed with a change in work program and methods of training the horse. However, long-term problems may be more difficult to resolve satisfactorily.

Paradoxically, it may be helpful to see a usually well-ridden horse with a subtle performance problem ridden by a less clever rider. The professional rider may be making subtle adjustments to the gait inadvertently and thereby masking gait irregularities reflecting low-grade lameness.

The fit of the tack and its suitability must be evaluated. The veterinarian should not assume that because the saddle has been fitted by a professional saddle fitter that it necessarily does fit. The position of the bit in the mouth, the size of the bit relative to the size and shape of the mouth, the suitability and the severity of the bit should all be assessed. The mouth should be inspected carefully to ensure that no lacerations of the tongue, gums, or corners of the horse's mouth have occurred and that the teeth have no sharp points or hooks.

In some instances the horse may appear to be clinically normal, and it is necessary to try to establish whether the complaint is indeed pain related. In my experience, phenylbutazone is the most effective analgesic drug for trying to determine whether a problem is pain related. The drug must be given at a high enough dose (minimum 4.4 mg/kg twice daily) for long enough (at least 7 days if the horse does not respond within 2 to 3 days). A comparison with records of the horse's attitude, behavior, and action before, during, and after treatment should be assessed as objectively as possible. A positive response confirms the presence of a pain-related problem, whereas a negative response does not definitively exclude a pain-induced problem. The potential exists to create a placebo effect for the rider, and in selected horses, "blinding" the rider to the treatment may be worthwhile.

If the problem seems to induce considerable anxiety and tension when the horse is worked, mild tranquilization with acepromazine or sedation with detomidine can be helpful to determine if the problem is pain induced or reflects tension. The tension actually may be induced by the rider or the environment in which the horse is worked. Working the horse in a different situation with another skilled rider can be helpful.

If the horse is fresh and exuberant, minor gait abnormalities can be difficult to assess, especially in a big moving dressage horse. In these circumstances mild sedation can be useful.

It is important to try to determine if a problem reflects pain or weakness caused by a neurological problem. Low-grade hindlimb toe drag may reflect a mild proprioceptive deficit rather than lameness. When in doubt, a comprehensive neurological examination should be performed (see Chapter 11). This is particularly important in areas (North and South America) where equine protozoal myelitis occurs. In other countries it is important to establish whether the horse has ever been to America.

It is also important to recognize that some horses that are mild wobblers, with mild hindlimb gait abnormalities seen when moving loose or in hand, may actually move loosely and freely when ridden, with apparent good coordination, and perform rather well in dressage despite a problem.

Diagnostic Analgesia

In some horses taking an educated guess about a potential source of pain and medicating the suspect joints with, for example, triamcinolone and assessing the response to medication (so-called diagnostic medication) may be necessary. For example, in a dressage horse taking uneven height steps behind in piaffe, or failing to maintain a regular two-time rhythm in piaffe, medicating the distal hock joints may be worthwhile. However, it is necessary to recognize that a negative response does not exclude definitively distal hock joint pain as the primary problem. Paradoxically, in some horses the response to intra-articular analgesia may be better than the response to intra-articular medication, whereas in others the response to medication is better than that after analgesia, even in the absence of radiological abnormalities.

Frequently through careful clinical evaluation of the horse, a low-grade, bilateral, symmetrical lameness can be identified that can be investigated further through local analgesic techniques and then appropriate imaging modalities. With low-grade bilateral forelimb lameness, blocking both front feet simultaneously, for example, may be more useful than blocking one at a time and then assessing the change in the horse's overall way of moving. With poor hindlimb propulsion, blocking the region of the origin of both hindlimb suspensory ligaments may be more useful than blocking one at a time. In some horses concurrent forelimb and hindlimb lameness may be detected. Alternatively, back stiffness may be identified, requiring further investigation by nuclear scintigraphy, radiography, and ultrasonography.

Diagnostic Imaging

Thermographic evaluation, particularly of the neck and back, is thought by some to be helpful in diagnosing low-grade performance abnormalities (see Chapters 25 and 98). However, others have found thermography much less rewarding. It is vital to recognize the normal thermographic patterns, the responses to exercise, and the many other external factors that may influence the results.

In horses in which no obvious gait abnormalities can be identified or in which subtle changes are noted that cannot be investigated further by local analgesic techniques, performing a comprehensive nuclear scintigraphic examination of the horse may be helpful (see Chapter 19). However, interpretation of the results can be notoriously difficult, because areas of increased radiopharmaceutical uptake (hot spots) are not necessarily synonymous with pain, and not all musculoskeletal problems are associated with hot spots. For example, bilateral proximal suspensory desmitis may result in a slightly restricted and stiff hindlimb gait, but nuclear scintigraphic examination may be normal. Great care must be taken not to overinterpret the results of scintigraphic examination. Results must be correlated carefully with the clinical signs, the responses where possible to local analgesic techniques, and the response to diagnostic medication.

Evaluation of gait and other aspects of performance on a high-speed treadmill are discussed in Chapter 101.

COMMON PERFORMANCE PROBLEMS

The most common causes of poor performance not related to readily recognizable overt lameness are these:

Possible clinical problems
- Bilateral foot pain
- Bilateral fetlock pain
- Bilateral proximal suspensory desmitis, forelimb or hindlimb
- Bilateral carpal pain
- Thoracolumbar or sacroiliac pain
- Intermittent upward fixation of the patella or delayed release of the patella
- Bilateral distal hock joint pain
- Tying up

Rider-induced problems
- Hand brake on (i.e., too much restriction by the rider's hands) and no legs
- Overweight; inability to follow the rhythm
- Lack of confidence
- Over horsed rider being too restrictive
- Misunderstanding of how to achieve "on the bit"
- Poor eye for a stride; therefore repeatedly placing the horse in less than ideal positions for take off for a fence, making the jump more awkward, or requiring more effort
- Sitting crookedly
- Lack of rapport with the horse

Trainer-induced problem
- Monotonous training program with no variation

Horse problems
- Lack of ability
- Unsuitable temperament
- Loss of confidence
- Lack of rapport with the rider
- Staleness
- Lack of focus in a dual-purpose breeding stallion and competition horse
- Inconsistency of a mare

These horses are time consuming to investigate, often requiring a multifaceted approach to diagnosis. Although it is often essential to see the regular rider riding the horse, separating the horse from the rider for several days can be helpful, particularly with horses ridden by amateurs or semi-professionals. It is essential to have access to adequate facilities to see the horse ridden (or driven) properly on a daily basis and to be able to perform local analgesic techniques and then re-evaluate the horse ridden or driven under the same circumstances.

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CHAPTER • 101

Experiences Using a High-Speed Treadmill to Evaluate Lameness

Benson B. Martin, Sue J. Dyson, and Mike W. Ross

The advent of the high-speed treadmill has led to many advances in evaluating equine poor performance in exercise physiology, gait analysis, cardiac disease, and lameness diagnosis.[1-5] This chapter describes our experience using a high-speed treadmill for lameness evaluation in the performance horse.

Lameness has been implicated as a cause of poor performance in several studies involving a complete physical examination, lameness examination, and where possible a high-speed treadmill examination.[1-3] In one study, 74% of horses were found to have lameness as a component of poor performance.[1] In another study, in which horses were examined for poor performance with no apparent history of lameness, significant lameness in 23.9% of horses precluded a high-speed treadmill investigation.[2] In a third study, 4.3% of horses sound enough to undergo a high-speed treadmill examination for poor performance were significantly lame after high-speed treadmill exercise.[3]

In our experience, convincing trainers and owners that lameness is a cause for poor performance can be difficult. Several studies using a shoe model to induce lameness to assess the metabolic cost of pain related to lameness suggested a trend that pain related to lameness may not increase the metabolic cost of exercise, but it does increase the heart rate in response to pain.[6-8] However, another study suggested that a metabolic cost of pain and exercise does occur.[9] Pain may alter a horse's ability or willingness to perform up to expectations or previous performance levels.[10]

CRITERIA FOR CASE SELECTION

The most common criterion for a treadmill lameness examination is lameness that occurs at only high speeds or after an extended period of exercise. The treadmill examination is used most commonly in Standardbred (STB) racehorses or endurance horses. However, a treadmill lameness examination can be used in any breed or discipline, although a horse in which lameness is only apparent when the horse is ridden may be unsuitable.

Horses in which a complete lameness investigation, including diagnostic analgesia and appropriate imaging technologies, fails to reveal a diagnosis are potential candidates for treadmill lameness examination. Additional candidates include horses that require considerable work at low, medium, or higher speeds before the lameness becomes apparent, such as endurance horses or STB racehorses. A treadmill lameness examination also may be useful in horses that are fractious or otherwise pay little attention when jogged in hand or ridden. Horses must pay close attention when trotting on a treadmill and therefore have a more even, rhythmical, relaxed gait that is easier to evaluate.

Rider-induced problems
- Hand brake on (i.e., too much restriction by the rider's hands) and no legs
- Overweight; inability to follow the rhythm
- Lack of confidence
- Over horsed rider being too restrictive
- Misunderstanding of how to achieve "on the bit"
- Poor eye for a stride; therefore repeatedly placing the horse in less than ideal positions for take off for a fence, making the jump more awkward, or requiring more effort
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HISTORY

Important historical facts need to be ascertained. Some questions are general and some are use related. Some may need to be repeated in a different order to elicit an accurate answer and may include the following.

General Questions

How long has the horse been lame?
When do you notice the lameness?
Does the lameness improve with exercise?
Does the lameness become worse during or after exercise?
Does the horse ever appear to become anxious or frantic?
Does the horse sweat?
Has the horse's performance level changed?
Has the horse changed how well it performs?
Has the horse changed the style of performance?
Has the horse had any behavioral changes?
Has the horse been treated for lameness?
Has the horse been "tapped"?
Has the horse ever had any joints treated?
Has the horse received any intramuscular treatments?
Has the horse received any other therapy or medication?
How much and for how long?
What has been the response?
Does the horse receive any special supplements (e.g., sodium bicarbonate)?
What type of shoes does the horse wear and why?
Does the horse have any special shoeing or trimming and why?

Use-Specific Questions

Standardbred Racehorses

At what speed is the horse the worst?
At which gait (trot or pace) is the lameness most easily seen?
Is the horse worse on the straight or on turns?
Is the horse worse when turned the correct direction of the track?
Is the horse worse when jogging?
What is the horse's normal gait?
Has any change in the horse's gait occurred, and what is the change?
Is the horse on a line?
Is the horse on a shaft?
Does the horse get into his knees?
Is the horse worse when jogging free legged or in full harness?
Is the horse worse when checked-up?
Does the horse use any special gaiting equipment?
Does the horse wear any head poles?
Does the horse wear any other special equipment and why?

Thoroughbred Racehorses

Does the horse bear in or out?
Does the horse break out of the starting gate as well as before?
Does the horse require any special bit or tack and why?
At what speed is the lameness best seen or felt?
Is lameness worse with or without a rider?

Endurance Horses

How far does the horse have to go before the lameness appears (time or distance)?
What does the lameness feel like or look like?
Does the horse have any bruising of the feet?
Does the horse feel or look like it is tying up?
Does the horse sweat normally?

Dressage and Show Horses

Is the horse worse when ridden?
Is the horse worse on a large turn or small turn?
Is the horse worse going up or down a hill?
Is the horse worse when collected or on a free rein?

EQUIPMENT

The treadmill must have a speed of at least 7 to 10 m/sec. Most horses will not canter until the speed exceeds 7 m/sec. Lameness is usually evident at lower speeds, but occasionally it is necessary to see a horse trot at up to 7 m/sec for lameness to become manifest. In addition, for further poor performance evaluation the treadmill needs a speed of at least 14 to 15 m/sec and the capability of being elevated to at least a 6° slope. Ideally the treadmill should be mounted in the ground, which is safer. In our experience clients and horses easily adapt to an in the ground treadmill. It is easier to use video equipment with a treadmill in the ground, rather than above the ground.

At least 4 to 5 m of space in front of, behind, and on either side of the treadmill is needed to allow for equipment, cameras, and personnel and to allow room for the horse to move off the front, rear, or side. The moving treadmill surface is relatively forgiving. In one study in which a horse had a strain gauge placed on the left third metacarpal bone, bone strain was measured as the horse galloped on a dirt track, wood chip track, and on a treadmill. Compared with the direct track bone strain was 75% less when the horses galloped on the wood chip track and an additional 75% less when galloped on the treadmill.[11]

Kinematic analyses include high-speed cinematography, videography, electrogoniometry, or accelerometry to quantify the movements of the limbs and torso during exercise. Kinetic analyses use instrumented shoes, force plates, and pressure-sensitive mats to measure forces that affect, arrest, or modify motion. The reader is referred to Chapter 22 and other references. Many of these systems are expensive, time consuming, cumbersome, and require considerable expertise to use. The ideal gait analysis system provides considerable data, which are obtained easily, visually and statistically analyzed, and not too expensive. The system must be fast enough to assess correctly diagnostic nerve blocks in a timely fashion and simple enough to not alter the gait of the horse.[12,13]

Simple systems can be assembled with a digital video camera, a tripod, and a computer, using simple video editing software to review and edit the images. If a computer is not available, video editing tape decks can be used. The camera or cameras are placed perpendicular to the side and front or rear of the treadmill. Caution must be used with the rear camera. Enough light must be available for adequate video viewing (at least two 1000-W lights or equivalent).[12,13]

The video camera can be a Hi-8 or digital camera, with a video stabilization system and should have a shutter speed of at least 1/2000 (digital) or 1/4000 (analog).[14] Most consumer-grade cameras film at 30 frames per second (fps). Therefore some video loss is apparent compared with more sophisticated (200 fps) and expensive systems. Digital and Hi-8 video create less video noise and provide a superior viewing experience. For rapid viewing the recorded tape can be placed into a Hi-8 or digital videocassette recorder that has a jog-shuttle device for stop frame viewing or frame grabbing. Alternatively, video editing tape decks, which are more expensive than traditional videocassette recorders can be used. Digital images also can be reviewed on a computer and thus more accurately analyzed and edited. Turnkey systems including hardware and software using universal serial bus or IEEE 1394 rapid transfer technology are available.

Using video technology allows the clinician to critically examine the baseline lameness and lameness after diagnostic analgesia, record them in real time, analyze the differences in real time slow motion, maintain a permanent record for follow-up examinations, and demonstrate to the owner or trainer the difference in gait before and after a successful block. Video analysis also can be useful in demonstrating initial footfall, footfall after trimming or shoeing, and the

effects of different types and weights of shoes. The technology gives one a new appreciation for lameness, motion, conformation, and gait at a walk, trot, or higher speed. The camera is much more sensitive than the human eye and can answer many questions and create new ones.[12]

PREPARATION OF THE HORSE AND SAFETY CONSIDERATIONS

It is important that a horse is reasonably fit to perform an accurate treadmill lameness examination. STBs should be trained down to at least a 2-minute mile. Endurance horses, in which lameness may not occur for several miles, need to be at least fit enough to accomplish this task easily. For other horses, they should be in regular work. To do otherwise places the horse and examination at risk. Unfit horses run the risk of exertional myopathy, thus obfuscating the results of any lameness examination.

We prefer horses to wear shoes when being examined for lameness. Tremendous friction and therefore heat are generated by the contact of the horse's foot and the treadmill belt. Whenever a foot contacts the belt, the foot slips and therefore causes friction. Many research horses that exercise daily on a high-speed treadmill have chronic foot problems secondary to the heat generated. This heat can be enough to melt the glue of glue-on shoes at higher speeds. If the horse is not shod, the toes of the hind feet can break off and bleed, leading to substantial lameness for several weeks. We do not place any type of tape on the horses' shoes. We recommend using flat shoes, with no toe grabs, stickers, borium, or turndowns. This minimizes wear to the treadmill belt, which lasts 9 to 12 months before replacement is required when used daily. We recommend that shoeing changes be made 3 to 5 days before a treadmill examination, in case foot problems related to shoeing are found that may mask the horse's actual primary lameness.

Examination for lameness on a treadmill requires at least three people. One person controls the treadmill and the horse's head; one person stands behind the horse to keep it moving forward if necessary, and one person is the clinician. The clinician is responsible for operating the video equipment and observing the horse at a walk, trot, and any other desired gait. Occasionally, two people are required at the head in fractious horses.

Schooling of the average animal usually takes about 30 minutes to one hour before the horse is ready to be examined for lameness. Most (>99%) horses can be schooled and examined for lameness or poor performance in the same day. This period includes acclimating the horse to the treadmill and the fan noise, to standing on the treadmill with the machine off, and to moving with the treadmill at the walk, the trot, or pace at medium speed, a canter at low speed (7 m/sec) for non-STBs, and then at rest. STBs then are harnessed and moved up to a speed of about 7 m/sec for 200 to 300 m. Most horses adapt easily using this method of schooling, which is safe and successful in 99% of horses examined for lameness or poor performance. Occasionally, horses are recalcitrant or scared and need to be reschooled a day later and usually adapt well. Some horses (<1%) cannot be examined because the examination would not be safe for the horses or personnel.[15]

Safety of horses and personnel is paramount. Personnel must be cautioned to be alert at all times and not to stand behind the horse when the treadmill is moving. Shoes and horses can come off the treadmill without warning. Substantial injuries (e.g., tendononitis) occur in less than 1.5% of horses. Lacerations, broken up hind feet, and myositis occur in less than 5% of horses.[15] All horses wear protective boots on all limbs. All STBs are schooled first without equipment, but with protective boots, and then they wear the normal equipment plus protective boots if they move at speeds greater than 7 m/sec.

To minimize the risk of injury to personnel and horses, it is important to have a trained team to examine each horse. Personnel should block any windows through which the horse can see out, eliminate as many noise distractions as possible, and ensure that no doors can be opened in front of the horse during schooling or exercise. This allows the horse to concentrate on staying up with the treadmill and minimizes frightening or distracting the horse during the examination.

LAMENESS DIAGNOSIS

Most lameness is seen best in all breeds with a minimum of equipment in place. STBs, which usually race checked up, are examined first with a minimum of equipment (i.e., no hobbles) and not checked up. Many lameness conditions in STBs can be obfuscated, changed, or eliminated when a horse wears its equipment and in particular, when checked up. Other horses are examined wearing just a halter unless some equipment change exacerbates the lameness (rare). A riding horse may need to wear side reins. Occasionally, STBs with carpal or stifle lameness are worse when wearing hobbles.

Horses are first examined at the walk and then at the trot and are recorded on video. Recording from the side is useful to evaluate stride length and frequency. Examination from the rear is helpful in evaluating the swing of the forelimbs and hindlimbs and the footfall.

Observation of lameness using a treadmill is different from the standard lameness examination because the horse is stationary relative to the observer, allowing viewing and video recording of the horse's gait from the side, front, and rear. The most useful gaits for observing lameness are the trot and pace. These are symmetrical gaits and allow for easier evaluation of lameness and gait rhythm.[13]

The horse should become comfortable at the trot, so that the lameness or gait abnormality can be established. An audible rhythmic pattern to the gait also becomes apparent. The horse is in rhythm (normal) or out of rhythm (lame). In addition to video recording for later viewing, it is important to evaluate the lameness concurrently to establish a baseline. If lameness is not readily apparent, reviewing the video immediately before performing diagnostic analgesia may be advantageous. Although detecting a difference in footfall is a standard part of any lameness examination, in our experience footfall may be exacerbated on a treadmill. Sound horses that trot have a symmetrical, diagonal, two-beat gait, and pacers have a symmetrical, lateral two-beat gait. The horse should be observed carefully for a change in how the horse swings its forelimb or hindlimb. In STB pacers, any change in how the horse moves from side to side at the poll or hips, when viewed from the rear after diagnostic analgesia, is important. These horses, whether pacing free legged (no harness) or when in harness or checked up, have a tendency to swing from side to side and land more heavily or with greater excursion on the sound side. An in-depth description of these gaits is available.[13]

In addition to eliminating lameness, a change in stride characteristics may occur, including stride frequency, length, and swing plane. Some horses not accustomed to the treadmill, or not comfortable on the treadmill, may have an increased stride frequency and shortened stride length, which also may be seen in lame horses.[13] Once accustomed to the treadmill, many sound horses exercised at medium to higher speeds on a treadmill have a longer stride and thus a lower stride frequency.[16] However, lame horses may have an increased stride frequency and shortened stride length.[17] This can best be demonstrated and seen using video.

Other more subtle changes include improvement in gait fluidity, improvement in use of the head and neck and shoulder, and change of an anxious eye expression, flexion of major joints, foot flight, width and rhythm of gait, and footfall.

Occasionally, horses may become stiff or anxious and lame or lamer behind during or after exercise. Exercise should be discontinued. Exertional myopathy (see Chapter 84) or aorto-iliac thrombosis (see Chapter 52) should be considered likely causes.[18]

ADDITIONAL TESTS

It is useful to measure creatine kinase and aspartate aminotransferase concentrations before and after exercise to monitor horses for subclinical myopathy, overt myopathy, or previous undiagnosed myopathy.[19] If aorto-iliac thrombosis is suspected, a rectal examination and an ultrasonographic examination per rectum should be performed. Horses with aorto-iliac thrombosis commonly become lame behind during or shortly after commencement of exercise.[18] They have an anxious look and can sweat more profusely than their level of exercise would dictate.

The treadmill provides a smooth, even surface at a constant, controllable speed and a horse that is stationary relative to the observer. Some horses are lame in hand, but not on treadmill, and some are lame on treadmill, but not in hand. Horses that are only lame on turns are difficult to evaluate on a treadmill, which only operates in a straight line. Some patients will not tolerate working on a treadmill.[20-22]

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SECTION • 2
The Racehorse

CHAPTER • 102

The Sales Yearling

PURCHASE EXAMINATION OF A THOROUGHBRED SALES YEARLING IN NORTH AMERICA

• Benson B. Martin, John C. Kimmel, and Mark W. Cheney

Veterinary inspection of a Thoroughbred (TB) yearling at public auction requires expertise and experience.[1-13] Previously, yearlings commonly were examined after purchase and if a problem was discovered subsequently, a lengthy, expensive, and frequently unsatisfactory arbitration for buyer and seller alike was implemented. Recently a shift has occurred toward a comprehensive examination of yearlings before purchase.

It is important that the veterinarian's role be defined in advance with all the necessary parties, such as the potential trainer, owner, and syndicate manager. Clinicians should determine whether they are prepared to offer advice concerning the pedigree or whether their advice will be strictly veterinary. It should be made clear that no guarantees of future performance can be given. Any potential conflicts of interest must be declared, including whether the veterinarian is involved as an agent, breeder, purchaser, or consignor.

Veterinarians should establish precisely for whom they are working and to whom information can be divulged. They should not give privileged, confidential information to anyone else. Provide a representative estimate of the fee for the examination. Sometimes a buyer may wish to gain access to information obtained by another. This is acceptable provided that the original client gives permission. In this way the fee may be split between two or more prospective purchasers. If clinicians work regularly with purchasers, they may be able to contribute to their buying policy, which may include consideration of pedigree information, budget, conformation type, sex, and potential resale breeding value. Excellent communication with trainers is important, because they ultimately have most control of the horse when it enters training.

It is also important to establish a good relationship with the consignor or agent. Veterinarians should be courteous and respectful and should arrange a mutually suitable time to examine the yearling and should request permission before carrying out endoscopic or radiographic examinations.

Experience is essential to make accurate interpretations of conformational or gait abnormalities, radiographic or endoscopic findings, and the results of examination of the eyes and heart. Knowledge of pedigrees also can be helpful.

Conditions of Sale

It is important to be aware of the conditions of sale for each sales company. These conditions usually differ with the age of the horse, use of the horse, and sales company. Sales are weighted in favor of the consignors and conducted with an attitude of caveat emptor. The warranty is limited, with no implied warranty for use or soundness other than those conditions published in the sales catalog. The repository is an area at the sales set up for viewing radiographs, and videotapes of the upper airway at rest, in selected horses. If veterinarians determine this information is not adequate or satisfactory, it is their responsibility to obtain the desired information. They should examine all available information carefully. If any problem is discovered after the sale that was evident in the repository information, the consignor is not liable. The sales company makes no warranty about the accuracy or completeness of repository information and makes no interpretation of it. Repository information is for the convenience of potential buyers and as a courtesy of the consignors and also decreases the exposure of yearlings to repetitive and stressful examinations. Other information, such as previous surgery, medical problems, eye problems, current medication, vaccination, Coggins and equine viral arteritis status, and presence of radiographs, may be available from the consignor, if the veterinarian asks.

Presale or Postsale Examination

Currently many persons prefer presale inspection. This eliminates the process of arbitration and places the onus of disclosure on the consignor and makes the buyer responsible for obtaining all available information before sale. If the buyer does not obtain such information (such as identification of a chip fracture), the buyer has no recourse. With examination after sale the only problems for which a horse may be returned are listed in the catalog as conditions of sale. The veterinarian is responsible for knowing these conditions and the terms under which arbitration can occur. Arbitration usually must be implemented within 24 hours after the sale of the yearling, and the yearling must still be on the sales grounds. Failure to comply with this negates any chance of arbitration.[14] Arbitration usually involves a veterinarian representing the sales company, the consignor, and the buyer.

Clinical Examination

A yearling TB is immature and its physical appearance may change considerably. Developing the skills to predict how each horse may develop by learning from experienced persons is worthwhile. Assess the general attitude of the horse, its eye, and presence. Be aware that many yearlings are tranquilized for ease of inspection, so one may not get a true picture of the animal's attitude, which can be important in training and racing. Evaluate the horse's conformation. Look at the horse's feet, because the old adage "no foot, no horse" is true. Assess the horse's shoulder, hip, top line, length of back, and then the lower limbs. A good shoulder and hip can accommodate many conformational defects lower in the limb. In our opinion the following conformational abnormalities may predispose to lameness and should be avoided: back at the knee, tied in behind the knee, offset knees, and exceptionally straight hindlimbs. Carefully evaluate the shoeing and trimming of the

feet, because a good farrier can make a horse that toes in or out appear to be almost normal, and they are most adept at repairing an abnormal hoof appearance. After examining the horse walking in hand, perform a more detailed examination in the stall, out of sight of the general public, as a courtesy to the consignor. Assess carefully any swellings and palpate the joints, tendons, and ligaments. Examine the eyes and auscultate the heart. Look for evidence of previous periosteal stripping or other surgery, evident as dermal thickening or white hair in areas where surgery is commonly performed.

Endoscopy and Echocardiography

The veterinarian should ask the consignor or his manager for his assistance and permission to perform an endoscopic, radiographic, and echocardiographic examination of the animal and should arrange a time to do so. A resting endoscopic examination of the horse's upper airway should include stimulating the horse to swallow and holding off the horse's airway. This allows more complete evaluation of the horse's upper airway function at rest. Echocardiographic examination is not performed routinely but can be used to assess heart size and left ventricular free-wall contractility or to detect abnormalities.[15]

Radiography

For many yearlings radiographs are obtained up to 30 days before the sales and are stored in the repository at the sale. Most states allow only veterinarians licensed in the state to examine these radiographs. The examining veterinarian is responsible for ensuring that all the desired views are present, that the radiographs are properly identified, and that they are of suitable quality. The radiographs should be interpreted carefully. If the radiographs are incomplete, of poor quality, or absent completely, the veterinarian should should arrange to have his or her own complete set of radiographs obtained. In our opinion a comprehensive examination consists of 46 views, plus any additional views needed on the basis of clinical examination. These views include the following:

- Front feet: lateromedial (LM) and dorsopalmar (DPa)
- Front fetlocks: DPa, LM, flexed LM, dorsolateral-palmaromedial oblique (DL-PaMO), and dorsomedial-palmarolateral oblique (DM-PaLO)
- Carpi: DPa, LM, flexed LM, DL-PaMO and DM-PaLO
- Hind fetlocks: dorsoplantar (DPl), LM, dorsolateral-plantaromedial oblique (DL-PlMO), and dorsomedial-plantarolateral oblique (DM-PlLO)
- Tarsi: DPl, LM, DL-PlMO, DM-PlLO
- Stifles: caudocranial, LM, and caudolateral-craniomedial oblique (CdL-CrMO)

However, the following 36 views are those required by the Keeneland sales repository in 2001:

- Front fetlocks: dorsal 15° proximal-palmarodistal oblique, LM, flexed LM, DL-PaMO, and DM-PaLO.
- Hind fetlocks: dorsal 15° proximal-plantarodistal oblique, LM, flexed LM, DL-PlMO, and DM-PlLO
- Carpi: flexed LM, DL-PaMO, and DM-Pa LO
- Tarsi: D10°L-PlMO, LM, and DM-PlLO
- Stifles: LM and CdL-CrMO

This provides a comprehensive, but not all-inclusive, picture of the horse's musculoskeletal system. Other sales companies may have different requirements. The veterinarian is responsible for ascertaining the requirements.

A number of common radiographic findings may eliminate a horse from further consideration.[16] Fractures of the carpus, distal phalanx, tarsus, proximal sesamoid bones, and the second or fourth metacarpal (metatarsal) bones generally are considered unacceptable, whereas small osteochondral fragments on the dorsal aspect of a fetlock joint may be acceptable. Osteoarthritis of the carpus, fetlock, or proximal interphalangeal joint is not acceptable. However, small osteophytes on the dorsoproximal aspect of the third metatarsal bone do not preclude necessarily a successful athletic career.

The relevance of osteochondrosis depends on the location of the lesion, its size and severity, the presence or absence of osteoarthritis, and the prognosis for racing after surgical treatment. A small fragment may be amenable to surgical treatment and should be discussed with the buyer. If a fragment has been removed previously and no evidence of osteoarthritis exists, the horse may be a reasonable risk for purchase. Horses with osteochondral fragmentation of the distal intermediate ridge of the tibia or small lesions of the lateral trochlear ridge of the femur usually are treated successfully by surgical removal. Horses with osteochondral fragments on the proximoplantar aspect of the proximal phalanx in the hindlimbs (which may actually be traumatic in origin) represent a reasonable risk.

If an osseous cyst-like lesion is identified, especially one involving the stifle or fetlock, the horse generally should be considered at high risk for developing lameness. Other radiographic abnormalities that may preclude purchase include sesamoiditis, laminitis, or active splints.

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PURCHASE EXAMINATION OF A THOROUGHBRED SALES YEARLING IN EUROPE

• David R. Ellis

Yearling sales occur in Europe later in the year than in the United States. The earliest, at Deauville in Normandy, is in early August, but in England and Ireland the main sales are conducted between the end of September and early December.

Monitoring and Sale Selection

Most higher-class farms that offer yearlings regularly monitor the growth rates, weights, and conformation of the young stock. Such monitoring often is done with veterinary help and is conducted in close cooperation with the farrier. This should ensure that any corrective farriery is undertaken as early as possible, thus gaining maximum benefit and not leaving correction to too late in the growth of the animal. The second growth spurt, between 9 and 12 months of age, is an important window of opportunity in which to make substantial changes through corrective techniques. Regular veterinary inspections also help to detect disease, such as acquired deformities or developmental orthopedic disease, early enough to allow successful remedial measures to be undertaken. As in the United States, selection of the sale at which the yearling is to be offered is made in the spring, usually April. A few consignors conduct radiographic surveys at this time to help this selection and to deal with potential problems such as bone chips and osteochondrosis.

Preparation

Yearlings are brought in from pasture 6 to 8 weeks before sale for increasingly intensive preparation. This involves walking exercise with the horse led by hand, sometimes using uphill work, or using a walker. Treadmills used to be used, but they have fallen from favor because they tend to give the animal a goose-stepping type of action. Lunging is introduced later in preparation, particularly for the heavier or fatter animals, and this will be short and at a sharp trot and canter, on both reins and often twice daily. Lunging is more important to sale preparation in Europe because most yearlings, particularly the more expensive, will undergo respiratory examination at the canter by the purchaser's veterinary surgeon immediately after sale. Education and fitness are important if this postsale test is to be conducted safely and satisfactorily. A substantial risk of injury exists during lunging if the exercise is not conducted carefully and on a suitable surface. Interference injuries, such as brushing or overreach wounds. can be prevented by using protective boots, farriery, and careful buildup of speed and fitness and particularly by using a sympathetic surface. Surfaces such as deep sand or dry wood chip may become too deep or slide away under the horse. Paddock turf can be too firm and slippery to be safe. Modern surfaces, such as Fibresand (Fibresand UK Ltd., Mansfield Sand Co., Nottinghamshire, UK), or Polytrack (Martin Collins Enterprises, Hungerford, UK) on a slight camber are safer. Some of the more successful vendors place more emphasis on lunging to achieve fitness and muscle condition than on walking. Excessive walking usually makes the person leading the yearling fitter and leaves the yearling bored and slovenly.

Yearlings usually come up from summer grazing in good, perhaps heavy, body condition. Converting the fat yearling into a well-muscled potential athlete by judicious feeding and exercise is an art. Overfeeding the leaner animal may lead to physitis (see Chapter 59). As the weather starts to cool, most yearlings will be rugged (blanket applied) in the stable to ensure that they do not start changing to a winter coat. This change in management means that less energy is required in the diet.

Using medication to promote growth or body condition is much less common nowadays, partly because certain vendors became renowned for these practices and suffered in the long term. Anabolic steroids are not licensed for use in the horse, now deemed in mainland Europe to be a food animal. Several years ago Tattersalls, the important Bloodstock Auctioneers, conducted a pilot program of drug testing yearlings for steroids at their premier sale to assess steroid use. Although the results were never published, the effect of this exercise was salutary.

During the preparation period, front shoes will be fitted, and some changes can be made to hoof trimming and balance to conceal slighter conformation faults, fill horn defects, and ensure that hoof wear during exercise is not excessive. Few yearlings will be fitted with hind shoes. It is important that the final shoeing is not too close to the sale date, so that tight nails or other shoeing problems do not cause lameness.

Orthopedic problems encountered in the yearling year can include physitis—mainly at the distal radial growth plate, but occasionally also the distal tibia—splints, sesamoiditis and strain of suspensory ligament branches, osteochondrosis, or bone cysts. Horses with bone cysts often have clinical lameness when forced exercise commences, as in sale preparation, or breaking in immediately after sale. Managing orthopedic problems in the months before the sale not only requires the ideal veterinary measures according to the disease, but also requires giving consideration to the importance that potential purchasers place on the problem (little perhaps in animals with small settled splints) and to alternative strategy if the chosen sale has to be missed. If, for example, a yearling develops a bog spavin that requires surgical removal of osteochondral fragments 3 months before sale, having the joint back to normal, the clipped hair regrown, and the yearling adequately fit to offer for auction would be a close race to run. The ethics of offering such an animal also have to be considered.

Conditions of Sale

Conditions of sale for yearlings at the main European auction houses do not include orthopedic conditions or surgery, with the exception of denerving at Doncaster (United Kingdom) and Goffs (Ireland). The latter auction house also deems wobblers to be returnable. Purchaser power and the example from the United States have introduced more pre-sale radiographic examinations at premier sales. These now mostly are conducted by the vendor, and films of standard views of the carpi, hocks, all fetlocks, and stifle joints are available for examination by purchasers' veterinary surgeons. No official repository exists, but viewing facilities are available at the sale paddocks.

Radiographs are not essential to a profitable sale, because some of the most successful vendors at Tattersalls have not allowed pre-sale radiography or endoscopy. They stand by their yearling on orthopedic matters and rely on the auctioneer's condition of sale regarding wind conditions. The latter is more stringent than in the United States regarding laryngeal hemiplegia but does not include other upper respiratory disorders, which are considered intermittent, treatable, or so rare (for example, rostral displacement of the palatine arch has been detected three times in 17 years) as to be dealt with on a single-case basis. Conditions of sale for English and Irish yearling sales state that a horse is returnable if it is found to make a characteristic abnormal inspiratory sound when actively exercised (a roar or whistle at the canter) and have endoscopic evidence of laryngeal hemiplegia that was not declared before sale. Horses are lunged for this examination soon after sale and before they leave the sale paddocks.

Orthopedic Conditions

The most commonly found orthopedic condition to cause significant difficulty after sale is the subchondral bone cyst in the medial condyle of the distal femur (see Chapter 47). Characteristically, lameness occurs soon after the horse starts

lunging exercise for breaking in. Clinicians experienced with yearlings always radiograph a yearling's stifles if lameness develops in a hindlimb under these circumstances, with no obvious clinical sign of origin of pain. Vendors usually are approached with a view to taking the animal back, particularly if the money has not changed hands, but not all will do so. No case has yet come to court, and it must be admitted that after conservative or surgical treatment, some such horses have gone on to race satisfactorily, but not as 2-year-olds.

Conditions commonly found during prepurchase veterinary examination include the following:

- Enlargement of the proximal sesamoid bones can be the most important condition, because enlargement may result from fractures sustained as a young foal that have healed by fibrous union (see Chapter 37). Enlargement is often multiple and can involve the apex or the base of the bone. Sesamoiditis, with or without suspensory branch desmitis, also is seen in yearlings (Fig. 102-1).
- Any effusion of a joint must be viewed with suspicion, particularly if forced flexion is painful. Purchase cannot be recommended, and even if good quality radiographs reveal no abnormality, a guarded opinion is given.
- Asymmetry of front feet is common, and a slightly narrow hoof need not detract, providing the hoof is not twisted or shear heeled. Feet are sometimes overcorrected, which leaves them unlevel when viewed from the front standing on a hard, level surface or when the foot is held up and the solar surface is inspected. These conditions can cause orthopedic problems in training, and only mild imbalances are acceptable. The narrow, boxy foot is probably a lesser evil than the broad, flat foot with collapsed heels.
- Lameness rarely is seen at sale examinations, but it can be difficult to spot because routine examination only involves inspection at the walk. Trotting can confirm or deny lameness. Ataxia can be difficult to discern because the frequently inspected yearling tires during the day and can appear weak. Also, some yearlings are given mild sedatives to control behavior, which may give a similar effect.
- Presale injuries are common. Yearlings travel by road or ferry, and knocks and abrasions often cause more distress to the vendor than the animal. Capped hocks are particularly common but rarely important.

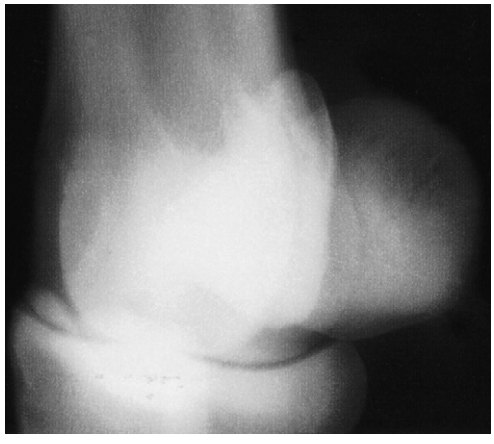

Fig. 102-1 Dorsolateral-palmaromedial oblique radiographic view of a metacarpophalangeal joint. Irregularity and enthesophyte formation on the abaxial margin of the lateral proximal sesamoid bone reflect sesamoidits. (Courtesy I.M. Wright, Newmarket, England.)

Ultrasonographic Examinations

Ultrasonographic examinations have been introduced by some purchasers. Ultrasonography has been used mainly to measure dimensions of the cardiac chambers, but some horses that have had injuries or other soft tissue anomalies may be offered with ultrasonographic images provided by the vendor or, with the vendor's permission, may be examined by ultrasonography by a purchaser's veterinarian. Conditions that may warrant such an examination to give purchasers confidence include thickening of suspensory ligament branches or fleshy superficial digital flexor tendons (SDFTs). The former is seen distally where the suspensory ligament branch attaches to the proximal sesamoid bone and usually results from a mild strain. Whatever the fiber pattern appearance in these horses, they often need considerable patience in training as 2-year-olds but ultimately can stand racing. Fleshy tendons are a thickening or bowing of the SDFT in the forelimbs that is seen in yearlings or 2-year-olds in training. They show minimal or no soreness on digital pressure and on ultrasonographic examination have an increased cross-sectional area of the middle third of the tendon and a coarseness of fiber pattern, but no core lesion or accumulation of fluid (Fig. 102-2). These horses do well if they are shown patience in training (horses with mild lesions are capable of light cantering exercise, but not galloping) and are not given aggressive veterinary treatment. With regular monitoring the tendon(s) usually thins and straightens to normal during the summer months, and many horses can be raced as 2-year-olds. This condition is probably an adaptive mild inflammation, because it occurs in yearlings that have had no managed exercise and in 2-year-olds in early training. Fleshy tendons should not be considered in the same serious manner that a strain or core lesion would demand in an older horse and pose only a slightly greater risk of tendonitis later in the racing career. Local anti-inflammatory or counterirritations are sometimes applied, but the efficacy is dubious.

NORTH AMERICAN STANDARDBRED SALES YEARLING

• Mike W. Ross

Veterinary inspection of the Standardbred (STB) sales yearling at the major sales is limited compared with that of the North American TB yearling. At most sales the number of veterinarians inspecting horses for their own potential purchases is similar to the number of those hired by owners and trainers to provide opinions.

Location and Time of Major Sales

The major sales take place in the fall in Kentucky and Pennsylvania, with smaller sales elsewhere. Small consignors may sell directly through the sales company, but large breeding farms or well-known agents sell most STB sales yearlings. Separate dispersal sales of broodmares, sucklings, and weanlings occur with the yearling sales or at a later date. Horses of racing age sell at various sales across North America, but no specific sales occur for 2-year-old horses in training.

Conditions of Sale

All STB sales yearlings sell as is, meaning when the hammer falls, the buyer owns the yearling in the condition in which it was sold and must pay according to the conditions of sale. The sales companies are not responsible for determining the suitability of the horse for racing or for unsoundness discovered after the sale. The sales company announces the yearling's sex and reproductive changes (gelding, spayed, ridgeling, cryptorchid), if the yearling was conceived by embryo transfer, and if the horse has been "nerved." Consignors sign a contract with

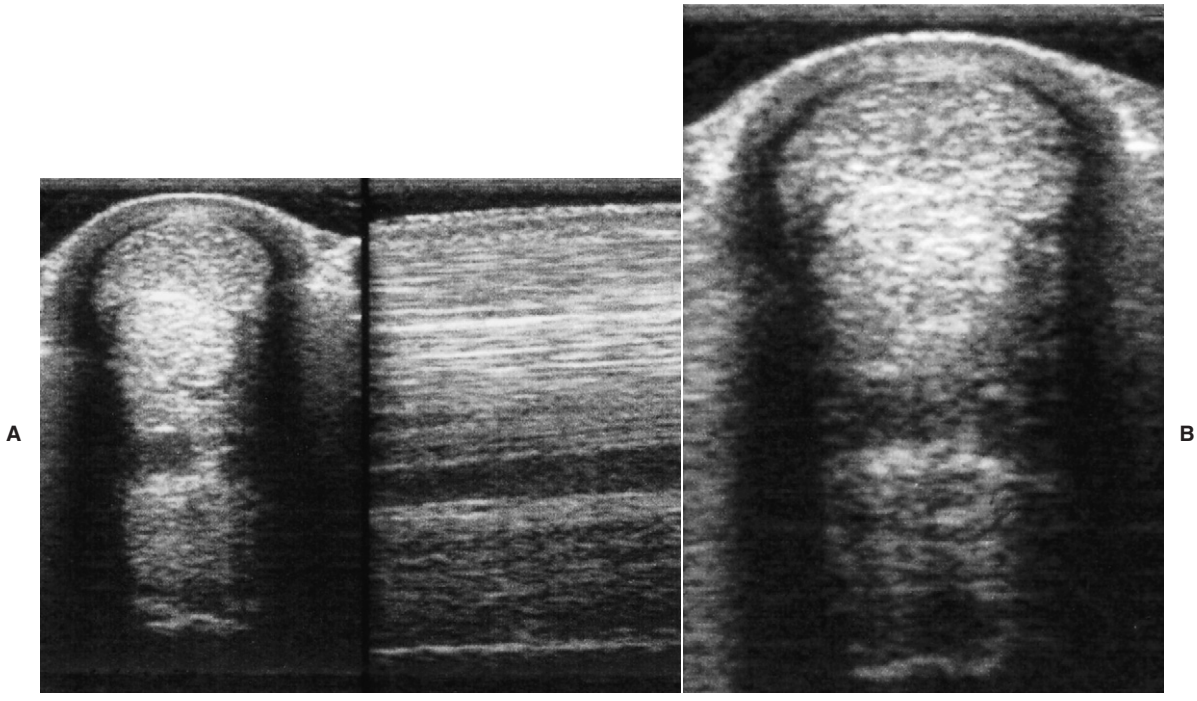

Fig. 102-2 **A,** Transverse (on the left) and longitudinal ultrasonographic images of the mid-metacarpal region of a 2-year-old Thoroughbred with fleshy tendons. There is subcutaneous edema and enlargement of the superficial flexor tendon. **B,** Enlarged transverse image seen in **A.** The entire cross-sectional area of the tendon has a diffuse reduction in echogenicity.

the sales company that includes details about remediation if a dispute regarding soundness occurs, but the legal responsibility is with the consigner, not the sales company. Arbitration, according to the laws of the state in which the yearling was sold, is used if a suitable arrangement cannot be made.

Consignors, usually large farms acting as agents or independent sales agents, have a separate contract with individual owners to represent them at a sale. Owners are required to give information about neurological status, cribbing, fractures, neurectomy, or unsoundness of sight or wind at least 30 days before sale. The consignor determines if information regarding these conditions is announced at the sale, but no contract stipulates that known conditions, such as lameness or previous surgery, be announced at the sale. To prevent disputes and to preserve their integrity, consignors may request that surgical procedures, such as arthroscopy to remove osteochondritis dissecans fragments, or other pertinent information are announced. Verbal announcements and the availability of presale radiographs and surgical reports may be detrimental to the sales price, despite resolution of the condition, although removal of tarsocrural osteochondritis dissecans fragments is usually acceptable. Philosophical differences exist between consignors and only some provide presale information. Thus little incentive exists to disclose information or to investigate problems before sale. Many owners prefer that problems are not investigated so that the yearling may be consigned without disclosure.

Resolution of Disputes

The consignor is primarily responsible for adjudicating conflicts about STB sales yearlings. Because sales companies stipulate yearlings are sold as is and arbitration is the final remedy for disputes, consignors often are forced to provide flexible remediation when disputes arise. If consignors are owners, they deal with problems directly with buyers. If consignors are agents for owners, they become the intermediary. In most instances consignor and buyer attempt to avoid arbitration. Problems arise when a condition is found after the sale, usually on postsale radiographs, that is judged to be detrimental to the yearling's future potential as a racehorse. The buyer refuses to pay and contacts the consignor. Influential large buyers can often stand their ground, and the consignors may refund money, give credit to the buyer for the future purchase of a yearling, pay for surgery if necessary, or lower the sales price. If agreement cannot be reached, arbitration ensues. The high prevalence of osteochondritis dissecans fragments means that most disputes concerning these are judged in favor of the consignor. Disputes regarding carpal chip or other fractures, osseous cyst-like lesions, extensive radiolucency of the proximal sesamoid bones, or unusual osteochondritis dissecans or traumatic conditions often are resolved in favor of the buyer. Fractures are considered important, and the term should be avoided if a condition is known by most to be a manifestation of osteochondritis dissecans. Failure to disclose a condition is most difficult to prove but often is suspected by disgruntled owners.

Role of the Veterinarian

Because repository radiographs are not available, the veterinary presence at major sales is minimal. Veterinarians are sometimes asked to accompany buyers to farms before sale, or to the sale itself, but are handicapped by not having the ability to review radiographs. Influential buyers may be able to convince consignors to allow radiographic and endoscopic examinations and may otherwise threaten not to bid on horses. Veterinarians often are asked to judge conformation and interpret what effect abnormalities may have on future race performance. They may be asked to review pedigrees, but in general, trainers and owners are extremely knowledgeable. Veterinarians are usually prohibited from performing radiographic and endoscopic examinations at a sale or, if they are allowed to do so, must perform the examinations after normal

sales hours. However, these examinations usually are allowed for horses of racing age.

Conformation

A definite relationship between conformation and the development of certain lameness conditions exits in the STB sales yearling (see Chapter 109). A difference in the relevance of abnormal conformation exists between a trotter and pacer. Sickle-hocked conformation is acceptable in a pacer, unless severe, but in a trotter this conformation leads to lameness. A sickle-hocked trotter is usually fast but lame. Mild sickle-hocked conformation is considered by some to be desirable in a pacer, but in horses of either gait, this abnormality predisposes to curb, osteoarthritis, and fracture of the distal hock joints (see Chapter 79). In-at-the-hock conformation is undesirable in the trotter and pacer. Calf-knee or back-at-the-knee conformation is prevalent in STB yearlings, and if mild and in a pacer the conformation is acceptable, but in a trotter this abnormality should be avoided. Any conformational abnormality of the carpus in a trotter should be considered carefully. Bench-knee or offset-knee conformation is undesirable in a trotter or pacer. Tied-in below the knee leads to superficial digital flexor tendonitis, especially in a pacer. I believe TB racehorses can tolerate clubfoot conformation better than STBs, and this abnormality should be avoided. Metacarpal and metatarsal exostoses are common, but usually of little concern, unless large or proximally located near the carpus and tarsus, respectively.

Videotapes of yearlings in action (loose exercise in paddocks or jogging on the track) are usually available and are thought to be much more informative in trotters than in pacers. Desirable characteristics of trotters include a wide gait behind and a narrow gait in front. An acceptable trotter can be base narrow and toed out in front, because hindlimbs often land lateral to the forelimbs (called *passing gaited*), and interference injury is minimal. Base-narrow, toed-out conformation predisposes a pacer to considerable interference injury. In pacers, base-wide forelimb conformation is acceptable, whereas in a trotter this predisposes to interference. Toed-in conformation, if mild, is acceptable in a pacer but not in a trotter, because interference injury and lateral branch suspensory desmitis are problems.

Radiographic Examination

Because no repository for radiographs is provided and many consignors actively discourage presale radiography, radiographs are usually unavailable. However, some farms examine all Standardbred sales yearlings radiographically before the sale, remove osteochondritis dissecans fragments, and have radiographs available on request. Because osteochondritis dissecans fragments of the tarsocrural joint generally cause effusion in late weanlings and early yearlings, these often are removed surgically before sale, but this may not be announced. Radiographs may be available for other types of injuries, such as lumps or bumps in the metacarpal or metatarsal region or elsewhere, to demonstrate that the injury is unlikely to affect future race performance.

Radiographs commonly are obtained after sale and may lead to questions about future soundness and, in some horses, arbitration. In general, radiographic changes that affect racing performance are radiolucent defects of the proximal sesamoid bones (sesamoiditis and osseous cyst-like lesions), large conglomerate osteochondritis dissecans fragments involving the plantar processes of the proximal phalanges (intra-articular and extra-articular fragments) in trotters, osseous cyst-like lesions of the distal aspect of the proximal phalanx and middle and distal phalanges, large osteochondritis dissecans lesions of the lateral trochlear ridge of the femur and subchondral bone cysts in the medial femoral condyle, single or multiple fragments located in the distal sesamoidean ligaments (rare form of fragmentation seen in the forelimb, thought to be a manifestation of osteochondritis dissecans), carpal chip fracture, and various forms of osteoarthritis. The mere presence of osteochondritis dissecans fragments does not necessarily affect sales price if the condition is known before the sale, and certainly in most yearlings, does not preclude future soundness and successful racing. The most common sites of osteochondritis dissecans involve the tarsocrural joint and the metatarsophalangeal and metacarpophalangeal joints. Once discovered, these fragments are often removed prophylactically before training begins. Management of fragments in the metatarsophalangeal joint is discussed elsewhere (see Chapter 43).

Future Direction

Many disputes occur between buyers and consignors concerning abnormalities in postsales radiographs, lameness, or neurological disease. Determination of when a lesion developed, if a true fracture exists, real or perceived effects on prognosis, and chain of custody (in whose care the yearling was when the problem was noticed) complicate final payment. These issues generally are resolved between the consignor and buyer (see the previous discussion), but occasionally arbitration is necessary and usually favors the consignor. Availability of presale radiographs in a repository would be preferable and a strong forward step for the STB industry, and although this change is not yet well accepted, mandatory radiographic examination is gaining support from some of the most influential owners and agents.

CHAPTER • 103

Biochemical Markers of Bone Cell Activity

Joanna Price

Bone is a complex tissue that undergoes change throughout life by the processes of bone formation by osteoblasts and bone resorption by osteoclasts. Bone remodeling is necessary for bone health and allows the skeleton to respond rapidly to changes in its internal and external environment.[1] Bone formation and resorption are coupled; the cycle begins with the recruitment of osteoclast precursors, which attach to the bone surface and resorb the subjacent matrix. Osteoblasts then differentiate from mesenchymal precursors and fill in the resorption pit with new bone. In the healthy adult, skeleton formation and resorption are balanced. However, changes in this balance occur during growth in response to increased or decreased exercise, as a consequence of hormonal changes, during aging, after therapeutic intervention, in metabolic bone disease, or in response to damage. One of the challenges facing researchers in equine orthopedics is development of sensitive and specific non-invasive methods to evaluate these changes in bone turnover in vivo.

Long-term changes in bone mass and structure can be assessed using techniques such as quantitative ultrasound, dual-energy x-ray absorptiometry, or quantitative computed tomography.[2-4] However, for changes in bone mass and architecture to be of a sufficient magnitude that they can be detected using these methods may take several months. Furthermore, quantitative computed tomography and dual-energy x-ray absorptiometry cannot be used in the conscious horse, and use currently is restricted to ex vivo or research studies. In contrast, biochemical markers measure dynamic changes in bone cell activity and can be measured in body fluids using straightforward methods, which are inexpensive.[5] Bone marker measurements can be repeated at frequent intervals and so are convenient to use in the field. Presently, biochemical markers of bone cell activity remain research tools, and most published studies reviewed in this chapter relate to fundamental aspects of bone marker research (such as assay validation and sources of variability). However, an increasing amount of work is being undertaken by several groups on the potential clinical applications of bone markers in horses. Progress in equine bone marker research has been facilitated greatly by the increased use of bone turnover markers in human clinical research relating to metabolic bone disease, particularly osteoporosis.[6,7] In fact, most equine studies have used markers originally developed for people, and many have involved successful collaborations between veterinary and medical research laboratories.

Biochemical markers of bone cell activity are classified as markers of bone formation or of bone resorption, although some reflect changes in both processes. In general the markers are enzymes expressed by osteoblasts, osteoclasts, or organic components released during the synthesis and resorption of bone.[8] Because each marker may reflect a different physiological process in bone, assaying for a combination of markers is preferable, because this provides more information on bone (re)modeling rates. Furthermore, studies in people have shown that in certain diseases individual markers yield more useful information than others.[9]

Box • 103-1

Abbreviations Used in Discussion of Bone Cell Activity

ALP	Alkaline phosphatase
BAP	Bone-specific alkaline phosphatase
BMC	Bone mineral content
CTX	Type I collagen C-terminal telopeptide
DEXA	Dual-energy x-ray absorptiometry
DYR	Deoxypyridinoline
GGHyl	Glucosylgalactosylhydroxylysine
Ghyl	Galactosylhydroxylysine
HPLC	High-performance liquid chromatography
Hyp	Hydroxyproline
ICTP/CTX-MMP	Carboxyterminal cross-linked telopeptide of type I collagen
OC	Osteocalcin
PICP	Type I collagen carboxyterminal propetide
PYR	Pyridinoline
QUS	Quantitative ultrasound
QCT	Quantitative computerized tomography
RIA	Radioimmunoassay

An ideal biochemical marker should be measurable in body fluids (urine, serum, or plasma) by a sensitive and specific technique and should be specific to its tissue of origin. Other criteria that determine the value of a biochemical marker are whether the factors that control its synthesis and metabolic pathway are understood and the factors that influence its biological variability. To date, little is known about factors that regulate the synthesis and metabolism of bone markers in horses.

Some common abbreviations used in discussion of bone cell activity are found in Box 103-1.

BIOCHEMICAL MARKERS OF BONE FORMATION

Bone formation markers are synthesized by osteoblasts and are all measured in serum or plasma (Table 103-1).

Alkaline Phosphatase

The enzyme alkaline phosphatase is associated with the osteoblast plasma membrane and is required for osteoid formation and matrix mineralization. Total serum alkaline phosphatase is derived from multiple sources and thus is not a specific marker

Table • **103-1**

Markers of Bone Formation

MARKER	METHODS USED	BODY FLUID	SOURCE	COMMENTS
Total alkaline phosphatase	Colorimetric	Serum	Bone, liver, kidney, placenta	Not bone specific
Bone-specific alkaline phosphatase	Elecrophoresis Precipitation ELISA	Serum	Bone	Some cross-reactivity with liver alkaline phosphatase
Osteocalcin	RIA ELISA	Serum	Bone	Specific osteoblast product
Type I collagen propeptide	RIA	Serum	Type I collagen in bone, skin, tendon, and other soft tissues	May be contribution from tissues other than bone

ELISA, Enzyme-linked immunosorbent assay; *RIA,* radioimmunoassay.

of bone formation. However, tissue-specific alkaline phosphatase is posttranslationally modified, and this provides the basis for a number of methods that have been used to separate and quantify different equine alkaline phosphatase isoenzymes. Bone alkaline phosphatase predominates in serum during growth, although the proportion of the bone isoform decreases in adults.[10,11] No studies have yet described changes in circulating bone alkaline phosphatase associated with equine musculoskeletal disease. However, alkaline phosphatase activity increases in subchondral bone in osteochondrosis,[12] which indicates that bone-specific alkaline phosphatase potentially may be a useful marker of this condition, and this requires further investigation.

Osteocalcin

Osteocalcin, otherwise known as bone Gla protein, is the most abundant noncollagenous protein in bone, and a small fraction of the protein is released into the circulation following synthesis by osteoblasts. Osteocalcin fulfils one of the main criteria for a bone marker: it has tissue specificity. Several studies have described the measurement of osteocalcin in horses, and the work of Olivier Lepage has made a substantial contribution.[13-16] Osteocalcin is the most labile of the bone markers, thus samples should be processed rapidly, and longer-term storage should be below –25° C.[17] Osteocalcin levels are affected by anesthesia; therefore sampling during surgery should be avoided.[18]

Carboxyterminal Propeptide of Type I Collagen

Type I collagen is the most abundant collagen in bone, and the procollagen molecule contains a carboxyterminal extension domain that is split off before fibril formation and released into the circulation. The carboxyterminal propetide of type I collagen provides a measure of newly synthesized type I collagen and is normally measured by radioimmunoassay (RIA).[19] However, type I collagen is not bone specific and synthesis in other soft tissues may contribute to carboxyterminal propetide concentrations. For example, increased levels have been observed following tendon injury,[20] and a peak occurs in carboxyterminal propetide levels during rapid weight gain in growing Thoroughbreds.[21]

BIOCHEMICAL MARKERS OF BONE RESORPTION

Most bone markers that reflect resorption of bone matrix are degradation products of type I collagen[22] (Table 103-2). Until recently these markers generally were measured in urine, but this clearly presents practical difficulties in the horse. Fortunately, several of the newer markers developed for people can be measured in serum, and this may increase the repertoire of resorption markers available for use in horses.

Hydroxyproline

Hydroxyproline is formed from the posttranslational hydroxylation of proline. However, hydroxyproline now is used rarely as a resorption marker because of poor specificity. Concentrations reflect collagen turnover in bone, cartilage, skin, and other soft tissues. Hydroxyproline was first measured in horses more than 25 years ago.[23]

Hydroxylysine Glycosides

Hydroxylysine glycosides occur in two forms, glucosylgalactosylhydroxylysine and galactosylhydroxylysine, which are released during collagen degradation. Bone contains mostly galactosylhydroxylysine and this has been used as a measure of bone resorption in people.[24] We have adapted a human high-performance liquid chromatography assay for measuring galactosylhydroxylysine in horse serum, and levels are correlated significantly with concentrations of another bone resorption marker, the carboxyterminal telopeptide of type I collagen.[25,26]

Pyridinium Cross-Links of Collagen

Cross-links of fibrillar collagen are formed extracellularly in the telopeptide domains and stabilize collagen molecules.[27] If the telopeptide interacts with a hydroxylysine residue in the triple helix, the mature cross-link is pyridinoline found in cartilage, bone, ligaments, and vessels. If the residue is lysine, the cross-link is deoxypyridinoline, which is found almost exclusively in bone. Pyridinium cross-links in body fluids mainly are derived from bone and for many years deoxypyridinoline, measured by high-performance liquid chromatography, has been considered the gold standard of resorption markers. Recently a more convenient enzyme-linked immunosorbent assay has been used to measure the free fraction of urinary deoxypyridinoline in horses and the assay also can be adapted for measuring serum deoxypyridinoline.[28,29]

Cross-Linked Collagen Telopeptides

Cross-links are located at both termini in the type I collagen molecule. A number of peptide assays measure telopeptides generated during resorption. The first of these assays to be developed measured the carboxyterminal telopeptide of type I collagen (ICTP or CTX-MMP), which has been studied extensively in horses, mainly because for several years it was

Table • **103-2**

Markers of Bone Resorption

MARKER	METHODS USED	BODY FLUID	SOURCE	COMMENTS
Hydroxyproline	Colorimetric HPLC*	Urine	All recently formed and mature fibrillar collagens	Not bone specific
Deoxypyridinoline	HPLC ELISA	Urine Serum	Mature collagen in bone and dentine	Good specificity "Gold standard"
Pyridinoline	HPLC ELISA	Urine Serum	Mature collagen in bone and cartilage	Some contribution from cartilage
Carboxyterminal cross-linked telopetide of type I collagen	RIA	Serum	Type I collagen in bone and skin	May be contribution from tissues other than bone
Carboxyterminal cross-linking telopeptide of type I collagen	ELISA ECLA	Urine Serum	Type I collagen	Good specificity Preliminary results suggest equine cross-reactivity
Hydroxylysine glycosides	HPLC	Urine, serum	Bone and soft tissue collagen but enriched in skeletal tissues	High proportion in bone

* *HPLC,* High-performance liquid chromatography; *ELISA,* enzyme-linked immunosorbent assay; *RIA,* radioimmunoassay; *ECLA,* enhanced chemiluminescence assay.

the only marker of resorption that could be measured in serum.[16,19] Recently an immunoassay has been developed that recognizes the C-terminal telopeptide of type I collagen containing an isoaspartyl peptide bond[30] (CTX, Grosslaps Osteometer, BioTech, ALS), and a preliminary report describes its measurement in horse serum.[29]

FACTORS THAT INFLUENCE BIOCHEMICAL MARKERS OF BONE CELL ACTIVITY

Because bone markers reflect instantaneous changes in bone cell activity, a large number of factors can be a source of preanalytical variability. These factors recently have been classified as controllable (e.g., food intake, circadian changes, or exercise) or uncontrollable (e.g., gender, age, or intercurrent disease).[31] A failure to define and appropriately manipulate controllable sources of variability and to account for uncontrollable variability limits interpretation of the results of bone marker measurements in clinical studies.

Circadian Variability

Circadian variability may have a significant effect on markers of bone turnover, particularly urinary markers of bone resorption.[31] Black et al.[32] described circadian rhythms in urinary pyridinoline and deoxypyridinoline excretion in adult geldings, with peak levels between 2 and 8 AM. Circadian rhythmicity in osteocalcin concentrations also has been described, although this remains somewhat controversial.[14,32] Until further data on the circadian changes in other bone markers is available, the safest approach is to assume that this is potentially an important source of variability, and in any study samples should be collected at the same time of day.

Diet

Little is known about the influence of diet on bone turnover markers in horses. Lepage et al.[14] did not observe a short-term effect of feeding on osteocalcin concentrations,[14] and a more recent study found no effect of dietary calcium on plasma hydroxyproline or osteocalcin.[33] However, food intake can influence bone marker concentrations significantly in people and may exacerbate biological variability. For example, fasting reduces the circadian variation in serum levels of C-terminal telopeptide of type I collagen[34] and, although this marker can predict osteoporotic fracture if samples are collected during a fast, the marker has no predictive value in samples collected under uncontrolled conditions.[35] Another potential influence on bone turnover is dietary mineral supplements, which are widely used in horses. For example, higher hydroxyproline levels were reported in ponies fed increased levels of aluminum, which may reflect altered bone turnover.[36]

Seasonal Changes

Some evidence shows that time of year may influence bone turnover, and this may be particularly important during growth. Maenpaa et al.[37] observed some monthly variability in alkaline phosphatase and osteocalcin concentrations in Finnhorse foals, and we have seen an increase in bone turnover markers in Thoroughbred yearlings between midwinter and early summer.[21]

Age

In horses, as in people, biochemical markers of bone turnover are higher during skeletal growth than in adults. Age-related decreases in several bone markers have been described, including osteocalcin,[13,38] bone-specific alkaline phosphatase,[10,11,21] carboxyterminal propetide of type I collagen, carboxyterminal telopeptide of type I collagen,[19] pyridinoline, and deoxypyridinoline.[38] The decrease is most significant during the first year of life and levels do not tend to plateau until animals reach 3 to 4 years of age. Markers of bone formation and bone resorption show a similar pattern of change, because bone modeling involves resorption of bone at some sites (e.g., the endosteum) and formation at others (e.g., periosteal surfaces and the metaphysis). Clearly any study of bone markers must control for the effects of age, and this is particularly important in the young horse, when serial measurements (i.e., use of the horse as its own control) are likely to be most informative. A number of developmental orthopedic diseases occur during growth, and finding a biochemical

marker that could be used to identify animals that have or are at risk of abnormal skeletal development would be of great value. Already some evidence shows that bone turnover markers may be altered in developmental orthopedic disease. Osteocalcin concentrations were lower in a foal with generalized dyschondroplasia and vertebral epiphysiolysis,[39] and in a small cross-sectional study of horses with osteochondritis dissecans, we found that carboxyterminal telopeptide of type I collagen concentrations were elevated, indicating increased bone resorption.[21]

Gender and Breed

Chiappe et al.[38] described higher osteocalcin levels in Thoroughbred fillies between 24 and 36 months of age, whereas no gender difference was observed in younger horses. However, Lepage et al.[13,16] found no influence of sex on osteocalcin concentration in Standardbreds, or on osteocalcin, or carboxyterminal telopeptide of type I collagen concentrations in Warmblood or Draft horses more than 4 years old.

Ethnic differences in bone turnover have been described in people,[31] and horse type also must be considered as a potential effect on bone markers. Lepage et al.[16] showed that concentrations of osteocalcin were lower and carboxyterminal telopeptide of type I collagen levels higher in Draft horses compared with Warmbloods and suggested that this may relate to different rates of bone remodeling in the two breeds.

Disease

At this stage little is known about the effect of different diseases on markers of bone turnover in the horse. Clearly any medical condition that affects bone metabolism influences marker concentrations (e.g., nutritional hyperparathyroidism), as does any disease that changes marker clearance (e.g., liver or kidney disease). In equine athletes, undiagnosed musculoskeletal conditions unrelated to the problem under investigation could lead to misinterpretation of results.

POTENTIAL CLINICAL APPLICATIONS OF BONE MARKERS

The studies reviewed in the previous section have shown that bone markers provide a sensitive, straightforward method for monitoring bone cell activity in horses in vivo and have made a substantial contribution to our understanding of cellular processes in equine skeletal tissues. Although the value of bone markers as clinical tools has yet to be established, a significant amount of research is being undertaken in this area. It is important that the studies designed to test the clinical validity of bone and cartilage markers address specific hypotheses, use carefully selected populations of animals, and standardize outcome measures. Furthermore the clinical validity of markers can be established only if their inherent biological variability is controlled. More work also is required to improve knowledge of the factors that influence the metabolism and clearance of biochemical markers in horses. Continued collaboration between basic scientists and clinicians is important if progress is to be made, and collaboration between different laboratories will ensure the standardization of assays and that protocols are established for sample collection and storage.

Because bone markers reflect turnover in the *whole* skeleton and are affected by numerous variables, in my opinion it is unlikely that any single bone marker measured on one occasion will be able to function as a diagnostic test with a high level of discriminatory power. However, in horses as in people, the most useful applications of bone markers, if measured serially in the same horse, are likely to be as methods for identifying horses at risk of injury and as objective measures for monitoring treatment.[40-42] A further research application for the markers is as a method for monitoring the effects of exercise on bone cell activity.

Injuries to the skeleton are a major cause of morbidity and mortality in the equine athlete, and any method that could help identify animals at risk of injury before clinical signs develop would have significant benefit from welfare and economic perspectives. One study has shown that bone markers can be used to predict osteoporotic fracture in women, although this issue remains controversial.[40] A pilot study that we have undertaken recently showed that a single bone marker measurement taken at the start of the training season did not predict fracture risk in 2-year-old Thoroughbreds.[43] However, the study included fewer than 200 horses and a variety of fracture types. Larger numbers of horses would need to be studied over a long follow-up period to address the question of whether bone markers can predict fracture. It is also likely that using bone markers with another non-invasive measure of bone structure (e.g., quantitative ultrasound) will improve prediction of fracture risk.

USE OF BONE MARKERS TO STUDY THE EFFECTS OF EXERCISE

The extreme responsiveness of the bone to changes in its mechanical environment ensures that the skeletal mass and architecture are appropriate to prevent injury.[44] Evidence that biochemical markers provide a potentially valuable tool for studying the effect of exercise on short-term changes in bone cell activity is accumulating. Lepage et al.[16] suggested that a higher ratio of osteocalcin to carboxyterminal telopeptide of type I collagen in Warmblood compared with Draft horses may reflect a positive remodeling response in horses having regular daily work. We observed increased carboxyterminal propetide of type I collagen, carboxyterminal telopeptide of type I collagen, and bone-specific alkaline phosphatase concentrations in 2-year-olds exercised on a treadmill for 18 months. These results may reflect increased remodeling, caused by accumulated fatigue damage, associated with a long period of training on a hard surface.[45] In contrast a shorter period of treadmill training was associated with decreased carboxyterminal telopeptide and osteocalcin concentrations.[46] A decrease in osteocalcin also was observed when Quarter Horses commenced race training.[47] Of particular interest is a recent observation that marker levels change as the intensity of work increases during race training.[43] However, if a training regimen is not sufficiently osteogenic, changes in bone turnover may not be revealed using bone markers. For example, lunging yearling Quarter Horses had no effect on osteocalcin levels,[48] and no change on deoxypyridinoline or osteocalcin concentrations was observed when previously stabled Arabian horses returned to training.[38]

Although increased loading has anabolic effects on bone, disuse leads to increased resorption, and this can occur when a horse is transferred from pasture to a stable or result from a clinical condition that leads to restricted exercise. Bone markers have shown that immobility can lead to altered bone cell activity, which may predispose horses to injury. For example, when Arabian yearlings were confined to a stable, concentrations of the resorption marker deoxypyridinoline increased, whereas levels of the formation marker osteocalcin decreased. These changes were associated with a decrease in bone mineral content.[28] A decrease in osteocalcin levels after transfer of foals from pasture to winter stabling also has been described.[37] In a study of horses with acute tendon injury we observed that urine pyridinoline and deoxypyridinoline concentrations were increased. This probably reflected increased bone resorption associated with disuse of the affected limb.[49]

MONITORING RESPONSES TO THERAPY

To date, monitoring responses to therapy has proved to be one of the most valuable applications of bone markers in human medicine. The newer bone resorption markers in particular provide a sensitive measure of the effects of antiresorptive agents on bone turnover in human metabolic bone disease.[42] In my opinion, using biochemical markers to monitor the effects of treatment may prove to be one of the most important applications for the bone and cartilage markers in equine orthopedics. In the twenty-first century the veterinary profession must embrace evidence-based medicine, and this will require using objective measures, which should include biochemical markers, to monitor the effects of different therapeutic regimens on skeletal tissues in prospective clinical trials.

Some evidence already shows that bone markers in horses may be useful for monitoring responses to therapy and also for assessing the potentially harmful effects of drugs on the equine skeleton. For example, bone markers have shown that corticosteroids have a negative effect on osteoblast activity, and in the long term this could lead to osteoporotic changes in bone. Osteocalcin levels were found to be decreased significantly after intravenous, intramuscular, and oral administration of dexamethasone and triamcinolone acetonide.[50,51] Levels did not decrease after intra-articular injection of methylprednisolone acetate, which suggests that this route of steroid administration may not have long-term adverse effects on bone.[51] A recent study showed that serum bone-specific alkaline phosphatase did not reflect decreased mineral apposition rate associated with phenylbutazone administration.[52] However, the study was short term, and studies in people have shown that changes in bone formation markers may not occur for several months after treatment. We recently showed that bone marker levels were increased after administration of growth hormone in horses and thus could be useful as indirect measures for detecting its abuse in racehorses.[53]

In conclusion, biochemical markers of bone turnover provide a simple, inexpensive, non-invasive method for studying changes in the activity of groups of cells forming and resorbing bone. To the basic scientist, biochemical markers can contribute significantly to an understanding of the cellular mechanisms that underlie normal bone development and remodeling. To the clinical scientist, bone markers offer an opportunity to study the pathogenesis of disease and improve strategies for reducing the morbidity and mortality associated with a variety of orthopedic conditions in horses.

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CHAPTER • 104

The Bucked Shin Complex

ETIOLOGY, PATHOGENESIS, AND CONSERVATIVE MANAGEMENT

• David M. Nunamaker

Conditions of fatigue failure of bone and inadequacy of bone remodeling of the third metacarpal bone (McIII) in the racehorse are part of a condition known as *bucked shins* or *dorsal metacarpal disease.*[1,2] Bucked shins start in young healthy racehorses, usually Thoroughbreds (TBs) and Quarter Horses, but occasionally Standardbred (STBs), that undergo intense training for racing, usually as 2-year-olds, while the skeleton is still immature and in the growth phase (Fig. 104-1, *A*). The true incidence of bucked shins is unknown and may vary geographically, but reports range from 30% to 90%. A North American questionnaire cited an incidence of 70%.[1] Stress fractures (dorsal cortical or saucer fractures) usually occur some months after initial signs of bucked shins and may be a potential life-threatening injury if a horse is raced or exercised at speed (Fig. 104-1, *B*). The diagnosis of bucked shins is easy and often made by the trainer or owner. The history of sudden tenderness or soreness of the left (in North America) or both

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CHAPTER • 104

The Bucked Shin Complex

ETIOLOGY, PATHOGENESIS, AND CONSERVATIVE MANAGEMENT

• David M. Nunamaker

Conditions of fatigue failure of bone and inadequacy of bone remodeling of the third metacarpal bone (McIII) in the racehorse are part of a condition known as *bucked shins* or *dorsal metacarpal disease.*[1,2] Bucked shins start in young healthy racehorses, usually Thoroughbreds (TBs) and Quarter Horses, but occasionally Standardbred (STBs), that undergo intense training for racing, usually as 2-year-olds, while the skeleton is still immature and in the growth phase (Fig. 104-1, *A*). The true incidence of bucked shins is unknown and may vary geographically, but reports range from 30% to 90%. A North American questionnaire cited an incidence of 70%.[1] Stress fractures (dorsal cortical or saucer fractures) usually occur some months after initial signs of bucked shins and may be a potential life-threatening injury if a horse is raced or exercised at speed (Fig. 104-1, *B*). The diagnosis of bucked shins is easy and often made by the trainer or owner. The history of sudden tenderness or soreness of the left (in North America) or both

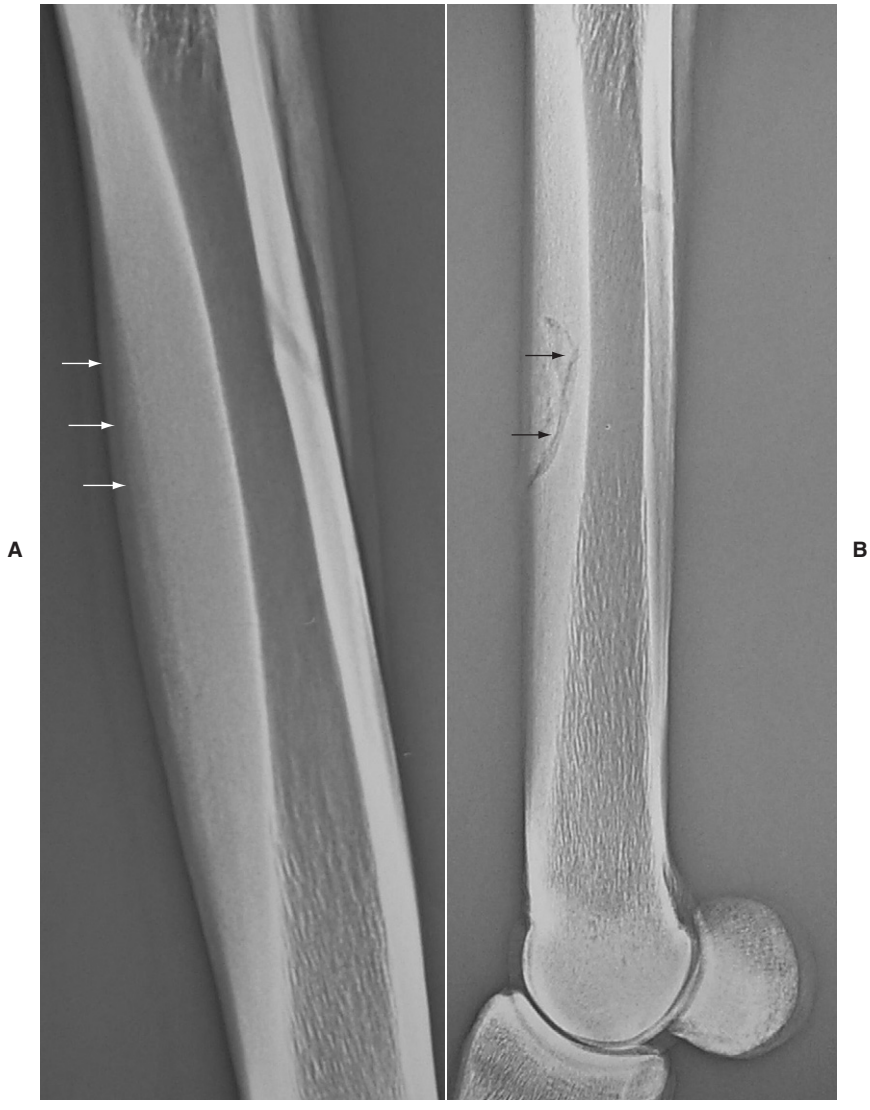

Fig. 104-1 **A,** Dorsolateral-palmaromedial oblique radiographic view of a third metacarpal bone. Periosteal new bone formation *(arrows)* over the dorsomedial aspect of the third metacarpal bone is evidence of bucked shins. **B,** Lateromedial radiographic view of the metacarpal region. This dorsal cortical fracture of the dorsolateral third metacarpal bone represents a common type of fatigue fracture *(arrows)* that usually occurs months after an episode of bucked shins.

McIIIs after high speed work or the first race, or soreness developing the day after, are cardinal signs of early bucked shins. Horses with severe disease manifest acute lameness and extreme sensitivity to palpation of the dorsal cortex of McIII and are unwilling to train or race. All gradations of pain or disability may be seen. Swelling and tenderness may suggest new bone proliferation in this area. Radiography is helpful to determine the amount of periosteal new bone formation, which determines prognosis. Large accumulations of periosteal new bone on the dorsal or dorsomedial surface of McIII suggest a serious imbalance between exercise and bone fatigue and may portend the actual stress fractures seen on the dorsolateral aspect of McIII some months hence.

RESEARCH FINDINGS

An understanding of the cause, pathomechanics, and pathogenesis of bone fatigue failure in the TB is helpful in determining prevention or treatment modalities and training regimens. It was formerly suggested that bucked shins resulted from microfractures on the dorsal aspect of McIII, caused by high-speed exercise. However, microfractures should heal without periosteal callus, which does not fit with the clinical observation of extensive periosteal new bone on the dorsomedial aspect of McIII. Work in our laboratory over the past 19 years has led us to propose a different cause of bucked shins[3] and an exercise program that may prevent bucked shins and stress fractures. The following summarizes our investigations.

Geometric Properties of the Third Metacarpal Bone: Comparison of Thoroughbreds and Standardbreds

The McIIIs of STBs and TBs of known age were examined, and comparisons were made between breeds of a particular age group and between the age groups of a particular breed. The second moments of area relate to bending stiffness in dorsopalmar and mediolateral directions and were used to determine the minimum and maximum principal moments of inertia (I_{min} and I_{max}). The most significant changes in the bone occurred at the midsections between the ages of 1 and 2, but

continued change occurred until age 3 or 4. I_{min} was smaller in the yearling TBs but larger in the adult TBs compared with STBs. During the first 2 to 4 years of life, I_{min} changed to a greater extent in TBs.[4]

In Vitro Comparison of Local Fatigue Failure of the Third Metacarpal Bone

Dumbbell-shaped specimens machined from adult McIII from TBs and STBs were tested in fully reversed cyclic bending experiments using a constant strain rotating cantilever model that measured load decrement. All tests were performed at 40 Hz and continued until the specimen broke or had a 30% loss of stiffness. Three different offsets were used to establish nominal strains of 7500, 6000, or 4500 microstrain in the specimens.

Data were analyzed using a power regression model for each horse and for each breed. Statistical differences were not found among the curves for individual horses of the same breed or for the curves between breeds. Pooled data then were used to describe fatigue characteristics of cortical specimens of McIIIs from TBs and STBs of various ages, subjected fully to reversed cyclic loading (Fig. 104-2).[5] The bone from young horses was much more susceptible to fatigue failure.

Because the in vitro fatigue was similar in TBs and STBs, other factors appeared to be important in the pathogenesis of fatigue failure of bone in the TB. This, together with the different inertial properties noted in STBs and TBs, led to the hypothesis that differences in training regimens or racing speeds between breeds might influence the incidence of disease.

Third Metacarpal Bone Stiffness Measurements

Whole bone stiffness measurements were made from horses of 2 months to 28 years of age using an Instron testing machine (Instron, Canton, MA) and a non-destructive three-point dorsopalmar bending test. The bones showed general increases in stiffness until they reached a plateau at about 6 years of age.

The material included McIIIs from twelve 2-year-old TBs, three of which had bucked shins. These three horses had differences in stiffness between the left and right McIIIs of 16% to 27%, respectively, whereas other trained or control 2-year-olds had considerably smaller left-right differences.[6]

In Vivo Strain Measurements: Relationship to Exercise

Bone strain in McIII was measured in horses of varying age, training at or near racing speed, by placing a rosette strain gauge on the dorsolateral aspect of McIII and recording using telemetry.[7] The mean peak compressive strain in four horses 2 years of age was –4841 ± 572 microstrain, compared with –3317 in a horse 12 years of age. One 2-year-old developed bucked shins, and its strain measurements were about 6 standard deviations greater than the other three.

After acquiring in vivo strain data, we correlated this data with in vitro fatigue data previously generated by determining the average number of cycles that a young TB would gallop in training before onset of bucked shins. The training records of six 2-year-old TBs that developed bucked shins were analyzed to determine the total distance worked before the onset of bucked shins. Stride length at canter, gallop, and racing speed was measured in a group of TBs to determine the number of strides (cycles) per mile. The total number of gait cycles was estimated based on the distances covered in a canter, gallop, and at work. The six horses were trained in these gaits between 10,000 and 12,000 cycles per month and developed bucked shins between 35,284 and 53,299 training cycles. This data was compared with the in vitro data described previously and showed good correlation (see Fig. 104-2).

Changing from the trot to the gallop changed the principal strain direction by more than 40° on the dorsolateral surface of McIII. Although trotting horses showed tensile strains in the long axis of the bone on the dorsal/dorsolateral surface, at racing speeds this same surface of the bone showed compressive strains.[8]

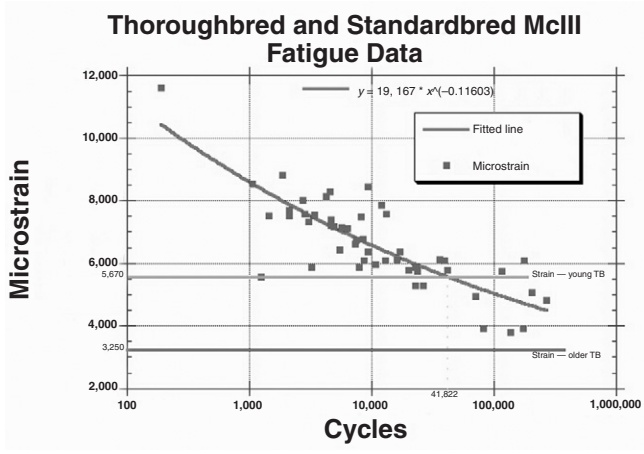

Fig. 104-2 In vitro fatigue data are plotted for the adult Thoroughbred and Standardbred. The regression line shows where the third metacarpal bone *(McIII)* will fail from repeated cycles. Superimposition of the strain levels of young Thoroughbreds shows that 41,822 cycles would result in fatigue failure. The superimposition of an older Thoroughbred shows more than a million cycles would be needed before bone failure.

Relationship of Exercise to Bone Fatigue

With the understanding that slow-speed gaits produce tensile strains on the dorsal surface of McIII, although high-speed exercise induces compressive strains in this same region, a study was undertaken to determine the effects of different training regimens and track surfaces on the modeling and remodeling of McIII in TBs.

Eight untrained 2-year-old TBs were divided into four groups of two horses each. Classical training methods were used for the horses in groups I and II. Group I horses trained on a dirt track. Group II horses trained on a wood chip track. Group III horses (control group) were not trained, but they were allowed free exercise in a large pasture. Group IV horses were trained using a modified classical training program on a dirt track.

The classical training program comprised daily gallops (~18-second furlongs, ~11.2 m/sec) of 1 to 2 miles per day (1.6 to 3.2 km), followed by shorter workouts or breezes at racing speed (~14-second furlongs, ~14.4 m/sec) once every 7 to 10 days that increased in distance from 2 to 6 furlongs (0.4 to 1.2 km) progressively over the course of the study. The modified classical training method used similar daily gallops, but the frequency of the high-speed workouts increased to three per week, while distances increased progressively from 1 to 4 furlongs (0.2 to 0.8 km). After 5 months the McIII was harvested in all horses. Microradiographs of bone sections were made to determine the extent of the remodeling activity (Fig. 104-3). Bone modeling on the periosteal and endosteal surfaces of McIII changed the cross-sectional geometry differently between the experimental groups. Classically trained horses (groups I and II) responded by appositional new bone formation on the dorsomedial periosteal surface, giving the impression that the medullary cavity, although reduced in diameter, was displaced laterally. Horses in the modified training group (group IV) had bone deposition dorsally and a slightly larger medullary cavity that was not displaced laterally. Control horses (group III) had new bone

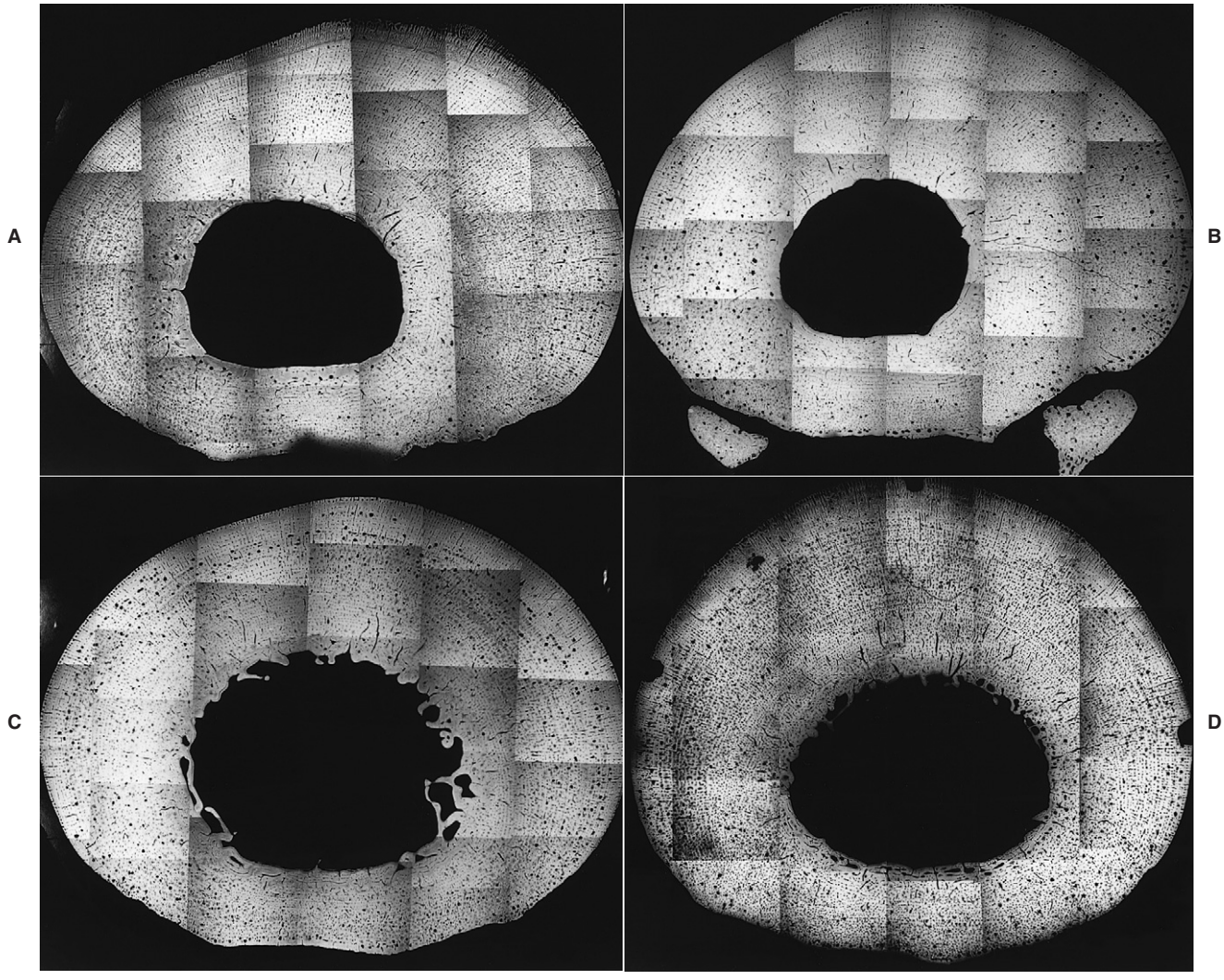

Fig. 104-3 Fifty percent length cross-sections of the third metacarpal bone were used to make these microradiographs. Individual photographs were taken with cross-sections magnified four times, and giant montages (~50 × 70 cm) were constructed to be able to evaluate individual haversian systems of each individual bone of each animal. Lateral is to the left. **A,** Group I horses: classically trained on a dirt track. **B,** Group II horses: classically trained on a wood chip track. **C,** Group III horses: controls. **D,** Group IV horses: modified training on a dirt track. Changes in modeling, remodeling, and shape can be seen easily by comparing different groups. Group II horses appear to be earlier in the remodeling cycle than do group I horses that already have remodeled in the medial and lateral cortices. All specimens are from the left forelimb, and increased new bone formation is seen on the medial surface of the classically trained horses (groups I and II). Modified training (group IV) and the control horses (group III) do not show this change. The lack of haversian remodeling in the dorsal-dorsolateral cortex of the classically trained horses in groups I and II is notable. In this area catastrophic stress fractures occur in racing animals.

formation on the medial, lateral, and dorsal surfaces. The medullary cavity remained large and centrally placed. Examination of McIII inertial properties showed that the I_{min} of groups I, II, and III were similar, but I_{min} of group IV horses was greater and was similar to the I_{min} previously reported for mature racehorses.

Microradiographic sections from the mid-diaphysis of McIII showed that bone remodeling occurred only medially and laterally in groups I and II. Filling of secondary haversian systems with new bone was most complete in group I specimens, indicating that the remodeling process was advanced further in horses exercised on a harder surface. A distinct lack of remodeling activity was apparent in the dorsal and dorsolateral regions in groups I and II. Horses in groups III and IV showed extensive remodeling throughout the cortex, including remodeling in the dorsal and dorsolateral aspects.

These data suggested that in horses training on a hard surface, bone remodeling occurred at a faster rate than in horses training on a compliant (wood chip) surface. Previous studies showed that classically trained horses on a hard surface have a higher incidence of bucked shins than do horses trained on a more compliant surface. One horse in group I developed bucked shins during the training period. Inertial properties (I_{min}) of McIII in the dorsopalmar direction were no different in horses trained on a hard or soft track. Horses in group IV had a significant change in I_{min} of the dorsal cortex of McIII,

similar to that seen in adult racehorses evaluated previously that were no longer at risk of developing bucked shins.[3] This supported the concept that exercise could be designed to optimize the shape and modeling/remodeling status of McIII and thus reduce the incidence of bucked shins.[9]

Exercise Programs Designed to Decrease the Incidence of Fatigue Failure

To determine the efficacy of an adapted training program to decrease the incidence of bucked shins, a prospective and retrospective study was performed using work training data from five commercial training stables. Two of the stables (2 and 5) were already using our modified classical training program, and the others (1, 3, and 4) were training in a classical manner. Horses in stables 1, 2, and 4 were trained on a commercial racetrack, whereas those in stables 3 and 5 were trained on private farm training tracks. All horses in the study were TBs, 2 years of age, and starting training for the first time and were followed for 1 year. Data collection stopped if horses developed bucked shins, were sold, or stopped training because of another event not related to bucked shins. The study included 11 years of training data from 226 2-year-olds. Fifty-six of the 226 horses developed bucked shins, and 170 horses completed the observation period or were sold.

Regression analysis and survival analysis techniques were used to explore the data. Horses in stable 2 had the best survival, and those in stables 1 and 4 the worst, and evaluation of data suggested that relationships between galloping and breezing were important. Horses in stable 2 had the highest breezing rate and the lowest incidence of bucked shins, whereas those in stables 1 and 4 had the highest galloping rates and the highest incidence of bucked shins.

Galloping increased the likelihood of bucked shins by 36.4%, whereas breezing short distances was protective, reducing the likelihood of bucked shins by 98.6%.[10] One should note that long-distance breeze rates are detrimental.[11] The winter of 1994 brought severe ice storms to the northeast. Horses in stable 2 could not be trained using the modified program and instead were trained using the standard classical program. An unintentional crossover design was created, and 62% of the horses trained that year developed bucked shins. Without using 1994 data only 9.3% of horses developed bucked shins.

DISCUSSION

Nineteen years of evolutionary experiments, based on an initial observation of a significant difference in the incidence of fatigue fractures between two breeds of racehorses, have led to a natural model for fatigue failure of bone. We can now compare in vitro and in vivo fatigue behavior and observe bone adaptation with different exercise regimens. Adaptive exercise has been shown to change the geometric properties of McIII, to influence bone modeling and remodeling, and to reduce the incidence of fatigue failure of McIII in the TB.

Comparisons of TBs and STBs show major changes in inertial properties of McIII resulting from growth and superimposed training. Comparisons of young TBs that are susceptible to fatigue failure, with older, resistant animals suggest that changes in bone inertial properties are an important factor affecting the incidence of bucked shins. Large McIII I_{min} values reflect probable increases in McIII stiffness in the dorsopalmar direction and thus reduced peak strain during high-speed exercise. The inertial properties of the proximal tibia have been shown to predict development of fatigue fractures in military recruits,[12,13] just as the inertial property measurement of McIII of 5-year-old TBs shows that these horses are no longer at risk for bucked shins.[4]

In vivo strain measurements of McIII demonstrated higher peak strains under physiological loading than ever had been reported previously in any species. Although in vitro test conditions differed from the in vivo loading, both involved significant bending components that can be expected to produce accumulated fatigue failure in bone. Superimposition of the in vivo strains reported for the young and older horses at racing speed produced a striking predictive relationship for risk of developing bucked shins (see Fig. 104-2).

Large surface strains, measured in vivo at high speeds on the dorsolateral aspect of McIII in young TBs in training, contrast dramatically with the smaller strains measured in adults that have raced successfully. Strains, under a given load regimen, measured on the surface of bone relate to the modulus and inertial properties (section modulus) of the bone. Because inertial properties have been shown to increase with age, and bone strains during high-speed exercise have been shown to decrease in older horses, the hypothesis was that changes in bone inertial properties and modulus serve to lower the peak bone strains as the young racehorse matures. However, training regimens possibly can outpace adaptive response. In fact, we observed that a certain percentage of young animals actually increased McIII surface strains after several months of training. Whole bone stiffness measurements showed right to left differences of up to 27% in horses in training, whereas no right to left difference was found in the non-trained control animals. Because bucked shins occur bilaterally but sequentially in TBs, usually on the left side before the right, the developmental stiffness changes in limbs possibly are not synchronized but may respond to the predominance of the left lead used by the horse in its racing gait as the horse works in a counterclockwise direction. Maximal strains at exercise and bone stiffness parameters probably change with time and may be declining on one side, while increasing on the other. Increasing bone strain measured at high speed during training, as seen in four of seven TBs, suggested rapid bone stiffness changes in vivo from exercise. Two possibly related explanations exist for this observation:

1. Bone stiffness decreases in vivo, much as it does in vitro, when the bone undergoes cyclic fatigue.
2. Bone stiffness increases from inertial property changes in McIII, but it may be overwhelmed by decreasing intrinsic material stiffness.

If Wolff's law is applied strictly, it follows that a bone that adapts to a particular peak tensile strain may not be prepared adequately to resist far larger peak compressive strains in the same location.

A recent in vitro fatigue study of equine McIIIs showed a difference in fatigue resistance to bending loads in different anatomical quadrants. Bone that was loaded in bending around the physiological bending axis had greater fatigue resistance than bone bent at 90° to this axis.[14]

We hypothesized that to adapt adequately for racing, McIII should be exposed during training to strains of the actual magnitude and direction experienced during racing. Furthermore, the high incidence of bucked shins in TBs suggested that loading to produce such peak strains and concomitant adaptive remodeling did not occur in a large number of TBs in classical training programs before racing.

Previous in vivo studies, using a functionally isolated rooster ulna, have shown that low numbers of loading cycles (four per day) were adequate to maintain bone mass. Thirty-six cycles were enough to stimulate bone formation.[15] The resulting periosteal new bone formation is the same type of bone reaction observed in TB McIII with fatigue injury.

Taking these observations into account, an exercise (training) regimen was developed that modestly increased the small numbers of high-load cycles using peak load magnitudes and directions that are seen during racing. Increasing the number

of short-distance workouts (breezes) from once every 7 to 10 days, as occurs with classical training programs, to three per week produced large changes in modeling, remodeling, and inertial property measurements of McIII. Classical training produced little progressive change in the inertial properties of McIII, seemingly no better than no training at all, whereas the new modified training program showed inertial property development that equaled or surpassed that observed in established older TBs, those horses apparently no longer susceptible to bucked shins.[3]

The idea of using exercise to produce adaptive bony remodeling is not new. Woo et al.[16] showed that 12 months of exercise in young pigs produced dramatic changes in the femur, increasing cross-sectional area by 23% and inertial properties (I_{min}) by 27%, without intrinsic bone property changes.[16] Most recently, Milgrom et al.[17] looked for exercise that can adapt bone and showed that playing basketball for 2 years or more was protective in reducing the incidence of bone fatigue failure in military recruits.

In the 11-year longitudinal study, we proved that adaptive exercise could be used to reduce the incidence of bucked shins. The relative incidence of bucked shins in classically trained TBs is probably higher than we found. The mean time taken for horses to develop bucked shins was about 25 days shorter than the mean time to loss for other reasons. Removing those stables where loss occurred before onset of bucked shins would have strengthened the data, but efficacy was shown including all the data. We would not expect horses in the modified training program to experience additional fatigue fractures after the observation period, because McIII should resemble more closely that of adult, non-susceptible racehorses.

Adaptive exercise has been shown to change the geometric parameters of a specific bone in a way that would be expected to reduce fatigue damage while, at the same time, significantly reducing the incidence of bucked shins. This correlation, although not explicit proof of the interrelationship between the factors measured, is convincing.

TRAINING TO PREVENT BUCKED SHINS

A training program has been developed to limit the incidence of bucked shins and can be modified by trainers to suit their situation.[18]

The principles of this training program are the following:

1. Bone changes its shape and structure based on its use (Wolff's law). It therefore follows that training adapts bone to training, racing adapts bone to racing, and training that mimics racing adapts bone to racing.
2. Short, high-speed workouts are introduced into the training program early and often (two per week) to introduce bone to the forces it will experience during racing.
3. Galloping is done in moderation (1 mile/day, 6 days/week). Long gallops result in high mileage that may induce high strain cyclic fatigue of bone.
4. Jogging is used for warm-up only and is not used to increase a horse's level of fitness, because jogging induces tension on the dorsal surface of McIII. (This principle is not supported by the survival analysis studies, but it did seem to be relevant when comparing 1994 data within stables 1 and 2.)
5. Once bone adapts to racing (6 to 8 months), this training program is no longer needed, because bone will not regress into its previous shape and architecture. Any successful program can then be instituted.

These principles were developed based on experimental studies that showed that at slow gaits the dorsal surface of McIII is under tension and at high speed is bending to produce compression. Structurally, materials are designed differently to support tension or compression. Think how strong a rope is in tension and how useless it is in supporting loads in compression. Short, high-speed exercise exposes the bone to compression on its dorsal surface and introduces the bone to the loads expected during a race. Experimental studies showed that this sort of training regimen changed the cross-section of McIII to the adult racing shape within 6 months.

Long, low-speed jogging only adapts the bone to jogging, creating tension on the dorsal surface of McIII. In stable 2 in the icy winter of 1994 the horses were jogged for 30 days, without any high-speed exercise, and then resumed the exercise program. The incidence of bucked shins increased fivefold compared with previous years and returned to normal the following year.

Because training is not always a cookbook recipe for success, the fundamentals of the program are described without an actual schedule. The horse should start galloping in December of its yearling year and be able to gallop a mile easily in 18-second furlongs. As training progresses, short, higher-speed events are included two times a week at the end of the gallops. Initially, horses are urged on to a faster speed for ½ or 1 furlong (15-second furlong). This higher-speed event is increased in length, and then speed is increased again. Two weeks elapse between speed or distance changes. The animals continue training and are breezing 12- to 13-second furlongs before the first race (usually by mid-June).

Horses with bucked shins can be trained using this program, provided that the horse is sound and periosteal new bone formation is not substantial. The secret is to decrease the horse's galloping distance (cut it in half) and add short workouts. For example, if a horse usually gallops 1½ miles/day, reduce the distance to ¾ mile/day and introduce short workouts twice weekly. We usually back the horse up about 1 month in its exercise program and get the high-speed workouts started over short distances. If horses are sore or lame, training is stopped and cold therapy and administration of non-steroidal anti-inflammatory drugs (NSAIDs) are begun. Once sound, horses are returned to training. In some horses NSAIDs may need to be administered for chronic soreness, but horses with frank lameness should be allowed to rest until sound.

If a horse's training schedule is interrupted by an unrelated lameness or upper respiratory tract disease, remodeling that starts with a resorption is sudden. When the primary problem resolves, if horses are returned to training at a level similar to that before rest but McIII porosity is increased, the horse is at risk of bucked shins. Therefore if a horse is out of the exercise program for 10 days or more, the horse should be backed up in its training schedule to minimize the risk of bucked shins.

Horses with bucked shins are at increased risk to develop dorsal cortical fractures or may have catastrophic failure of McIII. Horses without bucked shins are at low risk to develop fracture.

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STRESS FRACTURES OF THE THIRD METACARPAL BONE: SURGICAL MANAGEMENT

• Alan J. Ruggles

Most stress fractures of the third metacarpal bone (McIII) occur on the dorsolateral aspect of the left forelimb but may be bilateral (Fig 104-4). Treatment options include controlled exercise (see page 852), screw placement in the dorsal cortex, and cortical drilling around the fracture line. Recently extracorporeal shock wave therapy has been used with apparent early success (Editors). The placement of a screw does not result in interfragmentary compression, but it may have a local effect on bone modeling and remodeling. Cortical drilling is thought to improve vascularization and new bone formation at the fracture line.[1] Advantages of cortical drilling alone are that a second surgery is not necessary to remove the screw, and in some horses this procedure can be performed standing. However, drill breakage in a standing horse is a risk and, if the bit piece is not retrieved, may lead to chronic lameness. Clinical reports indicate that the use of cortical drilling and screw placement has a lower rate of fracture recurrence than cortical drilling alone. Success rates were 97% and 85%, respectively.[2,3] I use the combination technique.

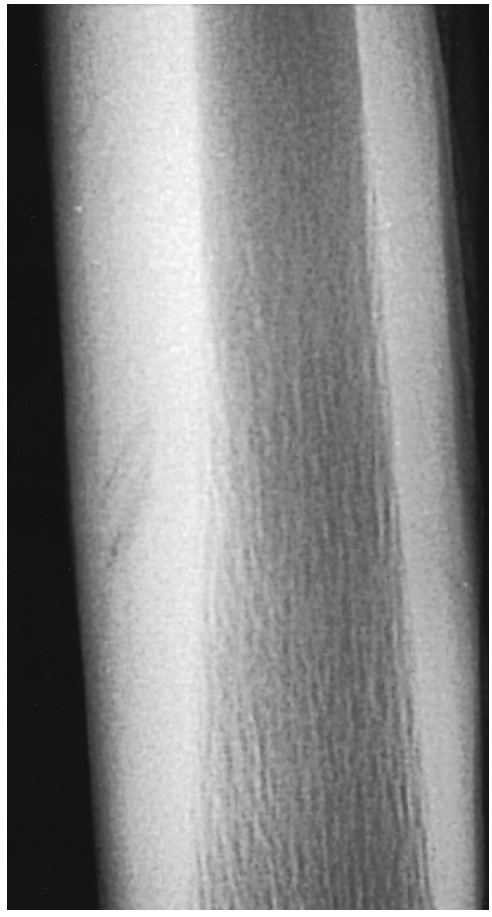

Fig. 104-4 Lateromedial radiographic view of a third metacarpal bone of a 4-year-old Thoroughbred. Dorsal is to the left. There is a typical dorsal cortical fracture of the third metacarpal bone, cortical thickening of the third metacarpal bone, and numerous oblique fracture lines. This horse is a candidate for drilling or screw placement.

Horses with radiographic evidence of substantial endosteal or perisoteal healing often heal satisfactorily without surgical intervention. Horses with severe fractures that are in multiple locations, or spiral around the bone, have a risk of catastrophic failure on recovery from general anesthesia and usually are treated conservatively (Fig. 104-5). To my knowledge no objective reports of conservative management of dorsal cortical stress fractures exist. In my experience conservative management is less reliable than surgical treatment, and does not result in a reduction in training days missed. In addition I have experienced a higher rate of recurrent fracture, or failure of fracture to heal, with conservative compared with surgical management.

Surgical Procedure

The horse is positioned in lateral recumbency with the affected limb uppermost. Both limbs can be operated on from this position. The periosteal new bone usually can be felt

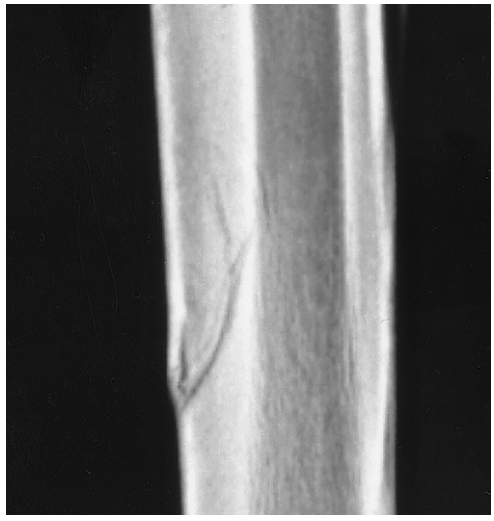

Fig. 104-5 Lateromedial radiographic projection of the third metacarpal bone of a 2-year-old Thoroughbred. Dorsal is to the left. There is a severe, dorsal cortical fracture of the third metacarpal bone. Conservative management in horses with severe fractures is recommended. This fracture is saucer-shaped in appearance and could propagate during recovery from general anesthesia.

through the skin, and a 6- to 8-cm vertical incision is made over the fracture, usually between the common and lateral digital extensor tendons. Radiographic control is used if necessary. The periosteum should not be elevated. A sharp 2.5-mm drill is used to drill a thread hole for a 3.5-mm screw perpendicular to the fracture line. Only the dorsal cortex is drilled. Overdrilling may result in impact on the palmar cortex and instrument breakage. During or after screw placement, radiographic control is necessary to determine proper screw placement. The screw is placed after countersinking, tapping, and measuring to determine appropriate screw length. Usually a single screw is used, but occasionally several screws are used for a long fracture. Five to 6 unicortical drill holes approximately 1 cm apart are made using a 2.5-mm drill bit in the region of the fracture, before routine closure and bandaging the limb.

If dorsal cortical drilling alone is performed standing, under sedation and perineural analgesia, a 3.2-mm drill bit is used to decrease the risk of drill breakage if the horse moves during the procedure.

Postoperative Treatment

Four weeks of stall rest with hand walking followed by 4 weeks of small paddock turnout, continued hand walking, or swimming are recommended. Screw removal is performed with the horse standing at 8 weeks, based on fracture healing. Return to light jogging can occur 2 weeks after screw removal, but more intense training should not start until at least 16 weeks after surgery and should be based on lack of clinical signs of lameness and radiographic signs of healing.

Prognosis

Prognosis is considered good, with 85% to 97% of horses returning to racing.[2,3] Recurrence of fracture occurs more commonly after cortical drilling alone, compared with cortical drilling and screw placement combination. Catastrophic failure has occurred after cortical drilling alone but not with the combined technique.

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CHAPTER • 105

On-Track Catastrophe in the Thoroughbred Racehorse

Ted Hill

Horses in racing and high-intensity training are subject to a variety of musculoskeletal injuries. For North American racing the overall incidence of musculoskeletal injuries ranges from 3.3 to 7.3 per 1000 starts, depending on variables such as reporting criteria and degree of follow-up. A much closer range of 1.1 to 1.8 injuries per 1000 starts is reported for catastrophic injuries resulting in euthanasia.[1] Catastrophic or fatal injuries are documented more reliably and are considerably less subject to bias or misinterpretation by the reporter. The rates for training injuries may be somewhat higher, although accurate acquisition and evaluation of these data is more difficult. To date, limited information is available for training injuries.[1,2] Several factors cannot be controlled during training, making information obtained inaccurate and incomplete. The absence of a veterinary observer during most training sessions allows many lameness incidents to go unreported. Often only injuries requiring an ambulance come to the attention of the track veterinarian. During training, no

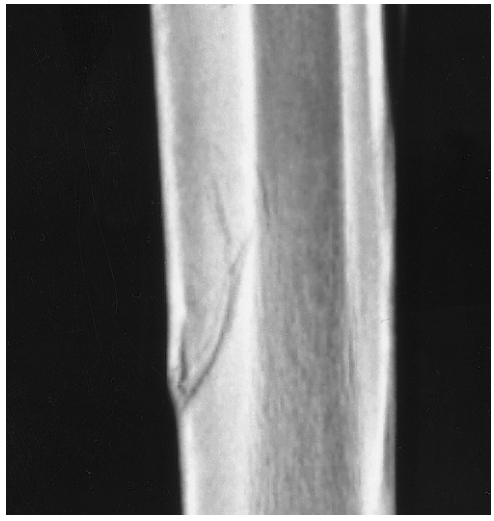

Fig. 104-5 Lateromedial radiographic projection of the third metacarpal bone of a 2-year-old Thoroughbred. Dorsal is to the left. There is a severe, dorsal cortical fracture of the third metacarpal bone. Conservative management in horses with severe fractures is recommended. This fracture is saucer-shaped in appearance and could propagate during recovery from general anesthesia.

through the skin, and a 6- to 8-cm vertical incision is made over the fracture, usually between the common and lateral digital extensor tendons. Radiographic control is used if necessary. The periosteum should not be elevated. A sharp 2.5-mm drill is used to drill a thread hole for a 3.5-mm screw perpendicular to the fracture line. Only the dorsal cortex is drilled. Overdrilling may result in impact on the palmar cortex and instrument breakage. During or after screw placement, radiographic control is necessary to determine proper screw placement. The screw is placed after countersinking, tapping, and measuring to determine appropriate screw length. Usually a single screw is used, but occasionally several screws are used for a long fracture. Five to 6 unicortical drill holes approximately 1 cm apart are made using a 2.5-mm drill bit in the region of the fracture, before routine closure and bandaging the limb.

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standard exists for soundness, medication use, and exercise rider skill. Differences between training injuries and racing injuries are documented when they exist.

Information about racing injuries presented in this chapter applies to racing worldwide. However, discussions of the regulatory veterinarian primarily refer to racing in America.

REGULATORY VETERINARIAN

The official veterinarian and staff members at an American Thoroughbred racetrack have many duties. The primary responsibility is monitoring the soundness of horses during racing and, to some degree, training. This task is accomplished by an efficient prerace inspection and observation of all horses before, during, and after a race. After the horse is identified, a visual assessment is made of the overall condition and attitude, and vital parameters are assessed. Ideally the temperature, pulse, and respiratory rate are recorded, and the eyes and mouth are examined closely. The mouth should be free of any inflammation or lacerations, which the bit would aggravate, possibly causing control difficulties for the rider. The forelimbs and hindlimbs are palpated, and attention is paid to the type of shoes worn and the condition of the feet. When bar shoes, aluminum pads, or quarter crack patches are noted, closer inspection of the foot is indicated. A palmar digital neurectomy is permitted in most jurisdictions. The clinician must always check for a neurectomy to ensure compliance with the rules. Usually a horse that has undergone neurectomy must be reported to the track veterinarian and its name conspicuously displayed for claiming races.

Any horse determined to be sick or unsound for racing is reported to the track stewards and withdrawn from the day's racing card. Only the stewards have the authority to officially scratch a horse, although in practice a track official rarely questions the veterinarian's recommendation. The veterinary department must function as an independent authority and neither management nor anyone connected to a horse should interfere with the veterinarian's decisions.

Once withdrawn for a veterinary reason, a horse is usually placed on an ineligible list and requires further evaluation before being permitted to race again. Depending on the nature of the illness or lameness, the evaluation may involve only a quick check, or if more serious, a monitored workout and thorough examination. The importance of the prerace inspection and follow-up examinations of identified horses cannot be overstated. Evidence exists that horses with prior pathological conditions are at increased risk of recurrent or more severe injury, possibly career ending or catastrophic.[3,4]

A veterinarian is present in the paddock, where horses are saddled, and at the starting gate. An injury or lameness requires prompt evaluation and contact with the stewards for a late withdrawal. The track veterinarian is the ultimate authority over all horses in the paddock and on the racetrack during racing and training.

Racing officials rely on the veterinarian for information and guidance. Objective data on injuries may assist the track superintendent to address concerns about the track surface and dispel rumor and exaggeration among horsemen. The stewards and judges need the veterinarian's input regarding the use, safety, and validity of new equipment such as shoes, bandages, protective eye covering, bridles, and therapy modalities. The official veterinarian also can be an invaluable source of information on medication for private practitioners, horsemen, and racing officials.

A brief comment on managing spills is appropriate. A *spill* is defined as a sudden fall of horse and rider that usually causes other horses and riders to fall. Ideally, one may call for immediate assistance from other veterinarians and experienced track personnel to assess and attend promptly to more than one horse injured in the incident. Horses may rise after a spill, although seriously injured, and move to other areas on the racetrack, thus further complicating the situation. A prompt overall assessment of the number of horses involved, the location on the track, and the major injuries are essential to prevent misallocation of resources. Triage and communication, with preferably one experienced veterinarian coordinating the activities, are key to an efficient professional outcome.

MANAGEMENT CONSIDERATIONS

Efficient management of the on-track catastrophe, while maintaining the horse's best interests, is the primary objective. The key elements required include trained personnel, equipment, and communication.

Communication is critical to make the best use of available resources. The track veterinarian must be prepared to respond to the injured horse and direct support personnel and equipment at the scene. The injured horse is under the care and direction of the track veterinarians until it is moved to a clinic or stable to be attended by the veterinarian employed by the owner or trainer. The stable veterinarian may be called to the scene for severe injuries and if time permits. The track veterinarian acts promptly to attend the injured horse on the track, thus facilitating an efficient transfer for further evaluation and treatment. Efficiency is important for the care for the injured horse and to allow the official veterinarian to be in position for subsequent races without undue delay. Ideally a communication network of two-way radios and mobile telephones works well to keep an orderly process in motion. Radio communication with track departments such as security serves to relay information as required.

The operator of the horse ambulance should have sufficient training to understand fully the ambulance features and how to use them. Knowing how to back up and position the loading doors properly is essential. Failure to accomplish these basic skills could cause further injury or delay proper treatment.

In most situations, track personnel are the first on the scene of the injured horse, but in other countries, such as the United Kingdom, a veterinarian follows the race and would be on the scene immediately. Track personnel routinely are equipped with radios and are crucial for initiating communication with veterinarians and providing the immediate care. Knowing who and how to call for assistance can save valuable time and distress for all involved.

Those individuals should be instructed in the basics of emergency care. The track veterinary department should provide such instruction to ensure a defined, reliable standard, without confusion and delay. Topics such as restraint, controlling hemorrhage, support of an injured leg, and when to apply a splint are well worth the time. Most if not all persons are willing to do whatever may be required to assist an injured horse, but they are often reluctant for fear of making a mistake. These first-line members of emergency care should be encouraged and recognized for their valuable assistance. Splint selection and application should be kept as simple as possible to avoid potential legal complications, which fortunately are uncommon. An overzealous and poorly trained person may assume too much responsibility, however, placing the horse at risk of further injury and subjecting track management to extensive liability. Outriders, who monitor training in the mornings, may be instructed in using a commercially available support, such as a Kimzey splint (Kimzey, Inc., Woodland, CA). The splint may be applied safely whenever loss of support in the fetlock joint is obvious, because the splint is simple, quick to apply, and generally well tolerated. Restraint is

often a critical issue for horse and handlers' safety. Usually keeping the horse quiet and still while supporting a distal limb injury is adequate until a veterinarian arrives to provide professional care. However, in some situations the horse is best kept recumbent (e.g., a horse with such a severe injury that it is unable to remain standing, even with assistance). Such horses may lunge repeatedly and fall, risking further injury to themselves and endangering all present. A fallen rider near the horse may be incapable of movement for several minutes while being attended, making control and restraint of the horse all the more imperative.

EQUIPMENT

Substantial improvements in the equine ambulance over the past decade have contributed to saving horses that otherwise would have been euthanized within minutes of injury.

The ambulance should be partitioned, well ventilated, allow ample daylight, and be equipped with interior lights. Sturdy, rubber floor matting and padded sidewalls, all easily washed, are required. A partition separating the horse holding area and a forward compartment provide a storage area and some protection for handlers. Ideally the ambulance trailer can be lowered hydraulically to facilitate loading of a severely injured horse and eliminating the need for a rear ramp. Side access doors with ramps provide convenient and safe off-loading, without having to back or turn the horse around. An extremely valuable feature is a movable middle partition, which serves as a squeeze chute to help support the horse during transport. A seriously injured horse will make excellent use of this feature, by leaning on it or the wall when turning, thus drastically reducing lateral movement and unnecessary weight bearing. The Kimzey Equine Ambulance is an excellent example of such a fully equipped vehicle.

The ambulance should be well equipped with splints and bandaging materials. Splints to support the metacarpophalangeal (fetlock) joint are essential, as are compression boots in two or three sizes. Bandage material should include sterile pads, gauze and cotton bandages, and elastic and adhesive wraps. Sufficient material to make a modified Robert Jones bandage always should be available. Duct tape is an excellent means to secure a heavy support bandage or splint, preventing shifting and providing axial stability. Inflatable compression splints are sometimes helpful to stabilize carpal fractures, but application of a cotton and elastic bandage is often faster and more effective.

Some of this material can be kept on the equine ambulance, but space and security considerations require that most of the splints and medications are kept in a track vehicle that transports the veterinarians. Emergency supply bags can be equipped and organized to facilitate quick access at the scene of the injured horse.

Containers of ice should be kept on board the ambulance during racing for prompt application to acute injuries. Rubbing alcohol is also useful for cooling horses in warm climates.

Although it is not frequently required, a portable oxygen supply with a simple flow delivery system and two endotracheal tubes should be readily available. A sling of suitable design to support a horse for extended periods is recommended (Liftex Inc., Warminster, PA).

MEDICATION

Security concerns may preclude keeping medications in the ambulance in some jurisdictions. An emergency bag with select drugs is an efficient means to attend the injured horse on the track and in the ambulance. A suggested inventory includes butorphanol, xylazine, flunixin meglumine, ketamine hydrochloride, detomidine, hydrocortisone sodium succinate, epinephrine, phenylbutazone, and euthanasia products, such as pentobarbital and succinylcholine. Although it is not an approved drug for humane euthanasia alone, succinylcholine can be a valuable adjunct with other drugs to control an extremely difficult horse. A dose of 5 ml (100 mg), given with an initial dose of pentobarbital acts, safely to drop an uncontrollable horse with a catastrophic injury, thus permitting administration of the required full dose. An assortment of syringes, needles, tourniquet, scalpels, scissors, hemostats, and blood collection tubes complete the emergency kit.

TRAINING INJURIES

Humeral Fractures

During morning workouts and gallops, horses are subject to the same injuries as those that occur in racing. A few injuries, such as humeral fractures, are not encountered commonly in racing, but they are often seen while the horse is only galloping. For the reporting period 1993-2000, 12 of the 15 humeral fractures occurred during morning training.[5] Horses with spiral humeral fractures, with or without displacement, almost always are considered candidates for immediate euthanasia. The injuries usually involve the horse falling suddenly and remaining down, although some do rise. The rider may have no warning before the fall, but some horses may change leads and bobble or shorten the stride.

The diagnosis is straightforward if the horse is standing. The horse bears little or no weight and the limb is in a hanging position, with the horse unable to advance the leg if encouraged to move. There may be obvious swelling of the area, and palpation or manipulation with auscultation often reveals crepitus. Horses that are down on the track when the veterinarian arrives present a diagnostic challenge. They may make some attempt to rise, particularly with encouragement, but with the fractured humerus on the down side they are rarely successful. Rolling the horse over can be accomplished with at least two people and the use of shanks or similar equipment to avoid being kicked in the process. Once the horse is turned over, the fracture is often apparent and the horse may rise with the good foreleg down. These horses often fall dramatically when the fracture occurs and may be slow to recover because of the shock of both the fall and the fracture.

Although some injured horses may be loaded and transported to the stable, no treatment is recommended other than analgesia and physical support. These horses are extremely difficult to load without the assistance of capable and readily available personnel. Loading becomes substantially more difficult with time, and the increased swelling and pain make the horse reluctant to move at all after several minutes. A horse is best left supported on the equine ambulance until the stable veterinarian arrives to confirm the diagnosis before euthanasia. At the request of an owner, or to satisfy insurance requirements, radiographing the limb may be attempted to document the diagnosis, particularly for horses with minimally displaced fractures. For most horses with this severe injury, radiographs are obtained more humanely post mortem. Humeral and scapular fractures do occasionally occur during racing and horses should be managed in the same manner.

Collision Injuries

Collision injuries usually are associated with training, because many horses are often on the track, exercising at various speeds, distances, and directions. Maintenance equipment and personnel moving on and near the training surface open gaps permitting access to the track, and insufficient outriders to monitor and control the congestion all predispose to collision

injuries with other horses and inanimate objects. Injuries may be of any type, but include trauma to the head, axial skeleton, shoulder, and pelvis. Bruising and lacerations from impact are not uncommon. The severity can range from minor abrasions and contusion to severe, life-threatening fracture and hemorrhage.

Location of Injuries on the Racetrack

Considerable anecdotal and published information exists regarding the location of injury occurrences on the track surface.[5,6] Any injury type can be encountered at any point in a race, and trained personnel should be prepared to respond to any location. From experience, it is safe to conclude that most catastrophic injuries occur in turns and in the stretch run to the finish. Most racing operations position the equine ambulance in the area of the ½- to the ¼-mile pole to be available for a prompt response. Many injuries are evident immediately after a race and when horses are pulling up and begin galloping back to be unsaddled. Horses with carpal chip or proximal sesamoid bone fractures and new or recurrent tendonitis or suspensory desmitis usually show lameness after they are pulled up. The jockeys should alert an outrider for assistance. Some riders may be hesitant to pull up and dismount because of inexperience and uncertainty, but the track veterinarian should make this policy clear to the jockeys and encourage conservative, prudent decisions. Any horse suspected of being lame, by a jockey or outrider, should be held at that location until evaluated by an official veterinarian.

Horsemen, riders, and officials often express concern about unsafe track conditions. Extreme conditions, such as freezing, washouts, and gross discrepancies in uniformity, may be potentially hazardous. However, muddy or sloppy conditions are not a significant risk factor for serious injury, but they may contribute to rundown injuries, the bruising and abrading of soft tissue at the palmar/plantar aspect of the fetlock from contact with the track surface. Random injuries can cause poor performance. Most catastrophic injuries occur on a fast racetrack.[3,5-7] Muddy and sloppy tracks usually produce numerous late withdrawals and smaller fields, especially if a race was originally scheduled for the turf. Thus some degree of selection of horses occurs in these races. In addition, horses that are not in contention and showing response entering the stretch on a poor track usually are not encouraged persistently by the riders. They are allowed to ease themselves, a situation that may not occur on a fast track with the rider more optimistic of a good finish.

PUBLIC RELATIONS AND MEDIA ISSUES

The track veterinarian must be keenly aware of humane considerations and prepared to respond to questions from the media, patrons, horsemen, and track personnel.

In situations that expose the public to catastrophic injuries in the racehorse, the track veterinarian also must be cognizant of aesthetic considerations, such as using screens and covers to eliminate unnecessary viewing of horses and the process of euthanasia. During racing, time constraints may become critical to management. Most seriously injured horses can be supported, loaded, and transported for further evaluation off the track. On-track euthanasia should be reserved for those few horses for which no other alternatives exist.

During the 1990 Breeders Cup at Belmont Park, catastrophic injuries resulted in the euthanasia of three horses and considerable media attention. The need for more efficient liaison with the media was demonstrated clearly. In response, the American Association of Equine Practitioners initiated the "On Call" program at the 1991 Kentucky Derby and has continued this program to date. The program consists of veterinarians with media training who donate their time to respond to questions from the media regarding equine health care concerns. The "On Call" veterinarian is available to provide accurate and expert commentary, answering vital questions during a busy event, thus releasing the event veterinarians to attend the injured horse. "On Call" covers more than 60 equestrian events each year.

RACING INJURIES

Fractures of the Forelimb

Fractures range in severity from relatively minor chip and non-displaced incomplete fractures to open, comminuted fractures of long bones, which are considered the most severe on the track catastrophes. Fortunately the latter are rare. Available data support the clinical findings that most fatal fractures involve the forelimb and that most of these fractures are of the carpus or more distal part of the limb.[1] This section provides an overview of the most common types of fractures that are encountered on the racetrack, beginning with the foot and working proximally in the forelimb and hindlimb.

Foot and Pastern

Fractures of the distal phalanx are often articular, but they may be non-articular and involve only the wing. An affected horse usually completes the race or begins easing in the later stages, rather than pulling up abruptly. Distal phalanx fractures commonly are diagnosed after the race while the horse is on the track or is cooling out. Lameness may vary in intensity, but it usually increases to non–weight-bearing status within 30 to 60 minutes. Moderate to severe lameness is present, without palpable swelling. Non-displaced metacarpal or proximal phalanx fractures should be considered as a differential diagnosis. If the lameness is apparent at a walk, a compression boot to stabilize the limb distal to the carpus may be useful until other fractures are ruled out. Distal phalanx fractures are not as commonly diagnosed on the track as those of the proximal phalanx, and some may be missed initially, only to be diagnosed later and go unreported.

Middle phalanx fractures are rare as a primary injury and usually are seen as part of a multiple injury breakdown of the fetlock and pastern.

Fractures of the proximal phalanx usually occur suddenly during a race or while the horse is pulling up. Such fractures range from an incomplete fracture to varying degrees of fragmentation and severe comminution. Most horses with these fractures are lame and require prompt analgesia and application of a supportive splint or bandage. These horses do not tolerate a heavy compression boot well. Displaced fractures are diagnosed readily on the track. These require considerable attention to provide adequate support for transport to prevent further bone and soft tissue damage, thus optimizing the chances of repair and recovery. Proximal phalanx fractures are the most commonly diagnosed injury of the pastern and occur equally in the left and right forelimbs.

Metacarpophalangeal Joint

The metacarpophalangeal joint is involved in most catastrophic injuries. Horses with simple, non-displaced third metacarpal bone (McIII) condylar fractures usually develop acute lameness at the end of a race or after the race. Lameness is noticeable at a walk by the time the horse is loaded. With minimal swelling, diagnosis is presumptive initially, and appropriate supportive measures are required. Although the lameness may be substantial, localizing the injury is often difficult when displacement and swelling are minimal. However, some condylar fractures are diagnosed after the horse has returned to the stable to cool out, without the assistance of an ambulance. An open, comminuted McIII condylar fracture causes severe lameness. The horse is pulled up during a race or immediately

after the race. The proximal aspect of the displaced fragment penetrates the skin with some mild hemorrhage, making the diagnosis readily apparent.

Because the potential for further displacement and contamination is great, considerable care must be exercised to support the fracture site. Preferably a sterile bandage should be applied to the area and then the leg supported with a heavy bandage or compression boot. Excessive movement of the horse should be avoided while the horse is loaded and transported on the ambulance. Newer ambulances are equipped with a sliding partition to support the horse firmly. The prognosis is guarded because of the compound nature of this injury, and extreme care in managing these horses on the track is essential to a successful outcome.

Proximal sesamoid bones The proximal sesamoid bones (PSBs), especially of the forelimbs, are a frequent site of racing and training injury. The PSBs of the forelimbs are primarily involved. Classically, a simple fracture of the PSB results in a progressive lameness while the horse is cooling out after successfully completing a race or workout. The official veterinarian may not see many of these horses and only learns of their injuries after receiving information from the test barn personnel, trainers, or attending veterinarians.

A more severe sagittal or mid-body fracture of one or both of the PSBs causes severe, acute lameness during a race or immediately after a race. If only one PSB is involved, the loss of support of the metacarpophalangeal joint is minimal. The limb should be held by the first attendant on the scene, to prevent weight bearing and further movement, before application of the Kimzey splint. Once supported, these horses are relatively cooperative and confident to move for loading and transport.

In horses with biaxial PSB fractures, loss of metacarpophalangeal joint support is immediate. A dramatic drop of the metacarpophalangeal joint occurs whenever the horse attempts to bear weight. The limb must be supported immediately and the horse controlled to allow prompt application of a splint. These horses may attempt to move on the injured limb and, if they are not restrained, may cause substantial secondary damage to the soft tissues of the fetlock joint (see Chapter 37).

Fractures involving the metacarpophalangeal joint frequently are complex and involve some combination of McIII condylar and PSB fractures, thus compromising the suspensory apparatus. Horses running at race speeds that sustain an McIII condylar or PSB fracture continue to gallop several strides on the injured limb before pulling up. Often they may unseat the rider and gallop considerable distances before being stopped. Additional fractures and disruption of the suspensory ligament (SL) and distal sesamoidean ligament may result, and the injury may become open, with subluxation and disarticulation of the metacarpophalangeal joint. Depending on the severity of the injury, some of these horses may be manageable for ambulance transport. However, many require immediate euthanasia because of the extensive damage. The metacarpophalangeal joint is involved in two thirds of all catastrophic racing injuries at the New York Racing Association tracks.[5]

Third metacarpal bone The McIII condylar fracture has been discussed previously. Horses with minimal displacement have a good prognosis for return to racing after surgery. Those with substantial displacement or skin penetration have a less favorable prognosis, but with proper on the track management, many can have a reasonable outcome in an alternative career.

The McIII diaphysis is the site of a common racing and training injury referred to as *bucked shins* (see Chapter 104). If not properly managed, horses with bucked shins may not stride out well, move with a choppy action, and trail the field during a race. These horses may be pulled up by the jockey, but they require little on-track care.

Dorsal cortical fracture of McIII may result from the bucked shin complex, an injury that is seen most frequently in late 2-year-olds or in 3-year-olds. Lameness may be intermittent and palpation of the affected area often provides inconsistent findings. Dorsal cortical fracture of McIII poses a serious risk to horse and rider if not detected and properly treated. If a horse with a fracture is returned to training and racing prematurely or the fracture is undiagnosed, a complete diaphyseal fracture may develop. This catastrophic injury causes a horse to fall without warning during morning workouts or a race, risking serious injury to the rider and others in close proximity. The track veterinarian is faced with an essentially unmanageable horse. The fracture usually is comminuted and open. The limb distal to the fracture site is often attached by only tendons and remaining skin. The horse may attempt to rise or manage to rise, but it is extremely unstable and usually falls again. For the safety of all assisting at the scene and for the riders who may still be down on the track, the horse should physically be kept down by capable track personnel. Immediate euthanasia is indicated. If the trainer or owner is present and requests another opinion, the attending veterinarian can sedate or anesthetize the horse long enough for another veterinarian to be summoned. Attempts to develop an anesthetic protocol to facilitate transport of these horses for potential surgical intervention have been unsuccessful. Historically, this catastrophic injury has been the cause of spectacular horse spills in racing, causing considerable loss to the horse industry, serious jockey injury, and negative media attention. Because essentially no treatment alternatives are available, all effort must be directed toward prevention. Accurate diagnosis and the removal of horses from high-intensity training and racing are essential.

Carpus

Although the carpus is a frequent site for lameness, it is seldom an area associated with catastrophic injury (see Chapter 39). Carpal chip and slab fractures cause a range of clinical signs. Small chip fractures may be difficult to detect and, without obvious effusion or pain on palpation, are difficult to differentiate from McIII condylar fractures. A horse with a carpal slab fracture, especially if displaced, is usually severely lame. If a slab fracture is suspected, the carpus should be supported with a cotton wrap and elastic bandage or an inflatable compression splint. Comminuted carpal fractures occur and cause collapse of the proximal and distal rows of carpal bones and obvious instability. Although a horse with multiple carpal fractures may fall, most horses pull up abruptly, stopping in just a few strides. The horse is in acute distress and non–weight bearing and usually difficult to load and transport. If possible, a horse with a comminuted carpal fracture should be examined by consulting veterinarians, because euthanasia may be highly likely. Such examination avoids unnecessary movement and distress for the horse. Some horses may be salvaged for breeding purposes with surgery and extensive nursing care.

Radius

Radial fractures are rare. For an 8-year period, four horses were destroyed because of comminuted radial fractures. Interestingly, all four horses suffered a fracture of the left radius.[5]

Shoulder

Fractures of the shoulder, though rare, are always catastrophic. Those of the humerus most often occur during training and have been discussed previously (see page 856). The scapula may fracture during a race, and in my experience the injury always involves the joint. The horse may fall suddenly, but some horses manage to pull up in the race without going down. Horses are severely lame, have rapid, severe swelling from hemorrhage, great difficulty in advancing the limb, and a

dropped shoulder appearance. This is an excellent example of the benefit of viewing the entire horse from a short distance before commencing a close-up examination.

Inexperience with this injury often leads the clinician to an early misdiagnosis of a lower limb problem, only to be frustrated and embarrassed when enlightened by lay assistants. Swelling over the area may become extensive in a few moments but be overlooked if one remains focused on assessing the distal limb. On numerous occasions, support splints have been applied to the limb distal to the carpus, with the shoulder recognized as the primary injury only when attempting to load the horse onto the ambulance.

Nevertheless, once the diagnosis is made, the horse may be difficult to load, even with abundant help. As a rule, horses are more willing to attempt to move shortly after the injury occurs. Once loaded, the use of a movable partition for support is a tremendous aid. Euthanasia is best performed while the horse is still in the ambulance.

Fractures of the Hindlimb

Pastern

Fractures of the hind phalanges occur considerably less frequently than in the forelimb. Similar injuries occur, but fractures of the proximal phalanx predominate. Only 10 proximal phalangeal fractures of the hindlimb were diagnosed for the period 1993-2000 at the New York Racing Association tracks, compared with 28 for the forelimbs.[5] Horses appear considerably more able to support on only one hindlimb, and protect the injured site from further weight bearing, than to support on only one forelimb. Open fractures are much less common and the prognosis is improved. A lightweight splint and bulky bandage are recommended, because horses do not tolerate a heavy splint or boot well. Complex fractures involving the proximal and middle phalanges and the third metatarsal bone may be seen, although these are rare.

Third Metatarsal Bone

Third metatarsal bone condylar fractures occur less commonly than those seen in McIII. Horses do not tolerate the weight of a compression boot on a hindlimb and often do better with a thick support bandage. It is important to obtain good-quality radiographs, because many of these fractures are located medially and have a tendency to spiral, making surgical repair a challenge.

Tarsus and Tibia

Fractures of the tarsus are an uncommon cause of acute training and racing injuries. Osteoarthritis of the distal tarsal joints commonly results in reluctance to break from the gate and overall poor performance, but it rarely causes an acute on-track incident.

Catastrophic tibial fractures are rare and generally result from pre-existing stress fractures (see Chapter 46). Complete fracture may occur hours after a race or training, when the horse is moving about and resting in the stall. In horses with obscure hindlimb lameness, scintigraphic examination is a valuable diagnostic aid for preventing catastrophic injury.

Stifle and Femur

Femoral fractures are rare in the racing Thoroughbred. Osteochondritic lesions of the distal femur are not uncommon and cause subtle hindlimb lameness that is often difficult to diagnose (see Chapter 47). Stifle lameness, including that caused by osteochondritic lesions, often leads to compensatory lameness conditions. Tarsal or stifle problems may not cause catastrophic injury primarily but rather lead to a compensatory forelimb lameness and secondary breakdown, often involving the PSBs and metacarpophalangeal joint. Accurate diagnosis and treatment of many mild to moderate lameness conditions often helps prevent more serious injury.

Acute hindlimb lameness, other than that caused by complete tibial or femoral fracture, is generally difficult to assess on the track. The horse may pull up abruptly or be lame at the completion of a race and often is reluctant to bear weight. Unlike those with forelimb lameness, horses with acute severe hindlimb lameness are often relatively easy to load into the ambulance and transport with minimal effort. The clinician must keep in mind that a horse may have an incomplete fracture, such as of the third metatarsal bone. Thus maximizing support while the horse is on the ambulance and walking the horse minimally should be standard care for these horses.

Pelvis

Pelvic fractures can cause acute hindlimb lameness and occur predominantly in fillies while racing or training (see Chapter 51). Between the years 1993 and 2000 at the three New York Racing Association tracks, a diagnosis of pelvic fracture was made in 20 horses, 17 of which were fillies.[5] A horse with a pelvic fracture is in obvious distress and reluctant to move, but with proper analgesia and physical assistance, most horses can be loaded and relocated carefully to a stall. Extreme care must be taken, because displacement of the fracture is a potential risk, specifically in those with ilial body fractures. Severe hemorrhage and death can occur even when a horse is in the ambulance or stall. This potential for fracture displacement and hemorrhagic shock keeps survival numbers discouragingly low. Of the 20 horses with pelvic fractures, 6 horses died within 1 hour, and 11 were euthanized at the scene or less than 1 week after injury.[5] With increased use of scintigraphy and early diagnosis of stress-related bone injury, the hope is that catastrophic injuries such as displaced pelvic fractures can be avoided.

Head and Axial Skeleton

Horses involved in collisions or spills may suffer any number of head or spinal injuries. Loose horses attempting to jump railings and run through gaps frequently fall, but they seldom sustain serious injury. Young horses not uncommonly become fractious while being saddled, rearing, lunging, and occasionally falling. Horses with obvious injury, regardless of the severity, should be withdrawn from the race immediately. The track veterinarian must take responsibility for withdrawing a horse after a fall, often consulting with the trainer or owner. The most serious head injuries usually result from flipping in the saddling paddock and striking the hard ground or a wall partition. Sinus hemorrhage, although dramatic and at times copious, is generally not a serious concern. Horses that sustain a hard blow to the head may become disoriented and show ataxia initially. Prompt treatment with corticosteroids and dimethylsulfoxide may be indicated while the horse is kept quiet in a reasonably safe area. Horses should be led to and held in a grass paddock away from trees and fences. If ataxia subsides, the horse may be moved to a stall by ambulance or by walking with attendants.

I find it interesting that some horses may appear normal immediately after a hard fall and head trauma, only to become dull and ataxic or reluctant to move after a short time. Managing all of these horses as if they had potential injury to the central nervous system trauma injuries is advisable. The horse should be monitored closely while moving to the stable, and track personnel should communicate their concerns personally to the attending veterinarian whenever possible. A horse that sustains a serious fall resulting in fractures at the base of the skull is often recumbent, has aural hemorrhage and unfortunately, dies quickly. Fractures or other injuries of the axial skeleton may occasionally result from falling, but a definitive diagnosis may be difficult to make. A horse may be stunned or winded and remain recumbent after falling and make no attempt to rise. Vital signs are usually normal. After ensuring the airway is unobstructed and the saddle removed, the horse is given time to rise. Oxygen from a continuous-flow mask and an

intravenous corticosteroid (hydrocortisone sodium succinate, 100 to 500 mg) are recommended. It may be half an hour before a winded horse is ready to get up. When ready, and with a mild prompt of a slap on the neck or rump, many horses then stand unassisted. Some may be slower and assume a sternal position briefly before rising. Those that are unstable and appear weak behind will need assistance, by helping lift on the tail.

If a horse attempts to rise unsuccessfully several times even with assistance, rolling it on its other side may be best. Injury to the down leg may have occurred. A horse with fractures of the cervical or thoracic vertebrae makes little or no attempt to move, depending on the level of injury.

Horses down on the track with normal vital signs and no apparent limb injury must be presumed to have injury of the axial skeleton. The horse can be positioned on a mat while still recumbent and carefully pulled onto the ambulance for transport to the stable or emergency clinic. An anatomical diagnosis may be established, but the prognosis is extremely poor for horses that have not stood within 12 to 24 hours.

Soft Tissue Injuries

Injuries sustained at the speeds of racing and training workouts are often complex, with soft tissue and bone injury. Fracture of a PSB or McIII causes abrupt loss of action and compensation by the horse and concomitant reaction by the rider. Several strides may follow with some weight bearing on the affected limb. What began as a simple fracture may progress to a catastrophic, life-threatening injury in only seconds because of progressive soft tissue and bony disruption.

The soft tissues supporting the metacarpophalangeal joint are particularly prone to injury. Disruption of the digital flexor tendons or SL can cause gross hyperextension of the metacarpophalangeal joint and potential damage to the digital vessels and nerves. Inadequate perfusion of the injury site may limit or prevent a successful surgical repair or other attempts at recovery.

The metacarpophalangeal joint may subluxate with a primary soft tissue injury. This usually results from a traumatic disruption of the suspensory apparatus, although complete disruption of the digital flexor tendons also may be involved secondarily. If luxation is closed, the joint may become locked in hyperextension, requiring manual reduction before a splint can be applied. If open luxation occurs, the distal aspect of McIII can be seen. Complete luxation usually results from a complete disruption of the SL or the distal sesamoidean ligaments. Immediate support of the limb and splint application is essential for the closed injury. The horse with an exposed, open fetlock injury must be euthanized on the track.

Digital Flexor Tendon Injuries

Digital flexor tendon injury (tendonitis) primarily involving the superficial digital flexor tendon (SDFT) is a substantial cause of economic loss in the Thoroughbred industry. Tendonitis varies in severity, but horses with even the most minor tear require time to recover and return to training. Racing data from the three New York Racing Association tracks supports a recurrence rate approaching 25% for horses with tendonitis.[3] The overall rate is clearly higher when one considers horses lost to follow-up or re-injured before reaching racing fitness.

Acute tendonitis is usually not apparent on the racetrack and usually is diagnosed by the attending veterinarian several hours later. Severe or total disruption of the SDFT causes obvious lameness at the completion of training or a race. Lameness usually increases rapidly and hyperextension (dropping) of the metacarpophalangeal joint is apparent.

Recurrent tendonitis is usually more severe than the initial injury. A horse may pull up before completing training or a race. Swelling is often substantial, the mid-metacarpal region lacks definition, and the metacarpophalangeal joint drops moderately. The degree of swelling may preclude accurate diagnosis of what soft tissue structure is involved. The prerace inspection is particularly important in horses with a history of tendonitis, because this may be the determining factor in preventing re-injury. Careful palpation for sensitivity and swelling can reveal subtle changes that may have gone undetected by the trainer and exercise rider, because these horses usually continue to train without obvious lameness. The track veterinarian may require an ultrasonographic evaluation before permitting the horse to race. Comparison with previous examinations may demonstrate a substantial lesion, although the horse is sound at the trot and gallop. Despite all efforts, tendon re-injury occurs to some horses during racing or in workouts. When re-injury does occur, the application of a compression boot or splint is required. Horses with severe tendonitis, even with loss of metacarpophalangeal joint support, can heal with time to become pasture sound.

Digital flexor tendonitis in the hindlimb is unusual, but tendons are vulnerable to severe laceration and blunt trauma by being cut down during a race. Injury may occur when the foot of a following horse strikes the tendons of the hindlimb. The shoe, particularly one with a toe grab, may lacerate and completely sever the SDFT and less commonly the deep digital flexor tendon. Wounds often are badly contaminated and prognosis is guarded because of complications from infection. A clean support bandage is applied initially, and light sedation and analgesia are given. Further splinting is usually unnecessary and is not well tolerated. Immediate transport to an emergency hospital is best.

Suspensory Ligament Injuries

Injuries of the SL and digital flexor tendons have many similarities, are relatively common, vary considerably in severity, and predominantly involve the forelimb. Branch or body desmitis often is detected after the morning exercise when the horse is cooling out. Moderate or severe desmitis causes acute lameness, possibly with some degree of metacarpophalangeal joint drop, but swelling may initially be minimal. A presumptive diagnosis is made while the horse is still on the track, but differentiating between the SL and digital flexor tendon injury may be difficult. An important principle in emergency management is providing immediate support. A splint that supports the metacarpophalangeal joint is always indicated whenever suspensory disruption is suspected. Obvious dropping of the metacarpophalangeal joint during weight bearing reinforces the decision to use the standard Kimzey splint. PSB fractures often may occur concomitantly, but they may not always be readily apparent. Progressive loss of metacarpophalangeal joint support occurs as the severity of suspensory tearing increases. If the injury is closed, the on-track management remains the same. Complete SL disruption and disarticulation of the metacarpophalangeal joint was discussed previously (see page 858).

The distal sesamoidean ligaments are a vital component of the suspensory apparatus and even mild desmitis should be considered a serious injury in the Thoroughbred. Horses with mild desmitis look similar to those with digital flexor tendon or SL injuries. These injuries may appear to heal, but recurrence is common and problematic. My strong clinical impression is that horses with previous distal sesamoidean desmitis are at risk to develop catastrophic injury, and close monitoring of the status of these injuries is imperative when horses return to training. Any indication of recurrence of desmitis is sufficient cause for retirement or at least additional time off for re-evaluation. Re-injury is often catastrophic, with complete rupture of the ligaments, proximal displacement of the PSBs, and disarticulation of the metacarpophalangeal joint with multiple secondary injuries. Euthanasia usually is required for these horses.

REGULATORY CONSIDERATIONS

Attending veterinarians must be aware of their responsibility to the regulatory body, that is, the state racing commission or its equivalent. Some jurisdictions require a written report to be filed for any horse euthanized on official racetrack grounds. In lieu of a formal report, a good policy is to contact the track stewards and state or association veterinarian to inform them of catastrophic injuries that occur during training or racing. A necropsy and samples for pathological examination may be commission requirements and are vital for statistical evaluation of injuries at the racetrack. Important information can be obtained relative to the nature of the injury, track condition, age and sex of the horse, and other possible risk factors. The official track veterinarian, whether state or association, is fully knowledgeable of commission requirements and appropriate procedures and so is a valuable source of information for the veterinary practitioner on the backstretch. Free exchange between professionals is mutually beneficial. The official veterinarian frequently requires the prompt assistance of the stable veterinarian to continue the management and treatment of a seriously injured horse.

EUTHANASIA AND INSURANCE

When attending a horse with a catastrophic injury, the veterinarian should instruct the owner to notify the insurance carrier, if applicable, as soon as possible. In the event that euthanasia is not immediately essential but is later considered, most insurance companies require a second opinion. Horses with catastrophic injuries may require immediate euthanasia at the scene on the racetrack when supporting and loading the horse onto the ambulance is not possible. Regardless of the circumstances, thorough documentation with photographs of the injury and specifics of the incident should be maintained. If possible, radiographs should be obtained to document the nature of the injury. It is always advisable to have a representative of the stable present to provide consent for euthanasia, but this is rarely the owner. When consent cannot be obtained, the veterinarian on the scene must do whatever is indicated to alleviate distress and protect the horse from further injury until a consulting veterinarian arrives.

On rare occasions the veterinarian may be confronted with an uncontrollable horse or a situation in which euthanasia is the only course of action remaining, consent and consulting opinion being unavailable. After exhausting reasonable attempts to stabilize the horse and obtain assistance, the professional decision for euthanasia can be exercised without excessive concern. Documentation in these horses should include necropsy examination. Although extremely unlikely to result in a legal challenge, thorough communication and documentation is always advisable. Euthanasia is the final option available to the clinician and is justified to prevent further pain and distress. Although the technical aspects of the procedure are simple, resulting in a fast and humane death for the horse, veterinarians must exercise discretion and sensitivity. As disturbing as the situation may be for the attending veterinarian, there is no way to gauge the emotional and aesthetic impact to others who might be present. Persons with direct connections to the injured horse, track workers, media representatives, and patrons may all be within viewing range. An effort to screen the horse from onlookers during euthanasia and while removing the body is recommended. Those immediately connected with the horse should be afforded the opportunity to leave, although some will wish to remain. Finally, the veterinarian must remain in control of the scene, being cognizant of not only the needs of the horse, but also the concerns and safety of all others present.

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CHAPTER • 106

Epidemiology of Racehorse Injuries

Calvin N. Kobluk

Veterinarians and trainers have long recognized that racing injuries can be caused by racetrack design and track surfaces, but little scientific data relate musculoskeletal injury to these factors or document the economic impact on the racehorse industry.

Early studies in the United Kingdom demonstrated that lameness was the most common reason that horses in training failed to race.[1,2] Premature retirement, reduced performance, and injury result in economic losses to racehorse owners. In North America, most emphasis has been placed on catastrophic breakdown injuries because of the dramatic nature of these injuries, controversy in the media, and public awareness.[3-6] Attempts have been made to define factors involved in the incidence of these injuries.[7-12] A limited number of studies

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correlate day-to-day injury with racetrack surface characteristics[13-15] and other risk factors associated with Thoroughbred racing.[16-20]

EFFECT OF RACETRACK SURFACE

Components and Geometric Design of a Racetrack

Thoroughbred (TB) and Quarter Horse (QH) racetracks are similar in design worldwide, except where horses race on turf (see Chapter 108). The dirt track comprises a roadbed, covered with crushed stone and sand, with an overlying cushion of fine sand and organic material (Fig. 106-1). Standardbred (STB) tracks are harder to accommodate the sulky and different nature of the trot and pace gaits compared with the gallop. Turf tracks in North America consist of grass grown on a sandy soil base covering a crushed stone layer and the roadbed. Some turf tracks in Europe are natural surfaces, sown with special grass mixes.

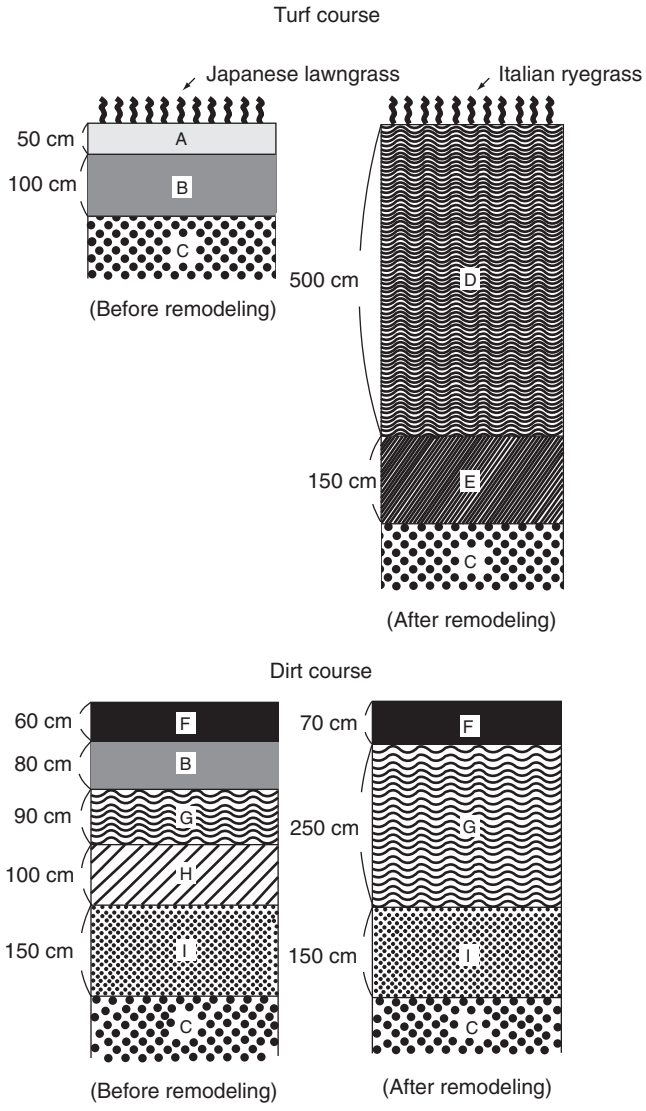

Fig. 106-1 Cross-sectional diagram showing the components of a section of the turf and dirt tracks before and after remodeling of the surface. Incidence of several racing injuries was significantly less after surface remodeling.[22] *A*, sandy clay; *B*, screened hill sand; *C*, road bed; *D*, sandy soil; *E*, single-sized aggregates of crushed stone; *F*, river sand; *G*, hill sand; *H*, volcanic gravel; *I*, crushed stone. (From Oikawa M, Veda Y, Inada S, et al: Effect of restructuring of a racetrack on the occurrence of racing injuries in thoroughbred horses, *J Equine Vet Sci* 14:262, 1994.)

Although racetracks in North America and many other countries are oval shaped and similar in size, with horses consistently racing in the same direction, in Europe each turf track is different, and horses race both left- and right-handed. However, the all-weather tracks in Europe are more consistent in design.

The discussion in this section on banking focuses on the purpose-built oval tracks. Banking in the turns is generally inadequate. A 6% grade is recommended for TB dirt racetracks, but this creates difficulties in track maintenance. The track surface material shifts along the rail, making maintaining a uniform depth of cushion difficult. An 8% grade is recommended for turf tracks. Anecdotal evidence and some research indicate that properly banked turns are extremely important in preventing breakdowns, and this is especially true at the end of the race when horses are fatigued. The banking of racetracks in Japan varies from 1.7% to 3% on dirt and 1.6% to 4.5% on turf, both far less than ideal.[21,22] A technique called *spiraling* also may improve track safety. The spiral curves improve the geometry of the racetrack, because no longer does the curve start at a single point, and a horse enters and leaves a turn in a more gradual manner.

Effect of the Physical Properties of the Racetrack and the Weather

A limited number of studies have attempted to document physical characteristics of the racetrack. The University of Minnesota American Association of Equine Practitioners Breakdown Study, hereafter referred to as the Minnesota study, conducted the first series of larger studies attempting to characterize racetrack conditions.[15,16] Weather conditions, the physical properties of the soil, including moisture content, composition, strength, cone penetrometer, and coefficient of friction between the surface and the hoof were documented at three successive racing meets at Canterbury Downs, Shakopee, Minnesota. The moisture content of the track was significantly different at various locations around the track and on different days. Rainfall contributed heavily to the moisture content of the track surface.

Track management can compensate for excessive dryness by adjusting the water management program. Racetracks located in the southwestern United States are far less affected by rainfall, and thus track management and moisture content become important management tools. Racetracks in California absorb about 136,500 L (30,000 gal) of water daily.[13] Excessive water in a track cushion eliminates air pockets between the soil materials and affects the load-bearing capacity of the track. Conversely, too little moisture prevents the soil materials from holding together. Studies at Washington State University suggest that high moisture content was the least efficient and indicate that intermediate moisture content is optimal (9% to 11%).

Organic matter content of the track is important because it enhances the moisture retention, decreases dusty conditions, decreases the ability of the soil to compact, and helps create a spongy surface. Excessive organic material results in increased drying time and stickiness of the track material. The Minnesota study monitored soil particle distribution, but no logical pattern was observed.[15,16] Diameter of the soil material is thought to be important. Too much soil matter of one diameter will pack, resulting in a firm track surface. Material of uneven diameters breaks up, causing traction problems for the horses. Further studies are necessary to determine the effect of soil particle distribution on track surface characteristics.

Shearing strength was measured using a hitch dynamometer. Some areas of the track were compacted, such as at points of high traffic from horse movement, starting chutes, maintenance equipment, water trucks, and general access to the track. These compacted areas in the Minnesota study were correlated with areas that had a high incidence of breakdown injuries. Changing the entry point for maintenance equipment and water trucks to a different area in the track with less racing traffic could have a positive effect on this compacted area. Shearing strength of the soil was determined by a cone penetrometer. The penetration force next to the rail was reduced, indicating that resistance in the soil was lower. The cone penetrometer also revealed differences in track surface between racing seasons. This may be attributable to increases in moisture content or organic matter in the track surface. Anecdotal evidence suggested that the jockeys learned that the soil next to the rail was looser and thus was not an ideal location for racing. By moving slightly farther away from the rail, where the cone penetrometer showed more resistance, the jockeys had a perception that the track was faster.

Anecdotal evidence suggests that harder tracks are faster tracks. The thought is also that harder tracks result in more mild to moderate injuries. Track surface studies at the University of Minnesota suggested the hitch dynamometer was useful in identifying compacted areas of the track where a cluster of injuries had occurred. Further research is necessary to determine whether the hitch dynamometer or the cone penetrometer is a better method of monitoring track surface hardness.

The Minnesota study indicated that the primary factor affecting the track surface is moisture content and the second most important factor is the organic material. Soil particle distribution should be evaluated to monitor the change in track composition, and cushion depth should be determined to ensure that areas of accumulation, especially near the rail, do not occur. Conversely, thinning of the cushion near the crown of the track could represent a significant increase in compaction and thus become a risk factor. Areas of high traffic access to the track need to be located where racing is minimal and not toward areas where racing traffic is high, at turns, or where fatigue becomes a major factor for the racetrack, such as the final turn.

The Japanese Racing Association demonstrated a seasonal effect, suggesting that cool weather has a strong influence on early morning training and potential for accidents. Whether cool weather influence is a track-related or a horse-related factor is unknown. The Japanese Racing Association conducted another study using horses with shoes that were fitted with accelerometers.[21] The higher the acceleration recorded by the accelerometer, the greater the force of impact to the limb. Thus any risk factors that can reduce acceleration of the limb reduce the forces exerted on the limb. Acceleration declined dramatically as the water content rose in turf courses. Lower acceleration was related to an increased depth of cushioning sand on dirt courses. Harrowing the racetrack reduced the acceleration rates by 38% compared with a track that had not been worked.[21,22] The shock wave patterns measured by the accelerometers were found to be a dangerous type when the track bed was exposed and the cushion was thin. Harrowing resulted in more uniform leg motion, and 94% of the shock wave patterns were of the safe type, indicating a stable running surface.

As a result the Japanese Racing Association developed new racetrack maintenance equipment, including a turf perforator to prevent hardness, a level harrow to even out the cushioning sand, a track hardness measuring van using a penetrometer, a dirt cleaner to remove pebbles from the cushioning sand, a water absorption roller to improve turf courses, an automatic water sprinkler system to ensure evenness in the water content of the track, a sand thickness gauge to ensure evenness of the cushion, and a side raker to ensure the cushion does not accumulate under the rail.

Synthetic Surfaces

Tracks composed of a large proportion of organic particles, such as wood fibers, wood chips, or sawdust, have the greatest dampening ability. Synthetic track surfaces were used on two major racetracks in the United States with a relatively low injury rate, but both have been removed and replaced with a conventional surface. Synthetic surfaces are still used in various training centers with success and at some all-weather racetracks in the United Kingdom. The synthetic tracks appear to be safe, with injury rates reported well below the industry standard, and also require less water and remain fast, even in adverse weather conditions. Unfortunately, synthetic tracks exhibit variability in the surface during hot, dry spells. Some portions were excellent, whereas other sections were uneven and fluffy. This lack of consistency resulted in the decision to remove the synthetic surfaces in the United States and replace them with conventional surfaces. The basic approach with a synthetic material is to add particles with the polymer coating and use this material for the track and the cushion. Synthetic materials also play a role in turf courses where they are used to anchor the roots of the grass, thus resulting in fewer divots and greater integrity to the turf course.

EPIDEMIOLOGICAL STUDIES OF RACETRACKS

A case-control study performed in New York identified one racetrack with a lower incidence of injuries.[20] The Equine Racing Injury Reporting System identified great variation in injury rates among participating racetracks, suggesting race surface is an important factor in the development of musculoskeletal injuries in North America. The reporting system information in 1992 showed the fatal injury rates for dirt starts on various tracks ranged widely, with a twofold difference between the lowest and the highest.[7] Great variability in track fatality rates also has been demonstrated in the United Kingdom.[23] Fatal injury rate at a given track varies from year to year. At some tracks racing injuries occur more frequently on certain days of the week and during specific race numbers. Racing on dirt surfaces represents 90% of all TB races in North America, and a higher fatal injury rate was observed on dirt versus turf. The Equine Racing Injury Reporting System study provided the first nationally based number for fatal racing injuries in TBs (1 death per 629.6 starts). The study also documented a range of fatal injuries between racetracks. The difference between dirt and turf racing has been demonstrated (1 death per 619.5 dirt starts and 1 death per 759.4 turf starts). More compelling evidence implicating the racing surface exists in the Japanese Racing Association study, which identified one particular racetrack with an increased incidence of injuries compared with the other tracks.[21,22] Changes in racetrack surface management were implemented and the injury rate was lowered. A strong perception exists in the racing industry that turf courses are safer than dirt courses. Analysis of this issue is clouded to some degree in North America because horses that race on turf still spend most of training time on dirt surfaces. The incidence of catastrophic breakdown injuries is much less in the United Kingdom, where many horses train and race on turf, but training and racing also take place on more varied terrain, including hills, and involve considerably fewer turns. The New York case-control study indicated a decreased risk of injury associated with racing on turf courses. An Australian study reported low catastrophic injury rates associated with turf racing.[24] The turf as a surface may be more forgiving for TB racing, but one of

the difficulties in the analysis is that most of the horses racing on turf are of higher quality, and thus factoring the comparison of turf racing versus dirt racing, without taking into account the quality of the horses, may yield invalid conclusions. A study comparing similar-quality horses racing on turf versus dirt is necessary. In addition, monitoring groups of horses that trained and raced on dirt versus horses that trained on dirt and raced on turf versus horses that trained and raced on turf would be interesting.

Many of the case-control and descriptive studies have included track condition and type as part of the study,[7-15,20] which is due to the reporting of track condition and type as assessed by the racetrack and commercially available on databases. In the New York study, horses that raced on firm turf had significantly lower risk of severe injury compared with those that raced on normal dirt.[14] Fast, sloppy tracks resulted in a higher risk of injury. Slower, muddy tracks resulted in a lower risk of catastrophic injury. The Minnesota study showed no apparent relationship between breakdown occurrence and the length of the race, and whether the race was on dirt or turf. Fifty-four percent of the major injuries occurred in the final turn (Fig. 106-2). This clustering of injuries was at an area where all the mechanized equipment was brought on to the track, was a transition point from a turn into a straightaway, and at a stage in the race when the horse is experiencing major fatigue.

The risk of catastrophic injury is not necessarily the same as the risk of other musculoskeletal injuries. Using other data collected from New York racetracks, the prevalence of fractures and soft tissue injuries could not be correlated to track conditions, environmental conditions, length of races, track location where the fractures occurred, or age of the horses. However, pooling of the data from four New York tracks showed that injuries increased from 2.12 injuries per 1000 starts on a fast track to 3.26 per 1000 starts on a muddy track and 2.76 injuries per 1000 starts on a sloppy track. Pooling also indicated that one track had twice as many fractures as another track in the study, strongly supporting the physical condition of the racetrack as a major risk factor in TB racing.

A Japanese Racing Association study indicated that the rate of accidents on turf courses was 1.58% compared with 1.65% on dirt courses.[21,22] Accident rates on turf courses were highest when the tracks were in fast condition, and the firmness of the track was thought to be a major factor. Dirt tracks had highest accident rates when the tracks were heavy, with high water content, possibly as a result of loss of the shock-absorbing effect of the cushion. The Japanese study also indicated that racetracks with higher injury rates had tighter turns and tended to be flatter courses.

A descriptive study involving 1100 racing injuries from 33 racetracks revealed that most musculoskeletal injuries occurred while horses are racing on dirt (948). Fatal injuries had slightly higher prevalence (27%) on dirt tracks compared with turf tracks (23%). Forelimbs accounted for most of the injuries. Stallions were overrepresented in the fatal proximal sesamoid bone fracture group. A prospective study of fractures in TB racehorses in England revealed an annual incidence of 9%, with 206 occurring during training compared with 21 during racing.[25] A seasonal distribution was identified and coincided when fast work was commenced. A German study showed that training failure was from lameness 57% of the time and again intensified with the onset of speed work.[26] An Australian case-control study found that horses running in stakes races were 2.3 times more likely to suffer a musculoskeletal breakdown than horses in a non-stakes race.[27] This is a substantially different finding from North American studies.

A recent Australian study demonstrated that fatalities are less common on Australian racetracks than those in the United Kingdom or in the United States.[27] The study demonstrated that the most significant factors associated with serious injury included track condition and location of the track. Horses racing on a fast, good track were twice as likely to suffer a serious limb injury as those running on a slow or heavy track. In addition the type of race had a large affect on the risk of injury, with horses in the hurdle races being 4 times more likely to suffer a serious injury and those competing in steeplechase races 7.5 times more likely to sustain a serious injury compared with racing on the flat.

The racing industry is keenly aware of the effect of the racetrack surface conditions and design contributing to musculoskeletal injuries at all levels. Information is limited, however, on how to correlate these data with the physical condition of the horse. Measurements of forces between the hoof and the ground indicate that peak vertical forces are 175% of body weight for the gallop, with forces 10% to 15% less on the hindlimb than the forelimb. Early studies used the drop-hammer technique to test the ability of the track surface to absorb kinetic energy from the hoof, as a model of the physical characteristics of the cushion.

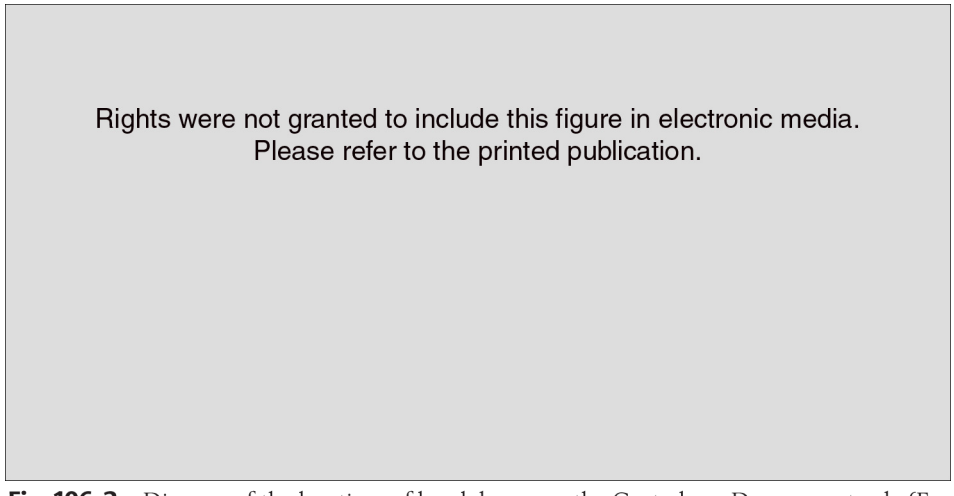

Fig. 106-2 Diagram of the locations of breakdowns on the Canterbury Downs racetrack. (From Clanton C, Kobluk CN, Robinson RA, et al: Monitoring surface conditions of a thoroughbred racetrack, *J Am Vet Med Assoc* 198:613, 1991.)

TRAINER-RELATED FACTORS AND EFFECTS

Information is limited about the effects of training programs on the prevalence of musculoskeletal injury. The Minnesota cohort study demonstrated large variations in training programs of the 11 participating trainers.[18] An arbitrary exercise score, problem score, and success score was assigned to each horse participating in the study. Only 17 of 95 horses did not develop any type of injury. Fifty-seven percent of the study horses were removed from training before conclusion of the study, 33% because of musculoskeletal injury. The relationship between the exercise and the problem score was linear. As the problem score increased, the exercise score decreased. As the exercise score decreased, the success score decreased. The trainer that had the highest average success score also had the highest exercise score and the lowest average problem score. As expected, the most successful training programs had a relatively low injury rate that allowed horses to be worked on a more regular basis and to compete more successfully. This trainer also routinely used ice whirlpool baths far more often than any other trainer in the study. A Kentucky study showed that injured horses had significantly less cumulative high-speed exercise than did control horses.[28] In my opinion, further cohort studies emphasizing the trainer as a risk factor are required to reduce the incidence of musculoskeletal disease in racehorses.

HORSE-RELATED FACTORS AND EFFECTS

Breed

The incidence of catastrophic injury in TB racehorses is much higher than in QHs and STBs, possibly from differences in training practices in QHs and the less stressful racing gait and slower speeds of STBs. The fatal injury rate in TBs was equal during training sessions and racing. QHs had much fewer training injuries. The Equine Racing Injury Reporting System survey had a low number of reports on QHs, but the injuries were more severe. Sixty percent of QHs died or were euthanized compared with 22% of TBs. No studies document the incidence of mild to moderate injuries in the three breeds.

Conformation and Quality

Preliminary conformation evaluations were carried out during the Minnesota study.[17] This suggested that horses with certain forelimb and hindlimb ratios were less likely to experience musculoskeletal injury. A subsequent study at Colorado State University on the conformation in racing QHs revealed an association between conformation and the odds of sustaining various racing injuries.[29] The study used 160 2-year-olds in race training and compared measurements taken from photographs with the prevalence of musculoskeletal injuries. This is discussed further in Chapter 4.

Pre-Existing Pathological Conditions

A study performed using data collected from New York racetracks was the first indication that pre-existing pathological conditions may be important in the development of catastrophic musculoskeletal injuries.[30] This study suggested that pre-existing pathological conditions and not racetrack conditions were the primary causes of catastrophic injuries. The authors proposed that pre-existing osteochondrosis, chondro-osteonecrosis, and tendon and ligament injuries predisposed to injury. The controversial conclusions also incriminated the overuse of corticosteroids.

The Minnesota study attempted to address the issue of pre-existing pathological conditions by a clinical, radiological, and ultrasonographic evaluation of the 97 horses in the cohort study. Prestudy and poststudy radiographic examinations revealed detectable lesions in the carpus in 72%, in the metacarpal region in 42%, and in the fetlock in 82% of horses. The incidence of definitive pathological lesions using pre-ultrasonographic and post-ultrasonographic examination was low. Post-mortem studies have shown a predisposition to complete humeral fractures often caused by pre-existing stress fractures of the humerus.[31] Seventy-seven percent of horses with complete fractures of the humerus had evidence of pre-existing stress fractures. Pre-existing stress related bone change was found in horses with pelvic fractures, scapular fractures, tibial fractures, and metacarpal fractures.[32,33] A descriptive study from Kentucky identified that a significantly greater number of horses with racing injuries and catastrophic injuries were in the last half of the racing group at the quarter pole, possibly indicating that pre-existing pathological conditions impaired racing ability.[8,10] Most studies strongly support a higher catastrophic injury rate in claiming horse compared with stakes and allowance horses. Many claiming horses were previously higher-quality horses, but because of musculoskeletal injury they dropped to a lower racing class. Anecdotal evidence suggests a much higher incidence of pre-existing musculoskeletal injuries in lower-quality horses, because many of these horses were previously racing in a higher class but were lowered because of injury.

Age

Racing in all breeds clearly is dominated by younger horses, with most of the purse directed toward horses in the 2- to 4-year-old range. The issue of racing younger horses, especially 2-year-olds, has long remained a controversial one. In some cases, scientific data supports the discontinuation of 2-year-old racing, but in other cases it does not. Further research is necessary to define whether 2-year-old racing is appropriate or at least how such racing is best accomplished to reduce the incidence of musculoskeletal problems. California studies showed that fatal injuries occurred most commonly in the 2-, 3-, and 4-year-old horses; but this reflects the largest group of horses in racing or training. More 2-year-olds were injured during training than during racing, possibly reflecting the stress of the change in environment when they arrive at the track. An investigation of 1100 reports of racing injuries (Equine Racing Injury Reporting System) indicated that racing injuries in 2-year-olds had a lower rate, 3.21 per 1000 starts, and that the fatal injury rate was lower than the other age groups, 1.43 per 1000 starts. In addition, more injuries occurred in the latter half of the year, suggesting more starts later in the year for the 2-year-old population or possibly an effect of fatigue and injury later in the racing year of these 2-year-olds. An Australian study showed that horses in the 4 to 5 years of age range were about 1.5 times more likely to suffer a serious catastrophic injury compared with those aged 2 and 3 years. In contrast a New York study identified a negative association between the risk of severe injury and the age of the horse.

Shoeing

The Minnesota study was the first study to investigate the possible effect of shoeing practices on TB racehorses.[17] In the cohort study of 97 horses, those shod at a higher angle were less likely to experience injury, earned significantly more money, and were more successful. A case-control study published from the California post-mortem program reported an association between toe-grabs and the increased risk of catastrophic musculoskeletal injuries, suspensory apparatus failure, and condylar fractures, demonstrating an association between the height of toe grabs and a twofold to sevenfold increased risk for these injuries.[19] Rim shoes were found to be associated with a decreased risk of catastrophic injury, lowering the odds of injury 2 to 3 times. This study has led to the intervention of racing regulatory officials in mandating certain types of shoeing for racehorses.

Post-Mortem Evaluations of Catastrophic Injuries

The New York study, suggesting that pre-existing pathological conditions were a primary cause for catastrophic accidents, was the first major post-mortem investigation of racing injuries.[30] The Minnesota study also attempted a post-mortem evaluation of horses with catastrophic racetrack injuries. Left forelimb injury predominated. The design of the case-control study used two randomly selected control horses from the same race in which the breakdown occurred. Lifetime earnings were significantly less for injured horses than control horses. Horses with bone injures were 7 times more likely than the control horses to have made less than 40% of race starts at Canterbury Downs and were 7 times more likely to have raced within the previous 12 days. Horses with soft tissue injuries were 2.5 times more likely to have been scratched by the track veterinarian. Fifty-four percent of the major injuries occurred during the final turn in this study. Fractures below the carpal joint accounted for 75% of all the injuries, with 60% of the horses having some post-mortem evidence of a previous injury, and 72% of the injuries involved the lead leg.

In Japan the Japanese Racing Association administers all racing and is able to document racing and training injuries. From 1985 to 1994 a greater number of fractures occurred during training than during racing each year, suggesting that training injuries are at least as significant as racing injuries in TB racehorses.[21] Fractures tended to be more severe during racing than during training.

The case-control study of racetrack breakdowns in New York associated a higher risk with years racing, racing in a later race, number of starts per year, and the total number of starts and age of the horse. Risk of injury was greater in the summer than in the winter or spring.[20] California studies showed the most common catastrophic musculoskeletal injury occurred in the proximal sesamoid bone and the suspensory apparatus. In a case-control study of racing-related risk factors, severe injuries in TB racehorses in California showed that stress-related injuries accounted for 83% of the deaths.[34] Complete fracture of the long bones accounted for 32% of the deaths. Seventy-seven percent of the complete fracture of the humerus had pre-existing stress fractures.

In a study from Kentucky the forelimb below the carpus was the most commonly injured area. The forelimb accounted for 90.2% of the racing injuries, with the suspensory apparatus the most frequently involved (44.7%). The right forelimb was injured more commonly after the wire, whereas the left forelimb was injured more commonly in the stretch turn. Many risk factors were assessed, including weight carried during the race, weight carried during the previous race, number of competitors in the race, duration of the race, changes in distance between the race in which the injury occurred and the previous race, number of career starts, interval between races, number of career wins, the amount of career earnings, sex, racing surface, race speed, track identity, status of jockey, and racing class or changes in racing class. None appeared to affect incidence of injury.

RACING ACCIDENTS

A descriptive study from the Japanese Racing Association, using video recordings of races, found that accidents from physical interactions or stumbling were predisposed by changing of the line of racing, oblique movements, or some action that upset the center of gravity of the jockey or horse.[35] The Kentucky case-control study demonstrated by video that horses had a 2.5 times greater chance of injury if a physical interaction occurred during the race.[8,10] The horses were at a 4.2 times greater odds of injury if they stumbled during the race, and a 7.7 times greater odds of catastrophic breakdown if stumbling occurred. An Australian study at two racetracks over a 10-year period found that horses with outside positions were twice as likely to suffer an injury versus those starting close to the rail. The Japanese Racing Association found that accidents during racing were common in the early spring, whereas training accidents were more frequent during the winter months.

FUTURE INITIATIVES IN THE STUDIES OF MUSCULOSKELETAL INJURY

In a report by the Equine Racing Injury Reporting System to the American Association of Equine Practitioners the value of accurate records documenting racing injuries has led to an injury monitoring system. Standardization of the reporting practices will facilitate comparison of databases from different racing jurisdictions. A national monitoring system implemented in 1992 provides a common format for regulatory veterinarians to record injury information and accumulate comparable data on a large scale. Routine racing and surface monitoring is being used at some level worldwide, and further techniques currently are being investigated. Some of the techniques are relatively easy to perform, such as monitoring cushion depth, moisture, and soil particle distribution. The development and use of modern technology allows the recording and maintenance of track parameters within desired guidelines and eventually will allow the correlation of surface condition parameters to the occurrence of musculoskeletal injury. Monitoring the following environmental conditions, including weather reports, track superintendent's logs, moisture content, cushion depth, bulk density, organic matter, shearing strength, and conditions at specific injury sites, will document the physical parameters of the racetrack. In addition, significant work on racetrack dimensions such as track slopes and configurations should be determined to facilitate comparison of injury rates with other tracks. Case-control studies will continue to allow the evaluation of these major racing injuries and hopefully will identify risk factors associated with them. In my opinion, cohort studies that follow a large number of racehorses on a day-to-day basis will be far more valuable in determining the risk factors on musculoskeletal injury. Such studies will identify better not only the risk factors associated with the catastrophic injuries, but also the mild to moderate injuries that have such a major economic effect on the racing industry. Many of the studies suggest catastrophic injuries tend to be random events, but certain risk factors clearly are associated with occurrence. The evidence for pre-existing musculoskeletal injury subsequently leading to catastrophic breakdown is compelling, and thus studies that identify these pre-existing injuries and correlate them to risk factors would be especially important in subsequently reducing catastrophic breakdown.

The Japanese Racing Association clearly has demonstrated that identifying the prevalence of racing injury rates, analyzing associated risk factors, and subsequently making management changes result in a reduction in injuries. Some of the risk factors are relatively easy to identify and make appropriate management changes; for example, toe grabs used on racing plates. Other risk factors, such as the effect of the trainer and the training program, are far more complex and will require large-scale cohort studies to demonstrate clear and useful conclusions. In my opinion the use of these types of studies, involving a number of trainers in a number of environments, will clearly define optimal training programs and methodologies that will significantly reduce mild to moderate injuries and subsequent catastrophic injuries.

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CHAPTER • 107

North American Thoroughbred

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DESCRIPTION OF THE SPORT

King James I of England established horse racing as the Sport of Kings in the early seventeenth century and the earliest English immigrants brought the sport to the American colonies. Colonials imported fine-blooded horses and staged informal race meets almost as soon as they hit land. Richard Nicolls took command of New Amsterdam, renamed New York, became the first governor of the colony in 1664, and established a racecourse.

Many of the founding fathers of the new country, including George Washington and Thomas Jefferson, raced horses. John Wickham, Aaron Burr's attorney at his treason trial, lost one of the top American stallions of the emerging Thoroughbred (TB) breed, named Boston, in a card game in 1835. Nathaniel Rives won the difficult 2-year-old colt and ignored advice to geld him. Boston not only won 40 of 45 starts, but he also sired Lexington. A foal of 1850, Lexington became the top racehorse in the country by winning from New York to New Orleans.

Lexington was named for the Kentucky town that became the center of TB breeding. Horsemen quickly realized that healthy horses could be raised on the limestone base of Central Kentucky, and the horse, Lexington, helped establish the area by becoming the top sire in the United States for 16 years.

Racetracks sprung up in many cities, and although the industry suffered during the Civil War, Saratoga was the exception. The New York social elite took in the waters at the Spa to escape the heat and humidity of the city, attended the races during the day, and partied through the summer evenings in spite of the war. A few years after the conflict, Col. M. Lewis Clark founded the Louisville Jockey Club to conduct race meets in Kentucky. In 1875 the Louisville Jockey Club opened a new racetrack, later called Churchill Downs, and that year Aristides won the first edition of the Kentucky Derby (the Derby).

The Derby did not begin as America's most famous race. Col. Matt Winn's genius at marketing in the early twentieth century was required to create the legend the Derby has become. When the filly Regret won the race in 1915, the Kentucky Derby rose to prominence quickly as a premier American sporting event and a major stop for 3-year-old TBs.

Fans were disappointed when owner Sam Riddle and trainer Louis Feustel deemed the Derby too early in the year for their strapping colt, Man o' War, the horse that had broken many records the previous year, winning 9 of 10 races. However, Man o' War added to his reputation without the Derby, going through a perfect season of 11 wins in as many races. To many he remains the finest TB racehorse the United States has ever produced.

A new moral climate was sweeping the nation after the turn of the century. Gambling, including on horse racing, was banned in many states as a precursor to Prohibition that began in 1919. Kentucky was the exception and horsemen and racing fans found many ingenious ways to skirt the law. The need for more state revenue from the depression in the 1930s prompted the ban on racehorse wagering to be lifted in many states. Until the 1970s horse racing was often the only legalized form of gambling in most states. From the 1930s to the 1960s horse racing, boxing, and baseball were the most popular sports in the United States.

War Admiral, sired by Man o' War, gained such a following in the 1930s that the whole country listened to the Seabiscuit–War Admiral match race. Because the older Seabiscuit had lost his first 17 races as a 2-year-old, everyone expected War Admiral to win. Yet Seabiscuit, "the people's horse," beat War Admiral that day, and in 1938 Seabiscuit was said to have had more newspaper space than Hitler.

As a 3-year-old, War Admiral won the Derby, the Preakness, and the Belmont Stakes, races that became known as the Triple Crown. Only 11 horses have ever won all three races. In 1948 Citation so impressively captured the series that some thought the horse replaced Man o' War as the greatest racehorse. That argument ensued for the next 25 years, despite names like Native Dancer, Swaps, Kelso, and Dr. Fager, none of which won the Triple Crown. However, in 1973 a bright chestnut colt, Secretariat, so captured the public imagination when he won the Triple Crown that *Time*, *Newsweek*, and *Sports Illustrated* all put him on the cover. He won the 1½-mile Belmont stakes by a spectacular 31 lengths.

Although the Triple Crown increased in stature, California developed a strong racing circuit. Santa Anita, east of Los Angeles, opened in 1934 and gained immediate attention by offering the then unheard of sum of $100,000 (U.S.) for the inaugural Santa Anita Handicap. Across town, Hollywood Park began attracting movie stars and established its own fixture, the Hollywood Gold Cup. Bing Crosby and friends built Del Mar about 100 miles south.

As transportation by air became more popular in the last half of the 20th century, horses from the East and West began meeting regularly. In fact, Affirmed, the latest horse to win the Triple Crown, in 1978, prepared for racing at Santa Anita and Hollywood Park.

Regional rivalries became popular, which added to the success of the Breeders' Cup series, inaugurated at Hollywood Park in 1984. Unlike the Triple Crown, which is solely for 3-year-olds, Breeders' Cup Day offers races for various divisions. Two-year-olds show what might emerge at the next year's Kentucky Derby. European horses often invade for the grass events. The Breeders' Cup Classic attracts elite runners on the dirt from coast to coast and from abroad.

The Breeders' Cup epitomizes the tenacity of the racing TB. Spectacular victories—including the triumph of Sunday Silence over archrival Easy Goer in the 1989 Classic, the victory of Personal Ensign over the Kentucky Derby–winning filly Winning Colors in the 1988 Distaff, and the devastating move Arazi made to capture the 1991 Juvenile after shipping to the United States from France—are legendary. American TBs regularly compete in Europe, Japan, Australia, and Dubai. Not even King James I could have envisioned the Sport of Kings developing into the international phenomenon it is today.

TB racing always has been an expensive sport. Since the introduction of parimutuel gambling, financial support and revenue has been derived from two groups, racehorse owners and the gambling public. The third leg of the horse racing industry is the racetrack operator. Racetracks are generally for-profit corporations, with the exception of Del Mar, Keeneland, and a few others. Racehorse ownership was once the domain of the wealthy, but now opportunities and partnership options are available for many. In the past, most owners maintained breeding operations to supply horses for their personal racing stables; commercial breeders were few. The focus of breeding was for successful racehorses and rigorous selection for soundness was as important as racing ability. Commercial breeders have now become a major source of racehorses and have a different goal. They produce horses to satisfy the commercial market, an entirely different objective, often unrelated to producing a racehorse.

Commercial breeders offer horses at public auction, primarily as yearlings but also as weanlings and 2-year-olds in training. The auction market is fashionable and in many ways fickle and trendy. Buyers favor precocious yearlings, relatively more mature than others. Sprinting bloodlines and well-muscled yearlings sell better than classic distance pedigree and conformation for all but the most expensive horses. Little is known of soundness of the sire and dam. Although these trends are worrisome, one positive consequence has been the ever improving quality of horse offered at public auctions. Now some of the best pedigreed horses in the world are sold at public auction, a fact not true 20 years ago, and even traditional owners and breeders have entered the lucrative commercial market.

The commercial market is a straightforward avenue for owners to buy a quality racehorse without maintaining broodmares. The September Keeneland yearling sale offers nearly 5,000 yearlings over a 10-day period. Numerous other sales are conducted throughout the year. Presale examination is an important part of veterinary practice at racetracks (see Chapter 102). The auction environment can be extremely high pressure for the veterinarian because the knock-down price may reach millions of dollars.

Owners can also buy horses in claiming races. In claiming races the prospective owner buys the horse for a predetermined price *before* the race, and the horses is considered sold once the starting gate opens, regardless of whether the horse wins or ever finishes the race. Purses and monies from purchase (claim) go to the previous owner. Potential buyers of claiming horses cannot examine the horse, but they can watch it walk into the saddling paddock. Buying a horse by claiming gives an owner a ready-made racehorse, while owners and breeders have to wait at least 3 years before racing a homebred horse, and yearling buyers have to wait at least a year. Unfortunately, the risks in claiming are considerable, because the claiming game has no honor and the new horse may already be at the end of its career.

Racetrack operators have changed to meet demands of the racing fans. The state, racetrack operator, and horsemen, through purse distributions, obtain income from a percentage of the moneys wagered, called handle. Revenue from simulcasting races across the United States and internationally is now an important source of handle, and Internet and at-home wagering are already realities. On the track attendance has dwindled and contributes only a fraction of what it did 20 years ago, when a fan had to come to the track to place a wager. The importance of simulcasting to the future of horse racing has led to consolidation of racetrack ownership.

Because track operators emphasize handle, races are run in a manner to encourage betting. Races must be competitive and the more horses the better. An anathema to the non-gambling race fan and many horsemen is that gamblers bet more money on a large field of poor-quality horses than on a small field of high-quality horses. In the United States, field size rarely exceeds 12 horses, because dirt racetracks are relatively narrow and have tight turns, and turf courses, usually inside of the main dirt track, are even narrower with tighter turns.

Races are made to be competitive by restricting which horses are eligible to run in a particular race. Most important are age, sex, distance, and surface (turf or dirt). Sex and age restrictions limit which horses are allowed to compete in a race. Age is a major factor, and 2-year-olds are never raced against older horses, because they would not be competitive. Distaff races are those restricted to fillies and mares, and although the distaff side is not excluded from open races, such races are seldom competitive, except early in the 2-year-old year.

Distance and surface are conditions selected to suit the horse. Another set of conditions, handicaps and allowances, can be confusing but work well. Handicap and allowance races are set up to even the race by varying the weight carried by the horse. Weight variation is a subjective value, determined by racing officials for handicap races. In allowance races, weight variation is determined by a set of published criteria. For example, 3-year-olds carry less weight than older horses, fillies less than colts, and non-winners may get additional weight off.

Most races in the United States are claiming races. In claiming races, horses of similar ability are pitted together. Because of the risk of loosing a horse by claim, owners and trainers are discouraged from running more valuable horses to steal a purse. The claiming aspect of American racing is popular, but it has several negative effects on racing.

Stakes races are for the best horses. Stakes races require an entry fee and some require an additional starting fee. Stakes races are raced according to sex, age, distance, and surface. Stakes races are graded, listed or restricted. Restricted stakes races, popular in recent years, restrict eligibility to the state of foaling or to conditions similar to allowance races. Stakes races are run even if only one horse is entered, but this is rare and is called a walkover. Stakes can be handicaps, allowances, or weight for age races. In weight for age races all horses carry the same weight, except for the well-established allowances for age and sex. The Kentucky Derby is a weight for age race for 3-year-olds. All horses are assigned the same weight (126 lb), but a filly receives a weight allowance and carries 123 lb. The best stake races are graded by a national committee and classified as Grade 1, Grade 2, and Grade 3; Grade 1 races are the top races. Graded stakes races are similar to the group or pattern race classification in Europe. The Kentucky Derby and Breeder's Cup Classic are examples of Grade 1 races.

Non-stakes races fall into several categories. Maiden races are for horses that have never won a race and can be allowance or claiming races. Races can be restricted to horses that have not won a certain number of lifetime starts. Other specific conditions for races can include eligibility for horses that have not won a race in a certain time period or over a certain distance. Claiming races also are restricted by age, sex, distance, and surface. Although races are devised to maximize competitiveness, the uncertain offering of races for non-stake horses greatly complicates training and veterinary care, because timing for future races can be difficult to judge.

Stakes races are predominantly for horses 2 and 3 years of age, whereas horses may continue to race in claiming races up to 10 to 12 years of age. Claiming horses may drop progressively in class and value, and the lowest level of TB racing in the United States is considerably below that of the United Kingdom. Much of the following discussion reflects our dealings with high-quality, younger horses. These situations are easier for the veterinarian to be in control and to dictate the diagnostic approach and therapeutic plan, working with the

trainer. With low-value claiming horses the trainer is more likely to dictate what is treated and with what. Frequently joints are treated with corticosteroids alone, rather than in combination with hyaluronan.

CONFORMATION

Many mild conformational defects appear to be well-tolerated by TB racehorses (M.W.C., P.J.M.). Mild toe-out and toe-in conformation causes few problems, but horses with moderate or severe toe-out conformation develop interference injuries. Toe-out conformation may predispose the horse to sub-solar bruising of the medial heel. The most severe conformational defect is calf-knee or back-at-the-knee conformation. This defect leads directly to carpal lameness. Short, straight pasterns increase concussion and long, sloping pasterns are undesirable because they may lead to soft tissue injuries or fractures of the proximal sesamoid bones (PSBs). Offset (bench) knee conformation leads to splint disease and injury of the antebrachiocarpal joint. Horses that are tied in behind the knee develop tendonitis. Sickle-hocked conformation leads directly to lameness of the distal tarsal region. Horses with straight hindlimbs develop lameness of the stifle region and are prone to upward fixation of the patella.

TRACK SURFACE AND LAMENESS

Track surface is a significant factor in the development of lameness and frequently is overlooked or neglected in lameness discussions (P.J.M., M.W.C.). Track surface dictates frequency and type of lameness. Surfaces that favor speed also predispose horses to catastrophic breakdowns. Quality of racing surface is much more important than whether the surface is dirt or turf. Hard racetracks with little cushion are the worst surfaces. One of us (M.W.C.) observed 38 horses with bone injury in a 4-week period when horses were training and racing on a hard racetrack. When sand was added to the track and the cushion increased from 6 to 11 cm, over the ensuing 4-week period only four horses developed bone injury. However, race times increased substantially. Horses need not train on hard surfaces to be able to race on them. Training on a forgiving surface and racing on a fast surface is safer than the opposite approach or working on a hard surface constantly. Uneven surfaces may predispose horses to injury.

Banking around turns can influence how horses negotiate turns and lameness distribution and expression. The hindlimbs of horses negotiating flat turns appear to slip out from under them and, in some instance, horses nearly go down.

Track surface can make a difference on how horses with injuries are managed or rehabilitated. Horses rehabilitating after fractures or other bone injuries should not train on hard tracks. Likewise, horses coming back after soft tissue injuries or racing with minor infirmities should not be trained on deep or muddy tracks.

More information is needed about how surfaces should be managed. For example, how should the track be harrowed and how frequently? What is the ideal moisture content of the cushion?

Many European horses that previously trained on grass now race in North America. Most race and continue to train on grass, but some make the switch to training and racing on dirt successfully (see Chapter 108). Our impression is that European horses appear to have fewer forelimb and more hindlimb lameness problems than North American TBs. European horses often train on long straightaways and this, combined with a forgiving surface (grass), is a situation different from training on dirt in North America. Horses bred to race on the turf appear to have an inherent conformational difference that allows superior performance on grass.

TEN MOST COMMON LAMENESS CONDITIONS

1. Lameness of the foot
2. Lameness of the metacarpophalangeal/metatarsophalangeal (fetlock) joints
3. Lameness of the carpus
4. Suspensory desmitis
5. Dorsal third metacarpal bone disease
6. Superficial digital flexor tendonitis
7. Tibial stress fractures
8. Distal hock joint pain
9. Myositis
10. Other stress fractures

LAMENESS EXAMINATION

A racetrack clinician has distinct advantages over other practitioners. A racetrack veterinarian is involved intimately in the day-to-day operation of a racing stable and becomes familiar with the normal gait, disposition, and general health care factors of each horse, such as appetite, coat condition, weight, training and racing status, and performance. Numerous examinations can be performed easily if needed. Horses can be examined before and after training to see if lameness worsens or improves.

History

The most important piece of information is the training history. What did the horse do today, yesterday, and the day before? How far is the horse from racing? When was the last work or race? Did any problems occur? Did the horse want to train? Did the horse lug in or out? Did the horse cool out normally? When did the trainer or exercise rider notice a problem? When was the last time the horse was shod?

Most fractures occur in horses with a history of a recent hard workout or race. Sometimes lameness may not become apparent until the horse returns to the track several days later, but the injury occurred in the previous workout or race. Humeral stress fractures are the exception, because they usually occur in horses returning to work after not working for 45 to 90 days. Horses that lug in (drift toward the inside rail) or lug out (drift toward the outside rail) usually are moving away from the source of pain. Many lameness conditions are insidious in onset. Subtle signs, such as a horse that is unusually nervous and prefers to break into the gallop instead of jogging, are easy to overlook. Lameness the day after a shoeing change may implicate a close nail or a drastic change in hoof angle.

The age of the horse is important. Dorsal third metacarpal bone disease and other stress-related bone injuries are unusual in older horses if they do not have problems at 2 or 3 years of age. Older claiming horses are much more likely to have chronic osteoarthritis, osteochondral fragmentation, or tendon injuries.

Palpation

The physical examination should be routine and complete. Horses are first examined in the stall, where they are comfortable and are more willing to tolerate manipulation. One of us (P.J.M.) feels strongly enough about thorough examination that if trainers want to start with a visual examination while horses are tacked up and proceed directly to injection, they should find another veterinarian. Many lameness conditions are acute and signs of inflammation are clearly useful. Horses

with chronic lameness can be challenging, but long-term association with a horse is of great benefit. Regardless, the examination is the same. With the horse standing squarely, all limbs are palpated for heat, pain, and swelling. During individual flexion of the front fetlock joints and carpi, pain response is important. Pain during carpal flexion almost always indicates a problem in that region. The clinician should remember that during lower limb flexion tests, joints other than the fetlock are being stressed. If concerned, we flex joints independently if possible. All aspects of the PSBs should be examined with the limb in flexion. Suspensory ligament (SL) branches, body, and origin and flexor tendons are carefully palpated along the entire length. The dorsal aspect of the third metacarpal bone (McIII) is palpated by placing the palm of the hand around the tendons and applying firm finger pressure. The forelimb is then brought forward while elevated and the dorsal cortex of McIII is palpated with firm thumb pressure. Careful palpation of the dorsal and palmar aspects of McIII is important in detecting fracture of this bone (M.W.C.).With the palm of the hand on the dorsal aspect of McIII the fingers can be used to palpate the proximal SL and each splint bone. Care must be taken when examining the dorsal and palmar metacarpal regions, because the clinician may inadvertently cause pain on the dorsal cortex by examining the palmar aspect of the metacarpal region and vice versa (P.J.M.). Proximal palmar metacarpal pain is important to detect, but false positives can occur (M.W.C.). Any suspicious response should be evaluated further with ultrasonography (P.J.M.). The carpus is flexed and the dorsal surfaces of the antebrachiocarpal and middle carpal joints are palpated with thumb pressure. An effort should be made to stretch the joint capsule around the borders of each carpal bone, because stretching often elicits pain if a lesion present. To evaluate the elbow and shoulder regions, the limb is pulled backward and forward. With the limb extended, a jerking, upward motion sometimes causes pain in horses with humeral stress fractures. The shoulder joint and intertubercular (bicipital) bursa should be palpated with firm digital pressure.

The hands should be run lightly over the back to assess for sore or tense muscles. The lumbar and sacroiliac regions, gluteal muscles, and greater trochanter of the femur should be palpated from each side. Although appearing hazardous, the gluteal muscles and greater trochanter also should be palpated from behind. If necessary a forelimb can be elevated. Firm pressure should be used to detect gluteal myositis or trochanteric bursitis. Standing behind the horse is the best way to compare effusion of the femoropatellar joints. Each hindlimb should be examined in the standing and flexed positions. The Churchill test should be performed bilaterally (see Chapter 6). A positive response suggests distal hock joint pain. Negative findings on palpation and manipulation do not eliminate a joint from consideration, but positive findings often point to the source of lameness.

Movement

It is mandatory to observe the horse during the first few steps out of the stall. Then the horse is usually walked down the shed row and back. Occasionally the horse is trotted immediately. It is important to trot the horse at a comfortable speed for the *horse,* not the handler, and the horse must be trotted far enough to reach an even speed. The head must be free, without allowing the horse to throw the head. Surface is important and the harder, the better; soft surfaces may hide lameness. The horse is trotted in a straight line and while circling. Trotting the horse in a circle is the best way to differentiate diagonal or ipsilateral lameness (i.e., left forelimb from right or left hindlimb) and exacerbates many lameness conditions. Horses with tibial stress fractures are much worse while circling. Horses with third carpal bone pain and medial foot lameness are worse with the affected limb on the outside. Horses with lameness of the fetlock joint are usually worse with the affected limb innermost. Although characteristics of lameness while trotting are important, they are subjective.

At times, observing a horse under tack is useful. Often, horses with hindlimb lameness are best examined on the racetrack, because many horses do not use themselves behind. Only when horses are absolutely sound in hand is examination at speed warranted, and even then, with the exception of understanding the complaint of the rider, obtaining useful diagnostic information is rare. However, one of us (P.J.M.) likes to examine horses with obscure lameness on the track, under tack at the trot.

The usefulness of flexion or other manipulative tests is debatable. Lameness may be exacerbated, but disagreement exists over what a positive response means. For instance, when performing a fetlock flexion, a positive response is common even in horses with lameness unrelated to the fetlock joint (R.M.A.). One of us (P.J.M.) finds fetlock and lower limb flexion tests worthwhile, but not carpal flexion, and finds hindlimb flexion tests non-specific. One of us (M.W.C.) finds forelimb flexion tests useful but finds all but the lower limb flexion test in the hindlimbs questionable. Each clinician needs to develop a protocol and be consistent.

Diagnostic Analgesia

Diagnostic analgesia is important to localize sources of pain and sequential, distal to proximal, intra-articular, and perineural blocks should be performed. One of us (P.J.M.) feels strongly that shortcuts, such as skipping immediately to a low palmar/plantar block rather than performing a palmar/plantar digital block first, lead only to misdiagnoses. Another (M.W.C.) feels confident in clinical examination and blocks only when unsure of his diagnosis but admits that when a trainer or owner wants a definitive answer, this approach is unacceptable. However, to diagnose proximal plantar metatarsal pain definitively, a block must be performed. Blocking horses with long winter coats can be difficult. For distal third metacarpal or metatarsal (MtIII) bone disease one of us (M.W.C.) uses specific palmar/plantar metacarpal/metatarsal analgesia. A novel approach is taken to desensitize the distal 50% to 75% of McIII. Five milliliters of local anesthetic solution is injected into the nutrient foramen. This block is not specific for bony disease, because the nearby accessory ligament of the deep digital flexor tendon can be affected; however, injury of this ligament is rare.

Whether to clip for intra-articular injections or analgesia is personal preference, but two of us do not clip (M.W.C. and P.J.M.). Although medicating and performing intra-articular analgesia simultaneously is acceptable, one of us (M.W.C.) prefers to wait at least 1 day after blocking to medicate.

Horses may be difficult to handle during analgesic procedures or during intra-articular injections, but usually a lip chain or twitch provides adequate restraint. Gentle, confident, sure hands with painless technique are best (P.J.M.). If sedation is necessary, a combination of 5 mg butorphanol and 200 mg xylazine or 0.15 mg (0.125 ml) detomidine is useful (M.W.C.).

IMAGING CONSIDERATIONS

Routine radiographic views often are supplemented with views looking specifically at problem areas of the TB racehorse. For instance, for the metacarpophalangeal and metatarsophalangeal joints, flexed dorsopalmar or dorsoplantar (DPa, DPl), the dorsal 35° distal-palmaro(plantaro)proximal oblique, a standard horizontal DPa or DPl, lateromedial (LM), flexed LM, and the dorsolateral-palmaro(plantaro)medial oblique and dorsomedial-palmaro(plantaro)lateral oblique views should be considered

routine. For the carpus the four standard views and a skyline each of the proximal and distal rows of carpal bones are routine.

Scintigraphic examination is important, because TB racehorses frequently develop cortical and subchondral stress-related bone injuries. Horses with obscure or undiagnosed lameness and those suspected of having stress-related bone injuries are candidates for scintigraphic examination. Because complete fracture often results from pre-existing stress-related bone injuries in cortical and cancellous bone, horses with signs consistent with pelvic, tibial, humeral, and McIII or MtIII stress fractures should undergo scintigraphic examination before continuing in work. However, interpretation is not always straightforward. False-negative results have been found in horses with pre-existing tibial stress fractures (P.J.M.) and false-positive results in those with modeling reflecting changes in work intensity. Therefore experienced interpretation is essential, and follow-up examinations are required if findings do not concur with clinical observations.

For ultrasonographic examination horses are routinely clipped. Ultrasonographic examination is valuable in diagnosing and staging tendonitis, suspensory desmitis, carpal tenosynovitis, lateral branch superficial digital flexor tendonitis, and other swellings of the pastern, swellings of the medial stifle region, and distal sesamoidean desmitis.

SHOEING

Trimming and shoeing of the TB racehorse are extremely important. In many practices, veterinarians are not involved in trimming and shoeing, and decisions often are made exclusively by trainers and farriers. Because recent evidence has linked catastrophic injury to toe grabs on shoes, now a general trend is to see horses shod with low grabs, or none at all. Toe grabs are thought by many to cause forelimb lameness. The rationale for using grabs is questionable, because the forelimbs are propelled by the hindlimbs and traction on the front feet makes little sense. Toes grabs may shorten the cranial phase of the stride, prevent forward sliding of the forelimbs, and cause lameness. One of us (M.W.C.) has observed many horses that are stiff and short up front, and by simply removing the grabs and applying Queens plates (aluminum shoes with a low toe grab; Victory Racing Plate Company, Baltimore, MD), lameness abates in 2 to 3 days. Lameness associated with toe grabs appears most common on dirt tracks with hard bases and cushions of less than 7.5 cm. For racing on dirt the Queens plates or variations are popular, and for turf the Queens plates and Queens XT shoes are required. Turndown shoes have received negative press, but they may be advantageous in horses with low heels. Slight turndowns without toe grabs on hind feet may actually benefit horses with hock and stifle pain. Many lameness problems may be created or exacerbated by poor shoeing, including proximal suspensory desmitis (PSD), tendonitis, and fractures of the PSBs. One of the major problems is shoeing horses with small shoes. Overzealous use of acrylic can cause severe foot lameness. Horses with thin walls are predisposed to nail bind and those with sheared heels suffer repeated heel bruising.

INABILITY TO MAKE A DIAGNOSIS

In many horses, despite taking a methodical and time-consuming approach to diagnostic analgesia, the source of pain cannot be identified (M.W.C.). These horses are referred for additional clinical and scintigraphic examinations (M.W.C. and P.J.M.). Horses with stress-related bone injuries may show few clinical signs, and scintigraphy is the imaging modality of choice. Lameness in young horses in training may be more difficult to diagnose than that in horses that are racing (M.W.C.). Many horses with undiagnosed lameness are confined to at least 6 weeks of walking.

SPECIFIC LAMENESS CONDITIONS

Lameness of the Foot

The most common cause of lameness in the TB racehorse is foot pain. Horses may be observed in the stall to pile bedding under the feet, presumably to cushion the feet or to change angles. Lameness is often worse with the affected limb on the inside of a circle. Heat at the coronary band and increase in digital pulse are frequent signs. TB racehorses have little hoof. Many are shod at least monthly, but most are shod more frequently, with thin, lightweight shoes. When shoes are changed frequently, little natural hoof is available with which to work if adjustments are necessary. The tendency is for the farriers to remove too much foot, particularly toward the heel, resulting in low heels. Thin feet have prompted the use of acrylics to augment the hoof wall, but overzealous use may also result in foot soreness itself (M.W.C.). Soreness and bruising are common, particularly on the medial quarter, just forward or at the bar. The center of load distribution for a TB racehorse is medial and palmar to the anatomic center of the foot, close to the medial bar.

Most racehorses react positively to the application of hoof testers because only a thin covering of horn protects the sensitive structures of the foot. Degree of sensitivity to hoof testers is important and is learned through experience. Hoof tester examination is best done when an assistant holds the foot (R.M.A.). Hoof testers can be placed carefully on front and hind feet, and slight differences in positioning make a significant difference. Left and right feet should be compared at least twice before reaching conclusions. The diagnosis of palmar heel pain can be confirmed readily by selective medial or lateral palmar digital analgesia. Horses with a naturally wide gait, or with carpal pain, bruise the medial quarters and can have concomitant foot pain. Acquired bruises of this nature are difficult to treat in horses with wide gaits without lameness and without managing primary lameness in those with carpal pain. In most horses with foot lameness, diagnosis is made with clinical findings and diagnostic analgesia. If abnormalities of the sole or hoof wall cannot be found, radiographs are occasionally necessary (P.J.M.). In most horses without fracture, lameness resolves in 2 to 3 weeks with specific therapy and the administration of non-steroidal anti-inflammatory drugs (NSAIDs).

Bruised Heels and Quarters

Lameness caused by bruised heels or quarters is often mild, and horses usually are kept in training. More often than not, little heat is associated with a bruised quarter. Mild dilation of one or both digital arteries and hoof tester sensitivity are the most common signs. The best treatment is rest, however. The foot should be examined for any conformational or shoeing faults; sheared heels or unbalanced feet are common causes of bruised quarters. Horses with toed-out conformation often bruise the medial heel (M.W.C.). Corrective shoeing is really the only treatment option. Steel shoes distribute force along the hoof wall evenly and can be applied to horses in training but not racing. Bonded shoes can be applied to horses that are racing. If the foot is warm, the horse should stand in iced water, but if the foot is normal temperature, the horse should stand in foot tub of hot water with Epsom salts. A poultice of warm, cooked flax seed or commercially available products such as Animalintex (3M Animal Care Products, St. Paul, MN) should be applied. A common practice of applying hoof-hardening agents such as iodine and turpentine should be avoided, because hard hooves bruise easily.

Abscesses

Foot abscesses are a common problem, particularly during the wet seasons. A foot abscess is the most common eventual diagnosis when a horse is found three-legged in the stall in the morning without a recent history of hard work or racing. TBs are prone to foot abscesses because of the manner and frequency of shoeing. Foot abscesses occur most commonly in the medial quarter, where bruising is also most common. Sole abscesses not involving the quarter are uncommon, take more time to resolve, and cause different clinical signs than do abscessed quarters. Although abscesses in the heel or quarter usually are located easily and precisely with hoof testers, a sole abscess can produce a large area of soreness. Identifying the specific area of pain is difficult.

Horses with foot abscesses are best managed with hot water baths with Epsom salts and poulticing. The longer the horse stands in hot water with Epsom salts, the better. Ideally, the shoe should be removed, and the abscess should be allowed to open naturally. Usually more damage is done to the hoof wall by attempting to open the abscess manually than by allowing it to open naturally. Probing the white line to identify an abscessed area can be worthwhile. Sole abscesses are more likely to require manual opening and debridement than are abscessed quarters. The integrity of the hoof wall needs to be evaluated and preserved, if possible, but wet, infected areas need to be exposed, opened, and dried. Foot abscesses or infections also can originate from grabbed quarters, which occur frequently in horses that stumble out of the starting gate. When grabbed, the hoof wall of the quarter may separate at the heel bulb or quarter, well down the foot. The opened area becomes contaminated with track dirt, and infection can occur several days to weeks after the original injury. Fortunately a pathway for drainage is already present.

Quarter Cracks

Quarter cracks are common for the same reasons as bruising and abscessation. The quarters and heels take continual pounding, particularly in a TB racehorse with poor hoof wall support. Quarter cracks may be caused by poor hoof balance but most commonly result from innate hoof weakness. Farrier-incurred hoof imbalance can contribute to the development of quarter cracks, but most commonly a single horse in a stable has numerous cracks. Horses with an initial crack are prone to develop subsequent cracks in the same hoof or other hooves. A horse ran successfully with seven quarter crack patches at one time (R.M.A.). If identified early, quarter cracks can be patched readily with acrylic or epoxy. The goal is to stabilize the hoof wall and eliminate uneven movement. Mild cracks should be patched if any movement occurs at all. As little patching material as possible should be used to stabilize the hoof wall. If a quarter crack is infected, a drainage path needs to be established. Some epoxies are much harder than the natural hoof wall and trapped infection eventually drains through natural tissues rather than the patch.

Osteoarthritis of the Distal Interphalangeal Joint

Most lameness conditions of the foot seen in other sport horses also occur in racing TBs. Considerable controversy exists over the frequency of osteoarthritis of the distal interphalangeal joint. Some veterinarians diagnose the problem regularly, whereas others never recognize it. In our experience, osteoarthritis of the distal interphalangeal joint is uncommon. Effusion is the most common clinical sign. Osteophytes may be seen on the extensor process of the distal phalanx. Frequently, horses with bilateral osteoarthritis of the distal interphalangeal joint have poor performance rather than overt lameness, and lameness is worse at the end of a race (M.W.C.).

Navicular Syndrome

Navicular disease and navicular bursitis are unusual to rare conditions in the TB racehorse (P.J.M.). Lameness can be localized using palmar digital analgesia, but most often horses have bruises, cracks, and abscesses, and those with navicular syndrome are unusual. One of us (M.W.C.) diagnoses navicular bursitis commonly. Successful management of this type of palmar heel pain has been achieved by injecting the digital cushion with orgotein (Palosein) and Sarapin and methylprednisolone acetate, applying a bar shoe, increasing heel angle, and giving NSAIDs. Intra-articular injection of the distal interphalangeal joint with hyaluronan and corticosteroids affects numerous structures in the foot and improves horses with navicular syndrome or osteoarthritis of the distal interphalangeal joint.

Distal Phalanx Fractures

Fractures of the distal phalanx occur occasionally. Most fractures are non-articular palmar process (wing) fracture, and horses do well with the application of a bar shoe. Articular wing fractures are more serious. Extensor process fragments are rare and can be removed arthroscopically.

Palmar digital neurectomy is permitted in most racing jurisdictions, but it must be reported to the race office. Indications for neurectomy are few, but occasionally a horse with a distal phalanx fracture and chronic pain is a candidate. Occasionally, palmar digital neurectomy is performed in horses with recurrent quarter cracks to eliminate pain, but the procedure does nothing to solve the original problem. Surgical neurectomy should be performed because percutaneous techniques are often ineffective.

Lameness of the Fetlock Joint

The metacarpophalangeal (fetlock) joint, carpus, and tarsus are the most important joints associated with lameness in racing TBs, especially the metacarpophalangeal joint. Clinical findings of fetlock joint lameness usually include effusion, heat, a positive response to flexion, and elimination of lameness using intra-articular analgesia. However, in some horses with subchondral bone pain, perineural blocks are most effective. Horses also can have substantial lameness associated with the fetlock joint without localizing signs. The most common lameness conditions of the fetlock can be divided into four categories: synovitis, distal palmar McIII or MtIII disease, fractures, and osteoarthritis.

Synovitis

Fetlock joint pain associated with effusion, pain on joint manipulation, a positive response to a distal limb flexion test, and moderate lameness is common, but horses generally respond well to intra-articular treatment with hyaluronan and corticosteroids, combined with systemic treatment with polysulfated glycosaminoglycans (PSGAGs). Intravenous hyaluronan may be useful in these horses. However, recurrence of clinical signs indicates that the horse should be removed from training, because continued training may result in chronic problems (M.W.C.). Firing and blistering may be useful.

Distal Third Metacarpal/Metatarsal Bone Disease

Stress and non-adaptive remodeling, a form of stress-related bone injury, of the distal aspects of McIII and MtIII is a common cause of lameness. During training and racing, these areas undergoe considerable modeling and remodeling, change shape, and are at risk to develop fracture and cartilage damage. Scintigraphic examination reveals focal areas of increased radiopharmaceutical uptake (IRU) and is the imaging modality of choice for diagnosis, because radiographs are often negative. Ultimately, damage to the distal McIII and MtIII leads to osteoarthritis and fracture. Early in the course of the disease pain originates from subchondral bone, without effusion or heat. However, intra-articular analgesia is usually effective in eliminating pain. In some horses all fetlock joints are affected simultaneously, and the horse appears sore all over. Once pain is abolished in one limb, lameness appears in the contralateral limb. Distal McIII/MtIII disease is so prevalent that some veterinarians first perform intra-articular analgesia of the fetlock

joint rather than beginning with palmar/plantar digital analgesia. As the disease progresses, radiographic changes can include flattening of distal McIII/MtIII, sclerosis and radiolucency, and when severe, pitting of McIII/MtIII. A form of severe flattening of the distal McIII has been recognized that may result in severe osteoarthritis by the end of the 3-year-old year (M.W.C.). Negative radiographic findings do not eliminate this disease as a source of pain, and scintigraphy is more sensitive for its detection. Areas of IRU can represent subchondral bone damage from non-adaptive remodeling or fracture, and a combination of imaging modalities is necessary to differentiate these conditions.

Osteochondral Fragmentation

Osteochondral fragments (chip fractures) of the proximal, dorsal aspect of the proximal phalanx are common in TB racehorses. Fragments are more often medial than lateral, but they can occur biaxially and bilaterally. Often disease of the fetlock joint occurs before fragments are recognized. Subtle radiographic changes in the silhouette of the dorsal rim of the proximal phalanx are important and indicate this area is experiencing stress. The distal dorsal aspect of McIII at the joint capsule attachment can be injured at the same time that chip fractures are seen.

Once fractures are recognized, arthroscopic surgery should be performed. After surgery the horse can be back in training as soon as 6 weeks if cartilage damage is minimal. Horses with small fragments can be managed conservatively using local ice therapy, NSAIDs, and a reduction in exercise intensity. Inflammation (effusion) subsides within 7 to 10 days and training resumes. Small osteochondral fragments appear to demineralize and no longer irritate the joint. If inflammation and lameness recur, the horse should be taken out of training, and surgery is recommended. Intra-articular injection of corticosteroids and hyaluronan is one option and is effective at reducing inflammation, but if training and racing continue, deep scoring of the articular cartilage of distal McIII occurs and leads to osteoarthritis. If the goal is long-term racing, surgery should always be recommended.

Osteoarthritis

Osteoarthritis is a common problem and usually is associated with fractures of the proximal phalanx or palmar/plantar McIII/MtIII disease, but it can develop as a primary condition. Arthroscopic examination often reveals considerable articular cartilage scoring and thinning, or complete erosions through the articular cartilage, without any evidence of an associated fracture. Because osteoarthritis is chronic and cartilage cannot regenerate, the disease must be identified early so preventative measures can be taken. Judicious use of NSAIDs and intramuscular PSGAGs is the best long-term treatment for osteoarthritis of the fetlock joint. If present, fractures need to be repaired or fragments removed. Young horses should be taken out of training and allowed to mature. Immature horses are particularly prone to overextension injury of the fetlock joint. Some horses may require PSGAGs throughout the racing career. Oral supplements may be beneficial, but intramuscular administration is best. Repeated use of intra-articular corticosteroids exacerbates chronic osteoarthritis, but whether this is a direct result of the medication or simply allows a horse to continue in training and racing is unknown. Unfortunately, heat, effusion, positive response to flexion, and lameness resulting from osteoarthritis need to be treated, because horses are expected to perform. Intra-articular injections are unavoidable, and a combination of hyaluronan and corticosteroids is preferred (R.M.A.). Horses with recurrent synovitis may need additional injections in combination with 2 to 3 weeks of rest (M.W.C.). In spite of information to the contrary, methylprednisolone acetate is the drug of choice, because if used prudently at well spaced intervals, the drug is preferable to repeated injections of shorter-acting corticosteroids (R.M.A.). Intra-articular injections of PSGAGs every 2 weeks are beneficial in horses with chronic osteoarthritis (P.J.M.).

Other Conditions

Distal McIII/MtIII condylar fractures, PSB fractures, sesamoiditis, and proximal phalanx fractures are discussed in Chapters 37 and 47. Distal McIII/MtIII condylar fractures often result from non-adaptive remodeling and may be more common in certain racetrack practices than others. One of us (P.J.M.) includes these fractures as a top 10 lameness condition. Prognosis is much better in horses with incomplete fractures, with or without surgery. One author (P.J.M.) recommends surgery in horses with fractures longer than 4 to 5 cm above the joint surface.

Lameness of the Carpus

The carpus is similar to the fetlock joint in several ways. The third carpal bone undergoes considerable modeling and remodeling with associated subchondral bone pain similar to distal palmar/plantar McIII/MtIII. Fractures and osteoarthritis are often the end-stage result of this process of stress-related bone injury. The response of the third carpal bone to stress-related bone injury is similar to that of cortical bone.

Non-Adaptive Remodeling of the Third Carpal and Radial Carpal Bones

The third carpal bone and other carpal bones such as the radial carpal bone become sclerotic (model) to withstand the stress of training and racing. The change in the third carpal bone is seen on the skyline radiograph as sclerosis of the radial facet. Sclerotic bone becomes painful if the condition of stress remodeling becomes pathological, called *non-adaptive remodeling.* If training and racing continue, bone loss or lysis, or fracture occur. Third carpal bone subchondral bone pain is common, particularly in young horses, but because pain involves bone, no treatment other than reducing training and racing intensity is available.

Diagnosis of third carpal bone subchondral bone pain can be challenging, because many young horses do not develop effusion. Heat may occur over the dorsal aspect of the carpus, and horses move wide and tend to abduct the limb during advancement (M.W.C.). Response to flexion varies, but in horses with subchondral bone pain, response is often negative. Diagnostic analgesia is essential for diagnosis. Careful interpretation of results of diagnostic analgesia is necessary because inadvertently blocking the proximal palmar metacarpal region with middle carpal joint analgesia and vice versa is easy (M.W.C.). The middle carpal and antebrachiocarpal joints always should be blocked separately. Radiographic changes of sclerosis and later radiolucency can be seen with good-quality skyline views, but they are missed easily with poorly positioned and exposed views. To assess radiographic exposure, the veterinarian should evaluate the second and fourth carpal bones. These bones rarely have alterations in trabecular pattern, and both bones should be clearly visible on the skyline view. Scintigraphy is an excellent tool with which to diagnose stress-related bone injuries of the third carpal bone and other bones, and although not needed in many horses, scintigraphy assists in early diagnosis and helps convince skeptical trainers and owners. Sclerosis of the third carpal bone changes the mechanical properties of bone, and sclerotic bone is brittle and at risk of fracturing. Few osteochondral fragments, small (chip fractures) or large (slab fractures), of the third carpal bone occur in normal bone. At the time of arthroscopic surgery to repair a third carpal bone frontal slab fracture, a wedge-shaped piece of sclerotic and often necrotic bone often can be found between the fracture fragment and parent third carpal bone.

Once sclerosis of the third carpal bone develops, treatment is difficult. Rest may not help remodel dense bone, so prevention is important. The training programs of young TBs with

evidence of third carpal bone sclerosis should be modified, but trainers are often unwilling. Although little evidence supports using isoxsuprine or aspirin to improve blood flow, some veterinarians prescribe these medications.

Carpal Fractures

Osteochondral fragmentation of the carpus is a common problem. Clinical signs are unique for each fracture type, and radiographic examination is most important. Horses with distal radial carpal bone fractures exhibit pain on direct palpation of the fracture site and often have effusion of the middle carpal joint. Horses with third carpal bone pain and fractures are unwilling to rock on the limb, evaluated by picking up the contralateral limb, and forcing the horse to rock on the affected limb by putting side-to-side pressure against the horse's body. Horses with distal, lateral radius fractures generally have pain on direct palpation. Horses with proximal intermediate carpal and radial carpal bone fractures may be surprisingly lame, without obvious palpable abnormalities.

A rare fracture, but one to keep in mind, is a sagittal fracture of the proximal aspect of the intermediate carpal bone (M.W.C.). Horses train and race but come back to the barn obviously lame (grade 3 of 5). When walking and jogging, they exhibit a stiff-legged gait and significant abduction during advancement. Horses respond positively to carpal flexion, and lameness is abolished using antebrachiocarpal analgesia. Intra-articular treatment does not improve lameness. The skyline view of the proximal row of carpal bones is mandatory for diagnosis, because fractures will be missed on other views. Early arthroscopic surgery is recommended, because osteoarthritis can develop if horses are continued in work with a fracture.

Arthroscopic surgery has improved the management of carpal joint lameness greatly. Removal of small osteochondral fragments and repair of large fragments substantially reduces the severity of osteoarthritis. PSGAGs can be useful in limiting the development of osteoarthritis, particularly if pain is originating from the third carpal bone. After surgery in horses with carpal osteochondral fragmentation, one of us (M.W.C.) recommends hyaluronan injection 2 to 3 weeks later. Horses with fractures of the middle carpal joint need additional rest compared with horses with antebrachiocarpal joint fractures (M.W.C.). One of us (P.J.M.) prefers to manage horses with incomplete or non-displaced third carpal bone slab fractures conservatively by giving 60 days of rest and re-evaluating the horse. The prognosis for TB racehorses with displaced third carpal bone slab fractures even with surgery is guarded to poor. Horses with fractures extending into the weight-bearing surface of carpal bones more than 0.5 cm have a poor prognosis. Horses with recurrent and chronic osteoarthritis appear to benefit from topical blisters and 3 or more months of rest.

Osteoarthritis

In young horses with early signs of osteoarthritis, rest is recommended. In horses with chronic osteoarthritis but that are able to race, intra-articular injection of a combination of hyaluronan and PSGAG produces better results than does injection of either product alone (M.W.C.). In horses with mild or moderate lameness, intra-articular injection of hyaluronan and triamcinolone acetonide 5 to 7 days before racing decreases clinical signs for 6 to 8 weeks. Most horses exhibiting carpal lameness appear to benefit from the intramuscular administration of PSGAG once every 4 days for seven to eight treatments, and intravenous injection of hyaluronan appears beneficial before racing.

Rest should be recommended for young horses with early osteoarthritis even though recurrence of signs is common. NSAIDs are useful in managing pain in horses with osteoarthritis of the carpus.

Intercarpal Ligament Damage

Because the complex carpal joint is highly dynamic and moveable, fractures and soft tissue damage can lead to osteoarthritis. Although fractures are more common, recently injury of the medial palmar intercarpal ligament has been recognized (see Chapter 39).

Suspensory Desmitis

Proximal Suspensory Desmitis

Suspensory desmitis is a most important cause of lameness in the TB racehorse and previously has been underestimated. Suspensory desmitis is much more common in the forelimb than in the hindlimb (P.J.M.). Hindlimb proximal suspensory desmitis (PSD) is difficult to diagnose because horses have a stiff, hopping-type gait similar to that in horses with tibial stress fractures, and heat and swelling are unusual (M.W.C.). Lameness in horses with forelimb PSD is similar to that in horses with carpal disease. Horses travel wide, with a shortened cranial phase of the stride, and are worse with the limb on the outside of a circle. Ultrasonographic examination has improved our understanding of many soft tissue injuries, but compared with superficial digital flexor tendonitis, which always has been well recognized, PSD is recently recognized. Conformational faults such as offset knees and upright pasterns and fetlock joints predispose horses to PSD. PSD is most commonly a 2-year-old lameness condition, perhaps caused by immaturity, and can be diagnosed by manual palpation in some horses, but that is the exception rather than the rule (M.W.C.). More often, horses with PSD require diagnostic analgesia, and one author (R.M.A.) uses the lateral palmar block, the site for which is just below the accessory carpal bone. Using the lateral palmar block it is unlikely to inadvertently cause analgesia of the carpometacarpal and middle carpal joints. In many horses lameness thought to originate from PSD after a positive response to high palmar analgesia, was later found to originate from the carpus (M.W.C.).

Considerable variation occurs in the ultrasonographic appearance of the normal and diseased proximal SL. Overt tears are easy to diagnose. However, hypoechoic areas can be identified in sound horses. Furthermore, bony pathological conditions of the proximal, palmar aspect of McIII can be seen in horses without obvious tearing of the SL. Avulsion fractures of McIII are diagnosed in horses that develop acute, severe lameness abolished by lateral palmar analgesia. Scintigraphy is the best way to diagnose bony injury of proximal McIII in horses without ultrasonographic evidence of PSD and negative radiographic findings. With avulsion fracture comes focal, intense IRU, but many horses have diffuse IRU of the proximal aspect of McIII. Whether these horses have chronic avulsion injury or whether this represents stress reaction or other injury of proximal McIII is unknown. Regardless, horses with bony injury of proximal, palmar McIII have a good prognosis if given a minimum of 120 days of rest. Horses with PSD often can be managed and allowed to continue racing. Local infiltration with corticosteroids or other products is of questionable benefit, and systemic therapy is more effective and rewarding (R.M.A.). Others disagree and recommend local injection with triamcinolone acetonide in horses with mild PSD (P.J.M.). The combination of Sarapin, methylprednisolone acetate, and in some horses Palosein has been successful, and 2-year-old horses may never take another lame step (M.W.C.). Systemic corticosteroids and NSAIDs reduce inflammation enough to relieve the lameness. Where regulations permit, phenylbutazone and triamcinolone acetonide (12 to 18 mg intramuscularly) can be administered 3 to 5 days before racing. A good alternative is two boluses of Naquasone (a combination of the diuretic trichlormethiazide [200 mg] and dexamethasone [5 mg]) 48 hours before racing, if permitted by the rules of racing. Triamcinolone lasts longer, but Naquasone contains a diuretic that is also useful for reducing swelling. An ice boot or an ice tub is beneficial.

Horses in which lameness is mild when trotted in hand or even under tack but refuse to give a full effort toward the end of a race should be given rest.

Corrective shoeing can make a difference in horses with PSD. In fact, PSD may be secondary to lameness of the foot (M.W.C.). The toe should be shortened, and the shoe should have only minimal toe grabs. Horses with PSD do not develop catastrophic disruption of the suspensory apparatus but return from the race sore.

Suspensory Branch Desmitis

Suspensory branch desmitis is a different situation and can easily lead to catastrophic breakdown if horses are managed improperly. Management of suspensory branch desmitis with infiltration of corticosteroids in a TB racehorse is a high-risk treatment, with no therapeutic benefit to the patient. Systemic anti-inflammatory therapy similar to the approach described for PSD is useful for horses with suspensory branch desmitis, but the horse should not be raced if the SL remains hot and painful. Suspensory branch desmitis is a dangerous injury, and current research is investigating the correlation between suspensory desmitis and condylar fractures in horses with catastrophic breakdown.

In horses with suspensory branch desmitis the splint bones should be evaluated carefully. Radiographs to check for fracture, flaring, or thickening of the second and fourth metacarpal bones should be obtained. A clear relationship exists between suspensory branch desmitis and pathological conditions of the splint bone. Splint ostectomy is recommended in horses with fractured, flared, or thickened splint bones, not just in those with fracture.

Mid-Body Desmitis

Horses with mid-body desmitis can be managed similarly to those with PSD, but if desmitis extends distally into the branches, the risk of catastrophic breakdown exists. Horses with suspensory desmitis may benefit from rest (45 to 120 days) and blistering (P.J.M.).

Bucked Shins: Dorsal Metacarpal Disease

Bucked shins or dorsal metacarpal disease (periostitis) is common but readily manageable. This condition is simply the mismatching of exercise with modeling and remodeling of McIII necessary for the bone to withstand the rigors of training and racing. Bucked shins are most commonly seen in young horses early in training. The simplest and best treatment is to modify the training program to match the horse. With bucked shins, horses are usually only mildly lame after exercise, unilaterally or bilaterally (P.J.M.). Bucked shins are common in young horses with other primary lameness conditions, particularly of the hindlimbs (M.W.C.). Heat and pain over the dorsal cortex are commonly found, but care must be taken not to place pressure on the palmar metacarpal structures while palpating the dorsal cortex of McIII (P.J.M.). One of us (M.W.C.) feels that careful palpation of the palmar cortex is useful in identifying McIII disease. Clinical signs are usually sufficient for diagnosis, but low or high palmar nerve blocks, infiltration of the nutrient foramen of McIII, or local infiltration of local anesthetic solution occasionally is required (M.W.C.). Radiographs reveal a typical modeling response, but in some horses with fracture xeroradiographs or computed radiographs should be obtained, because fractures may be missed on plain films (P.J.M.). Scintigraphic examination can be useful, mostly in horses with dorsal cortical fracture (M.W.C.).

Great strides have been made in changing training techniques to manage TB racehorses with bucked shins. This requires close monitoring of the dorsal cortex of McIII. Overall, the incidence of authentic dorsal cortical fractures has decreased greatly. Although all trainers have horses with bucked shins, those that still have major problems train in the old-fashioned style.

Once inflammation is noticed, ice, NSAIDs, and topical cooling muds, such as Uptite poultice (Uptite Co., Lawrence, MA), are often all that is necessary if training intensity is reduced and modified. Once periosteum becomes hot, thickened, and sore over a large portion of the dorsal cortex of McIII, modifying training is usually too late. Training should be stopped, and when necessary thermocautery may still be the best treatment, once inflammation has subsided (R.M.A., P.J.M., and M.W.C.). When done properly thermocautery is humane and effective. An old-time racetrack practitioner was asked a few years ago if he still fires horses and if so why? His response was simple: "I can tell you in two words: It works." The real problem with thermocautery is perception. Most persons who adamantly are opposed to thermocautery generally have no experience with the procedure. Regardless, alternative treatments are available, but none in one author's opinion work as well as thermocautery (R.M.A.). All treatments require extended periods of rest or light training. Others prefer a relatively recent technique called periosteal scratching. A 16-gauge needle is used to create linear incisions of the periosteum using aseptic technique. This procedure, as with thermocautery, is combined with rest and is done after the inflammation has subsided (P.J.M.). Prognosis after periosteal scratching is estimated at 70% (M.W.C.).

Bucked shins can progress to dorsal cortical (stress) fracture if horses with periostitis are forced to continue training. Stress fractures occur most commonly on the dorsolateral aspect of McIII, but those located in the distal third to distal fourth of McIII are most problematic. Fractures in this location under the extensor tendons can be difficult to palpate, but they may be extensive.

Horses with bucked shins should not be lame at the jog 1 to 2 days after the last workout, but if lameness persists, a dorsal cortical fracture should be suspected. Radiographs should be obtained. Success in treating horses with dorsal cortical fracture relates directly to the duration of fracture. Horses with acute fractures are treated more easily than those with chronic fractures. In horses with acute fractures, rest, with or without thermocautery, is successful. Chronic dorsal cortical fractures are essentially non-unions and heal slowly or not at all and require surgery. Cortical drilling (forage, osteostixis) using numerous small-diameter holes drilled across the fracture line is effective and is one author's treatment of choice (R.M.A.). Rarely a cortical bone screw placed in lag fashion or as a positional screw is needed. Whether compression is needed remains unanswered. Implants must be removed before training begins. Extracorporeal shock wave therapy appears promising for the management of dorsal cortical fractures of McIII (M.W.R.).

Superficial Digital Flexor Tendonitis

Superficial digital flexor tendonitis usually is advanced before lameness is observed and diagnosis is made. Lameness is one of the last clinical signs observed. Superficial digital flexor tendonitis is usually a career-threatening or career-ending injury, and estimates indicate that superficial digital flexor tendonitis is the single most common injury ending a TB racehorse's career. However, many horses with superficial digital flexor tendonitis return to racing or, on occasion, race through the injury. One author (R.M.A.) feels strongly that local infiltration of corticosteroids and continued racing are contraindicated. Intralesional injection of corticosteroids can cause tendon necrosis and result in complete rupture of the superficial digital flexor tendon (SDFT). Some horses may be able to race after treatment with systemic corticosteroids, NSAIDs, and ice therapy. The SDFT will set up on occasion, but that is certainly an exception, and horses fall in value.

Recognizing superficial digital flexor tendonitis before overt fiber tearing occurs has distinct advantages. To recognize tendonitis early, horses must be monitored closely, and ultra-

sonographic examination must be performed at the earliest indication of inflammation. Heat and swelling are early clinical signs but may not indicate necessarily the tendon is involved, because peritendonous injury can cause similar signs. *The key is pain on palpation of the suspect tendon*. Pain, even without obvious heat or swelling, is an important clinical finding. Ultrasonographic examination must include cross-sectional area measurements, because subtle enlargement of the SDFT precedes fiber tearing. Once recognized, horses with early superficial digital flexor tendonitis should be given rest, or at least the training intensity should be reduced.

One author (R.M.A.) prefers to manage horses with core lesions of the SDFT with tendon splitting. The surgery can be done with the horse standing and is inexpensive, and aftercare is minimal. To perform tendon splitting, a core lesion must be present, and the earlier the procedure is done, the better. A double-edged tenotome is used. The tendon fibers are split longitudinally with five rows of about 15 percutaneous stab incisions each. After surgery horses are surprisingly sound. β-Aminoproprionitrile fumarate also has been used successfully but requires a complex injection and exercise regimen, making the entire treatment expensive and impractical. Horses with superficial tearing along the edge of the SDFT have a poor prognosis with any treatment and may be the only candidates for peritendonous injection of corticosteroids, provided the horse is removed from training and rested.

Horses with superficial digital flexor tendonitis should have as much time off as possible. However, if owners and trainers are unwilling to give 6 to 8 months of rest before the horse returns to training, any treatment is a waste of time. One of us (P.J.M.) has stopped recommending any form of surgery or injections and simply recommends walking rest for 4 to 16 weeks. A blister is applied at least twice, and tendon healing is monitored with ultrasonography. Even with this approach, recurrence rate is 60% to 80%. The trainer plays a major role in rehabilitating horses with superficial digital flexor tendonitis. Those that are successful give the horse the most time off and are patient getting the horse fit, before starting hard work.

Tibial Stress Fractures

Tibial stress fractures are the most common cause of acute, unilateral hindlimb lameness in the TB racehorse. Lameness from tibial stress fractures usually is recognized after a hard workout or after breaking from the gate. Lameness can be severe, and the horse may not bear weight on the limb. Tibial stress fractures are usually unilateral, but bilateral fractures can cause unusual hindlimb lameness (P.J.M.). Similar to humeral stress fractures, tibial stress fractures often occur after a horse has had 60 to 90 days of training, often after a period of rest for an unrelated cause. Two of us (R.M.A. and P.J.M.) do not find manual palpation of the tibia useful diagnostically, but one of us (M.W.C.) finds deep palpation and tibial percussion helpful. Scintigraphy is the diagnostic modality of choice. Less than 50% of horses diagnosed with tibial stress fractures using scintigraphy have radiographically apparent fractures, even if follow-up examination is performed after 2 weeks. If scintigraphy is unavailable, even small areas of periosteal, endosteal, or cortical change should be considered important, if clinical signs suggest tibial stress fracture. Tibial stress fractures are located most commonly on the caudolateral cortex, proximally or mid-shaft, but can occur caudolaterally and caudomedially distally. Bilateral tibial stress fractures are occasionally seen, even in horses with unilateral hindlimb lameness. One of us (P.J.M.) has examined a number of horses with clinical signs consistent with tibial stress fracture but in which scintigraphic examination was negative. Horses returned to the track and developed complete tibial fractures. Now this author (P.J.M.) recommends re-examination in 3 weeks. False-negative results are unusual in horses with stress-related bone injuries unless fractures are located medially and only lateral scintigraphic views are obtained (M.W.R.).

Horses with tibial stress fractures are given 90 to 120 days of rest. The prognosis is excellent. The tibia is well-muscled and has a good blood supply and callus formation does not interfere with nearby structures. Complications develop when horses have spiral fractures and severe lameness or when horses with unrecognized tibial stress fractures are trained or raced. Catastrophic failure can result. Tibial stress fractures can recur if horses are given inadequate time for healing, but this is otherwise unusual.

Some horses with only mild lameness at the time of initial diagnosis and in which lameness rapidly resolved can be kept in light work. In these horses scintigraphic examination reveals mild IRU and radiographs are negative. Horses are given a minimum of 60 days without hard work, but if lameness recurs, 90 to 120 days of additional rest is recommended.

Tibial stress fractures were misdiagnosed as stifle lameness for years at some racetracks and the same error is still made today. Tibial stress fracture is a relatively recent diagnosis, but even with the introduction of high-quality non-portable radiography equipment at referral centers, the diagnosis was difficult to make because radiographs are often negative and clinicians have to isolate pain, a difficult task in many TB racehorses. Scintigraphy has taught us that tibial stress fracture is an important and frequent cause of hindlimb lameness in TB racehorses.

Distal Hock Joint Pain

Distal hock joint pain, or distal tarsitis, is a common cause of hindlimb lameness. One of us (M.W.C.) feels strongly that the hock joint is the most important articular structure in equine locomotion. Compensatory lameness caused by primary distal hock joint pain is important. The skeleton of the TB foundation sire Eclipse at the Racing Museum in Newmarket and the skeleton from an extinct breed from the La Brea Tar Pits in Southern California reveal extensive osteoarthritic changes. Radiographic changes in the tarsometatarsal joint of young horses are common and can be found in yearlings before training begins. The importance of osteophyte formation involving the dorsal proximal aspect of MtIII, often called juvenile spavin, is difficult to evaluate without signs of lameness.

Horses with distal hock joint pain have a typical gait. The hindlimbs travel close together and may even cross midline. Horses with bilateral lameness may not show overt lameness, but they are observed to be not right behind (P.J.M.). Some horses may break from the gait slowly, may not switch leads properly behind, or cross fire and develop abrasions on the medial aspect of the tarsus (M.W.C.). Horses often wear the outside aspect of the toe of the shoe. Horses that are positive to the Churchill test likely have distal hock joint pain involving the tarsometatarsal joint (R.M.A.). Some clinicians place emphasis on upper limb flexion, and certainly the response rate is high, but this test lacks specificity. Diagnostic analgesia should be performed to localize lameness to the distal hock joints. Distal hock joint pain can cause lameness without radiographic changes, particularly in young horses, but radiographic changes can be present in horses with pain originating elsewhere. Involvement of the centrodistal (distal intertarsal) joint is difficult to assess without radiographic and scintigraphic examinations. Nuclear scintigraphy is useful to identify slab fractures of the third and central tarsal bones. Oddly, horses with radiographically evident osteophyte formation may show little IRU.

The simplest and best treatment is intra-articular injection of methylprednisolone acetate (100 mg) into each affected joint. The centrodistal and tarsometatarsal joints are small, and injecting more than 3 ml of medication without encountering resistance is often difficult. Using hyaluronan gives little

advantage, because the joints are low-motion joints (R.M.A.). However, two of us (P.J.M. and M.W.C.) use hyaluronan (20 mg) and methylprednisolone acetate (40 mg), and one of us (M.W.C.) feels strongly that the combination of hyaluronan and corticosteroids prolongs the racing careers of affected horses by delaying progression of osteoarthritis. Rarely the cunean bursa is injected. There appear to be few complications following repeated injections of corticosteroids into low motion joints, such as the centrodistal and tarsometatarsal joints. If the diagnosis is correct, the response to therapy is rewarding. However, lameness recurring rapidly after one or two injections may indicate that the primary source of pain is elsewhere or that subchondral pain is a substantial component of lameness. Corrective shoeing may help horses with distal hock joint pain. A high hoof angle (≥54°) and the application of slight turndown shoes may help horses push off and prevent sliding. In horses with primary, chronic distal hock joint pain turnout or complete rest actually may worsen lameness, and better results are achieved in these horses by maintaining a low level of training (M.W.C.).

The tarsocrural joint usually is not involved, but if effusion exists, one author (R.M.A.) injects the joint with hyaluronan and corticosteroids, because the joint is a high-motion joint. If dark synovial fluid is obtained during arthrocentesis of the tarsocrural joint, cartilage damage should be suspected (M.W.C.). Repeated injections of corticosteroids into the tarsocrural joint should be avoided.

Myositis

Exertional rhabdomyolysis (ER) and non-specific localized myositis frequently are overlooked sources of lameness in TB racehorses. ER is a common problem, especially in young horses, and primarily is seen in fillies. Male horses can be affected, but much less frequently. Diagnosis usually is not a challenge if a veterinarian observes a hot, sweaty horse with obvious muscle cramps. Lameness is mostly bilateral, transient after exercise, and resolves within a few hours. Some horses have unilateral lameness. Colts often develop unilateral forelimb ER, resembling other causes of upper limb pain. Horses may have generalized whole body stiffness if ER is unrecognized and untreated. Serum levels of creatine kinase and aspartate aminotransferase are necessary to diagnose ER definitively. Horses at risk are given acetylpromazine (5 to 15 mg, intravenously) before exercise to prevent ER. Dantrolene (500 mg, PO) given 4 hours before exercise is used in horses that have had serious episodes of ER and have missed training.

Interestingly, some horses are found to have IRU during bone (delayed) phase scintigraphic imaging, and serum muscle enzyme levels may be normal or only mildly elevated. In some horses a history of previous ER is known, but others have no known history of ER. Large muscles or individual muscle bellies within muscles can be affected. Lameness associated with IRU of skeletal muscles may or may not be seen and depends on the muscles involved. Muscles of the hindlimb most commonly are affected, such as the gluteal, biceps femoris, and semitendinosus muscles, but forelimb or trunk muscles can be involved. Serum muscle enzyme levels are usually within the normal range.

Gluteal myositis occurs in TB racehorses. Lameness is characterized by a reluctance to reach forward during the cranial phase of the stride and to push off to extend the hindlimbs at the end of the stride. Lameness may result from a loose, cuppy racetrack that breaks away from the horse. Whether the trochanteric bursa is involved is unclear. Pain is best identified by using direct digital pressure over the greater trochanter of the femur. Local pain is eliminated with injection of corticosteroids (methylprednisolone acetate, 100 mg/site) with an 8-cm, 18-gauge spinal needle into the middle gluteal muscle. The needle is directed horizontally at a 45° angle to the sagittal plane toward the opposite tuber coxae. One author (R.M.A.) does not make any effort to inject the trochanteric bursa.

The lumbar region is prone to myositis. Diagnosis is made by palpation of pain and recognition of hindlimb stiffness. An effective treatment is methocarbamol (10 gm, PO, tid). This high dose is effective with no substantial side effects. The withdrawal time for methocarbamol can be long in some racing jurisdictions. Alternatively the affected muscles can be injected with corticosteroids diluted with Sarapin (2.5 ml/site of a mixture of 50 ml Sarapin and 200 mg methylprednisolone acetate).

Other Stress Fractures

TB racehorses are prone to stress-related bone injuries of several sites other than the tibia, including the humerus, pelvis, and scapula. Humeral stress fractures account for an estimated 5% of the fatal musculoskeletal injuries in California. Horses typically develop lameness between 45 and 90 days after returning to training, following rest for an unrelated cause. Acute lameness quickly abates within 1 to 2 days, typical of many horses with stress fractures. The cranial phase of the stride is shortened, and the horse may drag the forelimb. This fracture is dangerous. When humeral stress fractures displace, they spiral and invariably cause the destruction of the horse on humane grounds. However, when identified before displacement, fractures heal well without complications. Fractures are non-articular and rarely recur. Horses are given 90 to 120 days of rest. Scintigraphic examination is the best method to diagnose humeral stress fractures, because location precludes adequate radiographic examination in many horses. Humeral stress fractures occur medially and involve the proximal, caudal cortex immediately below the humeral head; the caudal, distal cortex; and the cranial, distal cortex. Radiographs may show proliferative changes and fracture lines in horses with distal, craniomedial fractures, but in other sites neither the fracture line nor proliferative changes often can be seen. Proximally, any loss of distinction in the proximal, caudal humerus should be considered diagnostic for a humeral stress fracture until proved otherwise.

Stress fractures of the scapula are unusual, but clinical signs are similar to those caused by humeral stress fractures. Nuclear scintigraphy is the only way to diagnose this fracture. Most scapular fractures occur where the spine of the scapula meets the neck, in the same location as scapular fractures that necessitate destruction on humane grounds.

Pelvic stress fractures are underestimated grossly, overlooked, and misdiagnosed as a source of lameness in racehorses. Pelvic stress fractures most commonly involve the ilium, anywhere from the tuber sacrale to the tuber coxae. Stress fractures at the base of the tuber sacrale frequently are misdiagnosed as strains of the sacroiliac ligament. Fractures involving the base of the tuber coxae can be incomplete or complete (knocked-down hip).

Pelvic stress fractures can be catastrophic injuries, if a fracture becomes complete and displaced and results in laceration of the iliac artery. All horses with stress fractures usually have a history of transient recent lameness. Considerable evidence indicates that catastrophic pelvic fractures develop from preexisting stress-related bone injuries in horses that were continued in training. We find it interesting that the incidence is higher in fillies. Nuclear scintigraphy is useful for identifying pelvic stress fractures, but several factors decrease sensitivity, including the overlying muscle mass. Even a minor area of IRU should be considered important. Motion-correction software recently has been introduced and improves accuracy and image quality. Ultrasonographic examination can be used to identify fracture if displacement exists but is less sensitive than scintigraphy. Ultrasonography is most useful in horses that cannot be moved. Pelvic stress fractures should be included in the differential diagnosis of any TB with hindlimb lameness. Horses returning from training with a severe, undiagnosed hindlimb lameness should be tied and not allowed to become recumbent until a pelvic fracture can be eliminated.

Horses with a pelvic stress fracture may not exhibit severe lameness and may be confused with those suspected of having sacroiliac pain or pelvic muscle pain.

Sacroiliac strain is diagnosed in horses when lameness results from slipping while the horse is coming out of the starting gate (M.W.C.). Horses with sacroiliac pain resent pressure applied between the tubera sacrale. Certainly, sacroiliac strain needs to be differentiated from pelvic fracture, because management may include local, deep injections of corticosteroids and Sarapin. Injecting the back or hip muscles in a horse with a pelvic fracture gives no benefit and incurs considerable risk.

The prognosis for horses with pelvic stress fractures is good, unless displacement exists. Even horses with complete, displaced fractures of the tuber coxae can return to racing if the displacement is not severe.

Other Lameness Conditions

Stifle

Stifle lameness causes unilateral and occasionally bilateral lameness in TB racehorses (M.W.C. and P.J.M.). Horses have mild or moderate lameness and appear to swing the limb. Other characteristics include a stiff hindlimb gait, not using the hindlimbs or weak behind, getting out in the straightaway, slipping behind, and locking up behind. The most common clinical finding is effusion of the medial femorotibial joint. Lameness is exacerbated by upper limb flexion. Pain on palpation of the medial collateral ligament is found occasionally.

Diagnostic analgesia is important, but eliminating the lameness totally in most horses is difficult. Radiographic findings are generally negative, unless osteochondritis dissecans or subchondral bone cysts are present. Horses with osteochondrosis usually are treated surgically before arriving at the racetrack. Ultrasonographic examination is useful in identifying soft tissue lesions such as collateral desmitis, and when identified, affected horses are given 7 to 10 days or more of hand walking.

Most horses with stifle lameness appear to have synovitis or early osteoarthritis. Differences of opinions exist regarding the type of intra-articular injections to use in the stifle. One of us (M.W.C.) uses hyaluronan and betamethasone, but avoids using methylprednisolone acetate, because synovitis may worsen. On the other hand, one of us (P.J.M.) uses hyaluronan (50 to 200 mg) and methylprednisolone acetate (160 to 200 mg) in the medial and lateral femorotibial joints.

Secondary Shoulder Region Pain

Horses with forelimb lameness characterized by a shortened cranial phase of the stride may have some degree of secondary muscle soreness in the shoulder region or mild intertubercular (bicipital) bursitis (M.W.C.). Horses often have primary lameness in the foot or fetlock joint, but management of the primary lameness conditions does not alleviate all clinical signs. Injection of a combination of methylprednisolone acetate, isoflupredone acetate, and hyaluronan into the bicipital bursa and 2 days of hand walking resolves remaining clinical signs in some horses.

CHAPTER • 108

The European Thoroughbred

Robert C. Pilsworth

HISTORY OF HORSE RACING IN THE UNITED KINGDOM

As long as man has ridden the horse, matches of speed probably have been held informally between proud owner-riders. Racing horses under saddle became established in England during the Roman occupation. The glorious Knavesmire racecourse at York has witnessed racing almost continually since then. Since the time of James I (1603-1625), a link has existed between royalty and the town of Newmarket in Suffolk, leading to its reputation as the headquarters of racing in the United Kingdom. Charles I, the successor of James I, established regular spring and autumn race meetings in Newmarket and built a palace and stables that have survived. Racing at that time consisted of matches between pairs of horses, over long distances by modern standards. Distances as long as 4 miles were not unusual. Over the succeeding two centuries the race distances declined, as did the age at which horses were allowed to race. Initially horses had to be older than 5 years of age, but this was reduced gradually until in 1859 even yearlings were allowed to race. This practice soon ceased, but 2-year-old racing had become established and became, unfortunately in the opinions of many, part of the Thoroughbred (TB) spectrum. During this time the breed itself changed considerably.

Throughout the seventeenth and eighteenth centuries, sea transport became reliable enough to contemplate moving a living horse from the Orient to the United Kingdom. This introduced the Arabian horse to our bloodlines and established the modern TB breed, producing profound changes in size and conformation from the native stock. Eight percent of the TB racehorses alive today are descended from just one of these stallions, the Darley Arabian, through his great, great grandson Eclipse.

As the distance of races declined, speed became an increasing factor and the fields of competitors increased in size. Eventually the handicap system was introduced in an attempt to avoid the dominance of the sport by a few exceptionally gifted individuals. All of this led to an increase in interest in racing as a spectator sport and a vehicle for gambling. From the nineteenth century onward, racing was more or less recognizable as the sport we enjoy today.

PATTERN OF RACING

The most prestigious races in Europe belong to a worldwide system of accreditation, which groups together races of similar standing. The best horses competing at top level meet each other in a group of internationally acknowledged races known

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PATTERN OF RACING

The most prestigious races in Europe belong to a worldwide system of accreditation, which groups together races of similar standing. The best horses competing at top level meet each other in a group of internationally acknowledged races known

as Group 1. This group includes all of the classics in the United Kingdom and the most prestigious races throughout Europe and North America. These races are open to all horses, but they have expensive entry fees and often have to be entered well ahead of time. This self-regulation prevents people from putting horses of limited ability into these races. Beneath Group 1 are two other groupings (Group 2 and Group 3) for horses that have excellent ability but are not up to the extreme rigors of Group 1 racing. Competitors in these races face a weight for performance penalty system. For instance, a Group 1 winner running in a Group 2 race carries a weight penalty in an attempt to equalize the competition.

The next tier down from Group races are the Listed races. Again the International Pattern Committee decides which races are of sufficient stature to belong to this list. Usually horses enter a Listed race when they have already won a maiden and possibly another race with specific conditions. Such horses have few other realistic options, because after two wins a horse carries a lot of weight in an open handicap. Often success in a Listed race increases potential breeding value far more than winning such a handicap. These limited opportunities are often the reason why horses that are good, but not quite good enough to be top class, are sold out of Europe to continue racing in North America, where races are more suitable. Horses are also often able to recoup in prize money the owner's investment, a situation that is often impossible racing in Europe, with its relatively lower prize money.

Beneath the upper echelon of horses racing in Listed and Group company is an open handicap system in which horses are allocated weight according to speed rating. These speed ratings are assessed by professional handicappers, who monitor the performance of the horses when they run in the first three maiden races. The horse's rating rises and falls during its racing career, depending on its most recent form. Although this system is obviously open to abuse by trainers running horses at inappropriate distances for the horse's ability, the system does allow horses of moderate or differing ability to compete against one another on more equal footing and produce an exciting finish.

Until recently all flat racing in the United Kingdom took place on turf. This led to a certain divergence in bloodlines and ability patterns of racehorses in North America and Europe. About 15 years ago all-weather racing was developed at two tracks, Lingfield and Southwell. Although initially introduced for hurdle racing as a way of keeping the betting public satisfied when the National Hunt cards had to be abandoned through bad weather, both tracks rapidly discontinued hurdle racing on all-weather surfaces because of the high injury rate. This left the way open for flat racing on the all-weather tracks to become established. Since that time another all-weather course at Wolverhampton has been established.

The all-weather track surface is not the same as the dirt commonly used in North America. The depth of the cushion is greater, and the material is a composite of oil, plastics, and sand. Few races with significant prize money are run on the all-weather surfaces, and the racing tends to be of a humble grade. However, the track does give horses of limited ability somewhere to race and also allows trainers with small stables to compete with each other in the absence of top-level horses and trainers with many horses. Several successful all-weather horses have made a transition to racing in North America to end up running in Group races on dirt, and the all-weather surface can act as a screening academy to pick out horses that seem particularly adept at performing on these artificial surfaces.

In the rest of Europe, racing takes place on American-style dirt in Norway and Sweden, and flat-racing artificial surface tracks are found in Germany and Belgium. Many other countries have all-weather tracks but for harness racing only.

Flat racing is popular in the United Kingdom, Ireland, and France but less so in the rest of Europe. Although training centers exist in Germany along with a significant number of high-grade races, flat racing has not really caught the imagination of the public in the same way as in Britain and Ireland and receives little media attention. Ireland always has had a strong tradition of horsemanship and racing, centered around the Curragh, is buoyant and popular. Standardbred racing is popular in France, Germany, and Scandinavia, where flat racing opportunities are limited. Many European countries enjoy racing in the absence of a substantial breeding industry, and this creates a market for the surplus racehorses produced and raced in the United Kingdom, Ireland, and France. Horses in the United Kingdom, Ireland, and France are raced at 2 and, ability permitting, 3 years of age, and at the end of the 3-year-old career many are submitted for sale if they have not shown sufficient ability to be retained for racing as older horses. This makes room in the yards for the incoming yearlings. These large dispersal sales at the end of the 3-year-old career supply the horses for National Hunt racing in the United Kingdom and for flat racing areas of the world lacking breeding programs. The need for yards to clear out the less gifted 3-year-olds to make room for the influx of yearlings also produces enormous pressure on trainers and consequently their veterinary surgeons to have a racehorse fit and able to race at 2 and 3 years of age, often without consideration for the long-term consequences of any treatments.

The small window of opportunity available to these horses impinges directly on many of the surgical and medical management decisions that need to be made when problems arise. The economics and practicality of any advice given has to be considered from the owner's viewpoint and the welfare of the horse.

COMPARISONS WITH RACING IN NORTH AMERICA

One of the major differences between training and racing in North America and Europe is in the geographical location and logistics of stabling and training of the horses. In the United States almost all horses train at the racetrack and are stabled there continuously. In Europe the horses live and train in yards often well away from the racetrack. These yards tend to be clustered around a training area, with gallops and conditioning canters available for use by local trainers. The horses travel on a daily basis to race at race courses that may be up to 200 miles away.

In the United States the horses, trainers, jockeys, and veterinary surgeons tend to move from one race course to another, but they stay at each track for long periods. Racing at each location takes place for many days or even weeks before horses and trainers move on to another track. Although some horses stay behind at one track, racing does not occur at that track when the primary focus is elsewhere.

In Europe racing seldom occurs at any one racecourse for more than 3 consecutive days. Horses are trained in traditional stables, some of which date back many centuries. Horses travel to race courses the day before racing, if the journey is particularly long, or even on the day of the race. Some horses make extremely long round trips. For instance, it is not usual for a trainer in Arundel on the south coast to send horses as far north as Ayr in Scotland, a round trip of 936 miles. Obviously the cost of transport has to be weighed against the potential gains, but the traditional system of training and traveling to the races in the United Kingdom seems to be holding up for now.

In 2000, two of the all-weather racetracks opened training barns adjacent to the track with a view to introducing

American-style training, track side. How popular and successful this system is going to be and whether it will spread to other racecourses remains to be seen.

In North America almost all training and racing takes place on a left circle, and this might be expected to have influences on the incidence rates of injury to the left and right limbs for many lesions, such as proximal sesamoid bone (PSB) fractures, third metacarpal/metatarsal bone condylar fractures, and tendonitis (see Chapter 107). In the United Kingdom much conditioning work and even race speed training takes place in straight lines (Fig. 108-1). Racing itself can be on straight tracks (e.g., 1000 and 2000 Guineas at Newmarket), predominantly to the left (Epsom Derby), or to the right (Doncaster St. Ledger). The tracks themselves divide into about one third right-handed and two thirds left-handed throughout the country. This has an important impact on the lack of specific incidence of injury to the left or right limbs. One large fracture survey in Newmarket showed few instances of left or right dominance for any injury.[1]

Making categorical comments about the impact the new all-weather tracks have made on specific lameness and injury in the United Kingdom is difficult for several reasons:

1. Although the racing is on a specific surface, because of the way British horses travel long distances to race, the horses in any race have trained on a variety of surfaces at home. Because it is believed that the accumulation of the daily work sets up stress-related injuries, the actual failure that may take place in a race often can have little to do with the track surface the horses are racing on that day.
2. Tendonitis is relatively rare in the populations of 2- and 3-year-olds in a large flat training yard, and I might expect to see one bowed tendon in a yard of 100 horses in any one season. Horses that race on all-weather tracks have a far higher incidence, but they often include many older horses (3 to 9 years of age) and a higher incidence of horses with chronic, low-grade gait abnormalities, both of which could help to raise the incidence of tendon injuries.
3. The incidence of fractures in horses racing on all-weather surfaces seems to be higher than on good grass, but turf described as firm or hard is just as testing. Generalizations about turf versus artificial surfaces in terms of impact on injury have to be more specific, because turf can vary from bottomless mud to baked clay in mid-summer. The older population of horses that race on the all-weather tracks, many of which carry a legacy of subchondral bone change from the previous training at 2 and 3 years of age for turf racing, could influence the higher incidence of fractures.

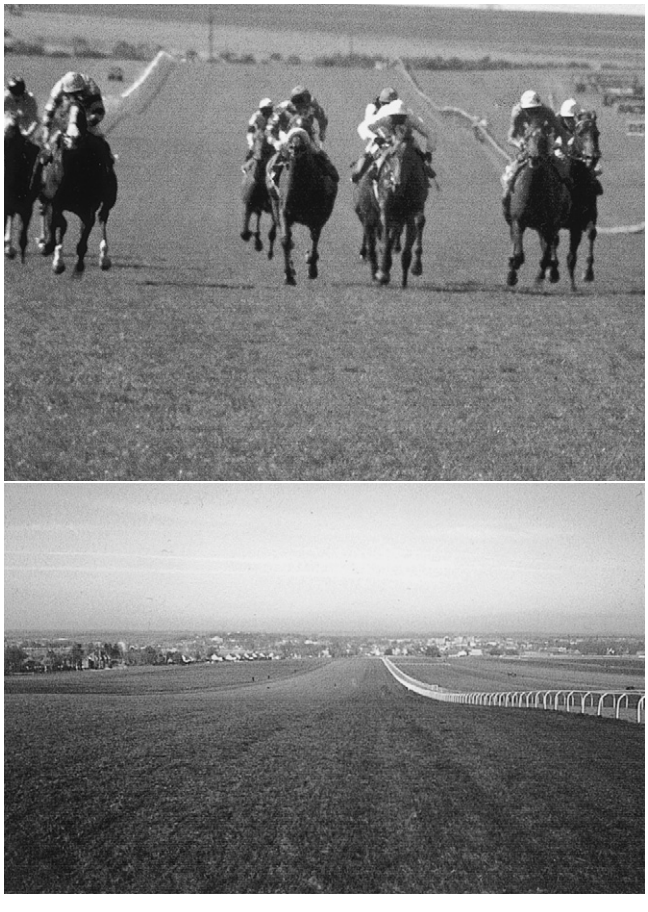

Fig. 108-1 **A,** Horses approach the finish of the long and lonely Rowley mile racetrack at Newmarket. This photograph illustrates the undulating nature of the straight 1-mile track over which both 3-year-old Group 1 classics are run. **B,** The long straight of the training track gallops up Warren Hill just outside Newmarket. An all-weather surface is between the white railings. The rest of the heath is used in strips, which are changed on a daily basis. Each strip of grass is used only once every 3 years.

TRAINING REGIMENS IN EUROPE

Yearlings arrive in the yards for breaking from the sales in September and October and, unlike the situation in North America, often go straight to the trainer for breaking rather than to a specialist pre-training center.

After breaking, the yearlings usually start steady cantering exercise until Christmas. As the racing season approaches, horses showing precocity and ability to withstand faster exercise step up in pace throughout February and March. The first 2-year-old races occur in April, encouraging hard training of skeletally immature horses. However, until the racing calendar changes and while these races are open and available, trainers will train horses for them.

All-weather racing has altered the seasonal impact of flat racing in the United Kingdom forever, and some flat race horses are now in training throughout the year. However, most of the high-quality flat race horses do not compete on the all-weather circuit and are put into a slow-speed maintenance program from November until January, involving light cantering and trotting. Fillies may be turned out for a period.

All of this is substantially different from the position in North America, where the horses often do not arrive at the racetrack until they are fully broken, in cantering exercise, and ready to do fast work. The breaking and pre-training often takes place in specialized pre-training centers, away from the city center tracks. This has important affects on the perception of racetrack veterinarians in North America concerning the incidence of developmental orthopedic disease linked to lameness. In North America developmental orthopedic disease often causes lameness leading to diagnosis and removal from training before a horse reaches the racetrack, and horses therefore are not commonly seen by track veterinarians. An example is a subchondral bone cyst in the medial femoral condyle. Subchondral bone cysts are a regular occurrence in the annual intake of yearlings in the United Kingdom, diagnosed by the trainers' veterinarians. Discussion with colleagues working at Californian racetracks reveals that they rarely see horses with subchondral bone cysts, presumably because of the effect of the earlier screening at the pre-training centers.

Once on the track in North America the racing is often less seasonal and less age specific, removing a lot of the pressure for success at 2 or 3 years of age within a short season. Whether a horse races on turf or dirt does not matter: all training takes place on dirt, and this should be borne in mind by veterinarians assessing horses for potential purchase to move from Europe to America. Asking potential purchasers whether the horse is intended for turf or dirt racing is largely irrelevant, because the daily training almost certainly will be on dirt. The dirt surface is more testing in many ways than anything these horses have seen before, and horses able to train and perform well on grass sometimes fail to make the transition to dirt.

CLINICAL HISTORY

Taking a detailed history is a prerequisite for a medical examination in any species. In racehorse practice one often is seeing a succession of lame horses during any morning in several different yards, and the detailed information may be limited. The trainer may not be present to supply it. However, I always try to ascertain the following information before examining a TB racehorse for lameness:

1. How old is the horse? This is probably the single biggest determinant for different types of lameness. The chronological age is important, but far more important is the skeletal naivete of the horse. An unbroken horse of 3 or 4 years of age behaves in essentially the same way as an unbroken yearling, irrespective of the difference in age, and may suffer similar problems when confronted with an inappropriate training program.
2. What stage of training has the horse reached? Has a yearling reached race-speed exercise yet, is it simply trotting, or has it entered normal conditioning cantering exercise? For a yearling that is lame while being broken or in early cantering exercise, one has to consider subchondral bone cysts and other osseous cyst-like lesions, osteochondritis dissecans, and associated osteoarthritis. Problems may be bilateral, resulting in a peculiar gait. Bilateral sacroiliac joint pain considerably affects the freedom of movement of the hindlimbs and may mimic the clinical signs of exertional rhabdomyolysis, but plasma creatinine kinase (CK) levels often will be normal or only slightly raised (500 to 1500 IU/L).

   Stress-induced bone injuries are more likely to become apparent as work intensity increases. Stress-induced bone injuries include all the long bone stress fractures of the humerus, tibia, third metacarpal (McIII) and third metatarsal (MtIII) bones, and the ilial wing of the pelvis. Similarly, in a 2-year-old that has reached advanced training speed, the carpus and metacarpophalangeal joint are common sources of forelimb lameness, and lateral condylar stress injuries of MtIII are common causes of hindlimb lameness. At 2 years of age the horse is less likely to have lameness associated with a subchondral bone cyst, osseous cyst-like lesion, or osteochondritis dissecans, because these lesions usually cause lameness when the horse is young and begins conditioning exercise. However, occasionally subchondral bone cysts and osseous cyst-like lesions may arise or show clinical signs for the first time at 3 years of age.
3. Has the horse raced yet? If a horse has raced successfully at 2 years of age, the horse is unlikely to have hidden osseous cyst-like lesions or osteochondritis dissecans. Also, the skeleton has probably been conditioned adequately, and the incidence of stress fractures can be expected to be lower. However, in countries where racing is seasonal, 3-year-olds can detrain through the closed season if they are not maintained in cantering exercise. Stress fractures then may occur in the following season if speed training starts too quickly. A Californian study showed an associative link between the occurrence of stress fracture of the humerus and previous long-term removal from training.[2]

   If a horse did not race at 2 years of age, then all of the differential diagnostic criteria used in the recently broken 2-year-old should be applied to the 3-year-old.
4. Has the horse had any previous orthopedic problems? Often in practice I know each horse personally and am able to recall the problems that affected the horse in its early years. The history of a horse with previous problems may guide the use of diagnostic analgesia in an unorthodox, but timesaving manner, to establish whether the current lameness is a recurrence of a previous problem.
5. What is the pattern of the lameness? One should ascertain whether the lameness is constant from day to day and for how many days the horse has shown a gait abnormality. The veterinarian should ask if the lameness gets better or worse within an exercise period. In some conditions, such as proximal suspensory desmitis (PSD), the lameness tends to get worse as the horse exercises, only to resolve with 24 to 48 hours of rest. Horses with lameness from other causes, such as osteoarthritis, often warm out of clinical signs considerably from the start of exercise.

   In horses with acute-onset, severe lameness, questions of clinical history become less important because the horse is often not bearing weight, and a suspicion of skeletal failure is raised. Knowing if this horse has been a good mover before onset of severe lameness is often helpful, because some injuries (third metacarpal/metatarsal bone condylar fractures, PSB fractures) often are associated with poor gait before the actual fracture takes place. The same is true of slab fractures of the third carpal bone, which often are preceded by a long period of subchondral bone sclerosis associated with bilateral third carpal bone pain (Fig. 108-2). One of the most useful questions to ask about a horse with severe lameness is what stage of training the horse has reached, because only advanced training to fast canter or gallop speeds usually results in bone failure. However, bacterial infection subcutaneously or within the hoof also can produce severe lameness.
6. Has the horse had a prolonged layoff for any reason? Horses brought back from a long period of box rest after illness or injury, without adequate prior bone conditioning, can get bizarre injuries from unusually low-speed exercise. These include complete, displaced scapular and humeral fractures, bilateral fractures of the PSBs and tibias, or displaced fractures of the proximal phalanx or condyles of McIII. This is a reminder of the vital need for time to allow adequate skeletal conditioning necessary to avoid injury.

Clinical Examination

The veterinarian always should get the horse out and see it walk and trot to determine which limb is lame. Riders are notoriously unreliable at detecting the correct lame limb. The veterinarian should determine whether the lameness is unilateral or bilateral by the way the horse moves. Many racehorses are slightly lame in all limbs and have a typical crouching,

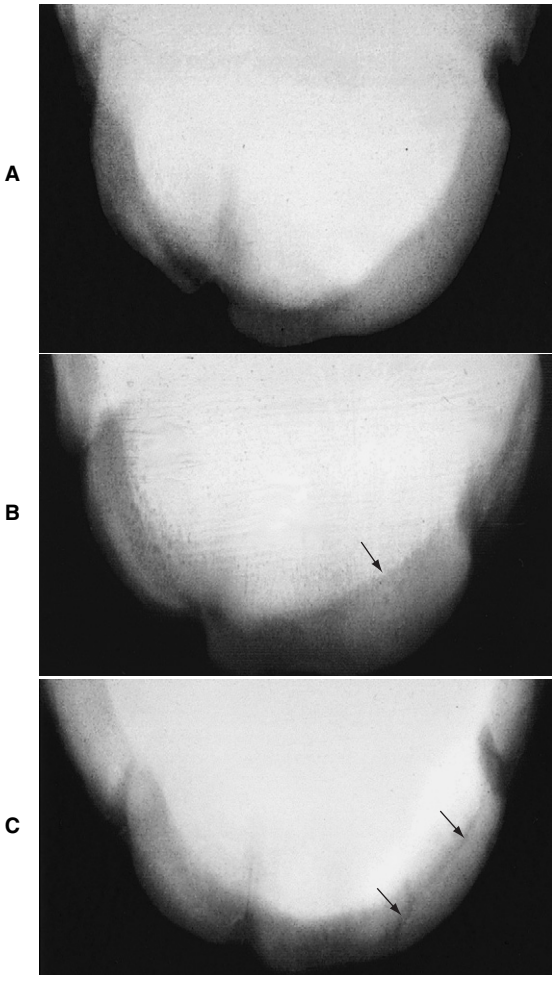

Fig. 108-2 Flexed dorsal 60° proximal–dorsodistal oblique projections of the third carpal bone of three Thoroughbreds. Lateral is to the left. **A,** Mild to moderate sclerosis of the radial facet of the third carpal bone. **B,** A short fissure fracture *(arrow)* can be seen propagating from the dorsal margin of the sclerotic radial facet of the third carpal bone. **C,** A non-displaced fracture is in the center of an area of intense sclerosis in the third carpal bone *(arrows)*.

shuffling trot or may try repeatedly to break into a canter because trotting is so uncomfortable. New lameness may be superimposed on a chronic level of unsoundness.

Increasing experience usually leads one away from the belief that any one injury is linked to a particular gait. Lameness typical of a shoulder or typical of a stifle tends to become a more remote belief, because so-called typical lamenesses so often are linked eventually to the most unexpected site. However, a few types of lameness do remain that seem to be linked to one particular pathological syndrome. Young TBs often show bilateral forelimb lameness related to carpal pain. These horses often trot with the limbs abducted from the midline and not fully flexed during forward motion. This gives a rolling, stiff-legged forelimb gait. The horse's attempt to get the contralateral limb down quickly to get off the sore limb as soon as possible shortens the forward phase of the stride, leading to a choppy, stilted action.

Pain associated with the metatarsophalangeal joint is often bilateral and leads to a fairly characteristic gait typified by low limb flight, often with dragging of the toe during protraction, and an exaggerated pelvic excursion dorsoventrally on both sides (in this clinic termed the *Marilyn Monroe trot*), because the horse dips off each painful limb. Horses with a humeral stress fracture often abduct the limb, with shortened cranial protraction at the walk.

Having ascertained which limb is the lamest, the limb should be examined in detail, using basic examination protocols (see Chapters 4 through 8 and 10). The following specific points apply to the TB racehorse.

1. Many racehorses show a pain response to squeezing of the foot with hoof testers, which is not related to a current lameness. The TB has a thin-soled foot, which often is shod incorrectly, allowing palmar third soft tissue bruising to occur. If the horse responds to hoof testers in the lame limb, I normally test the contralateral foot to see if the response is different. If the response to hoof testers is the same in both limbs, I would assume that the foot may not be the primary source of lameness.
2. Suspensory branches are best appraised by centering each branch between opposing thumbs and following the branch from the most proximal portion which is palpable down to the PSB. Subtle thickening in the suspensory branch may be detected as the thumbs separate running toward the PSB.
3. The metacarpophalangeal joint should be checked for distention of the palmar pouch of the joint capsule. Thumb pressure is applied to the dorsoproximal aspect of the proximal phalanx and the dorsodistal aspect of McIII, common sites for fragmentation and impingement lesions producing soreness. The limb then should be flexed partially and the response to flexion of the metacarpophalangeal joint monitored. Many young horses show a mild pain response to flexion, and the contralateral limb should be compared to gauge whether a difference exists. However, in a 2-year-old a bilateral pain response may reflect true problems in both limbs. A normal metacarpophalangeal joint should not be painful to flex.
4. The proximal suspensory ligament (SL) should be examined with the limb raised by squeezing the ligament between the thumb or fingertips and the palmar surface of McIII, first medially then laterally. Normal untrained horses show no pain response. However, many young racehorses show a variable degree of discomfort on SL palpation. Differentiating pathological and normal response to palpation associated with increased training can be difficult.
5. The dorsal surface of McIII should be squeezed with the finger tips with the limb raised and the pain response noted as an indication of sore shins. The surface of the shin should be palpated for periosteal thickening or even focal callus formation associated with a stress fracture.

   Running a finger down the shin with the horse bearing weight often causes a horse to buckle the limb for months or even years after shin soreness has subsided and is not a reliable diagnostic test.
6. To detect distention of the antebrachiocarpal or middle carpal joint capsules, pushing on the dorsal surface of the joint with the finger and thumb of one hand while observing the palmar pouches of the joints is useful. Mild effusion then may be detected.
7. Many young racehorses resent extension of the shoulder joint in the absence of a lesion.
8. Many racehorses mildly object to forced tarsal flexion equally in both hindlimbs unrelated to a lesion. A profound response to flexion of the tarsus of the lame limb, which is not matched in the contralateral limb, usually signifies a tarsal bone fracture or a distal tibial stress fracture. If pain is noted on flexion, thumb

Fig. 108-3 Application of torsion to the tibia. The metatarsophalangeal joint is rotated medially, the os calcis laterally, and firm shoulder pressure maintained with the stifle. Horses with incomplete linear fracture of the tibia often show a sharp pain response to this test.

pressure should be applied to the proximodorsal aspect of MtIII and the dorsolateral surface of the tarsal bones to determine a pain response. Pain response is often marked if there is a slab fracture of the third tarsal bone.

9. The tibia is a common site for stress fractures and is examined by curling the fingers around the caudal surface of the tibia from the medial aspect and squeezing on a common predilection site for stress fracture, the caudal surface of the distal third of the tibia. Torsion of the tibia also is attempted by flexing the limb, applying lateral traction to the os calcis with one hand while maintaining firm contact on the stifle with one's shoulder, and medially twisting the distal MtIII with the other hand (Fig. 108-3).
10. The pelvis is another frequent site of stress fractures and is examined by monitoring the position of the bony landmarks—the tubera sacrale, tubera coxae, third trochanter of the femurs, and tubera ischia—looking for displacement (see Chapter 51). Response to deep palpation of the muscles that lie between these sites is also noted. The most common clinical sign in horses with a non-displaced pelvic fracture is guarding of the musculature as a response to deep palpation. Guarding gives a hard, board-like texture to the muscle and often is accompanied by fasciculation of the gluteal mass. Similar clinical signs can be seen in horses suffering from exertional rhabdomyolysis, and this may be a difficult differential diagnosis to establish. Muscle enzyme concentrations may be elevated (creatine kinase 500 to 1500 IU/L) in horses with a pelvic fracture. The tail should be raised and lowered and tail tone monitored. A flaccid tail often indicates a sacral or coccygeal fracture.

Imaging Considerations

Radiography

Many lesions in the TB racehorse are associated with subtle changes in bone density and structure rather than overt fragmentation of bone. These variations result in subtle changes in radiopacity (increased radiolucency and or sclerosis), which often are seen best on high-quality radiographs using single-screen film and excellent radiographic technique or by using digital radiography.

These standards usually are not achievable in the field, and for this reason we encourage all trainers to bring horses to the clinic for radiography. Radiography is notoriously unreliable in detecting early fracture lines and subchondral bone collapse. For this reason, if lameness is linked to a particular site by diagnostic analgesia and nothing is visible radiographically, repeating the radiography 2 weeks later is often advisable. Any horse with subtle changes in bone density can be given time to develop to the point where additional damage can be seen. During investigation of individual joints, special projections are often most useful (Fig. 108-4). Radiographic examination of the metacarpophalangeal joints always should include the flexed 125° dorsopalmar projection, which is extremely useful for evaluating the palmar and plantar surface of the condyles.[3,4] In a hindlimb this view often is achieved best as a plantarodorsal projection. The limb is cupped loosely by a hand underneath the tarsus and allowed to hang in a semi-flexed position. The cassette is placed in a cassette holder and positioned dorsal to the metatarsophalangeal joint, and the x-ray machine is positioned above and behind to achieve the orthodox 125° projection (see Fig. 108-4).

Radiographic examination of the carpus always should include a flexed proximal 55° distal skyline view of the third carpal bone, which is sometimes the only projection in which lesions in this bone can be seen clearly. The projection is easy to do well but is still often done poorly. Small changes in the angle of projection make a significant difference to the amount of the third carpal bone visible in this projection.[5] One should not be satisfied unless the whole of the radial facet is seen clearly (see Fig. 108-2).

The relevance of changes in bone density should not be underestimated. Many stress fractures of the long bones are seen only by the occurrence of endosteal or periosteal callus. Periosteal callus may only be visible with the aid of a bright light. Fracture lines are relatively rare. Despite the difficulty in imaging incomplete fracture lines, some horses may develop complete, displaced fractures during periods of box rest.

Scintigraphy

Scintigraphy has allowed enormous advances to be made in the detection and understanding of stress fractures in the racehorse.[6,7] Scintigraphy should not be used as a first line of investigation in an obvious single-limb lameness, unless a strong suspicion exists that the pelvis or tibia is involved and radiography has been unrewarding. Examination of a whole TB racehorse that cannot trot and has a multilimb lameness is often useful. These horses are difficult to assess using diagnostic analgesia, because one is often racing against time, moving from one limb to another with the predominant lameness constantly changing. Scintigraphy in these horses often results in a list of lesions that can be investigated radiographically and by judicious use of diagnostic analgesia. The other roles of scintigraphy include examining a site identified by diagnostic analgesia but in which no abnormality can be found on radiography and safety screening of an initially severely lame horse that has become sound before a diagnosis has been reached. Making a diagnosis in these horses at post-mortem is thus avoided.

Ultrasonography

Ultrasonography is the imaging modality of choice for investigating soft tissue injuries in the metacarpal and metatarsal

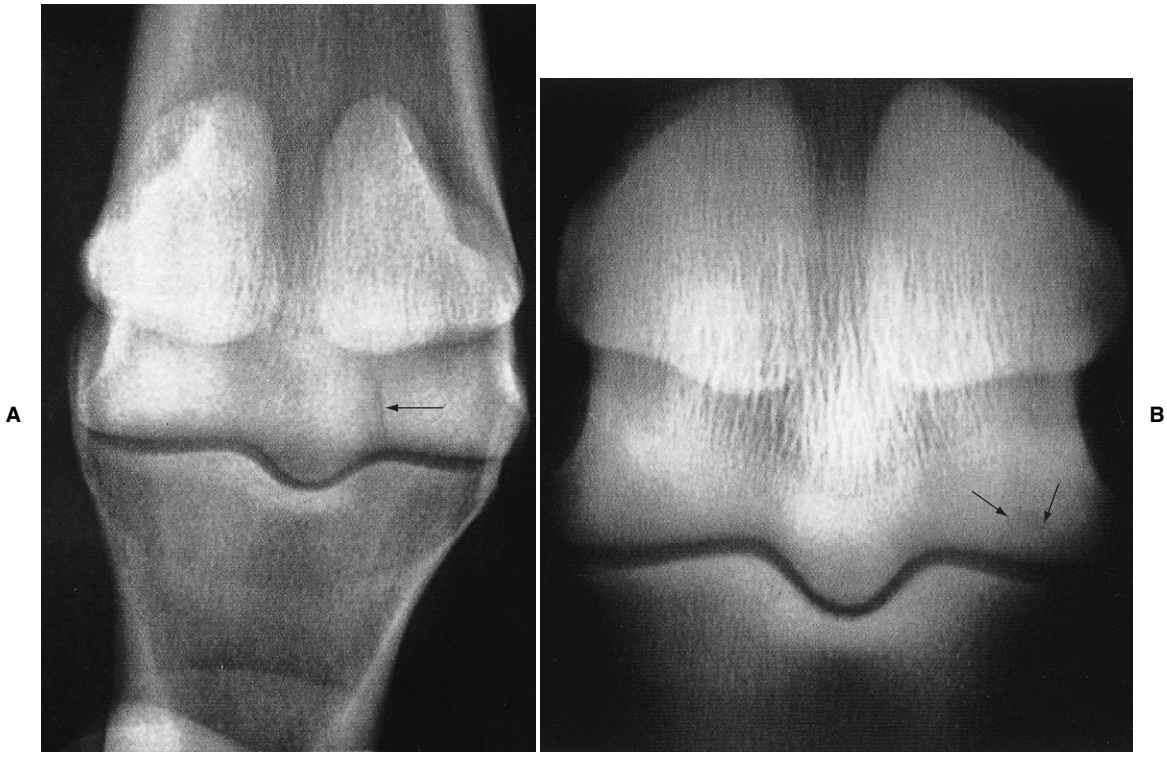

Fig. 108-4 Flexed dorsopalmar radiographic view of a metacarpophalangeal joint. Lateral is to the right. **A,** There is an incomplete linear fracture *(arrow)* in the lateral condyle that was not visible on any other projection. **B,** Flexed dorsal distal 125°–palmar proximal oblique radiographic view of a metacarpophalangeal joint. Lateral is to the right. There is a semi-lunar radiolucency *(arrows)* in the lateral condyle at the site of a chronic stress injury.

regions (see Chapter 16). The only observation pertinent to the young TB alone relates to PSD in horses of 2 years of age. Lesions are often subtle, with enlargement and loss of the neat rectangular shape of the normal SL.

The clinical relevance of bilateral enlargement of the superficial digital flexor tendons along with intratendonous edema in a 2-year-old may be difficult to interpret, because normal tendons increase in size in response to training[8] (see Fig. 102-2). If obvious distortion of the contour of the tendons exists, I advise that the horse not gallop or race at 2 years of age. The horse is maintained in alternate day trotting and cantering for 3 months. This treatment has not led to true tendonitis at 3 years of age in any horse. I refer to these horses as having juvenile tendonitis but have never been comfortable that this degree of caution is mandatory. Perhaps these horses could do more work without further injury.

Ultrasonography is also useful for detecting and monitoring some fractures of the ilial wing and shaft of the pelvis (see Chapter 51).

Diagnostic Analgesia

Having carried out the clinical examination described, one structure causing lameness may be obvious. For instance, the horse may show a sharp pain response on palpation of the SL or distention of the middle carpal joint capsules with pain on flexion. Diagnostic analgesia may not be necessary in these horses and diagnostic imaging of the site may be the next step. Many horses have a list of diagnostic possibilities: the horse may have shown a pain response to hoof testers and some tenderness to palpation of the SL and have some distention of the middle carpal joint capsule. To tease apart these findings, diagnostic analgesia is mandatory. Diagnostic analgesia is done most efficiently at the clinic rather than the training yard, because the procedure is time consuming. If a horse is fresh when admitted for diagnostic analgesia, the increase in muscle tone and general excitement can obscure a mild lameness, and acetylpromazine (2 to 5 mg per horse intravenously) can help settle the horse, without affecting its ability to trot.

If the horse is admitted to the clinic for a day, a positive block can be allowed to wear off, so that more localized blocks can be carried out. For this reason, my own first block is usually an abaxial sesamoid block so that the foot can be eliminated or investigated in greater detail later. Many horses with mild lameness exhibit a detectable deterioration in the degree of lameness if the foot is desensitized and is not the source of pain.

If this block renders the horse sound, the block is allowed to wear off and more detailed diagnostic analgesia is performed. A palmar digital block is carried out, one nerve (either medial or lateral) at a time, the lameness being re-evaluated after each branch is blocked. If effusion of the distal interphalangeal joint is present, an intra-articular block is performed.

If the abaxial sesamoid block is negative, a low palmar (four-point) block is used. This may alleviate lameness; not improve lameness, indicating the problem is much higher; or give a 30% to 50% improvement in lameness. Such results are observed commonly in horses with PSD.

The next logical step is the subcarpal block, which I perform using 1.6-cm needles and 2.5 ml of mepivacaine in the recess immediately palmar to the top of the small metacarpal bones. Short needles and small volumes of local anesthetic solution are used in an attempt to reduce the frequency of false-positive analgesia of the middle carpal joint. False positives can occur by inadvertent direct penetration of the distopalmar outpouchings of the carpometacarpal joint[9,10] or by diffusion of local anesthetic solution up the facial planes to a site proximal to the middle carpal joint.

A positive subcarpal block always is allowed to wear off, and the middle carpal joint subsequently is blocked to ascertain which site is responsible for the lameness. Unfortunately in some horses complete soundness follows both the middle carpal joint and subcarpal blocks. When this occurs, ultrasonographic examination of the proximal SL and a radiographic examination of the carpus are performed before an informed guess is made about the most likely source of lameness. If no lesions are found after imaging both sites, defining the source of pain categorically may not be possible, and the horse is a candidate for nuclear scintigraphy.

Negative subcarpal and middle carpal analgesia are followed by median and ulnar nerve blocks. Although these blocks leap-frog the antebrachiocarpal joint, median and ulnar nerve blocks usually abolish lameness associated with lesions of this joint. Intra-articular analgesia of the antebrachiocarpal joint may fail to abolish lameness originating from distal radial chip fractures in some horses.[11] The jump directly to median and ulnar blocks avoids the possibility of false negatives misleading the lameness diagnosis.

Negative median and ulnar blocks usually are followed by advice to carry out scintigraphy because scapulohumeral joint lameness is relatively rare and intra-articular analgesia does not alleviate lameness associated with humeral stress fractures, which are common. Although osteochondritis dissecans and osseous cyst-like lesions of the scapulohumeral joint are found in late yearlings and early 2-year-olds, these often show scintigraphically and can be confirmed later by radiography and diagnostic analgesia if necessary. Intra-articular analgesia of the elbow and scapulohumeral joints is performed if the median and ulnar blocks and scintigraphy fail to establish the seat of lameness.

Hindlimb investigation follows a slightly less rigorous approach for three reasons:

1. Some young TBs become difficult with repeated needle entry of the hindlimbs, resulting in a serious risk to the veterinary surgeon, the member of staff holding the horse, and the horse itself.
2. The common occurrence of stress fractures in the more proximal sites in the hindlimb, particularly in the tibia and the pelvis, means that many horses potentially would be blocked in detail from toe to stifle, without producing any improvement in lameness. For this reason in a hindlimb an abaxial sesamoid block is performed first, followed by a six-point (low plantar) block above the metatarsophalangeal joint. A positive response to a six-point block is allowed to wear off, and the response to intra-articular analgesia of the joint is assessed. If these blocks produce negative results, a subtarsal block is performed and then followed by a tarsometatarsal joint block. If the horse behaves satisfactorily, tibial and fibular (peroneal) nerve blocks are performed. If lameness is still present, the horse may be allowed to rest undiagnosed or the stifle is examined radiographically, or scintigraphy is performed, depending on the wishes of the trainer and owner. Only if lameness recurs after a period of rest and the absence of a scintigraphic diagnosis are the stifle joints blocked.
3. Bilateral lameness associated with subchondral injury to the lateral condyle of MtIII is common in TBs and often can be abolished by blocking the lateral plantar metatarsal nerve only, just dorsal to the button of the splint, using 2 ml of local anesthetic solution.[12] This often produces an overt lameness in the contralateral limb. This block is easy to perform and the problem is common, so it is tempting to use this block first in horses showing the typical bilateral gait abnormality often seen in this condition.

Shoeing Considerations

TB racehorses have notoriously bad horn quality and hoof conformation. They appear to have uniformly thin soles and a tendency to develop long toes and low heels. Medial to lateral hoof balance is vital to the TB racehorse remaining sound, but it is sometimes not pursued adequately. All horses should be shod wide at the heels to allow full weight bearing by the horn structures. However, in racehorses this tends to produce an abundance of pulled shoes by the trapping of the metal of one shoe underneath the other foot in the stable, particularly on rubber floors, which do not allow the trapped shoe to slip out. This encourages farriers to tuck the shoes out of reach under the foot, which leads to progressive collapse of the heels and long-toe, low-heel conformation. Farriers have to be reassured that they will not be held responsible for pulled shoes within the stable and that the health of the horse's foot is more important than the management problems sometimes associated with correct shoeing.

Interference injuries are not as common in the TB as in Standardbreds and are more common when the horses are on a reduced exercise plane or in 2-year-olds in early training. Interference injuries seem to become less common as the horse strengthens and becomes more fit. In every horse such injuries should be approached as a joint exercise, with the full involvement of the farrier. The following approaches are helpful.

Brushing Injuries of the Hindlimbs

Hindlimb brushing injuries are caused by the contralateral foot in flight and produce cuts on the medial aspect of the fetlock. Most of these horses toe out and are base narrow, and some swing the foot inward during protraction at walk and trot. Traditionally three-quarter round shoes have been used, with the medial branch missing. Over time, however, this shoeing results in progressive collapse of the medial hoof wall, pushing the limb even further out of line, and brushing may actually increase. An alternative is to use light steel or alloy plates behind. This lowers the weight of the foot and often stops the problem. If this fails, we use a 2-cm lateral trailer on the shoe. Half-round shoes are always a help, because they remove the cutting edge if interference does take place.

Brushing Interference in the Forelimbs

Brushing in forelimbs is difficult to treat. Affected horses usually toe out significantly from distal to the metacarpophalangeal joint and are base narrow. Horses often first show interference when galloping at speed and injury causes significant hemorrhage and swelling over the medial PSB. This swelling then makes future interference even more likely, and a downward spiral begins. The steps I usually take involve careful medial to lateral correction by the farrier, rest and anti-inflammatory treatment to reduce swelling, and application of closely fitted half-round section shoes. Topical application of dimethylsulfoxide containing flumethasone is indicated unless there is an open wound. If a full-thickness skin wound is present, topical antisepsis and systemic antibiotic treatment may be indicated to prevent undue swelling at the site.

Protection of the site of injury on return to training with a thin layer of cohesive exercise bandage can help, but great care should be taken in applying this type of bandage because the lack of padding can all too easily produce a bandage bow (bind). Even with all these measures, for affected horses to suffer repeated intermittent problems throughout their careers is not uncommon.

Scalping, Forging, and Over-Reaching

Scalping, forging, and over-reaching are produced by the hind foot arriving in the site occupied by the front foot too soon in the gait cycle.

Scalping produces cuts on the dorsal aspect of a hindlimb pastern, too dorsal to have been caused by the contralateral limb. Scalping is caused by the tip of the front shoe striking

the front of the advancing hind pastern, as the horse just misses forging. Such injuries often are avoided by a four-point trim or shortening the toe and using a rolled-toe shoe in front to speed breakover fractionally, thus getting the front foot out of the way by the time the hind foot arrives. If this alone fails, then using a light steel or alloy plate with a square toe in front and heavy steel shoes behind often eliminates the problem. The same measures are used to reduce true forging, when the hind shoe strikes the tip of the front shoe, producing the characteristic steel on steel noise from which the name is derived. Over-reaching, when the hind toe strikes the bulb of the front heel, can be approached in the same way. Over-reach boots can help but can only be used in slow work. Over-reaching is most common when the horse is fresh and being restrained hard in its exercise or is being exercised in deep sand, preventing easy limb clearance. For this reason these horses can be helped by judicious use of mild sedation before exercise (10 to 25 mg of acetylpromazine PO, 30 minutes before exercise) and by exercising on a non-impeding surface. In many horses all of these measures can cease as the horses become fully fit.

TEN MOST COMMON LAMENESS CONDITIONS

The following are the 10 most common lameness conditions; these are discussed in detail in this section.

1. Foot-related lameness
2. Suspensory desmitis
3. Lameness associated with the middle carpal joint
4. Subchondral bone injuries to the distal third metacarpal/metatarsal bone
5. Lameness subsequent to bacterial infection
6. Stress fractures of the long bones (third metacarpal bone, tibia, and humerus) and pelvis
7. Exostoses of the second and fourth metacarpal/metatarsal bones (splints)
8. Undiagnosed hindlimb lameness
9. Fractures of the proximal phalanx and condyles of the distal third metacarpal/metatarsal bone
10. Lameness associated with the tarsometatarsal joint

Foot-Related Lameness

TB feet are usually thin soled, and most racing TBs have a pain response to hoof testers, even in the absence of genuine lameness. To minimize laminar bruising, good shoeing technique is essential and horses should not be exercised on a surface that is too firm. Medial to lateral foot balance is also vital but often neglected. Farriers often are expected to shoe in the dark of the early morning, with the horse loose in the stable on deep bedding. This often leads to poor visual assessment of foot-pastern axis and balance. Clinicians should all help farriers to change this attitude in favor of a system that allows these skilled professionals to achieve their potential. Such a change involves providing staff members to lead out and hold the horses for the farrier to inspect.

Bruising usually is diagnosed when the horse shows an exquisite pain response to hoof testers in a fairly localized area, but no obvious tract leading to the deeper structures. Careful paring with a hoof knife often reveals pink discoloration in the horn layers immediately above the area of the bruise. Removing the shoe and applying a poultice to the foot for a day is usually successful in managing this condition. If the horse is close to a race and time is of the essence, the foot is iced morning and night in an attempt to decrease the pain associated with the bruise. Once the pain is clearly only from the bruising and no pus pocket has formed under the sole, poulticing is stopped to dry up the sole again before shoeing. If foot lameness is associated with infection, then draining the subsolar abscess is mandatory. Although traditional textbooks talk about radical curettage and establishing sufficient drainage, in the racing TB removal of too much horn is associated with an extended period of lameness because of laminar damage and pain from exposed soft horn. Initially the hole to the pus pocket should be as small as possible. Once drainage has been established, hoof testers can be used to expel pus and to pull in soaking agents or hydrogen peroxide. The hole often can be enlarged by a bent 16-gauge needle. The foot usually is soaked then in warm salt water for 10 minutes morning and night and in between is wrapped in saline-soaked cotton wool dressing contained within polyethylene. Walking these horses twice a day if the degree of lameness permits is a good idea to encourage extrusion of the pus, which leads to a quicker return to full work and decreases the risk of rhabdomyolysis on return to training. Some horses with infection within the foot are infuriating, because although an exquisite pain response occurs and a clear tract leads into the foot, blood in the sensitive laminae is reached before the infected focus is found. The horse then usually resents further digging in the sensitive laminae. Radiographic examination is justified in these horses to rule out infectious osteitis of the distal phalanx, usually evident as a radiolucent area. The condition is treated by radical curettage after sedation, local nerve blocks, and application of a tourniquet. A treatment (hospital) plate is then applied.

Traumatic corns can be one of the most frustrating conditions with which to deal. Affected horses usually have a long-toe, low-heel conformation and inwardly collapsing heels. Intense pain occurs when hoof testers are applied across the seat of corn and yet exploration simply reveals subsolar blood pockets or bleeding tissue. When these are exposed, localized prolapse of the sole may occur at the seat of corn. These horses take a long time to regain soundness and normally are shod with a bar shoe to bridge the weight bearing from the non-affected heel to the wall of the other quarter. The horse should be kept in walking exercise only for at least 2 weeks to allow some horn growth before an attempt at retraining. Training and racing on hard surfaces is contraindicated, which can be realistic advice in the United Kingdom. These horses may be more difficult to manage if sold to train on the tracks in North America. The farrier should be involved in attempting to bring up the heel angle in these flat-footed horses, and the four-point trim is often helpful in achieving this quickly. Medial to lateral balance should be checked, because traumatic corns often occur on the lower heel of a horse that has been allowed to become unbalanced. This seems to occur because of second contact concussion as the foot strikes the ground in a biphasic manner, which is corrected by lowering the longer of the two heels to make sure that the foot strikes the ground evenly.

Sometimes a breach in the horn at the seat of corn allows infection to enter, producing an infected corn. The condition often is linked to swelling and pain on palpation of the bulb of the heel above the corn. Drainage, poulticing, and remedial shoeing are the standard treatments.

In lameness associated with the horse being pricked by the farrier, with or without development of infection, using a glue-on shoe when reshoeing after the problem has been resolved is sometimes a good idea. Most farriers shoeing racehorses are extremely skilled, and if they have managed to puncture the sensitive laminae during a routine shoeing, the cause is usually because the horse has extremely little wall outside the white line. The direct glue-on technique, using a normal alloy racing plate stuck directly to the sole without nails, allows the horse 3 to 4 weeks of extra horn growth before further nailing is done. The more traditional tab-glue shoes are less satisfactory, because the shoes tend to become loose and the horse's foot shape may change. This is not the

case with direct glue-on shoes. Horses that repeatedly lose shoes and have increasing wall avulsion and damage are treated best using direct glue-on shoes to allow time for wall recovery.

Suspensory Desmitis

The SL can be injured at any location, but PSD is most common in a young racehorse. PSD is often bilateral, although the lameness observed is almost always unilateral. PSD is characterized by weight-bearing lameness of grade of 1 to 3 of 5, which seems to disappear rapidly after periods of rest. Even 24 hours of box rest often renders these horses sound. However, within an exercise period, the lameness is typified by becoming worse the more the horse does (warms into the lameness). Horses with PSD often show significant pain when the SL is squeezed between the thumb and the back of McIII with the limb flexed. The veterinarian should start at the apex of the PSB and work along the SL, squeezing it against the bony structures of the limb. The response should be compared with the contralateral limb. Most normal young TBs show some degree of tenderness to palpation of the SL, although unbroken and untrained horses of the same age usually do not. This may indicate that many racehorses suffer a degree of PSD during early training in the absence of observable clinical signs.

Local analgesia is discussed on page 885. When performing subcarpal analgesia, the needles should be applied to the syringe before injection to prevent the introduction of air into the fascial planes, which can preclude later ultrasonographic examination. This air appears as a hyperechoic band between the SL and the accessory ligament of the deep digital flexor tendon, obscuring evaluation of the SL.

The other common predilection site for injury of the SL is the interface of a distal branch and the ipsilateral PSB. These lesions usually occur when the horse starts faster exercise. In horses with conformational defects the same site can be affected in both forelimbs. For example, in a horse with offset carpi and toe-in conformation, the lateral branch of the SL and the lateral PSB are affected. Typical radiographic changes of sesamoiditis are seen with clear disruption of the SL/PSB interface. Central hypoechoic lesions are identified by use of ultrasonography. The SL branches often are enlarged palpably, but ultrasonographic imaging reveals this to be a zone of periligamentous fibrosis, rather than true swelling of the branch itself.

Horses with PSD are confined to box rest for 2 to 4 weeks until clinically sound and then walk on a horse walker for 4 to 6 weeks, followed by a month of walking and trotting exercise. Then ultrasonographic re-assessment is recommended to determine whether the horse should start alternate day cantering exercise or continue trotting for another month. Horses with distal branch lesions may have a guarded prognosis if substantial sesamoiditis exists. Most of these horses are taken out of training altogether as 2-year-olds and given the same rest program as horses with PSD. At the end of the period of controlled exercise, the horse usually is turned out and brought back in at the end of the year for further education, before resuming training as a 3-year-old.

Most young racehorses with PSD have an increase in size of the proximal SL and irregularity of contour, with bulging of the normally flat palmar border, detectable by ultrasonography. The fiber pattern may be heterogeneous. Ultrasonographic monitoring of these lesions may reveal a decrease in size of the SL, with more linear borders. However, in many horses the SL never returns to normal. Exercise level is increased when the ligament shows good echogenicity, although it still may be enlarged. Some horses seem to have long-term hypoechoic lesions within the SL that do not change with further rest.

Less commonly, a unilateral, acute, focal core lesion occurs in the SL after a race or gallop associated with significant lameness, swelling in the subcarpal region and an exquisite pain response to palpation.

The prognosis for horses with mild to moderate PSD in 2-year-old TBs is good, but in horses with acute core injuries the prognosis is more guarded. Some of these horses take 4 to 5 months before they are able to withstand training and have a risk of re-injury. The prognosis in older horses is also less good.

In a 3-year-old horse that had PSD at 2 years of age, mild lameness may recur as training speed increases, in the absence of any real changes that can be identified using ultrasonography, radiography, or scintigraphy. This lameness always can be abolished by subcarpal analgesia. These horses with subcarpal ache often can be managed by administration of 5 to 10 mg of triamcinolone acetonide in 3 ml of local anesthetic solution, injected in the same site as a subcarpal block. Lameness often is abolished for long periods after this treatment, without apparent degeneration of the SL, even with continued training. It is vital to complete a full diagnostic evaluation before treatment in these horses to eliminate the possibility of training horses tolerating incipient catastrophic lesions under the influence of corticosteroids.

Lameness Associated with the Middle Carpal Joint

Several distinct syndromes appear to be associated with the middle carpal joint. One of the most common syndromes is exemplified by a 2-year-old in early training that develops an increasingly short, choppy gait at the trot. The limbs are held abducted during the protraction phase, and the carpi are barely flexed. This gives a stiff, rolling action. Often the horse has bilateral middle carpal joint effusion and pain on carpal flexion. Injection of local anesthetic solution into one middle carpal joint produces lameness (grade 1 to 2 of 5) in the contralateral limb. The gait changes so that the desensitized limb often is brought back toward the midline. Some of these horses have mild sclerosis of the third carpal bone, seen on a skyline projection, but others have no detectable radiographic abnormality. These horses probably are suffering from pain produced by concussion of immature cartilage and excessive torsion on untrained ligaments within the carpus, and should be given rest. If excessive effusion occurs, the joint may be drained and medicated with triamcinolone acetonide (5 mg) and hyaluronan, but only if rest is to follow. Non-steroidal anti-inflammatory drugs (NSAIDs) may be administered for the first 4 or 5 days of rest. Most horses are restricted to box rest for 1 month, followed by walking exercise for 2 weeks, followed by a month of trotting, before resuming full training. Many horses never show lameness again during the second introduction to exercise loading.

The second syndrome, which may include some horses in the first category if training has continued, involves development of more severe sclerosis in the third carpal bone. Focal overloading of the third carpal bone results from a conformational defect (offset carpi with toe in is the worst) or from a mismatch between loading of the limb and time allowed for adaptation. These horses are usually bilaterally lame; often one lame limb dominates, and contralateral limb lameness is only recognized when the lamest limb is blocked. Radiographic signs include sclerosis in the radial facet of the third carpal bone seen on the 55° flexed skyline projection, associated with local areas of rarefaction around the nutrient foramina or with small comma-shaped fissure fractures extending from the dorsal cortex. Further training of these horses risks development of a full sagittal fracture (see Fig. 108-2).

Despite statements to the contrary in the literature, we believe that the early sclerotic changes in the third carpal bone are reversible after a period of box rest. However, there seems to be a certain degree of pathological damage from which there is no return. Once the radial facet becomes completely devoid of trabecular detail and has a ground glass

appearance on radiographs, the changes are often permanent. These lesions often are associated with recurrent lameness each time the horse reaches racing speed. Identifying sclerosis of the third carpal bone at an early stage is therefore vital. Screening radiographic examinations of the third carpal bone in a 2-year-old may be useful before starting full race speed galloping. Without radiographic information, lameness may be the first indicator of sclerosis, which may already be advanced.

Lame horses with third carpal bone sclerosis are confined to box rest for 2 months followed by a month of walking exercise. Radiographic examination is then repeated. We advise that these horses are not trained with a view to racing at 2 years of age, although they may re-enter the training environment so that they acclimatize to respiratory disease and other management problems that might influence the 3-year-old career.

Problems related to the distal radial carpal bone are also common, associated with lameness and distention of the middle carpal joint capsule. Radiographic abnormalities include spur formation on the distal aspect of the radial carpal bone and radiolucent changes in the same site leading to modeling of the dorsal margin. This usually leads to secondary sclerosis of the opposing third carpal bone, because the load becomes borne by the medullary bone, instead of the weight-bearing pillar of the dorsal cortex. These radiographic abnormalities result from overloading the carpus, leading to bone stiffening, cartilage damage, and secondary osteoarthritis. Debate exists over whether spurs should be removed surgically. Osteoarthritis of the middle carpal joint is already established. Although removing the spur is logical, it is not curative, and the joint still will require treatment the following season. With cheap horses and in yards where money is tight, many horses with mild carpal osteoarthritis can be treated once by intra-articular medication with hyaluronan and triamcinolone acetonide, followed by 4 to 6 weeks of rest and a graded return to exercise.

If a chip fracture occurs, then removal of the fragments is probably still mandatory. One should explain to the owner and trainer, however, that the horse has pre-existing osteoarthritis that will persist after surgery and that further problems should be anticipated the following season.

The antebrachiocarpal joint is more forgiving than the middle carpal joint in the amount of pathological change that can be seen radiographically in the absence of clinical signs. Large fragments can be removed surgically with a favorable prognosis for the following season.

A small group of horses with middle carpal joint pain remains in which lameness recurs after periods of rest, despite absence of radiological changes. These horses often have unilateral lameness associated with middle carpal joint effusion. Some of these horses have tears of the palmar intercarpal ligaments. Response to conservative treatment often is disappointing and, even after prolonged periods of rest, lameness recurs as soon as work intensity increases. I normally medicate these carpi with hyaluronan and corticosteroids, but in the absence of a firm diagnosis, this empirical treatment is illogical and usually unrewarding. Diagnostic arthroscopy probably is always indicated in these horses but is also often unhelpful in producing a permanent cure.

Subchondral Bone Injuries to the Distal Third Metacarpal/Metatarsal Bone

Subchondral bone injury is an extremely common cause of lameness in young TBs and is vastly underacknowledged. Subchondral bone pain seems to be increasingly implicated in much joint-related lameness in other classes of horses (e.g., navicular syndrome, tarsal pain, and third carpal bone pain). In the young TB subchondral bone injuries in the distal MtIII and McIII often are linked to bilateral or quadrilateral lameness, which limits performance. Horses with bilateral subchondral bone injuries of the distal aspect of MtIII fail to push from behind at exercise. Often these horses appear to bunny hop for the first section of canter. The rider reports that the horse feels wrong behind and does not perform as well as it previously did. With American-bred horses equine protozoal myelitis (EPM) is an important differential diagnosis, because the clinical appearance can be similar. Onset of clinical signs can be delayed, so that a yearling bred in America and sold in Europe could have EPM. The clinical history (see page 882) and response to diagnostic analgesia (see page 885) have been described.

Subchondral bone injuries are extremely difficult or impossible to identify radiographically. If radiographic lesions are visible, these are usually end-stage and warrant a guarded prognosis (Fig. 108-5). These lesions usually represent necrosis and collapse of subchondral bone and are visible as a crescent-shaped lucency in the middle of the lateral condyle, seen on the flexed plantarodorsal oblique or flexed dorsal distal 125°–palmar proximal oblique projections (see Fig. 108-4, *B*). Sclerosis of the lateral condyle also occurs and is most visible on the lateromedial view. Deliberate over-exposure may be necessary to demonstrate a triangular sclerotic region just palmar to the mid-point of the articular surface (Fig. 108-6). All racing TBs seem to develop some degree of sclerosis in this site as a normal physiological response to loading from the PSB as the limb assumes full extension.

On scintigraphic examination these stress injuries are remarkable because increased radiopharmaceutical uptake (IRU) is focal in nature and markedly increased. On a plantar view, IRU in MtIII is mistaken easily for IRU in a PSB, because the shape of the lesion is similar to that bone. However, on a lateromedial view, the PSBs can be seen clearly plantar to the region of the IRU, localized within the condyle itself (Fig. 108-7). In the forelimb, subchondral bone injuries in the metacarpophalangeal joint seem to occur in the medial or lateral condyle or both. Horses usually have bilateral lameness and trot with the head held low with a short, stumbling gait as they try to get off each weight-bearing limb as fast as possible. If all fetlock joints are injured, a horse may refuse to trot and attempts to break into a slow, rolling canter as speed

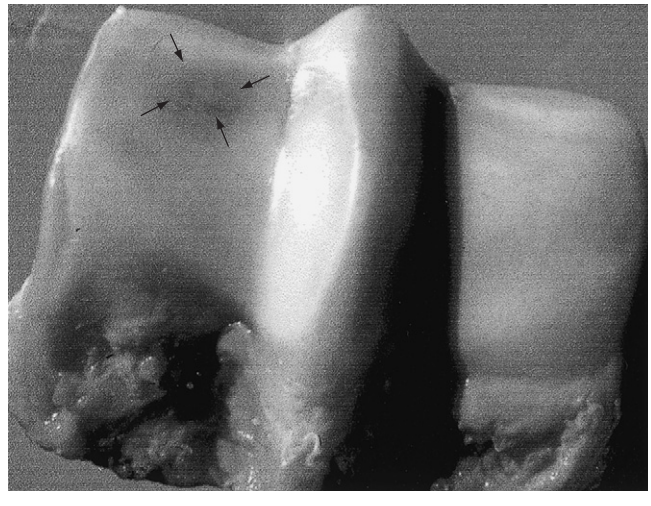

Fig. 108-5 Post-mortem specimen of the distal aspect of a third metatarsal bone from a horse with plantarolateral focal increased radiopharmaceutical uptake seen on a bone scan. Lateral is to the left. The specimen was collected when the horse was euthanized for unrelated reasons. The area in the center of the plantarolateral aspect of the condyle is dark colored, and the cartilage surface is irregular *(arrows)*. Histopathological sections cut through the site revealed abnormal irregular subchondral bone.

increases. Lameness often becomes apparent in the contralateral limb after local analgesia of one metacarpophalangeal joint. Radiographic examination is often unremarkable, but scintigraphy confirms significant focal bone activity.

Treatment is difficult, and we have no protocol that results in uniform resolution of lameness. Many different treatment regimens have been used, including intra-articular injection with hyaluronan or corticosteroids, systemic medication with isoxsuprine and aspirin and with polysulfated glycosaminoglycans, and rest alone. Often the lameness disappears rapidly after the instigation of rest but returns when the horse resumes fast galloping speed. Although some horses have been able to perform without further problems, predicting which horses may respond favorably is not possible, although those with subchondral lucency and sclerosis rarely remain sound. This does not mean that these horses cannot perform, and many racehorses seem to withstand training despite lameness. However, this may be a performance-limiting problem that directly interferes with racing ability and determines the level at which the horse can compete.

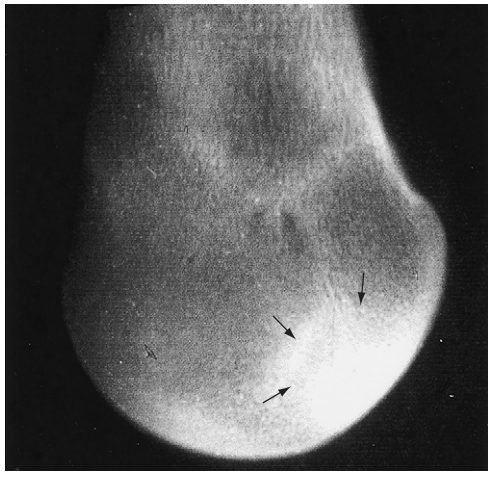

Fig. 108-6 Post-mortem lateromedial radiographic view of a thin bone section of the third metatarsal bone cut through the area shown in Fig. 108-5. There is intense sclerosis of the bone *(arrows)* adjacent to the site of the injury in the condyle. This change sometimes can be seen in radiographs of horses affected with condylar stress injuries.

Because horses with advanced lesions rarely return to sustained soundness, it is advisable to stop hard training as soon as the condition is noted in a 2-year-old and allow 3 months of rest. This is not a guarantee that the problem will not recur at 3 years of age, but going on with training these young horses in the face of this lameness seems illogical. Many horses can be trained at 3 years of age without problems. When the clinical syndrome appears in a horse of 3 or 4 years of age for the first time, the prognosis is much worse than when subchondral bone lesions occur in a 2-year-old and the horse is allowed to rest. In an older horse end-stage pathological damage in the affected condyle is more common. These horses may well have been trained through the problem at 2 years of age, with a low-grade bilateral lameness that was not recognized.

Some horses first show hindlimb lameness associated with the lesions in the lateral condyles of the MtIII after a race on unsuitably firm ground. They are described as jarred up. Although clinical appearance of a jarred-up horse has long

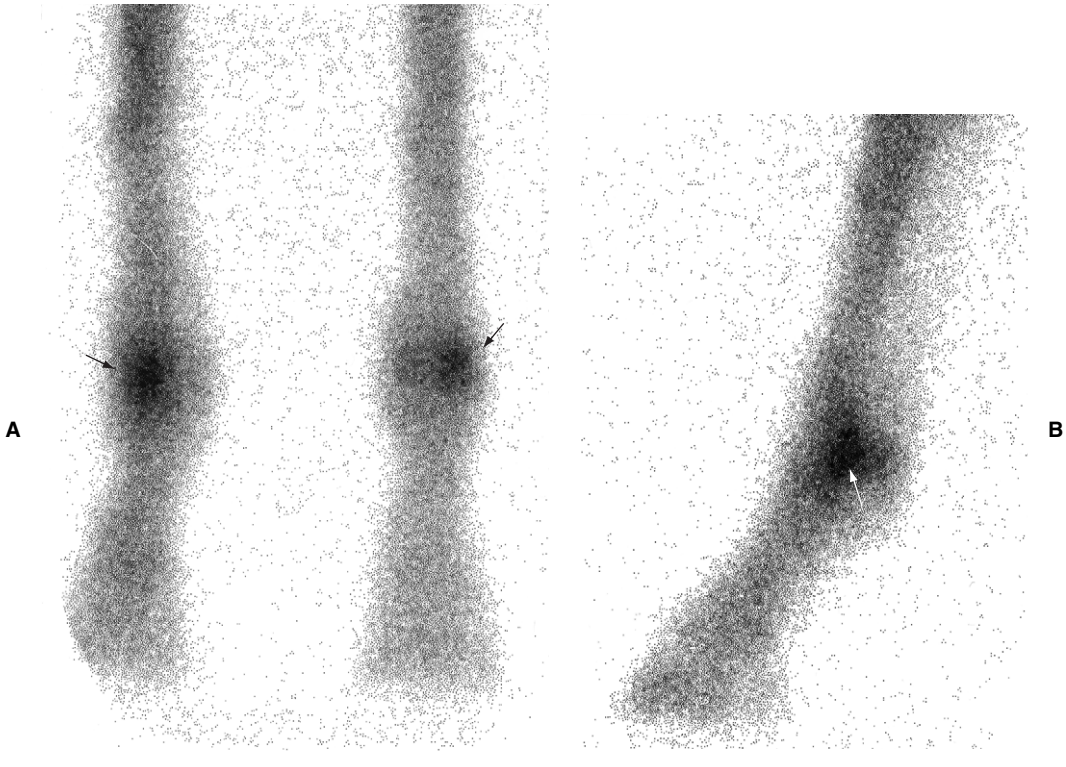

Fig. 108-7 **A,** Plantar delayed (bone) phase scintigraphic image of horse with bilateral stress injuries to the lateral condyle of the third metatarsal bone. The shape and distribution of the increased radiopharmaceutical uptake *(arrows)* easily can be mistaken for the proximal sesamoid bone. **B,** Lateral delayed (bone) phase scintigraphic image of the same horse clearly shows the increased radiopharmaceutical uptake to be situated in the plantar aspect of the condyle *(arrow)* rather than the proximal sesamoid bones, which are in a more plantar position.

been recognized, no precise definition of the syndrome has ever been made. The syndrome may well represent multifocal subchondral bone pain. Affected horses may never regain normal action. The reasons why some horses become progressively more lame while others can race with success and maintain a steady-state lameness are not understood.

A few horses with subchondral bone injury have been examined at postmortem (see Figs. 108-5 and 108-6). The degree of damage to the subchondral bone at the injury site easily explains why some horses are untrainable. We need to understand why some horses develop these lesions while others do not, whether the type of training surface may be a predisposing factor, what pathological mechanisms are involved, and why these lesions produce pain.

Lameness Subsequent to Bacterial Infection

Bacterial infection in a limb of a horse can cause a degree of pain greater than almost any other injury, including a displaced fracture. Although wounds in any part of a limb can become infected, leading to an obvious swelling and lymphangitis, some discrete syndromes in the racehorse deserve mention.

Staphylococcal Abscesses on the Palmar Aspect of the Metacarpophalangeal Joint

Horses exercising on sand or wood chip surfaces seem to be prone to bacterial infections around the ergot that produce small, focal abscesses, which can be difficult to find if the horse has a winter coat. The horse often initially points the limb, is severely lame (grade 4 of 5), with swelling on the palmar aspect of the metacarpophalangeal joint. Manipulation of the joint is resented. The abscess can mimic the appearance of an infected joint and must be differentiated, because synoviocentesis inadvertently may introduce infection into the joint. Careful evaluation shows that the metacarpophalangeal joint capsule is not distended. By working through the hair coat with the fingernails, localizing the point of a small abscess is often possible. The horse shows a severe pain response, and a pink pore-like hole may be observed discharging serosanguineous pus. Bacteriological culture almost invariably results in the growth of a hemolytic *Staphylococcus aureus*.

Treatment is by external application of kaolin paste or a similar open poultice, combined with systemic antibiotic therapy along with NSAIDs and forced walking exercise. Horses are normally sound after 12 to 24 hours, and if this is not the case, then the diagnosis should be re-established. Bandaging should be avoided because the limb often continues to swell and the bandage may become constricting, which can lead to ischemic necrosis and sloughing of the skin.

Focal Peritarsal Cellulitis

Focal peritarsal cellulitis is a syndrome in the hindlimb that appears to be related to the architecture of lymphatic drainage and is characterized by acute-onset severe lameness (grade 4 or 5 of 5), often with a shock-like state, hyperventilation, and sweating. Rectal temperature is elevated (39° to 40° C). Clinical examination of the limb reveals an exquisitely painful area on the medial aspect of the hock, with focal swelling (Fig. 108-8). Brushing this area lightly with the fingertips usually causes the horse to stagger sideways and abduct the limb sharply to get away from the pain. Almost always a small skin lesion of some sort is evident in the distal part of the limb, through which the initial infection may have entered.

Although the horse is severely lame in the stable, if forced to walk, the lameness usually improves after a few steps and the horse bears weight reasonably well. The focal swelling on the medial aspect of the tarsus appears to be linked to a nidus of lymphatic drainage because the swelling is so consistently positioned. The pain is so great because of involvement of the retinacular ligaments immediately beneath the site, causing a compartment-like syndrome. Some clinicians from North America describe emergency surgical drainage of these abscesses. However, conservative treatment using antibiotics (oxytetracyclines), along with NSAIDs administered morning and night, and forced exercise several times daily is usually adequate.

Often within 24 hours the swelling changes from a small egg-sized swelling on the medial aspect of the hock to involve the entire distal part of the limb. Swelling gradually resolves with exercise over the next month.

A great risk of laminitis in the contralateral limb exists in the early stages, because of the combination of severe lameness in the affected limb and toxin production from the abscess. It is therefore vital that the horse is bedded on deep bedding during its initial treatment and that regular exercise is encouraged in spite of obvious lameness in the affected limb.

Axillary and Scrotal Intertrigone Infections

Horses are affected by skinfold infections, particularly in the axilla and groin. The infection begins in the fold of skin and causes an exudative, purulent dermatitis. The swelling of the affected region results in more occlusion and development of anaerobic infections deep in the skin folds. Affected horses often stand with the limb abducted and appear as if they have a proximal long bone fracture. Because the swollen skin folds close themselves off, these lesions can be missed unless the limb is pulled away from the body to expose the raw pink exudative dermatitis. Treatment is by systemic administration of antibiotics and analgesics and topical cleansing of the affected area with a debriding agent, followed by application of an antibiotic ointment. Fusidic acid creams are often effective because most of these infections involve staphylococci. Topical treatment often requires profound sedation of the horse so as to be carried out safely, because of the pain involved. Exercise should be limited to a short period of hand

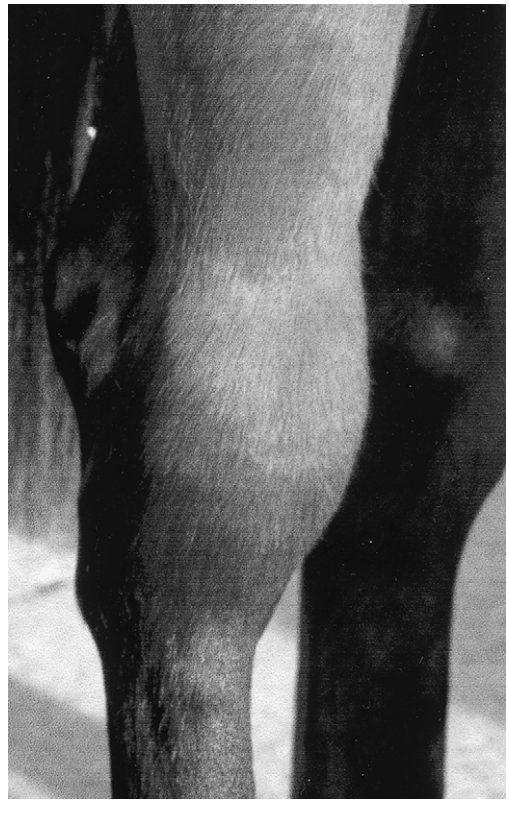

Fig. 108-8 A horse with focal peritarsal cellulitis. There is tight focal distention caused by swelling beneath the skin, retained at either extremity of the tarsus by the retinacular ligaments.

walking, until the edema of the skin folds of the axilla subsides, because walking produces friction that makes the condition worse. Less commonly the area between the scrotum and the medial aspect of the thigh can be involved, especially in horses exercising on dirt or sand. Affected horses walk in a crouching gait, with abduction of each hindlimb during protraction.

Stress Fractures of the Long Bones and Pelvis

Post-mortem studies carried out on racehorse fatalities in California have revealed that horses have evidence of previously undetected stress fractures of the long bones. We have been aware clinically of these fractures for some time, particularly when they became complete and displaced in horses in training. With the advent of scintigraphy, the early diagnosis of these stress fractures has been increased.

Certain rules apply to all stress fractures in the TB. Fractures usually occur after repeated cyclical loading of bone over time rather than as an acute incident. Lameness itself may be acute in onset, as the bone reaches the stage where it can no longer bear weight and the cortex begins to collapse. However, because the lesions are chronic, periosteal new bone formation, irregular ill-defined lucencies in the cortex, and occasionally cortical displacement may be detectable radiographically. Because stress fractures result from repeated focal over-loading, they tend to occur in predilection sites in the bone, reflecting the architecture of that bone and the manner in which weight is borne during locomotion. The repeatability of these sites makes looking for these injures on radiographs straightforward.

Skeletal bone seems to detrain almost as effectively as it can be trained. For this reason any horse that has been removed from training for longer than 3 weeks should be given sufficient time to allow reconditioning of the skeleton on return to training. Stress fractures may occur in horses of any age if they have been removed from training for a period of more than a month and have been returned to cantering exercise too rapidly.

Periostitis of the Dorsal Cortex of the Third Metacarpal Bone (Sore or Bucked Shins)

Sore shins are a common problem in 2-year-old racehorses or in older horses that have not reached race speed previously or have had a long layoff. Most of these horses are not shown to the veterinary surgeon. The stable staff and trainers know how to recognize sore shins and treat them in a variety of traditional methods. It is advisable to restrict the horse to trotting until the area is no longer painful to light fingertip pressure with the limb flexed. The horse is then re-introduced to the exercise level one step beneath the level that produced the soreness. This speed is maintained for 1 month before increasing. If the shin soreness returns as speed increases, the cycle is repeated.

Rarely, more invasive therapy is required for horses that have three serial episodes and for which normal management strategies have failed. The shin is pin fired under local analgesia, and the horse is walked for 2 weeks and then trotted for 2 weeks, before gradually increasing speed work. Freeze-firing is a local temporary neurectomy only and confers no long-term benefit. The shin is a barometer of what is going on in the rest of the skeleton, and when sore the shin indicates that the skeleton needs more time to adapt. Freeze-firing simply lets us ignore this and allows the horse to develop real damage elsewhere. This subject is controversial, and the views expressed here are not shared by all veterinarians.

Dorsal Aspect of the Third Metacarpal Bone

Fracture of the dorsal aspect of the third metacarpal bone presents as an extension of the sore shin complex. When a true stress fracture (dorsal cortical fracture) is present, the pain to palpation usually is localized to one area rather than being distributed across the entire surface of the shin. Normally a periosteal callus is present at the site of the fracture, causing a bump on the surface of McIII. Radiographic examination should include unorthodox lesion-oriented oblique projections because the fracture lines are often extremely difficult to see, particularly in the acute phase of the injury. Horses are usually slightly lame, but continued training results in a significant increase in lameness after each exercise period, which reduces rapidly. A lame horse with focal periostitis that is painful to palpation should be considered to have a stress fracture in that site, even if the fracture is not visible radiographically. Treatment is by rest, which can include walking exercise. These injuries normally take about a month to heal, after which light training may begin, but 3 months should elapse before full training speeds are achieved. If the injury has progressed to a clear tangential fracture line in the cortex, some veterinarians recommend surgical drilling (osteostixis), or even screw insertion across the fracture line, to encourage healing. This has never been necessary in my experience. Extracorporeal shock wave therapy also is being used to treat this type of fracture, but its clinical efficacy remains to be proved. Normally early diagnosis allows full healing with conservative treatment alone.

Tibia

Stress fractures of the tibia are one of the most common causes of hindlimb lameness in 2-year-old racehorses in race training. In the seasonal racing calendar, when yearlings enter training in October or November, peak incidence of tibial stress fractures is in April or May as speed work increases. The tibia has three main predilection sites. The most common, in naive horses entering training for the first time, is the proximolateral cortex, 7 to 8 cm distal to the proximal articular surface of the tibia. Stress fractures in this site are visible on radiographs as poorly defined callus, often associated with an oblique fracture line in the cortex itself. The second common predilection site, often seen in a 3-year-old or in a 2-year-old in more advanced training, is in the caudal distal cortex, 10 cm proximal to the tarsocrural joint. On radiographic examination endosteal callus can be seen as a subtle sunburst of increased radiopacity extending forward from the caudal cortex on a lateromedial projection. On a caudocranial projection a lucent line is usually visible, surrounded by an area of sclerosis in the middle of the distal tibia. The third common site is the middle of the medial cortex. In this site the injuries usually are seen as periosteal and endosteal new bone formation only, and rarely are fracture lines visible.

Occasionally a complete, comminuted displaced fracture of the tibia can occur during racing or training. The prognosis in these horses usually is regarded as hopeless. In this practice complete tibial fractures occurring at exercise have almost disappeared because of the advent of scintigraphy, suggesting that in the past some complete fractures were the end stage of chronic stress fractures, which are now being detected early.

Diagnosis of tibial stress fracture is difficult because these injuries are not affected by diagnostic analgesia. The typical history is of a 2-year-old being brought into faster speed exercise or of a 3-year-old that did not race at 2 years of age being prepared in the same way. The lameness is usually acute in onset and can be severe (grade 3 or 4 of 5) but usually diminishes rapidly with rest. Forced flexion of the hock and torsion of the tibia produces pain in horses with severe lameness (see Fig. 108-3). Scintigraphy is ideal to confirm these lesions, but the degree of IRU seen in the mid-shaft and distal shaft sites is often low.

Treatment is by rest and a gradual return to training. The horse is restricted to box rest initially for 2 to 6 weeks depending on the severity of the radiographic signs. Occasionally, stress fractures of the tibia do become complete and displaced during periods of convalescence after normal recumbency. The horse should be tied up by the head for the first 3 to 4 weeks if the weight bearing ability of the tibia is severely

compromised (for precautions, see Chapter 51). Less severely affected horses are exercised as soon as it is practical. If a treadmill or horse walker is available, horses can be walked as soon as clinically sound, allowing a total healing period of between 2 and 3 months. Trotting exercise can be included in the second and third months.

Humerus

A horse with a humeral stress fracture has an acute-onset forelimb lameness with features of proximal forelimb pain. The horse walks with a short cranial phase to the stride and circumducts the limb in protraction. Substantial weight-bearing lameness is evident at the trot. Horses often drag the toe of the affected limb in the bedding during protraction in the stable. The lameness rapidly diminishes, and in 2 days horses may be sound, only to go severely lame again after return to exercise.

Two main predilection sites exist for humeral stress fractures. One site is on the caudal cortex just distal to the humeral head, about 5 cm distal to the weight-bearing surface. Loose periosteal callus usually is visible, with sclerosis of adjacent bone and occasionally incomplete tangential fracture lines. The other site is the cranial cortex of the distal humerus about 5 cm proximal to the elbow joint surface. A focal sunburst of callus usually is visible on a mediolateral radiographic view, and a linear fracture line is sometimes seen. Treatment is as described for tibial stress fractures. Complete, displaced humeral fractures can occur in exercise and horses have a hopeless prognosis.

Pelvis and Axial Skeleton

Fractures of the sacral wing of the ilium are often stress fractures and are described in detail elsewhere (see Chapter 51). Recently laminar stress fractures of the vertebrae immediately adjacent to the pelvis have been described.[13] There is usually a unilateral lameness for which no other reason can be ascribed after routine diagnostic analgesia and survey radiography. Scintigraphic examination of these horses reveals focal IRU associated with the lumbar vertebrae on the same side as the lameness. Some horses simply show bilateral poor hindlimb propulsion, short stepping at the walk, and a hunched-up lumbar spine, coupled with poor performance without an obvious lameness. Being certain that the laminar stress fracture is the cause of the reduced performance is difficult. However, if scintigraphy reveals an active bone injury in the spine, continuing full training cannot make sense, and these horses subsequently are allowed to rest. Although this lesion is described as widespread in the Californian pathological survey, the clinical presentation is uncommon. I have examined the lateral lumbar spine routinely in all bone scans over the past 2 years and have not encountered the lesion as a common incidental finding.

Exostosis of the Second and Fourth Metacarpal/Metatarsal Bones (Splints)

Splints are more of a nuisance in a racehorse than a major problem. They are clinically obvious and a sharp pain response can be elicited by squeezing the area. Splints normally are associated with lameness in the initial stages, although this can subside rapidly. Splint lameness is one of the few conditions with which lameness is often more obvious with the affected limb on the outside of a circle. Splints are common in horses of 2 years of age, but can occur in horses of any age, particularly if they experience a change in loading because of a different type of training surface or a different angle of camber. In many horses a fracture line is not detectable radiographically, but most horses with obvious exostosis formation associated with lameness probably do have fractures within the body of the bone, which often can be identified scintigraphically. For this reason a period of stable rest is probably indicated until the horse is clinically sound. The horse then can recommence trotting for 2 to 4 weeks to allow the injury to heal fully. If pain on palpation and lameness immediately recur when the horse returns to exercise, then pin-firing may be considered to encourage more aggressive callus formation. This is one of the few orthopedic indications in which firing achieves a good clinical result in most horses. Horses with displaced fractures of the distal aspect of the second and fourth metacarpal/metatarsal bones are treated by surgical removal of the distal portion of the bone.

Multiple comminuted fractures of the second and fourth metacarpal/metatarsal bones sometimes result from external trauma and heal surprisingly well. Many fragments may be identified by digital palpation with a gloved finger through an open wound and confirmed radiographically. Most horses with fractures heal satisfactorily with antibiotic treatment, bandaging of the wound, and stable rest. If infectious osteitis does occur with sequestrum formation and recurrent fistulation, then affected bone should be removed surgically.

Undiagnosed Hindlimb Lameness

Despite all our modern diagnostic aids, the cause of hindlimb lameness in a significant number of horses remains elusive. Given the huge muscle mass involved in the quarters of the horse, some horses must have muscular origin and insertion pathological conditions. In human athletes lameness is commonly linked to strains, tears, and cramps in the semimembranosus and semitendonosus muscle groups and in the adductor and gluteal muscles. Horses are presumably no different. As yet, no definitive test appears to be available to confirm genuine muscle injury. Thermography is helpful in some horses but is disappointing in others. Increases in CK and aspartate aminotransferase levels, although pathognomonic for exertional rhabdomyolysis, do not seem to occur following genuine tears of muscle tissue. Presumably these tears result in little cellular disruption, the damage being more in the fascial planes and connective tissue.

If a horse remains undiagnosed after all available diagnostic analgesia and imaging modalities have been used, then the horse is confined to box rest for 1 month. Most horses become sound in this period. The horse walks for another month and then returns to walking and trotting exercise, before resuming a normal ascending exercise program. Although admitting defeat and being unable to give a definitive diagnosis is frustrating, we should try to avoid attributing a definite cause for the lameness if we have no evidence to support it. We have to educate our trainers and clients to accept the fact that ascribing lameness to injury of a specific structure in every horse is not possible.

Fractures of the Proximal Phalanx and Condyles of the Distal Third Metacarpal/Metatarsal Bone

The most dramatic example of a proximal phalanx fracture is a split pastern that is a frequent injury, particularly in a 2-year-old racehorse as it enters fast work. The length and configuration of these fractures include complete sagittal fractures involving both articular surfaces, multiple comminuted displaced fractures, and incomplete fractures extending short distances from the proximal articular surface. Diagnosis is usually straightforward with a complete fracture or an extensive incomplete fracture. With extreme injury a horse pulls up severely lame during or at the end of exercise. With less severe injuries, horses may finish exercise apparently sound but become increasingly lame on return to the stable. The horse is usually not bearing weight by the time of examination. Pain always occurs on flexion of the metacarpophalangeal or metatarsophalangeal joint and on pressure applied with the fingertips to the proximal phalanx along its sagittal dorsal midline. Radiographic examination is usually confirmatory.

Short incomplete sagittal fractures of the proximal phalanx can be notoriously difficult to see radiographically, when the fracture line only extends 1 or 2 cm into the bone. Clinical signs are of a severe lameness (grade 3 of 5) that rapidly improves so that the horse may be sound within 2 days.

Flexion of the fetlock joint is usually not painful. Firm squeezing of the proximal dorsal surface of the proximal phalanx usually is resented, particularly approaching the proximal articular surface, and this is a reliable clinical sign. A horse with this type of fracture should be given box rest for at least 2 weeks before further radiography. Short, incomplete fractures easily become complete comminuted fractures if a horse is exercised after 2 weeks of rest, because the fracture weakens by normal osteoclastic activity. Therefore although the horse may be sound, a premature return to work may result in a catastrophic fracture.

Fractures of the condyles of McIII/MtIII are a frequent injury, but they occur more often in 3- and 4-year-olds than in 2-year-olds. The lateral or medial condyle can be affected, and medial MtIII fractures often spiral proximally. The whole fracture may not be seen on one radiographic view, and several oblique views should be obtained to determine the extent of the fracture before contemplating general anesthesia for surgical fixation. Spiral fractures present a risk during anesthetic recovery.

A complete, displaced condylar fracture presents little diagnostic challenge. The horse is extremely lame and resents palpation of the lateral or medial condyle. Flexion of the metacarpophalangeal/metatarsophalangeal joint invariably is associated with sharp pain, and joint capsule distention and effusion usually occurs because of hemorrhage. Short, incomplete condylar fractures are more difficult to diagnose. Lameness is usually severe (grade 2 or 3 of 5) and diminishes rapidly. Horses often become lame again as soon as they are returned to cantering exercise. Diagnostic analgesia localizes lameness to the metacarpophalangeal/metatarsophalangeal joint, but nothing may be seen radiographically. A flexed 125° dorsopalmar view is vital if these lesions are to be seen adequately.[3] If radiography is performed in the field, using portable equipment, then an upright flexed view that moves the PSBs proximally is useful to identify the fracture[4] (see Fig. 108-4, *A*).

If a condylar fissure is suspected, the horse should be restricted to walking and trotting exercise for 2 weeks before follow-up radiography is performed. Many horses that eventually develop full, displaced condylar fractures are described as having been bad movers before injury, and almost certainly significant prodromal pathological damage contributes to the cause of these fractures.

Pain Associated with the Tarsometatarsal Joint

Sore hocks are probably much more common in the United States than they are in the United Kingdom. Tarsometatarsal joint lameness can be bilateral or unilateral and often does not resolve with rest, and the response to intra-articular administration of corticosteroids varies extremely. I treat horses with triamcinolone acetonide (10 mg) at the time of intra-articular analgesia to avoid having to re-enter the joint. After this therapy, many horses are sound, and lameness never returns. However, other horses require intermittent repeated medication, and others will not respond at all.

Horses with bilateral lameness are more difficult to diagnose. Bilateral plantar stress injuries in the distal aspect of MtIII should be eliminated first by diagnostic analgesia. If diagnostic analgesia is negative, local analgesia of one tarsometatarsal joint should be performed in the hope that an obvious lameness will become visible in the contralateral limb. Care should be taken if medicating both tarsometatarsal joints to avoid a laminitis-inducing dose of corticosteroid (e.g., no more than 20 mg of triamcinolone acetonide per horse).

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CHAPTER • 109

The North American Standardbred

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DESCRIPTION OF THE SPORT

The Standardbred (STB) was developed as a racing breed by registering any horse that could trot or pace a mile in a set time, called the standard. An early standard was 2:30 (2 minutes, 30 seconds, 0 fifths). Early STBs were diverse because they were different types with a large genetic pool. Crossing many types resulted in a strong, rugged breed that lacked refinement of the head, ears, and bone structure. Trotters were large and coarse, whereas pacers were smaller and more refined. The breed has progressed rapidly by selective breeding in the last 3 decades. Although trotters and pacers now are bred separately, the physical appearance has become more alike, and both lines have a racy appearance similar to Thoroughbreds (TBs). Early trotters were farm or buggy horses, selected for good disposition and durability, traits that are still characteristic. Early STBs were raced several 1-mile races in the same day, called heats, a part of the sport demanding stamina and gameness.

A TB stallion, Messenger, imported from England in 1788, was prominent in the formative period of the STB. Many of his offspring had a natural inclination to trot. In 1849 a descendent of Messenger sired Hambletonian 10, the foundation sire of the STB breed. The breed developed in the northeastern United States, where it was influenced by the Hackney and Morgan breeds and local mixed-breed horses.

The STB races at two different gaits, the trot, a diagonal two-beat gait in which the left forelimb (LF) and right hindlimb (RH) move together, and the pace, a lateral two-beat gait in which the LF and LH move together. High knee action and a lateral (left to right) nodding of the head are typical of a trotter (Fig. 109-1, *A*). A trotter travels straight ahead (Figs. 109-1, *A*; 109-2, *A*; and 109-3), but a pacer sways from side to side while moving forward (see Fig. 109-1, *B*).

Thirty years ago trainers had to spend several months determining at which gait a STB was more talented; some could not handle speed at either gait. As late as the 1970s and 1980s, for a horse to race one season at one gait and the next season at the other was not unusual. A few horses raced at different gaits weekly, but today most STBs race at only one gait. Many foals pace or trot naturally. Although while racing, trotters trot, when they are lame, they occasionally break into a pace. Pacers often jog, the term used for trotters and pacers when trotting the slow or wrong way (clockwise) of the track at the trot, but train and race at the pace. The pace is faster than the trot. The world record for the pace during a parimutuel race is 1:47.2 and that for the trot is 1:50.4. Many pacers race near record time, but trotters do not. Trotters and pacers do not race against each other in parimutuel races. A STB racehorse must qualify for racing, meaning the horse must better or match a minimal speed before being entered into a parimutuel race. Another form of speed test is called a time trial. At certain 1-mile long racetracks, horses compete against a prompter, usually a TB in harness, and go as fast as possible, but the prompter cannot pass the STB. The world record for the time trial is 1:46 set by a pacer. Mean race times of STBs are decreasing constantly (the horses are becoming faster) because of selective breeding, changes in track surfaces and banking, popularity of 1-mile racetracks (racing is faster because horses only negotiate two turns), refinement in race bikes (sulky), and advances in veterinary care.

Early trotters were raced under saddle and then with carts and wagons, with a time under 3 minutes being noteworthy. Sulkies appeared regularly in races in the 1840s, and the 2:30 barrier fell in 1845, becoming a time standard for the era. Race times fell steadily, and in 1897 a stallion named Star Pointer paced a mile in under 2 minutes. The first horse to trot the 2-minute mile was Lou Dillon in 1903.

North American STBs start a race behind a moveable starting gate, a vehicle carrying a barrier to keep the horses in line until the gate pulls away, at which time the horses accelerate to start the race evenly. In some countries horses start from standing, a staggered standing start, or a moving start without a gait. In Europe, races are only for trotters, and top 3-year-old and older North American trotters often are sold to European buyers.

STBs in North America are divided into four classes. The first is the International trotter that races in the top competitions in North America and Europe. The second is the Grand Circuit or stakes horse that travels from track to track, racing against the top 2- and 3-year-old horses. These horses may earn more than a million dollars a year. The third class includes the greatest number of horses stabled at or near a parimutuel track. These horses race repeatedly at one or two different tracks and are referred to as overnight horses. Overnight horses are raced in condition races, grouping similar quality horses by earnings, age, or sex, or in claiming races, where horses are grouped by price. The fourth class, county fair racehorses, popular mostly in the northeast and midwest United States, race for small purses at county fairgrounds, often under adverse track conditions, but they provide thrills for fans and owners beyond those of races with richer purses.

Amateur STB driving and racing under saddle are hobby type of events that are increasing in popularity. STBs compete at horse shows in the Roadster, hitched to a bike or wagon, and also as road horses under saddle. Former STB racehorses are not only popular in the Amish community, but they also can be found performing diverse sporting activities such as low-level dressage and jumping.

TRAINING

A classically trained STB has about 9 months of schooling and training before its first race. Most horses start training in the fall at 1½ years of age and begin racing the next summer. Many states have racing programs for 2-, 3-, and 4-year-old horses and require a horse to be nominated early in life. Eligibility then is maintained by periodic payments, called stake payments, creating a large pool of money divided among the winners of elimination and final events. Although some stake races receive track or corporate purse assistance, owners who put up their own money support most races. Stake races

are limited by age and sex. For example, 2-year-old trotting fillies race together. Stake payments must be made early in the spring, before talent and speed of individual horses can be confirmed, and owners, trainers, and veterinarians must decide which horses to stake. Emphasis on racing young STBs and the money necessary to maintain stakes eligibility place extreme pressure on trainers to race horses at 2 and 3 years of age. Horses often are pushed farther and faster than is reasonable, placing them at risk of stress-related bone injury, usually of subchondral bone. Many owners do not realize the difficulty of having a horse compete based on a calendar of scheduled events, expecting the horse to peak for the biggest purses, rather than on the horse's training and fitness level. The process of nominating and planning for stakes races is an art and science, and often a staking service is hired to organize periodic stake payments. Stake payments are a considerable expense and some owners spend several hundred thousand dollars a year. Horses not nominated, those dropped from stakes programs because of injury or lack of talent, or those in which payments were missed are still eligible for overnight racing. More and more late closer and entry-only stakes are becoming available for non-staked horses.

An STB is trained in a jog cart that is stronger, longer, and about three times as heavy as the 14-kg sulky or race bike (Fig. 109-1, *A*). A young pacer is soon equipped with hobbles, leather or plastic straps encircling the ipsilateral forelimbs and hindlimbs, to keep legs moving in unison (see Fig. 109-1, *B*). A horse capable of sustaining a pace without hobbles is called a free-legged pacer, but few race without hobbles. Hobbles can cause areas of hair loss on the cranial forearm and caudal crus called hobble burns, which are typical marks of a pacer.

Equipment is important in understanding gait and lameness. Head poles, boots, brace bandages, gaiting straps, and gaiting poles are used to improve gait and performance. A head pole keeps the horse from turning the head and neck to the opposite side. A horse with right forelimb lameness usually bears left, called bearing in or on the right line, because the driver pulls on the right line, causing the horse to turn the head to the right. This horse would wear a left head pole. Gaiting straps and poles run alongside the horse and attach to the cart beside the driver's seat and are used to keep the horse straight. Horses with unilateral hindlimb lameness often carry the hindquarters near the contralateral shaft, called being on the shaft. A horse with right hindlimb lameness goes on the left shaft and requires a left gaiting pole or strap. Boots are applied to minimize interference injury. Some horses chronically interfere, whereas interference in others is accidental. Soft brace bandages are used for protection and to widen the hindlimb gait.

Training the Young Standardbred

STBs are usually exercised 6 days a week at slow speed, jogging. Yearling STBs are introduced to the harness and bridle in the stall and then are line driven at a walk for several days before being hitched and jogged on the track. Once the horses can go to the track with a single driver, trainer, or groom, they are jogged 1 to 2 miles each morning, usually in a clockwise direction (wrong way) first. Young STBs are encouraged to jog at the intended gait as soon as possible. This is usually natural for trotters, but many pacers need hobbles or special shoeing. Weight usually is added to hind shoes of pacers, especially to the outside web, while the front feet may be left unshod. In trotters, weight is added to the front shoes. Shoeing and equipment changes begin early to produce the best gait possible.

Jogging is increased a mile every few weeks, up to 4 to 5 miles daily. Horses are taught to jog both ways on the track. Most young horses are jogged in groups, so horses overcome fear of traveling in close proximity. In large stables horses are trained in sets of three to five, to teach passing and racing in tight quarters, and to simulate racing. In addition to jogging, horses are trained fast every 3 to 4 days initially, but later twice weekly, going counterclockwise, the race or right way on the track. Horses are trained 2 and sometimes 3 fast miles on training days, but usually go back to the barn for a breather (to blow out) between fast miles (trips). STBs develop tremendous stamina compared with TBs, but fatigue and strain cycles predispose them to stress-related injury of the subchondral bone, not commonly of long bones as in TBs. When the fastest miles approach racing speed, young horses are ready for baby or schooling races to learn how to leave the starting gait. It is interesting that most STBs are trained, schooled, and qualified in the morning daylight and then make the first parimutuel start at night under the lights.

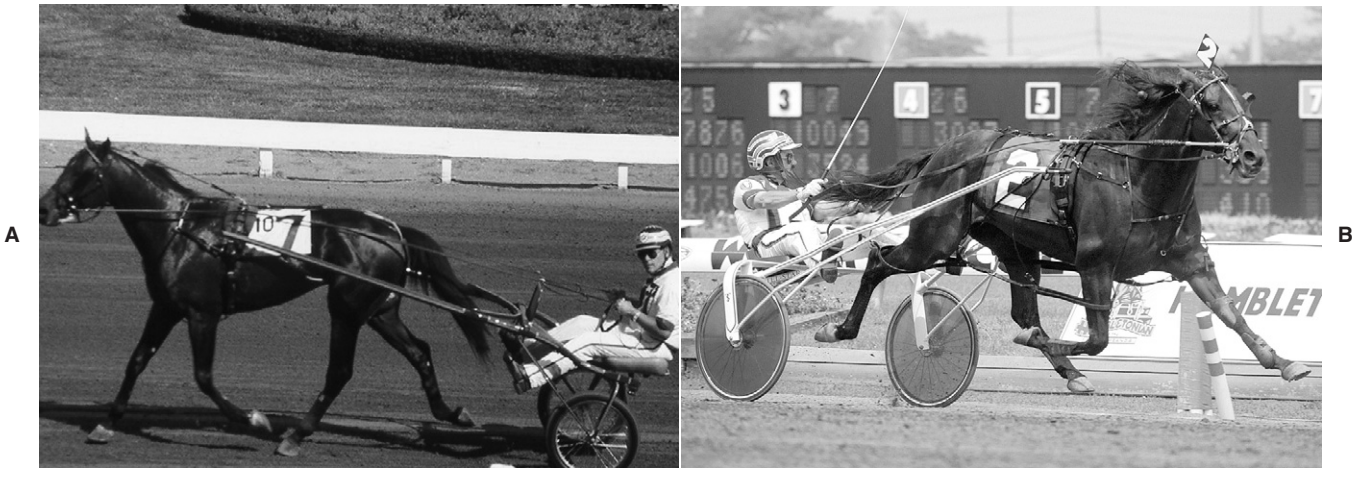

Fig. 109-1 The jog cart (**A**) is longer, stronger, and heavier than the race bike (**B**) and is used for training and jogging. **A,** Victory Dream is being warmed up by trainer Ron Gurfein in a jog cart. **B,** John Campbell and Real Desire on Hambletonian Day at the Meadlowlands (2002). The race bike is shorter and lighter weight than the cart. Note this pacer is in suspension, when all four limbs are off the ground. (Courtesy United States Trotting Association, Columbus, Ohio.)

Retraining a Racehorse

The classic retraining approach involves jogging 2 to 5 miles a day and additional training 1 to 2 times a week. A fast workout usually consists of two to three fast trips, which are simulated race miles taken at increasing speed. Horses usually are allowed to blow out between trips. Some are given double headers, two 1-mile workouts without returning to the barn, but they walk or jog slowly between trips. A break is when a horse leaves the expected gait and breaks into a gallop. Many training miles are given to obtain maximum physical fitness and to condition the horse to travel at speed without breaking. Many training miles are considered necessary to evaluate and adjust equipment and shoeing. Diagnostic analgesia can be performed between trips.

Alternative training methods produce good results and are customized to the disposition and physical condition of the horse. Trainers now use interval training, straight tracks, inclines and hills, swimming, towing behind a vehicle, and mechanical walker or joggers. With relocation of European trainers to North America, more alternative techniques have been introduced, including riding trotters between races.

Once racing, work is customized to each horse's needs. Hard training using the classic training method consists of one race and two training trips a week, but in this system horses are under considerable stress. Now that the breed is refined and heat races are fewer, racing horses are given much less training between races, and fast work may not be given at all. Reducing exercise intensity between races is important in managing STBs with chronic lameness.

LAMENESS AND POOR RACING PERFORMANCE

Lameness is the leading cause of poor performance and a major cause of STBs making breaks. Some overnight and some stakes horses are chronically lame but race weekly, although lame horses race slower, and as lameness progresses, horses drop in class. The major effect of lameness is seen during the last quarter of the race, called the last quarter time. Lower-class horses usually have a fast opening quarter, but slow last quarter time, whereas open and stakes horses have fast quarter times anytime during the race. Evaluation of last quarter time is useful to determine cause and severity of poor race performance. Lameness often costs a horse 1 to 2 seconds in last quarter race time, equating to 5 to 10 horse lengths. For example, a horse that normally paces the last quarter in 27.1, but for the last few starts has been coming home in 28.4 to 29.2 is probably lame, but concomitant respiratory and metabolic problems cannot be overlooked.

TRACK SURFACE AND LAMENESS

STBs usually race around an oval track, ½ to 1 mile in circumference; a 1-mile track is most common. Races are usually 1 mile in length, but a few are ⅝-mile sprints to 2-mile races. The track surface is a crushed rock base, covered with a packed, sandy soil and a thin sand or stone dust surface. STB racing requires a much firmer track surface than galloping horses. Sulky tires are more efficient if rolling over a firm smooth surface, and the STB gaits require a firmer and smoother surface than does the gallop. Track surfaces have become firmer, because year-round racing requires a surface suitable for racing in rain and sub-freezing temperatures. Hard tracks with loose surfaces require horses to wear shoes with added grabs and welded on spots of borium to avoid slipping. Excessive slipping or overzealous use of shoes with additives predisposes horses to lameness. A soft or deep surface can predispose horses to tendonitis and suspensory desmitis. Hard tracks with a covering of loose stone dust are slippery and may predispose horses to carpal synovitis, bruised soles, and muscle soreness. Racetracks get soft and sticky with small amounts of rain, and hard and unyielding with heavy rainfall that may wash the top surface into the infield.

TRACK SIZE AND LAMENESS

Track size has substantial implications in the development and expression of lameness. Tracks are 1 mile, ⅞ mile, ⅝ mile, and ½ mile, and in general, race times are lower (faster) on large tracks. Because most STB races are 1 mile, track size determines the number of turns during the race. During a race on a ½-mile track, an STB must negotiate four turns. Turns on shorter racetracks are much tighter than those on larger tracks (Fig. 109-2). Horses racing with mild chronic lameness are less

Fig. 109-2 **A,** A young trotter is on the left shaft coming out of a turn on a small farm track. On this track a horse has to negotiate four turns to train a mile. **B,** Horses approach the start of a race at the Red Mile, Lexington, Kentucky, a track that is 1 mile long. Compared with the turn in **A,** the turn on this track is substantially larger, and horses must negotiate only two turns for a mile race.

likely to sustain speeds necessary to be competitive on large tracks, so they may race on smaller tracks, but if lameness is worse in the turns, the horse may be less competitive on small tracks and better suited for a 1-mile track. If lameness is worse in the straightaway, the horse may be best suited for a small track. This is particularly important when lameness worsens as the race goes on, because large tracks have a long straightaway at the end.

During counterclockwise racing, right-sided lameness conditions exert more influence, even though the LF and LH are on the inside. Horses with left-sided lameness can race successfully on small tracks and apparently negotiate turns well. However, those with right-sided lameness cannot trot or pace the turns well, most likely from the effect of centripetal and compressive forces. Ideally, all racehorses should be sound, but many horses can race successfully with mild infirmity. Horses with substantial lameness should be treated or rested until the condition improves.

CONFORMATION

Forelimb and hindlimb faults lead to lameness and interference. Severity often depends on gait. A trotter should be as long from front to back as it is tall. Trotters are passing gaited if hind feet land outside the front feet, and in-line gaited if hind feet land in plane with the front feet. Wide hindlimb gait is advantageous because it allows a passing gait. Trotters can be mildly to moderately toed out in front without substantial interference problems, but they should not be toed in, because winging out leads to interference and lateral suspensory branch desmitis. Pacers can be wide and mildly toed in, but not toed out, because this fault predisposes the horse to hitting the knees. A club foot is less well tolerated by a STB than a TB and usually results in foot lameness. Low, underslung heels and long toes are acquired by attempts to increase stride length or to decrease interference. Back at the knee is undesirable and leads directly to carpal lameness in a trotter, but it is better tolerated by a pacer. Offset (bench) knee is a major fault and, although horses may race successfully, lameness of the carpus and metacarpal region often develops. Mild carpus valgus is tolerated by pacers but can lead to lameness of the carpus and metacarpal region. Pacers that are tied in behind the knee are at risk of superficial digital flexor tendonitis, but trotters are not. Short, straight pasterns and short backs are undesirable.

A serious hindlimb fault leading directly to moderate to severe lameness is sickle-hocked conformation (see Chapter 79). Mild sickle-hocked conformation typified many early pacers and is desired by some trainers. Sickle-hocked trotters are fast but develop curb and osteoarthritis of the distal hock joints, particularly severe in the dorsal aspect, and are chronically unsound. Straight hindlimb conformation is unusual but is a severe fault, leading to metatarsophalangeal and stifle joint lameness and suspensory desmitis. In horses with straight hindlimb conformation excessive extension of the metatarsophalangeal joint occurs. Long, sloping pasterns also predispose horses to fetlock hyperextension, but a primary metatarsophalangeal joint weakness is seen in some trotters with normal pastern lengths, in which the base of the proximal sesamoid bones (PSBs) is level with the mid-proximal phalanx. Abnormal elasticity or loss of support from the suspensory apparatus predisposes horses to run-down injury and suspensory desmitis. Cow-hocked conformation is prevalent in trotters but is not problematic unless severe. In-at-the-hock conformation predisposes horses to osteoarthritis of the distal hock joints, curb, and medial splint bone disease. Base-wide and base-narrow conformational faults, if severe, can lead directly to interference and lameness.

DISTRIBUTION OF LAMENESS

STB racehorses have a higher percentage of hindlimb lameness than TBs. The STB trains and races using a two-beat gait: two limbs bear weight simultaneously. Load is shared almost equally between forelimbs and hindlimbs. The caudal location of the cart and driver (load) and the addition of the overcheck apparatus shift the center of balance caudally, increasing hindlimb lameness (see Figs. 2-2 and 2-3). The distribution of forelimb to hindlimb lameness is approximately 55% to 45%.

TEN MOST COMMON LAMENESS CONDITIONS

The following are the top 10 lameness conditions in the STB racehorse:

1. Front foot lameness
2. Carpal lameness
3. Metatarsophalangeal joint lameness
4. Distal hock joint pain and other tarsal lameness
5. Suspensory desmitis: forelimb and hindlimb
6. Metacarpophalangeal joint lameness
7. Splint bone disease
8. Stifle joint lameness
9. Rhabdomyolysis and muscle soreness
10. Other soft tissue lameness: curb and superficial digital flexor tendonitis

LAMENESS IN THE YOUNG STANDARDBRED

The top 10 list contains those conditions seen in STB racehorses of all ages when grouped together, but unraced 2- and early 3-year-olds develop a subset of lameness conditions. Osteochondrosis (see Chapter 58), splint exostoses (splints), and curbs are common. Forelimb splints are usually medial but can occur laterally. Hindlimb splints are almost always medial and are more common in trotters. Splints can cause primary lameness or be secondary to carpal lameness, especially if exostosis is proximal. Forelimb splints develop when the limb is carried or lands abnormally. Young horses develop splints from difficulty learning the racing gait and by making breaks. While breaking, pacers often hit themselves, because galloping in hobbles is difficult. Curb is common in young pacers and may be recognized while horses are jogging, before training begins (see Chapter 79).

Carpal lameness is common in young horses. The middle carpal joint is the most common site, but synovitis can occur in the antebrachiocarpal joint. Carpal lameness is caused by early subchondral stress-related bone injury of the third carpal bone and is particularly common in trotters. Paddock turn out for 30 days is recommended. Middle carpal joint lameness must be differentiated from proximal suspensory desmitis (PSD). The thought once was that the distal radial physis was a source of occult carpal region lameness, but diagnosis was never confirmed. Physeal pain is a rare diagnosis today, but some veterinarians feel such pain is a possible source of colt soreness.

Bucked shins are rare but do occur in 2-year-old pacers in July and August. Most bucked shins occur in the forelimbs. Genuine bucked shins rarely occur in trotters, but interference trauma of the dorsal cortex of the third metatarsal bone (MtIII) is common.

Distal hock joint pain is a common cause of acute unilateral lameness, but if pain is bilateral, overt lameness may not be present. Horses appear stiff and sore when starting out and warm out of lameness. Gait abnormalities and repeated breaking may cause distal hock joint pain. Lameness may be difficult to abolish completely with intra-articular analgesia, because

pain is multifactorial, arising from peri-articular soft tissues and secondary gluteal myositis and trochanteric bursitis. Horses should be rested for 1 week and given non-steroidal anti-inflammatory drugs (NSAIDs) such as phenylbutazone (2.2 to 4.4 mg/kg PO bid). Intra-articular injections are avoided if possible, but treatment of the tarsometatarsal joint with hyaluronan alone, or in combination with corticosteroids, often resolves residual lameness. Using a less aggressive training schedule for 20 to 30 days is recommended. Shoeing should be evaluated and grabs and calks should be removed or minimized.

Interference injuries are common and plague many STBs throughout the racing career. Trotters interfere primarily by striking the toe of the ipsilateral front foot to the shin, pastern, or coronary band regions of the hindlimb (see Chapter 7). Interference from a front foot striking the medial aspect of the contralateral forelimb (cross firing) occurs occasionally in trotters but is a major form of interference injury in pacers. Injury to second (McII) and third metacarpal (McIII) bones, PSBs, carpus, heel, and hoof wall causes bruising and hematoma formation. Contusions can lead to soreness or frank lameness and can cause an altered gait. A trotter attempting to avoid striking the LH shin shortens the cranial phase of the stride, causing what appears to be a pelvic hike, mimicking LH lameness. Deliberate hiking can alter load distribution, causing compensatory lameness in the RH and RF. Local trauma is treated, and changes in equipment and shoeing such as applying brace bandages and boots are performed. A brief (5 to 7 days) reduction in exercise intensity is useful to allow a young horse to gain confidence in the corrected gait.

Young horses with poor gait should be examined carefully for neurological disease. Equine protozoal myelitis (EPM) is endemic in STBs and can be a real cause of gait deficits, or a catchall diagnosis (see Chapter 11).

CLINICAL HISTORY

Clinical history is critical for lameness diagnosis and to put lameness into context with other causes of poor racing performance. The attending veterinarian with an established relationship with the horse may know the horse's history. A trainer often refers to mild lameness by saying, "The horse is just sore, Doc" or, "The horse is off when jogging, but throws it away when going the right way." STBs have unusual resiliency and race rather well with chronic lameness, but lameness does not resolve with speed. Owners do not understand this concept. Signs of lameness become less visually apparent when horses go fast. Because STBs often race with numerous compensatory lameness problems, one must be open to the possibility that current lameness is not worsening of an existing problem but is an entirely new one.

A good starting place is to determine the chief complaint from the person(s) closest to the horse, usually the groom and trainer. What differs now from the horse's normal activity, gait, or performance? Has a change in training or racing schedule occurred? Any change in track surface? A horse noticed to be stiff and sore up front when walking to the track may be foot sore the day after the horse raced on a hard track. Have any equipment or shoeing changes been made? Additions of head poles, gaiting straps, and boots point to high-speed lameness and gait abnormalities. Shoeing changes, such as adding or removing bar shoes, are important because horses used to wearing bar shoes may have sore feet if bars are removed. Metacarpophalangeal joint lameness may be worse if bars are added. Stress-related bone injury of the metatarsophalangeal joint is seen soon after aluminum shoes with toe grabs are applied. Is the horse receiving any current medication and when was the horse last examined? What treatments, if any, were given? Although an acute injury or worsening of chronic osteoarthritis can cause acute synovitis, inflammatory and infectious arthritis can develop days to weeks after intra-articular injections, particularly if corticosteroids were used, because these drugs can suppress signs of inflammation and infection for weeks. Infectious arthritis is common 14 to 21 days after intra-articular injection, but often owners and trainers are incredulous because recent injections were not performed. What has the trainer and groom done recently to solve or treat the problem? Topical therapy may reduce or worsen inflammation and mask or augment clinical signs. Skin soreness after application of paints and blisters can cause a false-positive response to deep palpation.

Is the horse a trotter or a pacer? This information is critical. The usual distribution of pacers to trotters is about 3:1. Interference problems are different between gaits. Trotters develop contralateral or diagonal compensatory lameness, whereas pacers develop contralateral or ipsilateral compensatory lameness. A pacer with a left carpal lameness often develops LH compensatory lameness, but a trotter is more likely to develop compensatory lameness in the RH. The distribution of lameness differs between gaits. Fractures of the proximal phalanx and McIII/MtIII and superficial digital flexor tendonitis are less common in trotters. Trotters with stifle lameness, specifically osteoarthritis of the medial femorotibial joint, can cope better than pacers. Race times are different. Prognosis for certain problems such as osteochondral fragments of third carpal and radial carpal (RC) bones is worse in trotters. Pacers are more likely than trotters to start a race and to have five starts before and after surgical removal of osteochondral fragments of the carpus.[1] Osteochondrosis is common in certain bloodlines of trotter and pacers.

Is the condition worse at the trot or pace? Trotters and many pacers trot while jogging the wrong way, but assume the intended gait when turned the right way. Most forelimb lameness conditions are less obvious at the pace, but hindlimb lameness differs. In general, STBs with stifle lameness are worse at the pace than the trot. If trainers comment that the horse is worse on the pace than on the trot, the veterinarian should suspect stifle lameness until proved otherwise. Pacers wear hobbles, but trotting hobbles, those that encircle only the forelimbs and attach to a pulley caudally, are used if trotters are making breaks.

Is lameness different depending on what direction the horse moves? Problems on the right side are worse going the right way. Horses with medial foot and carpal lameness may show signs in the turns, especially entering the turning radius going the right way, but not when jogging.

What is the horse's last quarter time? Although high-speed lameness can cause a 1- to 2-second decrease in last quarter time, horses with mechanical upper airway disease and rhabdomyolysis have a 2- to 4-second reduction, and those with severe lameness or atrial fibrillation have a 5- to 10-second reduction in last quarter time.

Is the horse on a line? This is one of the most important pieces of information to obtain. Trainers sometimes dismiss young horses being mildly on the line, because nervous and inexperienced horses may change directions suddenly, but in reality most of these abnormal movements are caused by lameness. During counterclockwise racing of a horse on the right line, the driver has to increase the tension (pull harder) on the right rein to keep the horse straight and prevent it from bearing toward the infield. Just the opposite occurs when horses are on the left line (Fig. 109-3). Because of bit pressure, when a horse is on the left line, it turns or cocks the head to the left, toward the inside rail, and vice versa. If history is unclear, the veterinarian should watch the horse on the track and observe head position. Because most horses bear away from pain, a horse on the right line is most often lame on the right side, but exceptions occur. A common finding is metacarpophalangeal effusion

Fig. 109-3 This trotter is on the left line and the right shaft in the straightaway. The horse's head is turned to the left because the driver (Rich Ringler) has to pull harder on the left rein to keep the horse going straight. The horse's hindquarters are closer to the right shaft. This combination of left line and right shaft would be consistent with left hindlimb lameness.

and a positive response to lower limb flexion. The trainer states, "The horse was on the left line when finishing the mile, but is usually on the right line especially in the turns." Primary lameness may be in the right carpus, but chronic RF lameness has caused compensatory overloading and lameness of the left metacarpophalangeal joint, and both areas should be treated. Most horses on a line from forelimb lameness have foot or carpal lameness. However, problems located medially, such as medial sole bruising, quarter cracks, or carpal lameness paradoxically can cause a horse to be on the contralateral line. Therefore a horse with a medial LF quarter crack could be on the right line, whereas those with a right carpal lameness could be on the left line. All limbs should be examined, because not all horses read the book. "If a horse is on the left line, think right hind," one old saying goes, and rarely a horse with RH bruised or cracked heels will be on the left line.[2]

Horses on a line without overt signs of lameness are among the most difficult lameness mysteries to solve. Because compensatory lameness problems may be numerous in a horse in which primary lameness cannot be ascribed, deciding on which limb to begin diagnostic analgesia is a dilemma. Most horses need to be examined at speed after blocking, a time-consuming and frustrating process. In many horses on a line, obscure sources of pain such as subchondral bone can be difficult to identify, and horses often are referred for scintigraphic examination. Most horses chronically on a line have had many intra-articular or other injections, and trainers will state, "We've done his knees, hocks, stifles, and feet and nothing helped." Often forgotten are the metatarsophalangeal joints. In many horses numerous sites of pain must be managed simultaneously rather than sequentially. Recommending numerous intra-articular and regional injections, a shoeing change, and a change in exercise intensity in a horse that is on a line is not unusual.

Is the horse on a shaft? A horse with RH lameness drifts to the left (see Fig. 109-2) and positions the hindquarters closer to the left shaft of the sulky and is thus on the left shaft; the reverse is true with LH lameness (Fig. 109-3). Not all hindlimb lameness problems cause horses to be on a shaft. Horses with osteoarthritis of the medial femorotibial joint may not be on a shaft, but those with distal hock joint pain will be. A horse may be falsely on a shaft if it is hard on a line and the driver pulls hard to keep the horse straight, compelling the horse to twist its entire body, positioning the hindquarters close to one shaft to maintain balance.

Is the horse worse in the turns or on the straightaway? Although nothing is pathognomonic, a few findings are consistent. Horses with front foot lameness, splints, and curbs get worse as the mile progresses and so appear worse on the final straightaway. Horses with distal hock joint pain appear to be worse when going into or coming out of a turn but usually can pace and trot the turns successfully. Horses with carpal, metatarsophalangeal, and stifle joint lameness are worse in the turns. If a horse gets rough in the first turn just after leaving the gate, a medial RF problem such as a splint or carpal lameness should be suspected.

On what size track does the horse race? The relationship of track size to expression of lameness and the differences between left- and right-sided lameness problems were discussed (see page 897).

Is the horse making breaks? Unsound trotters often break stride and gallop but occasionally break into a pace. Lame trotters may pace when trotted in hand. Interference injury causes breaks and current shoeing or recent changes should be evaluated. A different way to hang up a horse, meaning how to shoe and equip it, may be all that is needed.

The veterinarian must be current on all medication rules in the racing jurisdiction.

LAMENESS EXAMINATION

The lameness examination can be divided into three stages: palpation, observation while moving on a lead shank, and observation under harness. The veterinarian should avoid narrowing the examination to the area suspected by the groom and trainer but should first stand back and observe the horse and watch the horse moving around and coming out of the stall. Subtle neurological signs may be apparent. Asymmetry, such as muscle atrophy, may point toward chronic lameness or neurological disease.

Palpation

Careful palpation is the art of diagnosis in the racehorse but often is sacrificed because palpation is time consuming. Palpation is critical in STBs because of numerous compensatory lameness problems. The veterinarian needs to be able to read the horse to make a diagnosis. A successful lameness detective respects what the horse is trying to say. The veterinarian should move over the entire horse with a light touch to see if a withdrawal response is elicited. A light touch along the neck and gluteal regions may elicit pain from secondary muscle soreness or painful previous intramuscular injection sites. Horses often exhibit a withdrawal response when a limb is first picked up, indicating a problem even before the specific region is manipulated. Horses may exhibit false-positive responses in areas that have been painted recently, blistered, or freeze fired. Once horses have been freeze fired for splints and curbs, many trainers assume the problem has been solved and are incredulous if the veterinarian suggests the area is still the source of pain. Cryotherapy is not a panacea and may have to be repeated, or another form of management may be needed (see Chapters 79 and 90).

Palpation should be done in a quiet place, before the horse is trotted in hand, so that all potential lameness problems are detected. Careful and detailed examination of the front feet should be performed. Hoof tester examination is critical, but

most horses show a painful response for 1 to 2 days after racing, particularly if the track was hard. Effusion of the distal interphalangeal joint capsule often accompanies early osteoarthritis and sore feet. Shoes on the front and back feet should be evaluated critically for wear, type, weight, and the presence of additives. The proximal, dorsal aspect of the proximal phalanx should be palpated for pain associated with mid-sagittal fractures of the proximal phalanx. Palpation findings in the metacarpophalangeal and metatarsophalangeal joints often do not correlate well with degree of lameness, response to diagnostic analgesia, and results of scintigraphic examination. Pain on palpation of the PSBs may indicate sesamoiditis. Normally the horse responds little to compression of the suspensory ligament (SL), deep digital flexor tendon (DDFT), and superficial digital flexor tendon (SDFT). Pain is often the first sign of desmitis or tendonitis. Chronic enlargement of the SL and SDFT are common in lower-class horses, and although the horse may not react to palpation, it may have associated high-speed lameness. Splint exostoses are most painful after training or racing but may be non-painful several days later. Standing and flexed subcarpal palpation may reveal pain from PSD, longitudinal and avulsion fractures of McIII, proximal superficial digital flexor tendonitis, and a dorsal medial articular fracture of McIII.

No palpable abnormalities may be associated with carpal lameness. The veterinarian should carefully assess for warmth and compare the limb with the contralateral limb. Effusion occurs in 2-year-old horses early in training, but it often is minimal in horses with subchondral bone pain. The antebrachiocarpal joint is rarely a site of carpal lameness in older racehorses, but in young horses, especially trotters, effusion may occur. The medial carpus and distal antebrachium are common sites for interference injury. Carpal tenosynovitis is unusual to rare, but occasionally hyperextension injury and hemorrhage or, rarely desmitis of the accessory ligament of the SDFT, occur. Pacers develop hobble burns and cellulitis in the proximal antebrachium. Elbow and shoulder joint lameness is rare, but horses may exhibit pain when the biceps brachii, intertubercular bursa, and other muscles are palpated or the shoulder joint is flexed, from muscle soreness secondary to primary carpal lameness. The neck, back, and rump regions should be palpated for symmetry and muscle soreness. Young horses will occasionally manifest sore withers from an improperly fitted harness. Because intramuscular injections of counterirritant and anti-inflammatory solutions are common in STBs, careful palpation is necessary to discover deep abscesses causing pain, lameness, and fever. Disease of the thoracic dorsal spinous processes is rare. In an 8-year period, none of 1020 STBs undergoing scintigraphic examination had increased radiopharmaceutical uptake in the dorsal spinous processes.[3]

Secondary muscle soreness of the gluteal region is a common problem, and the diagnosis of trochanteric bursitis often is made based on a painful response to compression. Trochanteric bursitis (whorl bone disease) may be overdiagnosed and difficult to authenticate, but injections often improve hindlimb gait and performance (see Chapter 48). Rhabdomyolysis also can cause pain on palpation of the rump region. Young horses with loose stifles often knuckle behind, but palpation is often negative. Pain associated with the medial aspect of the stifle may be present, but abnormalities when manipulating the patella are absent. Femoropatellar effusion in young horses can signal osteochondrosis. The medial femorotibial joint capsule should be palpated, because osteoarthritis is the most important lameness of the stifle. The crus is a rare site of pain. Hobble burns on the caudal aspect can cause soreness in young pacers.

The tarsocrural joint is the most common site for osteochondrosis and effusion in young horses should prompt radiographic examination. Peri-articular pain in the distal tarsus and proximal metatarsal region often accompanies distal hock joint pain. The Churchill test is done routinely, but in addition to those with distal hock joint pain, STBs often respond positively with other common distal limb lameness. Bony enlargement (bone spavin) is rare. Plantar soft tissue swelling and pain associated with curb is a common cause of lameness (see Chapter 79). The plantar metatarsal region commonly is overlooked, but it should be palpated carefully for the presence of suspensory desmitis. Signs of inflammation may be difficult to detect, because the SL is enclosed within the bony confines of the metatarsal bones. The first evidence of suspensory desmitis is mild enlargement and pain of the suspensory body in the mid-metatarsal region. Compression of the splint bones puts indirect pressure on the SL and explains why the Churchill test is non-specific. The dorsal aspect of MtIII is a common site for interference injury in trotters. Palpation of the fetlock region is critical, because the region is an important source of lameness, but often no localizing clinical signs are apparent. Pain from interference injury should be differentiated from that associated with mid-sagittal and dorsal frontal fracture of the proximal phalanx. The hind foot is an unusual source of lameness, but fractures of the distal phalanx, bruising, navicular disease, and osteoarthritis of the distal interphalangeal joint are diagnosed occasionally. STBs normally respond positively to hoof testers placed across the heels.

Movement

The horse should be examined at a walk and trot in hand. The pace is a forgiving gait, and head and neck nod and pelvic hike can be difficult to see. Evaluating lameness in pacers is easier while they are trotting, but correlation between lameness seen in hand and during pacing at high-speed is questionable. Forelimb lameness is less obvious at the pace. Degree of pelvic hike is comparable at the pace and trot. Baseline lameness should be determined before the harness is applied, because horses that are fracture lame should not be taken to the track, and in horses with obvious lameness, a good correlation exists between lameness seen in hand and in harness. The ability to trot horses in hand at the racetrack may be limited because of too much activity, lack of room, and often poor or slippery surfaces. Flexion tests are performed but lack specificity. The carpal flexion test is the most reliable and specific of all flexion tests. Direct compression of a painful splint, proximal SL, and curb followed by trotting is useful. Subtle hindlimb lameness is best evaluated in harness, because little correlation may exist with lameness seen in hand. Pulling a cart and different surface likely explain this observation. Subtle (less than grade 1 of 5) hindlimb lameness at the trot in hand should be taken with a grain of salt. Observation on the track puts lameness into perspective and allows evaluation of compensatory lameness. Lameness is most pronounced when horses are jogged slowly and is much less obvious with speed. High-speed lameness is evaluated by communication with the trainer and by resolving signs such as being on a line.

DIAGNOSTIC ANALGESIA

Diagnostic analgesia is essential to pinpoint lameness, but trainers and owners often prefer treatment of a suspected area rather than lengthy investigation. With weekly racing no time may be available for blocking, because levels of local anesthetic solution will be detectable. Diagnosis often is made based on response to treatment. Many common lameness conditions involve subchondral stress-related bone injury, and often horses can be blocked sound but do not improve with intra-articular treatment. Perineural analgesia is more effective than intra-articular analgesia for diagnosing subchondral bone pain.

Controlling as many variables as possible is important when observing STBs in harness. The same gait, equipment,

driver, approximate speed, and track direction should be used. Sometimes horses exhibit lameness when driven by the groom but not the trainer, because trainers expect a higher level of performance and often carry a whip. A false positive occurs when a horse exhibits lameness at the pace but after diagnostic analgesia returns to the track without hobbles equipment and trots soundly. The diagnostic analgesia procedure should be the only variable. STBs often warm out of lameness and should be evaluated immediately when starting out after each block. Over zealous use of a twitch and rough handling may compromise evaluation, and administration of drugs can add another variable. If horses are observed in harness after diagnostic analgesia, the person driving the horse should be instructed to return the horse to the stable as soon as improvement is recognized, because training a horse after blocking risks further injury.

Complete description of blocking techniques is found in Chapter 10. A few points about diagnostic analgesia in the STB must be kept in mind. Palmar or plantar digital analgesia desensitizes most of the foot. Horses with metacarpophalangeal or metatarsophalangeal joint lameness can be sound after palmar or plantar digital analgesia, particularly those with mid-sagittal fracture of the proximal phlanx.[4] The abaxial sesamoidean block is avoided, because inadvertent analgesia of fetlock pain can be misinterpreted as lameness of the foot or pastern. Individual carpal joints should be blocked separately, and differentiation between carpal and proximal metacarpal region lameness is necessary. The median and ulnar nerve block is underused.

A low plantar block must be done routinely, or many lameness conditions will be diagnosed erroneously as high up. The fetlock region is a major source of hindlimb lameness. The high plantar block is often overlooked, but PSD cannot be substantiated without it. The centrodistal joint is difficult to enter but should be blocked separately from the tarsometatarsal joint, because communication occurs in only 8% to 39% of horses. The medial femorotibial joint should be blocked separately from the femoropatellar joint, even though they communicate in a high percentage of horses.

IMAGING CONSIDERATIONS

Radiography

Routine radiographic examination of STBs does not differ from that of other sport horses. However, a few specific views should be kept in mind. Down-angled oblique (dorsolateral 45° proximal-palmaromedial distal oblique and dorsomedial 45° proximal-palmarolateral distal oblique) and horizontal oblique (dorsolateral-palmaromedial oblique [DL-PaMO] and dorsomedial-palmarolateral oblique) views should be obtained to evaluate the distal interphalangeal joint. The tangential (palmaroproximal-palmarodistal oblique) view of the navicular bone is less valuable in the STB than in other older sport horses. To evaluate the distal aspect of McIII/MtIII for subchondral stress-related bone injury and the space between the proximal plantar aspect of the proximal phalanx and the base of the PSBs for the presence of fragments, down-angled oblique (dorsolateral 15° proximal-palmaromedial distal oblique and dorsomedial 15° proximal-palmarolateral distal oblique) views should be obtained (see Chapter 43). The most important views of the carpus are the DL-PaMO view and the tangential (skyline) view of the distal row of carpal bones. In STBs it is essential to have a well-exposed and well-positioned skyline view to evaluate the third carpal bone for sclerosis, radiolucency, and osteochondral fragments (see Chapter 39). Radiographic changes of osteoarthritis of the centrodistal and tarsometatarsal joints are seen best on the lateromedial and dorsomedial-plantarolateral oblique (DM-PlLO) views. A common misconception is that changes associated with bone spavin occur medially, but not in young STBs. The DM-PlLO view is essential in evaluating the tarsocrural joint for osteochondritis fragments, because the two most common locations, the cranial intermediate ridge of the distal tibia and the lateral trochlear ridge of the talus, are evaluated best using this view. The caudocranial view is essential when evaluating the medial femorotibial joint for narrowing, osteophytes, and subchondral bone cysts. Inadequate exposure and positioning often result in views that cannot be interpreted.

Computed radiography and xeroradiography are useful in evaluating stress-related bone injury of subchondral bone and incomplete fractures. Computed tomography and magnetic resonance imaging have limited availability.

Ultrasonographic Examination

Suspensory desmitis, superficial digital flexor tendonitis, curb, and desmitis of the distal sesamoidean ligaments are common in the STB, and ultrasonographic examination is essential for diagnosis and assessing prognosis (see Chapter 16).

Scintigraphic Examination

Referral for scintigraphic examination is common, and horses with poor performance often have numerous areas of increased radiopharmaceutical uptake (IRU), indicating high-speed lameness is a part of the problem. Common areas of IRU include the distal phalanx, metacarpophalangeal joint (medial condyle of McIII and PSBs), proximal aspect of McIII, carpus, especially the third and radial carpal bones, metatarsophalangeal joint (proximal aspect of the proximal phalanx and distal plantarolateral aspect of MtIII and PSBs), the proximal plantar aspect of MtIII, tarsometatarsal and centrodistal joints, and medial femorotibial joint (see Chapter 19).

PROCEEDING WITHOUT A DIAGNOSIS

Proper identification of lameness as the cause of poor performance and localizing the site(s) of pain can be challenging and relies heavily on experience. It is particularly important to differentiate lameness from interference, because interference can cause signs that mimic lameness, lameness can result from interference, or lameness can cause interference. Horses on a line without obvious lameness and a negative response to diagnostic analgesia can be frustrating. Horses can become stubborn and react to equipment and twist and bend the head and neck, causing signs similar to being on a line. Occasionally, horses with tooth-related pain get on a line. An ocular problem is a rare cause of a horse being on a line. Trainers may use bridles with partial blinds or vision-restricting cups to control a horse on a line.

Simultaneous RF and RH or LF and LH lameness, or even primary hindlimb lameness, can be difficult, because hindlimb lameness can cause a head nod mimicking ipsilateral forelimb lameness. Diagnostic analgesia should start in the hindlimb. In trotters with foot and carpal lameness the caudal phase of the lame forelimb is longer, and to avoid interference, the cranial phase of the ipsilateral hindlimb is shortened (hiking), mimicking hindlimb lameness. Pelvic hike abates when the source of forelimb lameness is localized. The clinician should obtain a fresh perspective and ask a fellow veterinarian or refer the horse for advanced imaging.

To differentiate lameness from interference gait abnormalities, phenylbutazone (4.4 mg/kg bid for 5 to 10 days) is administered (the bute test). Horses with pain should improve, whereas those with pure interference gait abnormalities should not. However, not all horses with musculoskeletal pain respond to NSAIDs, particularly those with chronic lameness or numerous compensatory problems. Alternatively, the veterinarian

should medicate the suspected area and see if performance improves, because mimicking the maximal exertion of racing is difficult. A trotter on a line only going into the first turn may be helped by injection of corticosteroids around a mildly painful medial splint or into the middle carpal joint. Sometimes treating a different area between several races may allow a fortuitous discovery, but this requires good record keeping, and it may be expensive. Occasionally, horses with subtle or nondescript lameness or a rough gait are managed using a shotgun approach, and numerous intra-articular treatments are given simultaneously. This approach should be reserved for horses in which normal management procedures have failed and an important race is rapidly approaching. Sometimes a positive response to intra-articular corticosteroid injections results from systemic absorption and effect on a distant, rather than the intended, site.

A brief turnout period of 7 to 10 days is sometimes useful. In 2-year-olds a decision often is made to stop training and give 3 to 4 months of turnout rather than risk compensatory lameness or fracture. The decision to stop training a horse should not be taken lightly. Getting an older horse back to the same level of racing is often difficult, even with a planned turnout period, and jogging the horse 3 to 4 times a week to maintain condition and range of joint motion may be best. The older the horse is, the more difficult returning the horse to racing is. Older horses with chronic lameness do not thrive with rigorous training and often come out of a solo training mile lamer than after schooling or qualifying races. Older horses often are raced into fitness, rather than trained rigorously. A common mistake is to train or race these horses too often.

A complete neurological examination should be performed in any horse with gait deficits and even some with overt lameness, if neurological signs are noticed concomitantly. EPM is frequently diagnosed, but positive identification of the source of neurological signs is difficult to make, because serum and cerebrospinal fluid analyses are not accurate (see Chapter 11). A common method to manage horses when all else fails is to treat for EPM and observe the results. Some horses improve despite having no overt neurological signs.

SHOEING AND LAMENESS

Shoeing to obtain ideal high-speed gait is an art, especially in trotters that are difficult to balance. The farrier must be a part of the lameness team. Veterinarians have to take a back seat to the trainer and farrier and cannot be overtly critical of the shoeing approach. Sore front feet are common and many approaches are used. Sometimes a simple shoeing change redistributes load away from a sore area, and a common approach in trotters is a switch from a half-round to a flip-flop shoe (see Fig. 110-11). Classically, trotters are shod with more weight up front, whereas pacers are shod with more weight behind. Recently the tendency has been to lighten shoe weight in trotters and pacers in front and behind. The typical hind shoe of a pacer (a half-round, half-swedge) has been replaced by aluminum shoes with a low toe grab. Aluminum shoes are used commonly in front in pacers. In trotters, heavy front shoes may predispose to interference injury and carpal lameness, and now aluminum shoes often are used behind, allowing a lighter shoe to be used in front. Light aluminum shoes with grabs allow maximal track purchase and increase speed, but they may worsen existing osteoarthritis of many distal joints behind and in front. A STB can jog and train abnormally with a sprung shoe or a broken bar shoe. Special attention should be given to the medial half of the front feet because interference bruising is substantial.

A low-heel, underslung foot is not common in modern day STBs, although low-heel, long-toe conformation was previously favored in the erroneous assumption that it would increase stride length. When combined with heavy shoes, long toes prolong breakover, increase stress on the dorsal aspect of the forelimb, and predispose the horse to lameness. Using a shorter toe and more upright front foot is a major improvement in trimming trotters.

Quarter cracks and other hoof wall defects are common. Acrylic and composite repair of hoof defects have aided many sore-footed STBs. Acrylic often is applied to augment the hoof wall. In horses with thin walls, shoes are difficult to maintain, especially when shoes may be changed every 2 to 3 weeks. Overzealous application of acrylic can weaken a normal hoof wall.

SPECIFIC LAMENESS CONDITIONS OF THE STANDARDBRED RACEHORSE

Front Foot Lameness

Front foot lameness is the most common lameness condition. Palmar heel pain abolished using palmar digital analgesia is a common finding, but the veterinarian should keep in mind that palmar digital analgesia also abolishes most of the pain associated with the distal interphalangeal joint and toe and lower pastern regions. Palmar heel pain can be caused by bruised heels, corns, sheared heels, hoof cracks, contracted heels, wall separations and gravel, stress remodeling and stress fractures, traumatic fractures, osteoarthritis, or various combinations of these conditions.

Bruises, Corns, and Abscesses

Bruised feet can be caused by faulty conformation such as that seen in a large, flat-footed horse that develops bruised heels and bars. Small, narrow-footed horses are prone to quarter and heel cracks and sore heels. Young STB feet are trimmed and pared and subjected to daily concussion on a firm and unforgiving surface. Aggressive paring can predispose to palmar sole bruising. Overt lameness may not be seen. Horses are often sore when starting from the barn and warm out of lameness after jogging a short distance. Because early carpal lameness can produce similar signs, careful hoof tester examination is necessary and often reveals profound sensitivity across the heels.

Base-wide, toed-out conformation causes overload of the medial heel and quarter, and long, sloping pasterns cause overload of the palmar aspect of the foot. Shoes that are too narrow or those with short branches can cause sole bruising and corns. Horses that interfere (cross firing or forging) are often shod with the inside branch of the shoe turned in to prevent grabbing and pulling of the shoe, and this can cause corns. Shoeing to correct interference is important but so is management of lameness that may lead to interference. Tubbing and soaking may compound the problem, creating a softer, more easily bruised sole. Poulticing may reduce inflammation initially, but continuous poulticing has the same undesirable result. The sole should be hardened with daily applications of an iodine-based paint, such as Rites paint, an iodine-ether preparation. Attention should be given to the stall bedding, since overly dry stall may lead to dry, cracking hoof walls. Digging out the bruise prolongs lameness and predisposes the hoof to abscessation. A wide web, concave inner surface steel bar shoe is applied if the horse can tolerate this much weight. A trainer may prefer a lighter aluminum bar or egg bar shoe. Full pads may afford protection for an upcoming race, but bruises cannot be re-assessed, and sole paint cannot be applied. In trotters the flip-flop shoe is advantageous, because the pad can be lifted and the sole painted, and without shoe branches the heels can spread. Deep-seated bruises can become corns, corns can fissure and crack, and bacterial invasion can cause infection. With abscessation, judicious paring to establish drainage is necessary, but the more

sole that is removed, the longer it takes to heal. Soaking in warm water with Epsom salts (magnesium sulfate) and then wrapping with ichthammol (an iodine-based drawing salve) promote drainage.

Sheared Heels

Quarter and heel cracks, wall separations, and contracted heels can result from sheared heels. Improper and asymmetrical rasping over time results initially in lowering of one heel and later to vertical heel walls, contracted heels, and flaring of the wall on the side of the lower heel. With mediolateral hoof imbalance, the vertical heel strikes the ground first, and uneven impact causes structural breakdown between the bulbs of the heel. The bulbs are painful and little resistance is offered to digital displacement of the bulbs in opposite directions. Corrective shoeing involves shoeing the steep side full and floating the quarter. The flared wall is shod tight and rasped off over several trimmings. For a horse with a long-toe and underrun heel predisposed to sheared heels, the toe should be shortened and the heel maintained. Recurrent quarter and heel cracks are common sequelae.

Hoof Cracks

Hoof cracks, depending on location, are known as heel or quarter cracks. Cracks may begin at the coronary band or at the bearing edge of the hoof wall. If the quarter crack extends to the sensitive laminae, local tenderness and variable lameness result. Horses with dry, shelly feet or vertical walls are predisposed to quarter cracks. Untreated chronic inflammation, inappropriate rasping of the periople and application of excessive acrylic predispose horses to dry feet, because exposure of horn tubules results in loss of moisture. Indiscriminate cutting of bars predisposes horses to contracted heels and results in vertical walls. A vertical wall is more likely to crack than a normal one. Quarter cracks can develop if horses are shod with the web of the shoe set in. To manage quarter cracks, weight bearing is prevented by floating, and artificial support to the weakened and cracked wall is applied, using a full or egg bar shoe. Extensive, deep, or painful cracks are repaired and stabilized. Acrylic patches are now preferred, but the crack must be prepared by paring and drying with iodine paint or gentian violet, or infection may develop.

Contracted Heels

Contracted heels can result from palmar heel pain rather than cause it. Dry, brittle feet cannot expand normally and may contract, as does a foot that has a long toe and low heel. When faced with a choice, farriers too often pick a shoe that fits tight (narrow), rather than full (wide), when the horse really needs a size in between. The wall inevitably grows to the small shoe and contracts. The indiscriminate use of acrylic results in heel contraction, because the material restricts expansion. Acrylic should be placed on the weight bearing wall surface only to repair broken or brittle walls for nail placement or for rebuilding low heels. The last shoe nail should be placed at the bend in the foot quarter, rather than in a more palmar location, to prevent restriction of heel movement. Other causes of palmar heel pain reduce weight bearing at the heels, leading to contraction. To manage contracted heels, the quarter is softened, the toe is shortened, and an extended bar shoe that fits full (wide) is applied. Lowering the heel to achieve frog pressure is contraindicated.

Wall Separation and Gravel

Gravel is caused by separation of the white line at the quarter and heel and bacterial invasion. Lameness, insidious at first, progresses to nearly non–weight bearing. Digital pulse amplitude is increased, and the horse responds intensely to hoof tester examination over the affected area. The coronary band or heel bulb become sensitive just before infection breaks out. Local therapy such as soaking and poulticing may hasten coalescence and proximal migration of exudates. If pastern cellulitis occurs, broad-spectrum antimicrobial agents should be given. The horse is generally sound within 24 hours after drainage, but a full pad may be necessary to avoid repeat infection during wall healing. Wall separations and gravel are common complications to sheared heels and dry, brittle feet. Seedy toe is an infected wall separation at the toe.

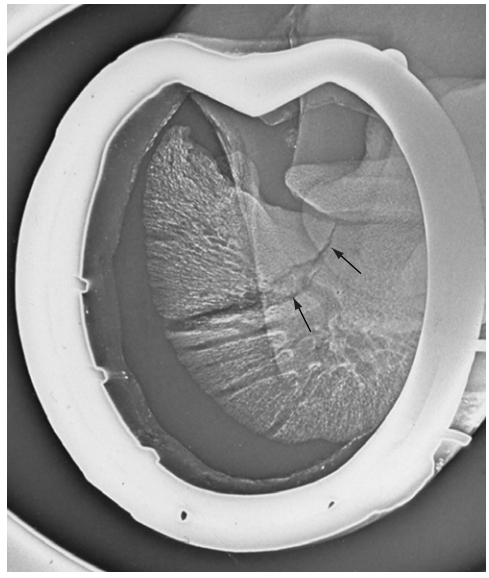

Fig. 109-4 Dorsomedial proximal-palmarolateral distal oblique xeroradiographic view of right front foot of a Standardbred racehorse. There is a typical articular fracture of the medial palmar process *(arrows)*. The horse is shod with a bar shoe with clips.

Sidebone

Ossification of the cartilages of the foot (sidebone) is a rare cause of lameness. Ossification may be hastened in horses with poor hoof conformation or in those in which interference injury occurs. Sidebone is found more often radiographically in aged horses with large, round, flat feet, but it may be seen incidentally in yearlings. If sidebone is painful to digital palpation, a bar shoe with heel clips and local subcutaneous injection of corticosteroids and Sarapin may provide temporary relief. Interference injury may cause fracture and infection of a cartilage of the foot.

Fractures of the Distal Phalanx

Fractures of the distal phalanx occur most commonly in the LF lateral and RF medial palmar processes and are usually articular (Fig. 109-4). Clockwise training and racing may account for asymmetrical location of fractures. Non-articular palmar process and mid-sagittal fractures are rare. Predisposing factors include hard racing surfaces (most fractures occur during a race, more often in winter months), foot imbalance, impact on an uneven surface, and overloading. Fractures of the distal phalanx may be the end result of stress-related bone injury of the distal phalanx, because IRU often is seen in the lateral aspect of the LF and medial aspect of the RF distal phalanx in horses with lameness abolished by palmar digital analgesia but in which radiographs are negative (Fig. 109-5).[5] Most fractures of the distal phalanx occur in aged horses. Treatment includes application of a wide web, concave inner surface steel straight or egg bar shoe with two side clips on each side. Horses are given 3 months of stall rest and then 3 months of turnout in a small paddock. Most fractures are complete, but non-displaced; mild displacement results in a step in the articular surface. Fractures take months to heal, and some appear to develop chronic nonunions, but many horses return to racing soundness even with a radiographically apparent non-union. Osteoarthritis of the distal interphalangeal joint often occurs, particularly in horses with displacement or non-union, but unless severe, prognosis

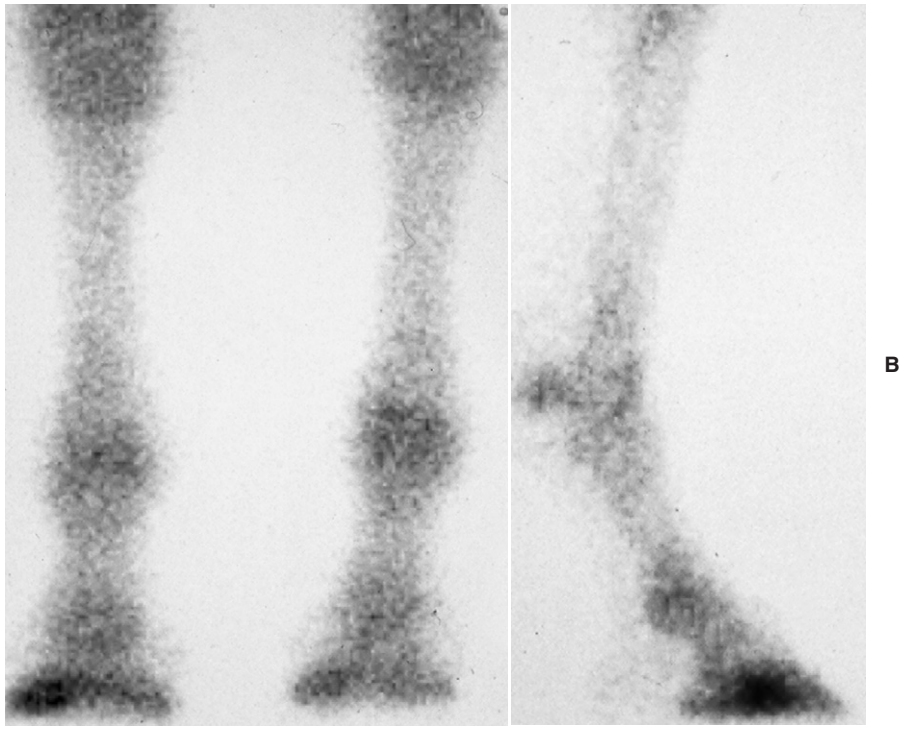

Fig. 109-5 **A,** Dorsal delayed (bone) phase scintigraphic image showing a typical distribution of increased radiopharmaceutical uptake of the Standardbred front feet. The left forelimb is on the left. (**B**) Lateral delayed (bone phase) scintigraphic image of the left forelimb. This Standardbred was lame in the left forelimb, and lameness was localized to the lateral aspect of the distal phalanx. In the left forelimb, stress remodeling of subchondral bone of the distal phalanx was diagnosed. Increased radiopharmaceutical uptake involving the lateral aspect of the left forelimb and medial aspect of the right forelimb distal phalanx is thought to be caused by counterclockwise training and racing, and stress-related bone injury can lead to fracture in these locations.

for racing is good. Osteochondral fragments of the extensor process are unusual, but arthroscopic removal of small fragments and repair of large ones are indicated. Prognosis is fair, but osteoarthritis of the distal interphalangeal joint can affect prognosis negatively.

Osteoarthritis of the Distal Interphalangeal Joint

The earliest sign of primary osteoarthritis of the distal interphalangeal joint is effusion. Effusion is also common in horses with nondescript foot soreness. Intra-articular distal interphalangeal injections of hyaluronan and corticosteroids are often performed. Progressive osteoarthritis can cause severe lameness, chronic fibrosis and thickening of the distal interphalangeal joint capsule, radiographic evidence of proliferative changes on the dorsal aspect of the middle phalanx and extensor process of the distal phalanx, and narrowing of the distal interphalangeal joint space.

Other Foot Lameness

Navicular disease is a rare cause of palmar heel pain. In 2-year-olds and early 3-year-olds IRU in the navicular bone is seen in delayed (bone) phase scintigraphic images, indicating abnormal bone modeling, but rarely do radiographs show substantial changes, except for mild medullary sclerosis. Navicular bone fracture, cysts, or acute separation of bipartite navicular bone are diagnosed rarely in forelimbs and hindlimbs. Horses with bipartite bones or fractures have a poor prognosis and palmar digital neurectomy is recommended.

Undiagnosed foot lameness, sore feet, is common. Horses have sensitivity to hoof testers and likely have soft tissue trauma, but a definitive diagnosis cannot be made. Treatment of the distal interphalangeal joint with corticosteroids and hyaluronan, perineural injection of anti-inflammatory agents, and shoeing changes are recommended.

Carpal Lameness

The carpus is the most common articular location for lameness and conformational faults. In 2-year-olds, carpal synovitis is common. High prevalence of carpal lameness is caused in part by racing young, immature STBs around tight turns on firm surfaces. The medial aspect of the middle carpal joint and the right carpus are predisposed to injury. Lateral injury of the carpus is unusual. Heat, effusion, and pain on careful palpation of the dorsal surfaces are common. Lameness varies with severity of injury, and horses carry the limb in an abducted position during advancement (see Chapter 39). Response to flexion varies, and diagnosis should be confirmed using intra-articular or median and ulnar analgesia. Secondary shoulder muscle soreness often develops. Horses warm out of lameness and weeks to months may pass before consistent lameness is seen. Radiographs are usually negative or equivocal initially, but sclerosis of the third carpal bone and mild marginal osteophyte production can predispose the horse to small osteochondral fragments or slab fractures of the third carpal bone. Scintigraphic examination often reveals focal areas of IRU, usually involving the third carpal bone or the medial aspect of the middle carpal joint collectively; bilateral lesions are common. Stress-related bone injury results in a continuum of subchondral and overlying cartilage damage, eventually leading to osteoarthritis or osteochondral fragments. Rest is the best form of management but is unacceptable to trainers and owners. Topical counterirritation with iodine paints is still

popular and is used with apparent success, because blistering usually is accompanied by a reduction in exercise intensity. The administration of NSAIDs for 3 to 5 days and a brief period of reduced exercise (2 weeks) may be an acceptable compromise between the veterinarian and the trainer. Intra-articular injections of hyaluronan or polysulfated glycosaminoglycans (PSGAGs) may reduce inflammation and theoretically may be useful in cartilage healing, but these often are used in a palliative rather than therapeutic manner. Injections may be of little benefit when pain originates from subchondral bone. Ten 2-year-old STBs with mild carpitis were treated with 3 to 5 weekly intra-articular injections with PSGAGs (Adequan) and were compared with a control group receiving NSAIDs and topical counterirritation.[6] The treated group had substantially less carpal lameness during the ensuing racing season. All horses raced, but horses in the control group required intra-articular injections to maintain soundness. Early aggressive intra-articular therapy may forestall development of progressive osteoarthritis in young STBs.

Palmar heel pain may play a role in the development of carpal lameness in young STBs. Early carpal lameness is easier to treat if palmar heel pain is recognized early and managed aggressively. In horses with bruised bars and poor mediolateral hoof balance the application of wide web bar shoes reduces inflammation and signs of carpal lameness. In all ages of trotters, the flip-flop shoe appears to be effective in reducing clinical signs of carpal lameness. Small osteochondral fragments of the carpus are the most common fractures and involve the third and radial carpal bones and rarely other bones in the middle carpal and antebrachiocarpal joints. Arthroscopic surgical removal and PSGAG therapy after surgery are recommended. Frontal and sagittal slab fractures occur commonly, and repair using 3.5-mm screws placed in lag fashion is recommended. Small, frontal slab fracture fragments can be removed. Although rest is an option in horses with non-displaced fractures, internal fixation may shorten recovery period and reduce risk of recurrence (see Chapter 39).

Metatarsophalangeal Joint Lameness

The metatarsophalangeal joint is a common source of pain causing lameness in STBs, and diagnostic analgesia is usually essential for diagnosis.

Stress and Non-Adaptive Remodeling

Stress or non-adaptive (maladaptive) remodeling of the metatarsophalangeal joint most often involves the distal, plantarolateral aspect of MtIII, PSBs, and the proximal aspect of the proximal phalanx (see Chapters 19 and 43).[7] Initially the horse has a variable, subtle, high-speed lameness, most commonly manifested around turns or by horses making breaks. Later, grade 1 to 2 of 5 hindlimb lameness is seen at the trot in hand. Lameness can be localized to the metatarsophalangeal joint, but it does not respond to intra-articular treatment. Palpation findings are negative or equivocal. Horses show variable responses to flexion and have negative radiographic findings but positive responses to diagnostic analgesia. The low plantar block is most effective, but horses may respond partially to intra-articular analgesia (and may require 12 ml). Later, sclerotic and radiolucent changes of plantarolateral MtIII are seen on down-angled radiographic views. Ultrasonographic evaluation may reveal a defect in articular cartilage late in the disease process. Scintigraphic examination is the best way to establish diagnosis and shows focal IRU involving the distal MtIII (see Fig. 43-2). The disease can affect both hindlimbs. The distal aspect of MtIII appears at risk to develop stress-related bone injury before other sites in the metatarsophalangeal joint, possibly from anatomical variations or uneven loading during the full extension and landing phases of the stride. Shoe additives such as calks or grabs may play a mechanical role. The metatarsophalangeal joint is a high-motion joint and subject to high-strain cyclic fatigue (repetitive loading) that is encountered during intense exercise around turns and on hard surfaces. This continuum of stress-related bone injury can lead to osteoarthritis and fracture (see Fig. 43-7 and accompanying text).

Exercise intensity is reduced for 1 to 3 weeks. If lameness is prominent, a 3- to 4-month rest period is necessary. Exercise reduction allows the modeling and remodeling processes to equilibrate and microdamage to heal. A flat shoe is applied. Cold water therapy and the administration of NSAIDs (phenylbutazone or acetylsalicylic acid) appear helpful initially. Intra-articular injections of hyaluronan and PSGAGs may help, but early in disease, overlying cartilage damage and synovitis are not prominent, and response to intra-articular injections is minimal. When radiolucent changes and synovitis develop, response to intra-articular injections is better, but prognosis is worsened.

Fractures

Acute, severe lameness is seen most commonly in STBs with fractures of the metatarsophalangeal joint, but some horses can race with incomplete fractures. Horses shorten the cranial phase of the stride, land on the toe, and rapidly compensate with other limbs. Often no effusion or pain on flexion is apparent unless a fracture is displaced. Diagnostic analgesia and examination in harness are avoided if fracture is suspected, because comminution or displacement can occur. Radiographs, including a flexed dorsoplantar or 125° dorsoplantar and flexed lateromedial views, should be obtained.

Mid-sagittal fractures of the proximal phalanx are most common, causing severe intermittent or severe unrelenting lameness, depending on fracture length and configuration. Fractures can be short, incomplete with mild periosteal proliferation on the dorsal aspect of the proximal phalanx (Fig. 109-6) or long, incomplete or complete mid-sagittal fractures, fractures that break out of the lateral cortex, and comminuted fractures (see Fig. 36-1). Horses with short, mid-sagittal fractures often race or train, because lameness is intermittent and often abates after several days. Clinical signs precede radiographic evidence of fracture for 7 to 21 days and scintigraphic examination reveals focal IRU of the proximal aspect of the proximal phalanx (see Fig. 43-4). Horses with short, mid-sagittal fractures can be rested or have internal fixation and have a good prognosis with either treatment.[8] Long incomplete, complete, displaced, or comminuted fractures should be repaired. Prognosis depends on degree of comminution and displacement and whether a strut of bone is intact to which to attach fragments. Horses with comminuted fractures rarely race again and are at risk of infection, fracture displacement, and osteoarthritis of the metatarsophalangeal and proximal interphalangeal joints and contralateral laminitis (see Chapter 36).

Dorsal frontal fractures of the proximal phalanx can be unilateral or bilateral, are most common in the RH, and occur in trotters and pacers (see Figs. 43-12 and 43-13 and accompanying text). Fractures of the PSBs are common and most often involve the lateral PSB. Small medial abaxial fragments can be difficult to identify. Apical PSB fractures are most common, but abaxial, mid-body, and rarely basilar fractures occur, and the combination of suspensory desmitis, splint bone, and PSB fractures is not uncommon. Horses show acute lameness after training or racing, with grade 3 to 4 of 5 lameness, and prefer to bear weight only on the toe. Effusion and a positive response to flexion are usually present, and the diagnosis is confirmed radiographically. Ultrasonographic examination of the SL should be performed to establish baseline information and assess prognosis. Surgical removal of apical, abaxial, and basilar fragments and surgical repair of displaced mid-body fractures using bone screws or wiring techniques are recommended. Prognosis is guarded in trotters and fair in pacers that have raced before fracture. Horses in which fractures occur early in training have a poor prognosis.

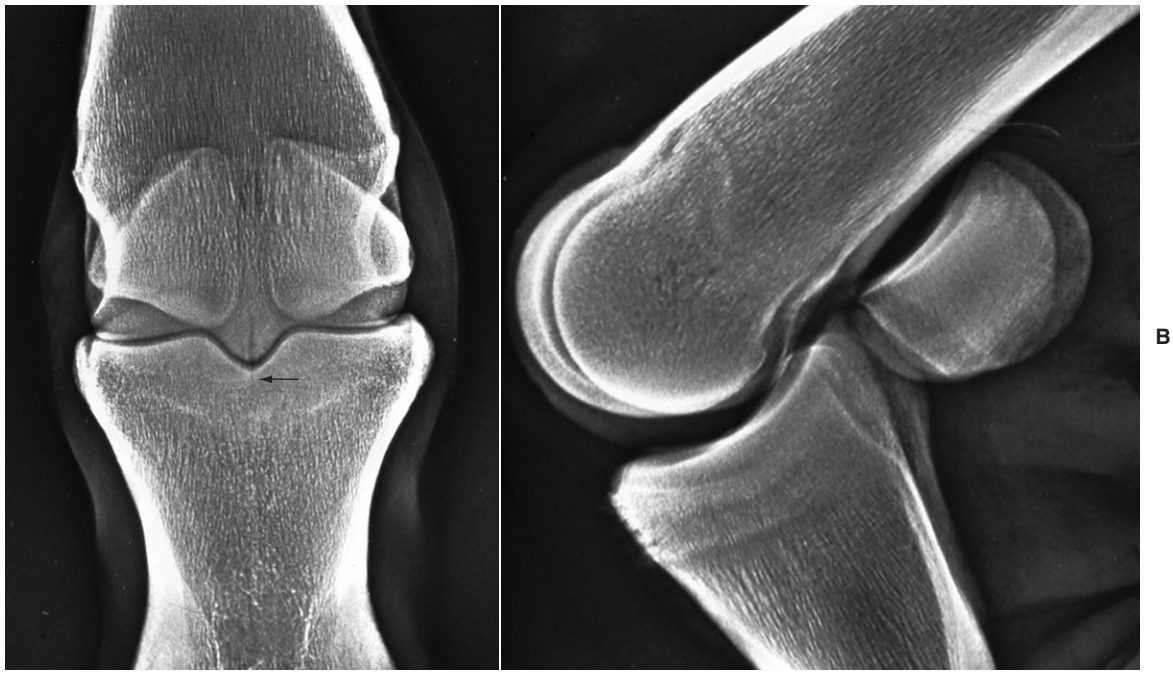

Fig. 109-6 **A,** Dorsoplantar and **B,** flexed lateromedial xeroradiographic images showing a short, incomplete, mid-sagittal fracture of the proximal phalanx *(arrow)*. There is slight periosteal new bone on the proximodorsal aspect of the proximal phalanx seen in the flexed lateromedial view. Radiographic changes often lag behind clinical signs and follow-up radiographic and scintigraphic examinations often are needed to establish a diagnosis.

Condylar fractures of MtIII occur, with similar distribution of short medial and lateral fractures. Clinical signs are similar to those caused by mid-sagittal fracture of the proximal phalanx, but diagnosis can be difficult unless good-quality radiographs and proper views are taken. A flexed dorsoplantar or 125° dorsoplantar view should be obtained. Medial condylar fractures often spiral proximally and should be repaired.

Osteochondrosis

The metatarsophalangeal joint is a major site for osteochondrosis, and the most common manifestation is plantar fragmentation. A complete discussion of axial, articular fragments and abaxial, non-articular fragments and other forms of osteochondrosis can be found in Chapter 43. Some debate exists over the management of STBs with axial, articular fragments and abaxial, non-articular fragments and other fragments. Horses with axial, articular fragments and dorsal fragments respond well to arthroscopic removal and post-operative intra-articular injections of PSGAGs or hyaluronan. We believe that surgical removal prolongs racing careers and decreases metatarsophalangeal joint lameness. Some horses, particularly pacers, race successfully without surgery.

Proliferative synovitis, radiolucent defects of the PSBs, traumatic small osteochondral fragments of the proximal phalanx, and osteoarthritis are discussed in Chapter 43.

Distal Hock Joint Pain and Other Tarsal Lameness

The hock joint is under considerable stress and strain, especially going into turns and with quick acceleration. Distal tarsitis, progressive osteoarthritis of the tarsometatarsal and centrodistal joints, is common. Horses warm out of lameness, often dramatically, because they may come out of the barn or paddock on the toe and eventually be able to train or race successfully. Horses show a mildly shortened cranial phase of the stride and a stabbing type of gait, with medial deviation of the limb during protraction and landing first on the outside toe (stabbing laterally), but this gait is not pathognomonic for distal tarsitis. Palpation in a flexed position often reveals pain of peri-articular soft tissue structures, and the Churchill test is often positive. Upper limb flexion is positive, but diagnostic analgesia is essential for diagnosis. Radiographs may show evidence of osteoarthritis in older horses, but in most 2- and 3-year-olds radiographs are negative or equivocal. Changes are seen most often in the DM-PlLO view, but dorsoplantar, lateromedial, and slightly off dorsoplantar views can be helpful. Scintigraphic examination confirms that osteoarthritis of the tarsometatarsal joint is most common, and changes are lateral and dorsolateral (Fig. 109-7). Focal IRU is sometimes seen involving only the centrodistal joint in horses that failed to respond to intra-articular analgesia of the tarsometatarsal joint, emphasizing the need to block and treat each joint separately.

Treatment comprises intra-articular injections, NSAIDs, shoeing changes, and rest. Intra-articular hyaluronan is useful to decrease inflammation, and concomitant use of NSAIDs such as phenylbutazone (4.4 mg/kg PO sid for 5 days and 2.2 mg/kg PO sid for 5 days) is recommended initially. As osteoarthritis progresses, methylprednisolone acetate (80 mg per joint every 6 to 8 weeks) is necessary to manage inflammation. A series of intra-articular and intramuscular injections of PSGAGs also can be given. In horses with severe osteoarthritis a period of 4 to 6 months of rest is successful, but recurrent lameness often mandates a limited racing schedule (10 to 15 starts). The toe is shortened, and a flat aluminum or steel squared-toe shoe is applied to allow easy breakover. All shoe additives are removed to decrease shear stress.[8]

Slab fractures of the third tarsal bone and proximal aspect of MtIII result from stress-related bone injury or from pathological fractures in horses with osteoarthritis. Third tarsal bone fractures are the most common and are best seen on a DM-PlLO view. Fractures of the central tarsal bone are usually

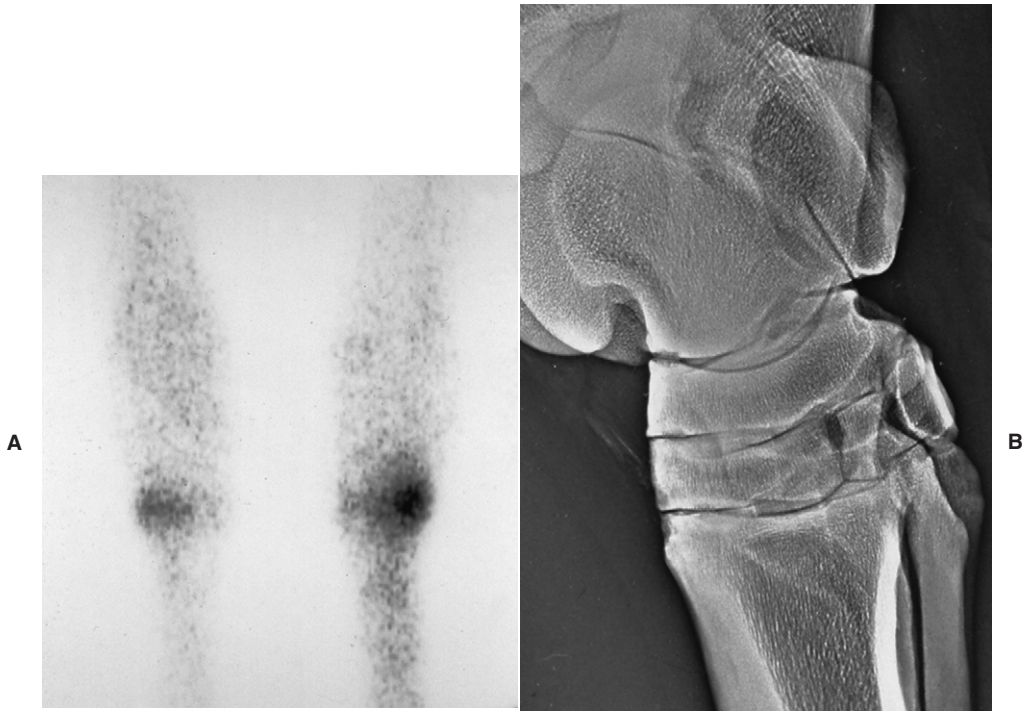

Fig. 109-7 **A,** Plantar delayed (bone) phase scintigraphic image of the hocks. There is focal, intense increased radiopharmaceutical uptake in the dorsolateral aspect of both tarsometatarsal joints (left hindlimb is on the right) in this 2-year-old Standardbred filly. **B,** Dorsomedial-plantarolateral oblique xeroradiographic projection of the left hindlimb showing minimal changes. Lameness and scintigraphic evidence of distal tarsitis precede radiographic changes by many months.

oblique, involve the dorsal aspect of the bone, occur without concomitant osteoarthritis, and are best seen in lateromedial views. Conservative management is recommended in horses with non-displaced fractures, but displaced fractures should be repaired. Ninety-two percent of horses with slab fractures of the third tarsal bone and 88% with slab fractures of the central tarsal bone raced after conservative management.[9]

The tarsocrural joint is often injected, but tarsocrural joint lameness is uncommon, except in young horses with osteochondrosis. Trainers often request injection of the upper and lower hock joints (tarsocrural and tarsometatarsal joints), but lameness most likely is caused by distal tarsitis. Bog spavin occurs most commonly in weanlings and yearlings, and radiographic identification of osteochondritis dissecans fragments prompts surgical removal. Most yearlings begin training without fragments. Although debate exists about the significance of tarsocrural osteochondrosis and lameness, we recommend surgical removal to resolve current inflammation, if present, and to prevent future lameness. However, osteochondrosis fragments are seen in some older racehorses in which neither lameness nor effusion are present. Tarsocrural lameness may occur without effusion and diagnostic analgesia is necessary to differentiate the site of hock pain. Good-quality radiographs are essential to discover occult osteochondritis dissecans fragments and fractures. The DM-PlLO view is best for evaluating the cranial intermediate ridge of the distal tibia and an off dorsoplantar view (dorsal 5° to 7° lateral-plantaromedial oblique) is best to evaluate for medial malleolar fragments. The most common sites for osteochondrosis are the distal intermediate ridge of the tibia, lateral trochlear ridge of the talus, medial malleolus of the tibia, medial trochlear ridge, and lateral malleolus. Osteochondrosis fragments can be large, numerous, loose, and free floating, and cartilage damage can be prominent even in yearlings, but with removal prognosis is good. If distention of the tarsocrural joint is unexplained, osteochondrosis of the medial malleolus of the tibia should be suspected, and diagnostic arthroscopy is recommended. Small osteochondrosis fragments (dewdrop lesions) of the distal medial trochlear ridge may be incidental, but can cause lameness if displaced, or if impinging on the central tarsal bone. Surgical removal is recommended.

Tarsocrural osteoarthritis occurs in older horses (usually >5 years) and may be associated with osteochondrosis, because fragments often are seen concomitantly. Osteoarthritis causes progressive lameness and effusion, but intra-articular analgesia should be performed to validate the source of pain. Large volumes of local anesthetic solution (30 to 50 ml) are necessary, because pain is severe; false negatives occur if 10 to 15 ml is used. Radiographs may reveal sclerosis and narrowing of the tarsocrural joint space, but end-stage osteoarthritis can be present with only subtle radiographic findings. Scintigraphic evaluation reveals diffuse IRU on both sides of the tarsocrural joint and is useful to differentiate osteoarthritis from sagittal fracture of the talus or subchondral injury of the distal tibia. Prognosis is grave.

Sagittal fracture of the talus is unusual. Acute, grade 3 to 4 of 5 lameness is localized to the tarsocrural joint by diagnostic analgesia, but often subtle tarsal lameness precedes fracture. Focal IRU is seen involving the talus, and off dorsoplantar views reveal subchondral lucency or fracture (Fig. 109-8). Of eight horses managed conservatively, seven raced and one was sound in training.[10]

Enthesitis of the long, lateral collateral ligament of the tarsocrural joint, on the lateral aspect of the calcaneus, results in mild to moderate lameness and mild tarsocrural effusion (Fig. 109-9). Soft tissue swelling is easy to miss initially, and radiographic changes lag behind clinical signs for at least 14 to 21 days, but lameness abates with intra-articular analgesia.

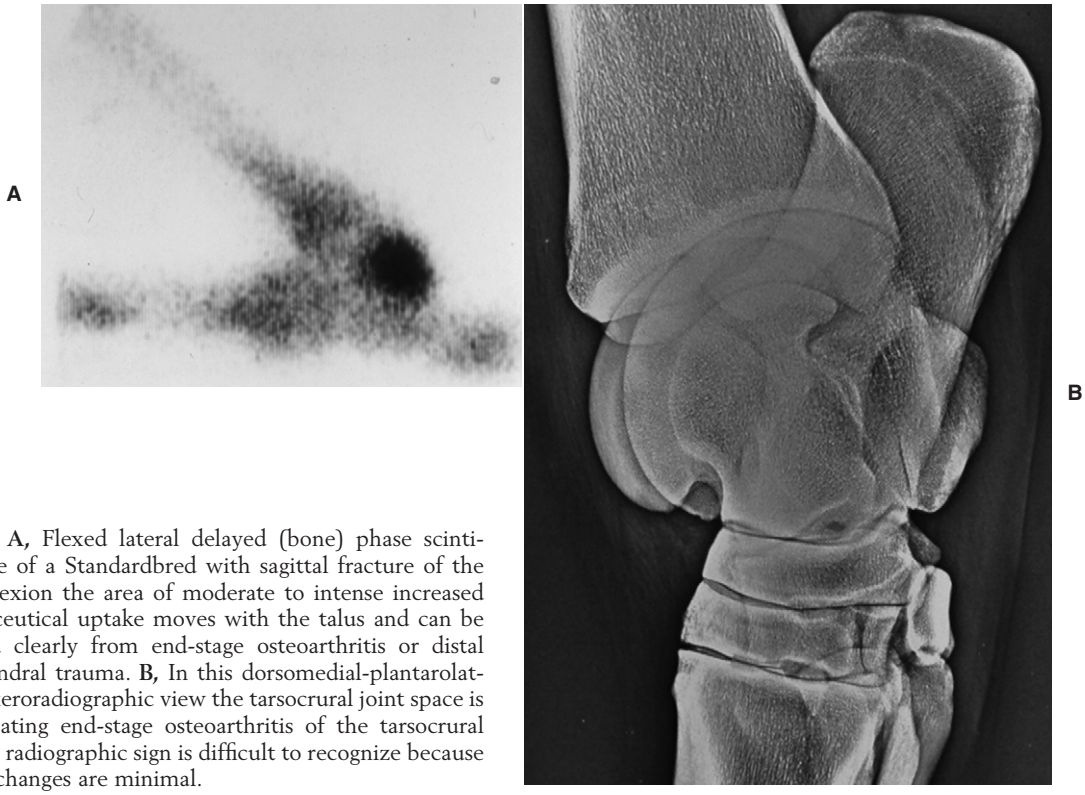

Fig. 109-8 **A,** Flexed lateral delayed (bone) phase scintigraphic image of a Standardbred with sagittal fracture of the talus. With flexion the area of moderate to intense increased radiopharmaceutical uptake moves with the talus and can be differentiated clearly from end-stage osteoarthritis or distal tibial subchondral trauma. **B,** In this dorsomedial-plantarolateral oblique xeroradiographic view the tarsocrural joint space is narrow, indicating end-stage osteoarthritis of the tarsocrural joint, but this radiographic sign is difficult to recognize because proliferative changes are minimal.

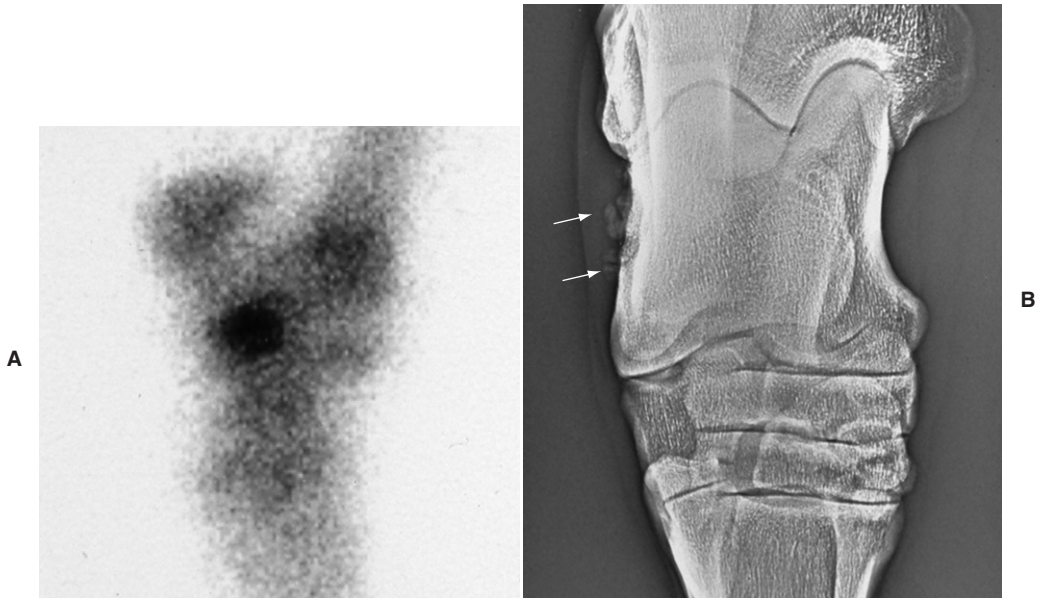

Fig. 109-9 **A,** Lateral delayed (bone) phase scintigraphic image of a hock showing focal, moderate to intense increased radiopharmaceutical uptake of the calcaneus. **B,** Dorsoplantar xeroradiographic view showing proliferative changes associated with the collateral ligament attachment on the lateral aspect of the calcaneus *(arrows)*.

Scintigraphic examination reveals focal IRU in the lateral calcaneus. Local injections and radiation therapy may allow continued racing, but rest to allow enthesitis to subside is often necessary.

Injury of the fibularis tertius tendon anywhere from the mid-crus to the distal tarsus can occur, but it is most common at the insertion on MtIII and the third tarsal bone. Soft tissue swelling is present, and the hock can be pushed forward without concomitant extension of the stifle with the limb in standing position. Horses require 4 to 6 months of rest.

Inflammatory synovitis or infection of the tarsocrural joint is not uncommon. Prognosis depends on the extent of existing cartilage damage and the financial ability to manage infection aggressively (see Chapter 66).

Suspensory Desmitis

Suspensory desmitis is the most common soft tissue injury and can be classified as unilateral branch desmitis, bilateral branch desmitis, body desmitis, and PSD. Suspensory desmitis is often an overload compensatory lameness condition, because fatigue and improper landing occur in lame horses. Suspensory desmitis is seldom a single-event injury but is caused by accumulated damage over weeks to months (see Chapter 73). Diagnosis is made by careful palpation from the origin to the insertion of the SL with the limb in standing and flexed positions. The splint bones and PSBs are examined for associated injuries. One of us (J.B.M.) has found that horses with core lesions are lamer, but exhibit fewer outward signs of inflammation, than those with peripheral lesions. Horses with suspensory branch desmitis are common and heal quicker (6 to 8 weeks) with lower recurrence than do those with body desmitis and PSD (4 to 6 months). Core lesions recur frequently. Because suspensory desmitis is often a compensatory injury, the primary lameness needs to be resolved. For example, osteoarthritis and osteochondral fragments of the left carpus frequently cause suspensory desmitis of the RF in horses of both gaits, RH suspensory desmitis in trotters, and LH suspensory desmitis in pacers.

Ultrasonographic examination is critical in assessing the SL and its entire length should be examined systematically. In hindlimbs, enlargement of the origin and body is more common than core lesions, and cross-sectional area measurements of the affected and contralateral limb are mandatory. Branches are evaluated for change in shape, loss of margin definition, central or marginal hypoechoic lesions, subcutaneous hemorrhage and edema, fibrosis and calcification, irregular contour (step line defects), and avulsion fragments of the PSBs. The intersesamoidean ligament rarely is involved.

Forelimb unilateral branch desmitis results from toed-in or toed-out conformation, hoof imbalance, and uneven landing. To correct interference injury with knee knocking resulting from toed-out conformation, the hoof is commonly lowered on the inside, causing uneven loading and medial unilateral branch desmitis, whereas in horses with toed-in conformation, lateral unilateral branch desmitis occurs. Improper impact can occur from fatigue or if a horse is stopped suddenly. Lameness varies with severity of injury and presence of associated splint and PSB disorders.

Forelimb bilateral branch desmitis is caused by acute or chronic hyperextension of the metacarpophalangeal joint. Prognosis depends on the degree of fetlock drop seen when the contralateral limb is held in flexion. Concomitant PSB fractures can occur. Dorsal subluxation (flexion) of the proximal interphalangeal joint is seen in horses with bilateral branch desmitis and body desmitis, whereas palmar subluxation (extension) is seen in horses with distal sesamoidean desmitis and loss of palmar support. If the axis of the proximal interphalangeal joint is altered, the prognosis for racing is poor.

Forelimb body desmitis often begins just proximal to the bifurcation of the SL, and careful palpation reveals pain and mild enlargement. The first sign may be mild swelling, without lameness, and training and racing are often continued, leading to further suspensory desmitis. Horses often race successfully with chronic suspensory branch desmitis, but if desmitis progresses into the body, lameness becomes pronounced. Chronic enlargement and thickening of the SL often causes bowing of the splint bones, and concussion leads to fracture. Horses with recalcitrant swelling of the SL should be examined with ultrasonography, because exuberant exostosis from fractures may encroach on the SL and cause swelling and pain.

Proximal palmar metacarpal pain is common and often called a check ligament problem, although genuine desmitis of the accessory ligament of the DDFT is rare. Palmar metacarpal pain can be caused by avulsion or longitudinal fracture of McIII and stress reaction/stress fracture of McIII, but PSD is most common. STBs are often painful to palpation of the proximal palmar metacarpal region and may have compensatory PSD, with primary lameness elsewhere. Lameness is noticed in the first half of the support phase of stride. An exaggerated head nod is often noted (J.S.M.). A carpal type of gait may be seen. Lameness is most prominent when the affected limb is on the outside of a circle. Local analgesia and ultrasonographic examination should be performed. Radiographs are necessary to identify bone injury. Scintigraphic examination is useful to differentiate proximal metacarpal from carpal injury.

In hindlimbs unilateral branch desmitis, bilateral branch desmitis, body desmitis, and PSD occur but less frequently than in forelimbs. Degree of lameness varies and similar concerns exist about MtIII, splint bone, and PSB involvement. Horses often land on the toe and are reluctant to put the heel down, especially if a concurrent pathological condition of the PSB exists. Suspensory branch desmitis often occurs first and can be managed successfully with continued work in some horses, but proximal progression toward the bifurcation and body causes substantial increase in lameness and may involve the splint bones, and often horses lose support of the metatarsophalangeal joint. Racing can no longer continue. Hindlimb suspensory desmitis is particularly important in young trotters. Many top 2- and 3-year-old trotters have been retired because of hindlimb suspensory desmitis. Second to carpal lameness, hindlimb suspensory desmitis is the most dangerous, career-limiting cause of lameness in trotters.

Early PSD can go unrecognized because of lack of obvious swelling. Local analgesia is critical for diagnosis. Once desmitis involves the body, swelling may be recognized, but damage is now extensive.

Inflammation can be suppressed, but rest is essential for healing. Rest is not well received by owners and trainers, who often exert pressure to keep a horse in work. Cryotherapy is used to manage horses with suspensory branch desmitis and distal body desmitis. Horses with marginal lesions respond well, but not those with core lesions. Cryotherapy decreases pain and seems to promote more or better-organized scar tissue. Periligamentous injections of sclerosing agents such as sodium oleate promote fibrosis and can be used to cause a swollen SL to set up. Periligamentous injections of corticosteroid mixtures reduce inflammation and lameness, but if training and racing continue, further damage occurs. Many naive owners lack understanding that the latest popular injection does not promote healing but simply masks inflammatory signs. Although horses may continue to race a few more starts, further damage inevitably occurs. Horses with PSD often can be managed using injections of corticosteroids, internal blisters and, if available, radiation therapy and, if lameness is mild and support is not lost, racing may continue. Shock wave therapy has gained prominence and shows promise.

Rest is the best approach, and between 3 to 6 months of rest may be necessary to allow healing. A combination of stall rest, hand walking and walking in the jog cart, and swimming is recommended. We do not recommend turnout, but many horses are blistered and turned out, although this has been shown to be detrimental to healing. Healing progress is assessed by ultrasonography. Surgical management using a modified Asheim approach and ostectomy of fractured splint bones and PSB fragments are recommended. One of us (M.W.R.) has performed bone marrow injection and fasciotomy on a limited number of top trotters with hindlimb PSD and body desmitis with modest success.

Prognosis depends on level of injury, gait (trotters worse than pacers), severity of injury (poor prognosis with fetlock

drop), and associated bony pathological conditions. Unless the SL is totally disrupted, prognosis for racing is good, but racing class is an important consideration, because few STBs return to the same or improved race class after sustaining suspensory desmitis.

Metacarpophalangeal Joint Lameness

Fatigue from underconditioning and other factors causing overload may lead to hyperextension of the metacarpophalangeal joint. Hyperextension leads to dorsal injury, such as small osteochondral fragments of the proximal phalanx and proliferative synovitis and to palmar injury, such as fractures of the PSB and suspensory branch insertional injury. Traumatic disruption of the suspensory apparatus is rare but usually involves rupture of the SL. Stress-related bone injury of subchondral bone leading to osteoarthritis and fracture occurs in the metacarpophalangeal joint, as described for the metatarsophalangeal joint. In a STB with normal conformation, metacarpophalangeal effusion should be a red flag (a barometer), often a sign of compensatory lameness. Young horses often have bilateral metacarpophalangeal synovitis secondary to palmar heel pain, because toe-first landing overloads the joint. Corrective trimming and shoeing often helps. In older horses unilateral carpal lameness often causes contralateral metacarpophalangeal synovitis. Primary lameness of the metacarpophalangeal can begin when horses break stride, stumble, or stop quickly.

Osteoarthritis and stress-related bone injury are the most common problems of the metacarpophalangeal joint. Early radiographs are usually negative, but radiolucency and sclerosis of distal medial McIII develop later. Scintigraphic evidence of IRU is more prominent medially, unlike in the metatarsophalangeal joint in which IRU is more common laterally. In older horses (about 4 years of age) with progressive severe osteoarthritis, narrowing of the medial joint space indicates severe cartilage damage. Owners are incredulous that the horse has end-stage osteoarthritis, because lameness can be acute and severe, and fracture often is suspected.

Early osteoarthritis is treated using intra-articular injection of hyaluronan alone or a combination of hyaluronan and PSGAGs. The latter is preferred and 3 to 4 weekly injections are combined with a reduction in exercise. Horses that do not respond may have proliferative synovitis, usually seen in older horses with chronic osteoarthritis. Injecting the joint in the dorsal pouch may be difficult and little synovial fluid may be present. Surgical resection of synovial pads and post-operative intra-articular injections of PSGAGs are recommended.

Fractures and osteochondritis dissecans occur, but palmar fragments are rare (see Chapter 37).

Splint Bone Disease

Exostoses and Fractures of the Second and Fourth Metacarpal Bones (Splints)

Splints are a common problem in 2- and 3-year-olds (see page 898). Poor conformation and hard track surfaces predispose horses to splints. Whether tearing of the interosseous ligaments occurs is unknown. Medial splints are most common. Yearlings can begin training with dormant, so-called field splints that become active as training advances. Splints rarely cause severe head nodding lameness, but put a horse on a line and signs become worse as work progresses. Splints on the abaxial surfaces of the McII and McIV are visible and can be palpated readily, but those on the axial surfaces require the limb to be lifted to relax the SL. Axial splints cause more obvious lameness than do abaxial splints.

Management includes reducing inflammation and limiting callus. Cold-water therapy and poultice should be applied, and horses are given NSAIDs. Blistering and external heat therapy may be contraindicated if reduction in the size of the callus is desirable. Ionophoresis with corticosteroids is successful. Peri-lesional injection of corticosteroids and cryotherapy are used successfully. If signs fail to abate, a fracture may be present. Improper toe-heel impact and mediolateral hoof imbalance predispose horses to splints. Chronic splint disease in older horses is almost always related to palmar heel pain. Aggressive therapy for palmar heel pain and restoring normal hoof strike is critical. Direct trauma from interference and breaking must be prevented.

Fractures of McII and McIV can be caused by direct trauma. Interference injury can cause fracture of McII, and accidents involving carts and other equipment predispose horses to fractures of McIV. If fractures are located proximally and displacement exists, surgical fixation using small plates and screws is best. McIV fractures can heal functionally without fixation. Most distal splint bone fractures are secondary to suspensory desmitis. Exuberant callus can impinge on the SL. Fracture callus and fragments should be removed.

Exostoses and Fractures of the Second and Fourth Metatarsal Bones

Exostoses most commonly involve MtII, but chronic painful splint exostoses are not as common as in the forelimb. Perilesional corticosteroid therapy is recommended (80 mg methylprednisolone and 8 ml of Sarapin). Fractures of MtII and MtIV occur occasionally and usually result from direct trauma, but distal splint fractures can be secondary to suspensory branch desmitis. Fracture fragments should be removed.

Stifle Joint Lameness

The stifle joint often is incriminated as a source of pain, but diagnostic analgesia identifies a problem in the more distal part of the limb. Osteoarthritis of the medial femorotibial joint is the most important stifle lameness, but in young horses osteochondrosis should be considered. Osteoarthritis is a progressive disease beginning in late 2-year-olds, and degree of lameness varies from grade 1 to 4 of 5. Distention of the medial femorotibial joint capsule is an important finding. The limb may be carried forward slightly lateral or outward during the cranial phase of the stride. The cranial phase is shortened and lameness is worse in the turns. Radiographs are usually negative or equivocal in 2-year-olds, and arthroscopic evaluation reveals generalized cartilage thinning and fissure formation. Later, in caudocranial radiographic views, narrowing of the medial femorotibial joint space and mild osteophyte formation on the proximal medial tibia and distal medial femur are visible (Fig. 109-10). Scintigraphic examination can confirm extensive osteoarthritis. Diffuse IRU involves both sides of the medial femorotibial joint. Arthroscopic evaluation at this time reveals exposed subchondral bone and severe synovitis, but therapeutic value of arthroscopy is negligible. If the diagnosis of osteoarthritis is made in a 2-year-old, a period of 3 to 4 months of rest is recommended. The stifle joint often is injected in horses without stifle lameness and few untoward effects are seen. Treatment of osteoarthritis with methylprednisolone acetate is contraindicated early in the course of the disease. Although the drug is effective and allows continued racing, osteoarthritis can progress rapidly, resulting in severe lameness. Hyaluronan (40 mg) and isoflupredone acetate (4 to 6 mg), or PSGAGs (750 to 1000 mg), are preferred, and although they are less effective than methylprednisolone acetate, they decrease inflammation and prolong the racing career. Systemic administration of PSGAGs or hyaluronan is helpful. Methylprednisolone acetate is necessary for older horses with advanced osteoarthritis, but if no improvement is seen, the racing career is usually over. Once severe lameness occurs, rest may improve comfort slightly, but horses will not tolerate training again. Trotters can tolerate racing with more advanced osteoarthritis than can pacers.

In young horses osteochondrosis can cause lameness and effusion in early training, prompting radiographic examination, but signs of osteochondrosis often are seen in weanlings and yearlings, and surgery is performed before training begins. Osteochondritis dissecans of the lateral trochlear ridge of the femur is most common, causes considerable effusion of the femoropatellar joint, and if bilateral may cause a bunny-hopping gait. Surgical treatment, 4 to 6 months of rest, and intra-articular therapy are recommended. Prognosis for racing is good, unless lesions are severe or cartilage damage of the patella is advanced. Subchondral bone cysts most commonly involve the medial femoral condyle but can occur in the proximal tibia. Lameness is often progressive, with horses first being on a line. Surgical debridement of the cyst is recommended. Horses need a minimum of 6 to 8 months of rest to allow healing and for lameness to abate. Prognosis is only fair (50% to 70%) for successful racing.

Rhabdomyolysis and Muscle Soreness

We divide muscle lameness into metabolic disease (recurrent exertional rhabdomyolysis, RER) and non-metabolic disease (muscle soreness). Signs vary, but horses appear stiff and hindlimbs are not fully engaged in the stride unilaterally or bilaterally. Palpation often reveals muscle pain but must be done carefully, because aggressive, sudden, and deep palpation elicits false-positive responses.

RER occurs more commonly in females and can cause swollen muscles. Overt clinical signs of tying up after racing and training can be seen, but subclinical RER can be a cause of poor performance. Myoglobinuria and elevation of serum creatine kinase (CK) and aspartate aminotransferase (AST) levels are diagnostic. Horses are managed initially with intravenous fluids, NSAIDs, tranquilization, and methocarbamol (22 mg/kg IV or 33mg/kg PO bid). Care must be taken when using acetylpromazine and methocarbamol simultaneously, because effects may be additive and horses may act severely tranquilized (J.S.M.). Walking is recommended in horses with mild RER but is contraindicated in those with severe RER. In horse with severe RER dantrolene sodium (2 mg/kg IV or via nasogastric tube) and intravenous dimethylsulfoxide solution are recommended. Large quantities of intravenous or oral fluids should be administered. After an acute episode, signs may recur or subclinical myositis may persist, reflected by elevations in CK and AST concentrations. Enzyme levels should return to baseline before intense training or racing resumes.

In horses with subclinical RER or those with recurrent acute episodes, we recommend a management protocol that includes a reduction in carbohydrate intake, especially before exercise, a high-fat diet, vitamin E and selenium supplementation, supplementation with oral potassium, and a regular 7-day exercise program combining jogging, brief periods of intensive training, and paddock exercise, if feasible. Chronic administration of dantrolene sodium (2 mg/kg) or phenytoin (8 to 10 mg/kg PO bid) is recommended, but racing jurisdiction rules must be followed.

Non-metabolic myositis is common, and gait is similar to that described for stifle lameness. Signs are similar to RER, but serum CK and AST levels are usually within or just above normal limits. The middle gluteal muscle and associated tendons commonly are affected unilaterally or bilaterally. Myositis may be a secondary problem or a primary source of pain after traumatic injuries such as accidents during transport, paddock injuries, slipping while racing on muddy or other undesirable surfaces, or by making a break. Horses are managed initially with muscle relaxants, NSAIDs, and rest (5 to 7 days). Horses with chronic soreness are treated with an internal counterirritant or corticosteroids injected strategically in the sore areas (see Chapter 89).

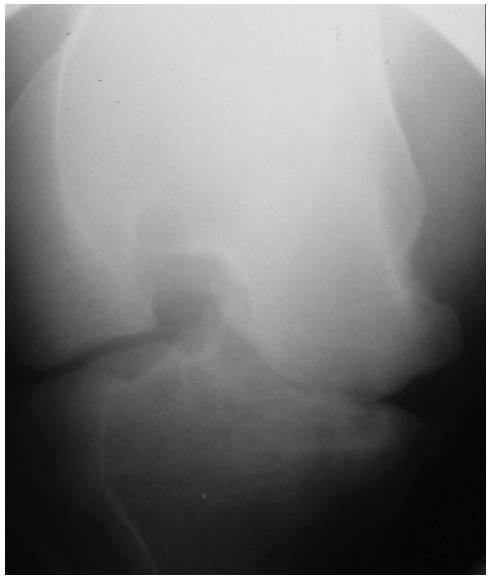

Fig. 109-10 Caudocranial radiographic view of a stifle showing advanced narrowing of the medial femorotibial joint space in a pacer with advanced osteoarthritis. Medial is to the right. Narrowing of medial femorotibial joint is often the only radiographic sign of advanced osteoarthritis of this joint. Subchondral lucency of the medial femoral and tibial condyles and osteophyte formation also are seen in this pacer, and unfortunately this horse will not race again.

Curb and Superficial Digital Flexor Tendonitis

Other soft tissue injuries such as curb and superficial digital flexor tendonitis occur commonly. Curb in the STB is discussed in detail in Chapter 79. Superficial digital flexor tendonitis is discussed in detail in Chapter 70.

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CHAPTER • 110

The European and Australasian Standardbreds

THE EUROPEAN STANDARDBRED

• Fabio Torre

DIMENSIONS AND CHARACTERISTICS

The European Standardbred (STB) is historically the result of different crosses between the classic American bloodlines and European families. The French STB is known for its stamina and aptitude for long distances. The American-derived families, however, have been selected for speed and racing at a fixed distance of 1 mile and are represented more widely in Italy and Sweden. Several countries (especially France, Italy, Germany, and Scandinavian countries) have developed individual bloodlines and today are able to produce good-quality STB trotters. In Europe no races are available for pacers. In Table 110-1 the STB population and racing activity of different European countries are listed.

Europe has a large number of racetracks for STBs, and they are characterized by different circuit lengths, surfaces, and designs. Half-mile tracks have been replaced progressively by 1-km (⅝-mile) tracks, and no 1-mile racetracks (with the exception of training centers) are present in Europe. The most characteristic and popular racetrack is the Plateau de Gravelle in Vincennes, Paris, where the Prix d'Amerique is held. The track surface is made in pressed charcoal powder, slopes and descents are present, and the circuit is egg-shaped, which is unique in Europe where other racetracks have a regular design. Another famous French racetrack, Cagnes-sur-Mer in Nice, is well known for being 1200 m in length and for hosting the Criterium de Vitesse, one of the main European free-for-all races. In Sweden, Solvalla racetrack in Stockholm represents the heart of the Swedish STB racing, and in late May the multiple-heat race Elitlopp is held. In Norway, next to well-developed STB racing (the main racetrack being Bjerke near Oslo), races for Warmblood horses (a breed historically devoted to hard work but now genetically selected for speed) are still popular and meet the special interest of the local public.

A variety of track designs and purposes are present in Europe. For example, a large number of small country tracks are found in France and local races are organized in a fair-like fashion, whereas the main racing activity is concentrated in Paris. In Italy, however, important races have a more even distribution among racetracks, Milan and Rome being the main places. In Table 110-2 the distribution of racing tracks in the main European countries is summarized.

Differences exist in availability of prize money among different countries, and this makes France and Italy the most attractive countries for trainers and owners. A number of well-known trainers started to move in the early 1980s toward France and Italy, especially from Northern Europe, and this has enhanced the exchange of experiences between trainers and veterinarians.

Prize money distribution among horses of different ages has strongly influenced racing, especially in Italy, where 2- and 3-year-olds have the richest races. Higher purses for younger horses have also affected breeding programs and have stimulated an interest in many of the orthopedic problems affecting young horses, especially osteochondrosis, which has been studied widely in Northern Europe.

Table • **110-1**

Distribution of Standardbreds Born in Different European Countries, Summary of the Racing Activity, Earnings, and Betting

COUNTRY	NEWBORNS PER YEAR (NO.)	RACING DAYS (NO.)	RACES (NO.)	RACING STANDARDBREDS (NO.)	TOTAL EARNINGS (EURO)	MONEY FOR BETTING (EURO)
Denmark	870	404	3,702	3,367	5,963,019	52,414,070
Finland	1,750	585	3,716	5,159	8,230,345	174,133,572
France	12,062	1,773	9,980	13,188	133,880,408	2,851,925,331
Germany	2,049	811	9,609	7,242	26,137,229	168,811,395
Holland	548	170	1,506	1,508	3,422,242	38,163,810
Italy	4,700	1,606	13,823	10,158	73,504,710	1,336,481,000
Norway	890	502	2,281	2,753	18,724,000	253,123,200
Sweden	5,040	937	8,529	12,942	63,781,182	1,045,013,432
Austria	310	124	1,206	1,144	3,060,177	9,748,299
Belgium	706	190	1,628	1,836	2,414,831	37,602,040
Spain	372	289	1,918	970	686,334	1,187,325

Data from UET (European Trotting Union), update December 1999.

Table • **110-2**

Racetrack Distribution in Different European Countries

COUNTRY	RACETRACKS (NO.)*	HALF-MILE RACETRACKS (NO.)	1-KM RACETRACKS (NO.)	>1-KM RACETRACKS (NO.)
Denmark	9	1	7	1
Finland	43	0	43	0
France	240	0	0	4
Germany	16	3	3	5
Holland	7	1	3	1
Italy	25	11	13	0
Norway	9	1	7	0
Sweden	26	1	25	0

Data from UET (European Trotting Union), update December 2000.
*The total number includes all racetracks in which at least 1 day of racing is held, but less important, occasional, and fair-like tracks (which are especially popular in France) are not considered in the subsequent columns.

APPROACHING LAMENESS PROBLEMS

Only STB trotters are allowed to race in Europe, and this makes the trainer's work more challenging. Any possibility to switch gaits or to move an unnaturally fast gaited trotter to a potentially good pacer is precluded. When the fast gait is compromised by biomechanical problems and significant interference between limbs (unlike in the pacer), corrections aimed at avoiding interference are attempted. Finding a solution for mechanical problems is not always possible, and corrective shoeing that alters the natural gait may cause compensatory lameness in different locations.

A series of mechanical limitations may be present at the beginning of training, and relatively soon they are followed by pain-related problems, usually affecting joints and less frequently digital flexor tendons and the suspensory ligament. A less than 100% natural trotting athlete is more likely than a natural trotter to have its gait totally compromised, even by a mild subclinical lameness problem, and consequently gait limitations increase. However, anything that is attempted by the trainer to improve gait (corrective shoeing, special equipment that prevents turning the head and neck toward one side, or shifting the hindlimbs toward one shaft) forces the horse to a non-natural fast gait and often results in lameness. In fact, when a non-natural gait is forced, the end result is usually that a single limb (or a biped) is overloaded, and the uneven loading is exacerbated by the progression of training.

When young horses start training, they frequently have been subjected to basic lameness and radiographic evaluations. This allows trainers the opportunity to treat conditions such as osteochondrosis or to be aware of other abnormalities. Prepurchase radiographic examinations and, when needed, preventative arthroscopic surgery (mostly for osteochondrosis of the tarsocrural joint and osteochondral fragments in the fetlock joints) are now practices that have received general acceptance. The reason to operate early is to have the horse rested before any training program is started. In yearlings eligible for autumn sales, it is important to perform surgery early to have presale radiographs without osteochondral fragments and to decrease effusion before the sale.

When moderate gait anomalies are present, experienced trainers usually give the horse time and keep going with a light exercise program instead of making radical changes. This allows, in many horses, a complete maturation of the equine athlete and, when the growth is complete and the muscular function well conditioned, the gait in many horses automatically improves without injuring the immature skeleton. Shoeing is also central in early training. Light plastic shoes are ideal to allow foot growth and expansion and to minimize trauma in the early phases of fast training.

TEN MOST COMMON LAMENESS CONDITIONS

The following are the 10 most common lameness conditions:

1. Hoof or foot pain
2. Osteoarthritis of the distal interphalangeal joint
3. Osteoarthritis of the metacarpophalangeal joint
4. Lameness of the middle carpal joint
5. Proximal palmar metacarpal pain including proximal suspensory desmitis (PSD)
6. Sesamoiditis
7. Suspensory branch desmitis
8. Lameness of the metatarsophalangeal joint
9. Superficial digital flexor tendonitis
10. Osteochondrosis of the tarsocrural joint

LAMENESS EXAMINATION

Horses with acute, severe lameness should be allowed to rest. Radiographs are frequently diagnostic, revealing the most common severe musculoskeletal injuries affecting trained STBs, such as incomplete sagittal fractures of the proximal phalanx, fractures of the proximal sesamoid bones, splint bone fractures, fractures of the third or radial carpal bones, fractures of a palmar process of the distal phalanx, fractures of the lateral condyle of the third metacarpal bone (McIII), slab fractures of the third tarsal bone, and stress fractures of the palmar aspect of McIII or the plantar aspect of the third metatarsal bone (MtIII). Apical fractures of the proximal sesamoid bones (PSBs) are a common injury in young STBs, and the lateral sesamoid in the hindlimbs is the most common location.[1]

The conditions mentioned previously represent injuries of the racing STB requiring rest or surgical repair. More commonly the veterinarian is consulted for mild or obscure lameness, gait disturbances, or poor performance. In any case a thorough history is mandatory before the lameness examination is initiated.

A basic lameness history must include the following:

- What is the trainer's complaint?
- What is the horse's gait (naturally born versus artificial trotter)?

- Describe shoeing management: recent changes, difficulties in combining appropriate shoeing and fast gait, ideal shoeing for the horse, and attempts at correcting the problem
- What is the horse's recent performance?
- When was the onset of the problem and correlation with previous lameness, if any?
- When does the problem arise during the race? Does the horse worsen in the turns or on the straight? Is the horse better at the beginning of the race and does it worsen at the end?
- Is the horse lame after the race?
- What about the day after the race?
- How is the horse during daily jogging, a question best addressed to the groom or assistant trainer?
- How does the horse behave when trained clockwise (though races are counterclockwise)?
- Is the horse better when trained on a straight track, when available?
- Does the problem worsen on a particular racetrack and specifically on hard tracks?
- Is the horse on a line?
- Is the horse on a shaft?
- Does the horse break stride? If so, when? At the start, approaching turns, in turns, coming out of turns, or in the straightaway?
- Does the horse deviate left in the turns and right on the straight lines? Does the tendency worsen at the end of the race?
- Has the horse been submitted to any previous lameness examination?
- Was the horse subjected to any previous treatment with paints, ointments, or local injections?
- Did previous therapy lead to any improvement?
- Was any other problem diagnosed or suspected (exercise-induced pulmonary hemorrhage, rhabdomyolysis)?

Concerning conformation, the clinician should check the following:

- Foot conformation (club foot or low heels, toed in, toed out, hoof wall angle, correction of the lateral-to-medial balance, quality of the horn, characteristics of the sole and quarters, type of shoeing). Club feet may indicate osteoarthritis of the distal interphalangeal joint. Toed-out horses have the most frequent gait disturbances because they tend to hit the contralateral carpus or the ipsilateral metatarsal region. Toed-in horses have less significant gait problems, but the uneven distribution of the weight is likely to produce lameness of the middle carpal joint or suspensory desmitis.
- Torsional defects proximal to the hoof (fetlock and carpus and, more rarely, the tarsus), uncorrected angular limb deformities, offset (bench) knees, tied in behind the knees, straight conformation of the hindlimbs. Any of these abnormalities invariably produce secondary injuries such as suspensory desmitis, superficial digital flexor tendonitis, and middle carpal joint lameness.
- Conformation of the foot is important. Asymmetrical foot size is often a consequence of reduced weight bearing and lameness on one side and the smallest foot is generally on the lame side.

Palpation

Many clinicians spend little time palpating a lame horse, a practice that I feel is a mistake. Areas of warmth (heat), especially in the hoof wall, must be detected, and regions of special interest include the fetlock joints, the metacarpal and metatarsal regions, carpus, hock, stifle, and back.

In the forelimb the distal interphalangeal joint capsule just above the coronary band is palpated to detect effusion. The digital arteries are located abaxially at the base of the PSBs, and the character of the pulse is evaluated and compared between limbs.

Hoof tester examination can be considered part of palpation. When possible, feet are first tested without removing the shoes, and ideally the horse should be kept shod until any examination in movement is completed. When diagnostic analgesia is needed, hoof testing must precede palmar digital analgesia, and shoes may be removed temporarily if bar shoes or pads prevent accurate testing. These six points are tested in each horse: lateral and medial quarters, lateral and medial middle sole, and lateral and medial toe. Testing the frog rarely produces useful information, and squeezing the quarters from lateral to medial with hoof testers may cause pain unrelated to the primary lameness.[2] Pain arising from quarters, especially mild pain medially, should not be overevaluated because this region is frequently sensitive to hoof testers in normal horses. The contralateral foot may serve as a reference. In my experience the right medial quarter is the most common area where clinically important pain is elicited by hoof tester examination in STBs, and this is probably secondary to the counterclockwise direction of racing. Generally speaking, a painful response is considered more important when it arises from the toe or from the lateral side of the sole. When shoes are removed, a further evaluation of the lateral-to-medial balance is performed. The sole itself is observed, and when it appears flat and painful, this may correlate with type of shoes and padding that are used. Overzealous padding may add to, rather than relieve, pressure on the sole. A leather or rubber layer may allow sand to pack quickly under the pad and create pressure and secondary bruises. These horses are better managed with shoes in which the contact is limited to hoof wall and no contact is made with the sole.

In the forelimb the fetlock joint is examined for effusion, alteration of the dorsal outline, and enlargement of the suspensory branches. The latter are palpated carefully with the joint flexed. Each branch is pressed gently axially, and alterations in consistency and pain response are noted. Range of motion of the fetlock joint is assessed. In a normal STB the fetlock joint can be flexed up to 90° (angle between the metacarpophalangeal region and the proximal phalanx) without eliciting a painful response. With one hand holding the dorsal pastern region, the dorsal aspect of the fetlock joint can be palpated further by using the other hand to compress the dorsal joint capsule against the bony prominences of the sagittal ridge and condyles of McIII. Horses with osteochondritis dissecans or a hypertrophic synovial pad exhibit a response. The bony profile of McIII must be followed with fingers to detect painful areas in the dorsal, lateral, and medial aspects. Palmar soft tissues are evaluated with the limb in a weight-bearing position and while being held off the ground. Each structure is palpated accurately to detect heat, pain, and edema in horses with acute lesions or fibrotic consistency and adhesions in those with chronic conditions. Fingers must be pressed firmly, deep in the proximal palmar metacarpal region, where pain originating from the proximal suspensory ligament (SL) is hard to detect. The carpus is better evaluated by holding the leg in a moderate degree of flexion. Careful digital palpation along the dorsal aspect should be performed. Of particular importance is the dorsomedial aspect where a thickened joint capsule and painful response corresponds to the common finding of osteoarthritis of the middle carpal joint. Palpation of the forelimb proximal to the carpus is rarely helpful. Elbow and shoulder lameness are rare, and bicipital bursitis has seldom been reported.

In the distal hindlimb, palpation is similar to that described for the forelimb with the exception that foot lameness is less

important. The metatarsophalangeal joint region is best evaluated while holding the limb in a semi-flexed position. When the regions of proximal metatarsal region and distal tarsus are palpated, some painful reactions frequently are elicited, especially on the medial aspect. This pain frequently is overemphasized in horses with back pain, especially when the trainer thinks the horse has primary lameness in the tarsus. Effusion of the tarsocrural joint often indicates osteochondrosis. The stifle must be palpated deeply and carefully, because the joint is complex and palpation is limited. The femorotibial joints seldom appear distended, but increased pressure in the medial femorotibial joint is an important sign. More frequently, joint effusion is limited to the femoropatellar joint. Distention of the latter must lead to specific examination of both femorotibial joints, because inflammatory processes involving the femorotibial joints may cause distention of the femoropatellar joint. With the exception of acute trauma, painful responses to palpation are rare in this area, even from the patellar ligaments that are identified easily. On the medial side, scar tissue and irregularities or enlargement of the medial patellar ligament may suggest previous desmotomy. When associated with femoropatellar effusion, the latter sign may suggest apical patellar fragmentation, and radiographic examination is indicated. The stifle can be flexed only in unison with most other hindlimb joints, so a painful response is not specific. With moderate flexion the medial collateral ligament can be stressed by the veterinarian pulling the tibia in a lateral direction with both hands and pressing the shoulder against the femur.

Palpation of the back is aimed at evaluating pain arising from joints (intervertebral, lumbosacral, sacroiliac, and sacrococcygeal), nerve roots, and muscles. The latter are the most likely origin of pain elicited by palpation, but other problems must be ruled out if associated clinical signs (atrophy, asymmetry) are present. Pain in the gluteal area may be secondary to many problems, including straight hindlimb conformation, hock or stifle pain, sore feet, gait imbalance, and stiff corrective harness equipment. So pain in this area should not be treated as a primary problem unless a thorough clinical examination has ruled out other sources of pain.

Movement

After palpation, the horse is examined during movement. I commonly tranquilize each horse I examine for lameness. Tranquilization (10 mg acetylpromazine maleate intravenously) improves the possibilities of handling the horse and lowers the risk of injuries to the veterinarian. Furthermore, the horse appears less stiff, mild lameness becomes somewhat more obvious, and the horse stands better for radiographic or ultrasonographic examination. The trainer must be consulted before injecting a tranquilizer, because this practice may preclude racing because of doping (the term used in Europe for a positive blood or urine drug test of a prohibited substance). Lameness is rarely detectable at the walk, but it is important to observe the way the horse lands with each foot to identify lateral-to-medial hoof imbalance. The horse is then trotted in a straight line on a firm surface, and the character of movements is observed. Abduction or circumduction of forelimbs is considered characteristic of carpal lameness, because the horse appears to attempt to avoid flexion. In the hindlimb, stiffness has been related anecdotally to distal tarsitis but in fact is not a specific sign. In horses with pain arising from the distal part of a hindlimb (frequently the metatarsophalangeal joint) the horse tends to moderately overflex the hock and stifle to shorten the weight-bearing phase of the stride.[3] In horses with more severe lameness, drifting forward of the sound limb is observed and a drop of the fetlock joint is easily seen. Abduction of the hindlimb is thought to be related to stifle lameness.

Flexion tests are used to supplement findings during movement and are similar to those used in other sport horses. Flexion of the carpus is accomplished by pulling the metacarpal region laterally, and the clinician's elbow may act as a lever against the horse's radius to stress the medial aspect. In young horses affected by carpitis, this maneuver frequently elicits pain.

DIAGNOSTIC ANALGESIA

Diagnostic analgesia can be performed in sequence from distal to proximal, or to save time, selective analgesic techniques can be used. For example, European trotters with a positive response to forelimb lower limb flexion inconsistently respond to palmar digital analgesia, so analgesia of the metacarpophalangeal or distal interphalangeal joints may be the first option. Perineural analgesia is preferred, however, to avoid minor risks of joint infections or to save the opportunity to provide intra-articular therapy immediately (when working in stables, trainers frequently are interested more in treatment than in diagnosis). I frequently start with low plantar analgesia in the hindlimbs, because lameness of the digit is rare. In young STBs with forelimb lameness and a positive response to carpal flexion, I block the middle carpal joint first.

In horses with obscure lameness or when lameness is only apparent during fast exercise, examining the horse on the track may be useful. The horse is rigged in full harness and first examined trotting in a clockwise direction. Speed then is increased, and the horse is turned to train in a counterclockwise direction, the same as racing. Clinicians can drive the horse themselves, sit in a two-seat wagon, or observe the horse from a car or from a distance. In my opinion, watching the horse during exercise is important, especially when routine training and racing can be simulated, and I prefer the horse to be driven by its usual trainer. Having the horse fully equipped, mimicking the stress of racing, and observing the horse in turns are important advantages to this form of lameness examination.

Diagnostic analgesia is useful, but the clinician must be aware of related risks and make the trainer and owner aware as well. Diagnostic blocks should be avoided in horses suspected of having incomplete fractures. Radiographic examination should precede diagnostic analgesia in these horses. When examining a horse on the track after local analgesia, the trainer should be told to limit the speed as much as possible, avoiding any sudden stop or sharp turn. An experienced trainer normally is able to appreciate the benefits of a block quickly and without stressing the horse. Examination at speed is needed to make a diagnosis in horses with plantar process osteochondrosis fragmentation of the proximal phalanx and fragmentation of the distal border of the distal phalanx.

DIAGNOSTIC IMAGING

Sophisticated equipment for diagnostic imaging is now available in most European equine clinics. Scintigraphy is available in Europe but is limited to large referral hospitals. Radiography remains the mainstay of equine diagnostic imaging, and the availability of excellent portable units has improved the radiographic examination under field conditions. Ultrasonographic examination commonly is performed.

Common pathological findings in the distal limb of European trotters include osteophyte formation on the distal aspect of the middle phalanx, sometimes associated with remodeling of the extensor process of the distal phalanx, as seen on a lateromedial view of the foot (Fig. 110-1). This radiographic pattern often is associated with heel growth proceeding faster than toe growth (club foot). When associated

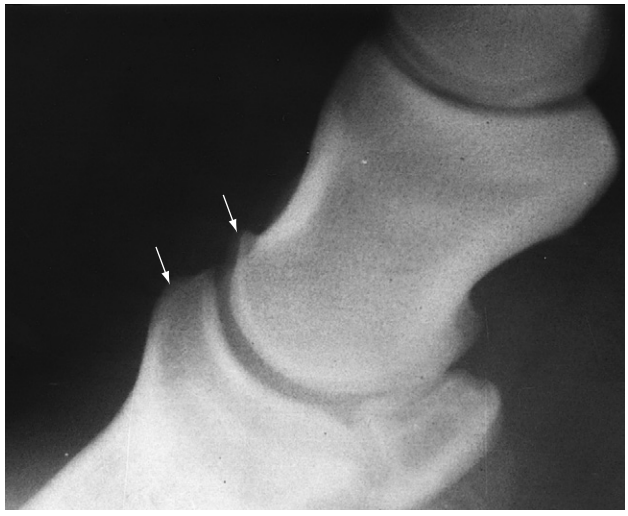

Fig. 110-1 Lateromedial radiographic view of the distal interphalangeal joint of a Standardbred trotter. Osteophyte formation on the distal border of the middle phalanx associated with moderate remodeling of the extensor process of the distal phalanx *(arrows)* is a common finding in European trotters.

with a positive lower limb flexion test and lameness abolished using analgesia of the distal interphalangeal joint, this radiographic finding is important and indicates osteoarthritis. Fragmentation of the extensor process of the distal phalanx has controversial clinical significance, but in my experience these fragments cause synovitis of the distal interphalangeal joint and are removed best by arthroscopy.

The solar radiographic view of the foot, unlike scintigraphy, rarely helps in diagnosing pain arising from stress remodeling of the distal phalanx, so the diagnosis of pedal osteitis is limited to horses with chronic lameness characterized by substantial radiolucency.[4] Fragmentation of the lateral and medial palmar processes of the distal phalanx, although controversial as a cause of acute lameness, may be an important source of pain best managed by shoeing.

The flexed lateromedial view of the metacarpophalangeal or metatarsophalangeal joints is useful in evaluating lesions of the distal aspect of McIII or MtIII. In the hindlimb the oblique views must be taken in a proximal to distal direction (downangled) to see better the area between the proximal phalanx and the base of the PSBs. Fragmentation of the proximal plantar aspect of the proximal phalanx represents a major cause of subtle hindlimb lameness (Fig. 110-2).

A dorsopalmar view of the carpus helps to evaluate the proximal suspensory attachment, and focal or diffuse palmar cortical sclerosis may be found (Fig. 110-3). In yearlings and young horses, this view allows examination of the distal radius, and pathological modifications of the growth plate on the medial side (physitis) are a frequent cause of early lameness. The most common lesions of the carpus are seen with the dorsolateral-palmaromedial oblique view and the dorsal 33° proximal-dorsal distal oblique (skyline) view of the flexed carpus to highlight the distal row of carpal bones. The latter must be of excellent quality, and the appropriate projection must be performed, because false-negative radiographs are frequent. Abnormal findings include areas of radiolucency in the dorsoproximal articular border of the radial fossa of the third carpal bone, radiolucent lines suggestive of fractures, and sclerosis of the third carpal bone.[5] A moderate degree of sclerosis in the radial fossa of the third carpal bone is considered normal in racing horses, but more severe sclerosis associated with radiolucent areas represents a pathological finding.[6,7]

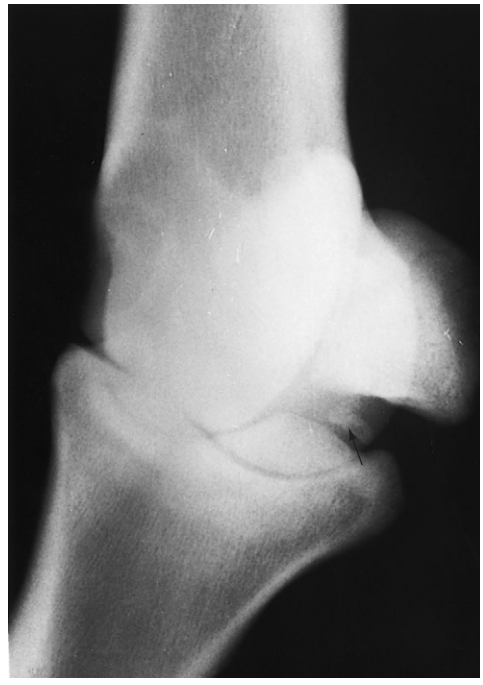

Fig. 110-2 Dorsoproximal 45° medial-plantarodistolateral oblique radiographic view of a hind fetlock of a 2-year-old Standardbred. There is plantar fragmentation *(arrow)* of the proximal medial aspect of the proximal phalanx.

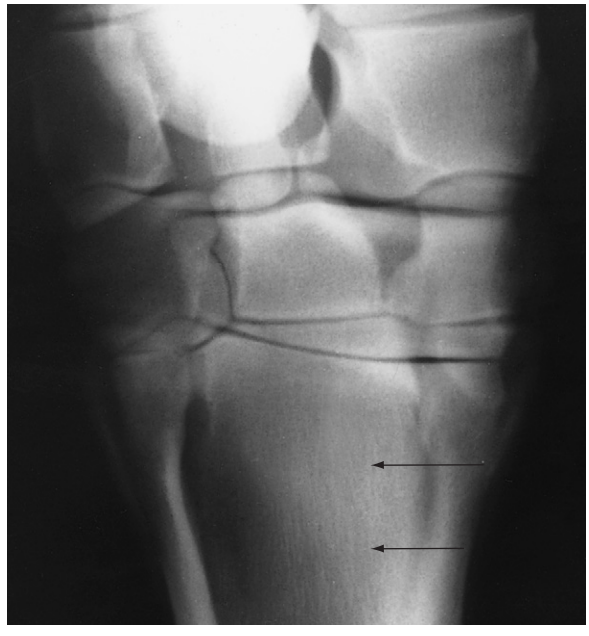

Fig. 110-3 Dorsopalmar radiographic view of the proximal metacarpal region of a Standardbred trotter. Sclerosis of the palmar cortex of the proximal aspect of the third metacarpal bone *(arrows)* is a common finding in trotters with chronic proximal suspensory desmitis.

In the hindlimb the centrodistal and tarsometatarsal joints frequently appear normal radiographically even when distal tarsitis is diagnosed clinically as the source of pain. Scintigraphy is an excellent tool for diagnosing osteoarthritis of these joints.[8]

The tarsocrural joint is a predilection site for osteochondosis in STBs, and fragments associated with effusion may represent an indication for arthroscopic surgery. Lesions affecting the lateral trochlear ridge of the talus and medial malleolus of the distal tibia more frequently cause lameness and effusion than do those of the cranial intermediate ridge of the distal tibia. In horses with effusion but without obvious fragmentation a specific dorsal 20° lateral-plantaromedial oblique projection is indicated to evaluate the axial aspect of the medial malleolus. Subtle osteochondral fragmentation or radiolucency easily can be overlooked (Fig. 110-4).

Curb is not seen frequently but can develop in the early stages of training in young horses. Curbs represent thickening of the plantar aspect of the hock and must be differentiated from the soft swelling caused by distention of a tarsal sheath. Abnormal stress to the plantar soft tissue structures can be predisposed by sickle-hocked or cow-hocked conformation. Accurate palpation differentiates curb from tarsal tenosynovitis. Ultrasonographic evaluation helps characterize the soft tissue structures involved in curb and assess severity (see Chapter 79). In the European STB, curb nearly invariably represents inflammation and thickening of the plantar ligament, but ultrasonographic evaluation is necessary to differentiate plantar desmitis from other soft tissue injury.

Trainers frequently ask for radiography or ultrasonography of the stifle because they seem to incriminate this joint as the source of pain in horses with obscure hindlimb lameness. Radiography of the stifle is important in foals and yearlings when femoropatellar effusion is present. Lateromedial and caudocranial views must be obtained. Osteochondritis dissecans of the lateral trochlear ridge and, less frequently, subchondral bone cysts of the medial femoral condyle can cause effusion and lameness in young STBs. Ultrasonographic evaluation of the stifle is useful in detecting soft tissue injuries, but these lesions are rare in STBs. Mild dimples or cartilaginous defects on the articular surface of the medial femoral condyle can be detected using ultrasonographic examination by holding the stifle in a semiflexed (90°) position.

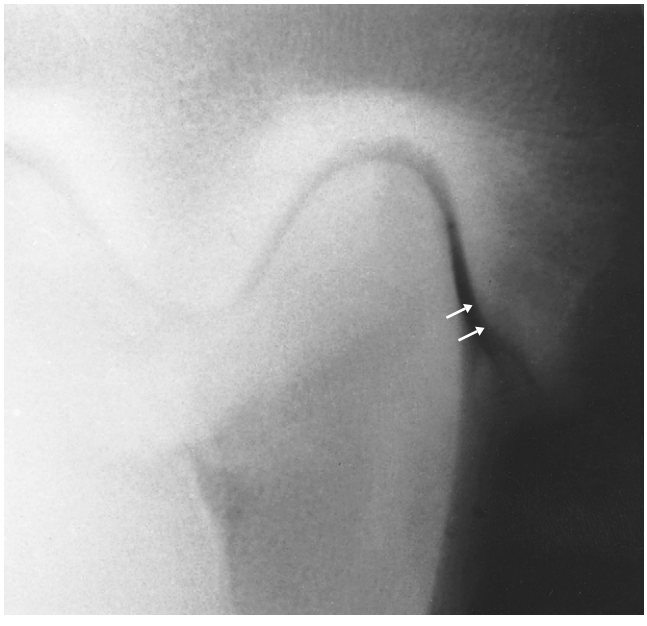

Fig. 110-4 Dorsal 20° lateral-plantaromedial oblique radiographic view of the hock of a 2-year-old Standard bred colt. There is axial fragmentation of the medial malleolus *(arrows)*, which is difficult to detect unless oblique views are obtained. The condition can cause effusion of the tarsocrural joint and lameness.

FINAL DIAGNOSIS, PROGNOSIS, AND TREATMENT OPTIONS

A final diagnosis is made easily when the horse has obvious severe lameness, as is the case in horses with the more common fractures and tendon or ligament injuries (incomplete sagittal fracture of the proximal phalanx, proximal sesamoid bone fracture, carpal chip or slab fractures, superficial digital flexor tendonitis, and acute suspensory desmitis). Surgery or rest most often is recommended for these horses. In some horses, rest is the best option, and accurate monitoring of the healing process must follow (fracture of the distal phalanx, stress fractures of the proximal aspect of the third metacarpal bone, and proximal suspensory desmitis).

Osteoarthritis of the distal interphalangeal, metacarpophalangeal/metatarsophalangeal, and middle carpal joints (particularly in young horses) can cause chronic lameness. Racetrack clinicians frequently inject the tarsometatarsal, centrodistal, and tarsocrural joints, but the true incidence of problems arising from these joints has not been substantiated using diagnostic analgesia. Problems affecting the proximal hindlimb, in the absence of visible lameness, frequently are diagnosed, but definitive diagnosis is difficult to substantiate.

Intra-articular therapy includes steroids or non-steroidal anti-inflammatory drugs (NSAIDs). A series of four intra-articular injections of polysulfated glycosaminoglycans (PSGAGs), 250 mg every fourth day and then at weekly intervals, is helpful in treating STBs with juvenile osteoarthritis, especially in the carpus. PSGAGs also have been useful parenterally (500 mg every 4 days for seven treatments).[9] High molecular weight hyaluronan (20 to 40 mg) may be used alone or with corticosteroids. Hyaluronan (20 to 40 mg) may be used intravenously.[10] The most popular corticosteroids for intra-articular therapy in horses include methylprednisolone acetate (40 to 60 mg), betamethasone disodium phosphate or betamethasone acetate (3 to 9 mg), and triamcinolone acetonide (6 mg). A basic treatment includes three intra-articular injections, using 60 mg methylprednisolone acetate for the first injection, followed by two injections of 40 mg at 2-week intervals. A similar protocol may be applied to betamethasone disodium phosphate and betamethasone acetate (6 to 9 mg the first time followed by two injections of 6 mg each). This treatment is usually helpful in horses with chronic osteoarthritis of the distal interphalangeal, metacarpophalangeal/metatarsophalangeal, and distal tarsal joints and sometimes resolves acute synovitis in young horses. When using triamcinolone acetonide (6 mg), clinicians must be aware of systemic effects and problems (laminitis and rhabdomyolysis) if several joints are injected at the same time. Although the cause is unsubstantiated, these complications may develop because of iatrogenic hyperadrenocorticism.

Liniments, paints, and blisters are still popular, and despite lack of scientific support, they appear strongly tested by time. Iodide-based light paints diluted in dimethylsulfoxide may be helpful in improving circulation to the distal limbs and help to remove edema. Stronger blisters (with iodide mercury, cantharids, cedar oil, or turpentine) may play a role in improving circulation in some areas, but trainers must be made aware of possible secondary effects of the chemical induced inflammation (scars, adhesions, cellulitis, and excessive joint inflammation) and that using most irritant blisters is not justified anymore. Thermocautery (pin firing) is now less popular in Europe but is still used to treat STBS with curbs and proximal splint exostoses. Paints and blisters are used more frequently in France and Italy than in northern Europe.

CORRECTIVE SHOEING

Corrective shoeing is important for many forelimb lameness conditions. Generally speaking, a good lateral to medial

balance and an ideal dorsal hoof wall angle must be provided before any shoe is applied. Then a large-based (wide web) shoe is ideal but not always possible because of gait characteristics. Many trainers prefer thinner and lighter shoes, because they are associated with increased speed. In horses with a flat sole the shoe must be in contact only with the wall border, and any rubber, leather, or silicon pad must be avoided. In these horses a rigid (aluminum) sole may prove helpful, but frequent cleaning is required to avoid sand accumulation under the sole. In horses with osteoarthritis of the metacarpophalangeal or metatarsophalangeal joints, bar shoes must be avoided because the bar prevents the natural slipping of the foot when landing and increases stress on these joints. A bar shoe may prove helpful in horses with superficial digital flexor tendonitis, but I prefer to leave this shoe on only during the recovery phase and during light training, whereas an open shoe is preferred for fast training and racing.

TRAINING PROGRAMS

Alternative training programs can be a valuable adjunct therapy, especially when dealing with unnatural fast gaited trotters. Clockwise jogging and training, training on straight tracks (using interval training schedules), and swimming are preferred. Training programs aimed at reducing speed and stress on the large upper limb muscles, such as fast trotting in a circle in deep sand, use of heavy wagons or wagons with preselected brake sets (power carts) have been used, but scientific studies and objective data to support use of these alternatives are lacking.

PROCEEDING WITHOUT A DIAGNOSIS

Progress in diagnostic imaging has made the situation rare in which an clinician cannot determine a diagnosis. When available, whole body scintigraphic examination in horses with occult lameness is useful. Results of scintigraphy must not be overinterpreted, and clinicians must be aware that conditions may be subtle or difficult to detect. Bone remodeling around osteochondritis dissecans or osseous cyst-like lesions may be subtle. When detailed clinical examination has failed to reveal a diagnosis, I suggest the following options:

1. The horse should be re-assessed 10 to 15 days later. This may help in horses with acute lesions without initial radiographic signs and when scintigraphy is not available.
2. A second opinion can be considered, especially if the horse can be referred to a center that is equipped with advanced imaging equipment.
3. The horse should be treated with NSAIDs (phenylbutazone, 2.2 mg/kg a day orally for 5 days and then every second day for 10 days) and then re-evaluated.
4. The clinician should treat the most likely site or sites of pain with short-acting corticosteroids. Some trainers prefer this approach because they feel the horse does not lose time and often feel something has been done. In some horses, experienced horsemen may help the clinician effectively by asking for a specific therapy that may help the situation.
5. The clinician should give the horse a period of controlled exercise or rest. This decision is particularly helpful when dealing with young horses, in which early training may have promoted bone and soft tissue adaptation and remodeling that requires time to heal. Before resting the horse, a complete radiographic examination is a good idea because horses with advanced disease may have a poor prognosis or may require additional rest.

LAMENESS IN THE EUROPEAN TROTTER

Hoof Pain

This non-specific definition refers to a number of conditions, including what most clinicians and trainers call foot pain. Lameness varies and the response to flexion may be equivocal. Negative flexion tests in a lame horse may suggest nonarticular hoof pain, and the same applies to horses in which the trainer's complaint is pain (with compromised gait) at the end of the race. Horses may break stride on the last turn or may get on a line in the straightaway.

Physical examination includes visual inspection of the feet (club foot, lateral-to-medial hoof imbalance, or toed-in or toed-out conformation), accurate palpation of the coronary band, appreciation of alterations in the digital pulse, and hoof temperature. A complete hoof tester examination is essential. A series of selective diagnostic analgesic procedures follows. Palmar digital analgesia commonly improves lameness by 75% to 90%. If a less than 50% response is obtained, an abaxial sesamoid block is performed. I avoid analgesia of the distal interphalangeal joint, because I may choose to medicate the joint. Recent evidence suggests analgesia of the distal interphalangeal joint is not specific, and pain originating from the sole may be abolished.[11]

Diagnostic imaging includes radiography and scintigraphy. Stress remodeling of the distal phalanx is a common finding in trained STBs. Radiographs are usually negative, although occasionally marginal changes of the distal phalanx suggestive of pedal osteitis are present.

Management includes corrective shoeing, controlled exercise, distal interphalangeal joint injections, and local application of iodide ointments or blisters. Temporary pain relief has been reported anecdotally after perineural injection of cobratoxin, alcohol, or other preparations and after percutaneous cryotherapy. Horses with mild lameness respond to corrective shoeing: rubber or leather soles may be added to wide web shoes, or different types of rubber flaps may be used temporarily. Light training for 2 to 3 weeks is suggested, and the horse is best trained on a soft track or on a straight track if available. Swimming is a good alternative method of training.

Osteoarthritis of the Distal Interphalangeal Joint

History, character of the lameness, response to flexion tests, and shape of the hoof are points to consider when osteoarthritis of the distal interphalangeal joint is suspected. Horses can be lame or reported to be intermittently lame. The tendency is for horses to develop osteoarthritis of the distal interphalangeal joint and club foot because the hoof growth in the heel region is faster than in the toe region. Evaluation and trimming of the hoof wall to lower the dorsal angle may help prevent this conformational change. Palpation of the area over the coronary band may elicit pain, especially medially where the condition must be distinguished from chronic, distal interphalangeal collateral ligament strain. Mild pain also may be detected by palpation of the proximal SL in the forelimb and muscles of the back, two common compensatory lameness conditions. Diagnosis can be frustrating because horses are not always improved with distal interphalangeal analgesia (this may suggest pain originating in the subchondral bone), and the gait may be abnormal because of other problems frequently secondary to pain originating from the distal interphalangeal joint. An 80% to 90% positive response to the perineural analgesia must be considered clinically relevant.

Lateromedial radiographs of the distal limb frequently show a prominent osteophyte on the distal aspect of the middle phalanx, sometimes associated with secondary remodeling of the proximal aspect of the distal phalanx (see Fig. 110-1). Fragmentation of the extensor process of the distal phalanx may also be observed, in which case the hoof wall frequently has a grossly triangular shape. Osteoarthritis of the

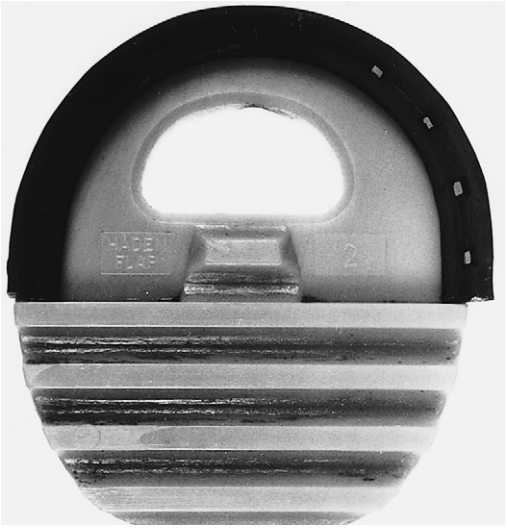

Fig. 110-5 A flap or flip-flop shoe can be useful in trotters with foot pain.

distal interphalangeal joint may be bilateral but is most commonly unilateral.

Suggested therapy includes intra-articular therapy with corticosteroids, PSGAGs, high molecular weight hyaluronan, or a combination of these drugs 3 times, at 7-day intervals (14 days when using corticosteroids). Corrective trimming to decrease excessive heel growth and maintaining the hoof angle at about 50° appear important. Wide-web shoes distribute load on a large surface area, and when possible, a rubber or leather pad is needed. The shoe should have a rolled toe to ease breakover. A thick rubber pad (flap or flip-flop) is used sometimes to replace the classic shoe (Fig. 110-5).

Osteoarthritis of the Metacarpophalangeal Joint

Osteoarthritis of the metacarpophalangeal joint may be acute or chronic. Acute synovitis is seen in young horses when training is intensified or when the track surface changes. Clinical signs include mild to moderate lameness, pain on palpation, and lameness after flexion. Radiographs are usually negative in young horses, but flattening or more severe changes of the sagittal ridge of McIII in older horses with chronic osteoarthritis often are seen. Contrast radiography reveals a filling defect corresponding to the hypertrophic synovial pad in the dorsal aspect of the distal, dorsal aspect of McIII. Ultrasonographic examination reveals various degrees of dorsal joint capsule thickening and increase in echogenicity of the synovial pad. Intra-articular analgesia abolishes the lameness in most horses.

In horses with acute synovitis, training program modulation (2 to 3 weeks of light jogging), corrective shoeing (wide-based shoes and pads), and intra-articular corticosteroids (a series of three injections at 2-week intervals) may resolve the problem. In these horses, concurrent pathological conditions of the articular cartilage, subchondral bone, and the synovial pad usually are lacking or mild. Horses with hypertrophic synovial pads benefit from arthroscopic surgery to remove the thickened tissues, because response to medical management is poor. This is frequently the case in older horses, in which advanced osteoarthritis is often present. Prognosis after surgery is only fair to guarded, however.

Middle Carpal Joint

The middle carpal joint is the most common site of lameness in young STBs. Typically, affected horses tend to trot with a wide gait, abducting the affected limb or limbs in an attempt to minimize carpal flexion.[12] Visual inspection from a dorsolateral perspective reveals abnormal contour of the dorsomedial aspect of the carpus. Palpation often elicits a painful response over the dorsal aspect of the radial and third carpal bones. Usually the response to carpal flexion is positive, and intra-articular analgesia abolishes lameness in most horses. A negative result from intra-articular analgesia does not rule out the middle carpal joint as the source of lameness, because subchondral bone damage under a relatively normal cartilage layer may cause pain that may not be desensitized completely. Radiographs are usually diagnostic, especially the skyline view of the distal row of carpal bones. Radiographic findings range from complete slab fracture of the third carpal bone to more subtle signs of radiolucency of the radial fossa of the third carpal bone. Scintigraphy is a sensitive and excellent tool in the early diagnosis of middle carpal joint lameness.[8]

Training program modulation and intra-articular injections of PSGAGs, hyaluronan, or small amounts of corticosteroids are the first steps in the treatment program. Arthroscopy is suggested when evidence of more severe bone damage exists. Chip fractures in STBs most frequently involve the third and radial carpal bones. Counterclockwise racing, especially in the turns, concentrates forces along the medial aspect of the right forelimb, and this makes the right middle carpal joint more predisposed to injuries.[13-15] The proximal border of the third carpal bone is involved more frequently than the distal border of the radial carpal bone in the STB, unlike in the Thoroughbred (TB) and in the pacer.[13,15,16] Arthroscopic surgery and rest generally yield a fair to good prognosis.[15] Incomplete slab fractures of the third carpal bone may heal with rest, possibly preceded by diagnostic arthroscopy and curettage of the lesion. Horses with complete slab fractures are best treated by internal fixation. Thin fragments are removed. When arthroscopy shows substantial loss of articular cartilage, the micropick technique may augment the possibilities of cartilage repair.[17] Arthroscopic findings in young STBs have been shown to correlate poorly with radiographic findings, and lesions appear frequently more severe than expected.[16] In horses without radiographic changes, I frequently find depressions and discoloration of articular cartilage (especially in the radial fossa of the third carpal bone), loss of articular cartilage in focal areas of the third and radial carpal bones, and partial or complete tearing of the medial palmar intercarpal ligament. Ligament injuries, especially affecting the medial palmar intercarpal ligament must be suspected in the absence of radiographic findings.[18] However, medial palmar intercarpal ligament injuries are rare. These horses are treated using arthroscopic trimming and are given 4 to 6 weeks of stall rest followed by 4 weeks of stall rest with hand walking exercise, and joints are injected with short-acting corticosteroids.

Proximal Palmar Metacarpal Pain Including Proximal Suspensory Desmitis

Proximal palmar metacarpal pain affects many more horses than we once thought. Mild pain in the proximal palmar aspect of the metacarpal region may be secondary to other gait disturbances and may reflect attempts to maintain balance when trotting fast. In this case the lesion rarely is substantiated by ultrasonography, but pain may be detected with accurate palpation (racetrack clinicians used to diagnose and treat blind splints in this area).

PSD is usually acute in onset, and direct palpation of the SL elicits pain. A carpal flexion test is often positive. Intra-articular analgesia of the middle carpal joint produces a variable amount of improvement, but some degree of lameness still is elicited by flexion. Subcarpal analgesia (4 mg of local anesthetic solution placed axial to each of the second and fourth metacarpal bones or direct infiltration of 2% mepivacaine over the proximal suspensory ligament) usually abolishes lameness.

Diagnostic imaging includes radiography, scintigraphy, and ultrasonography. Scintigraphy is useful in identifying horses with bony injury, including horses with an avulsion fracture at the attachment on McIII or those with a longitudinal fracture of McIII, or with enthesopathy. Horses with PSD without bony involvement may have positive pool-phase images, but delayed images are usually negative. Radiography (dorsopalmar and flexed lateromedial views) may reveal associated longitudinal or avulsion fractures of the palmar aspect of McIII or, in horses with chronic lameness, sclerosis of McIII at the SL origin (Fig. 110-3). Ultrasonography is useful in identifying patterns of ligamentous injury, palmar cortical bone damage, and monitoring the healing process.

The prognosis is fair to good, provided the affected horse is subjected to walking exercise and controlled training with concurrent monitoring of the healing process. STBs may tolerate an acceptable level of training with chronic injuries of the SL compared with TBs, possibly because of the increased percentage of muscle fibers present in the SL.[19] The prognosis in horses with PSD is better than those affected with lesions in the body of the ligament.

Therapy includes 6 to 8 weeks of rest followed by 4 to 6 weeks of 20 to 60 minutes of walking exercise or swimming. Local application of blisters and injections of corticosteroids or PSGAGs have been suggested, but they rarely are able to shorten the healing period. Cryotherapy has gained some popularity in treating STBs with PSD in racetrack practice, but its usefulness has not been substantiated scientifically. Intralesional injection of liquid bone marrow is a new, promising therapy that can be combined with fasciotomy.[20] More recently, shock wave therapy has been used to treat horses with PSD (see Chapter 99).

Sesamoiditis

Sesamoiditis may be defined as enthesopathy at the attachment of the branches of the SL to the abaxial surface of the PSBs. The condition also may affect the base of PSBs, but this form is rare and is best defined as distal sesamoidean desmitis. Two types of sesamoiditis are recognized. Type 1, or juvenile sesamoiditis, is characterized by radiolucent lines (vascular channels) in the proximal third of the PSB, radially oriented as seen in lateromedial or oblique radiographic views, and is a frequent feature in young (2- and 3-year-old) STBs (Fig. 110-6). This radiographic pattern is not associated constantly with lameness, and no link has been observed between the presence of these lines and PSB fractures.[21] Affected horses tend to be lame after training, but pain subsides with rest. Intra-articular analgesia does not abolish lameness. Lameness disappears after perineural analgesia of the medial and lateral plantar (palmar) nerves. The condition usually involves one or both hindlimbs, and the lateral PSB is affected more frequently. Scintigraphic findings indicate increased bone metabolism, but radiographic changes are seen in only 50% of STBs showing increased radioisotope uptake.[8] The lateral PSB represents one of the most frequent locations for abnormal scintigraphic findings in racing STBs.[8,22]

Type 2 sesamoiditis is chronic, frequently affects a single PSB, and is found more commonly in the forelimbs of older horses. Radiographic signs of type 2 sesamoiditis (evidence of radiolucent lines in the proximal half of the bone, irregular palmar/plantar and abaxial outline of the bone, enthesophytes, and mineralization of the adjacent intersesamoidean ligament) are associated with ultrasonographic evidence of suspensory insertion desmitis. One or both suspensory branches often are involved. Focal loss of echogenicity in horses with acute disease and increased echogenicity in horses with chronic lameness are common ultrasonographic findings. The insertion on the PSB (enthesis) becomes grossly irregular.

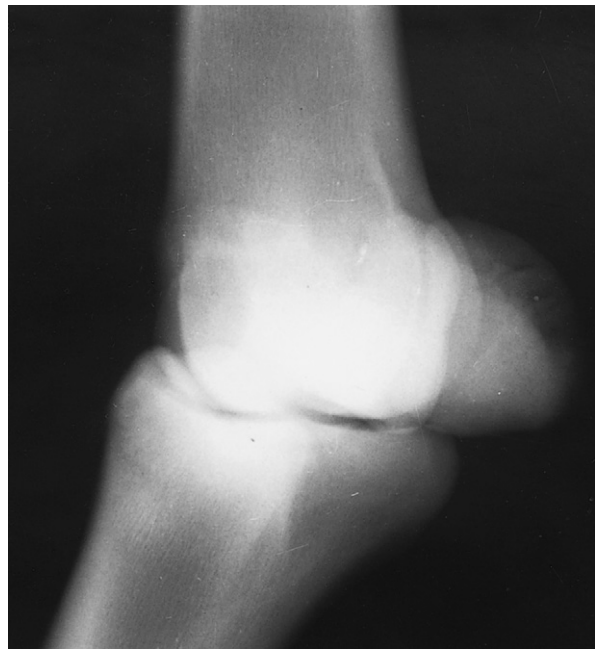

Fig. 110-6 Dorsolateral-plantaromedial oblique radiographic view of a metatarsophalangeal joint of a lame 2-year-old Standardbred colt with juvenile sesamoiditis. Note the radiolucent lines on the abaxial aspect of the lateral proximal sesamoid bone.

In horses with type 1 sesamoiditis, treatment involves rest, slow training, or turnout for 30 to 90 days, depending on the degree of lameness present. Radiographic monitoring of the lesion is probably not helpful, because lameness may improve substantially despite the persistence of radiolucent lines. More information is provided by scintigraphy initially and during follow-up examination. Medical treatment includes local application of paints or mild blisters, corrective shoeing (bars must be avoided, the quarters must be lowered moderately, and shoes must provide a wide base in the hindlimbs). Medical treatment is aimed at improving local blood flow, and isoxsuprine hydrochloride (0.6 to 1.2 mg/kg per os bid) and sodium acetylsalicylate (10 mg/kg PO bid) are recommended for 45 to 60 days. The efficacy of this therapy, however, is questionable. The prognosis is fair if lameness is not severe and if the horse tends to warm out of lameness. If severe lameness is observed and associated radiographic changes are pronounced, the prognosis is guarded. These horses are best given long periods of rest and paddock turnout (6 to 8 months). Treatment of horses with type 2 sesamoiditis includes rest, corrective shoeing (wide web aluminum shoes, leather pad, with particular attention to lateral-to-medial hoof balance), local application of dimethylsulfoxide, and paints and blisters. Pin firing is no longer justified. Cryotherapy has become popular in recent years, but in my opinion its clinical efficacy is poor. The prognosis is fair, but hoof balance must be monitored to prevent recurrence. Low-level or alternative (swimming) training is indicated.

Suspensory Branch Desmitis

Suspensory branch desmitis can be acute or chronic and is caused by lateral-to-medial hoof imbalance, exercise over uneven track surfaces, strains, and chronic fractures of the second and fourth metacarpal/metatarsal (splint) bones. Acute desmitis can be associated with metacarpophalangeal/metatarsophalangeal joint effusion. Shoeing changes, particularly when the hoof angle is modified (usually increased), frequently precede the condition, and when treating the condition, hoof

imbalances must be identified and corrected. Ideally the dorsal hoof angle in the forelimb should be kept between 48° and 52°, with a shoe providing good support to the heels. The routine training regimen, track conditions, and the counterclockwise direction of racing may affect the distribution of suspensory branch desmitis. In my experience the right forelimb and right hindlimb are most commonly affected, and lesions of the medial branch are twice as common as those of the lateral branch. Radiography and ultrasonography are performed to assess ligament damage and bone involvement. Enlargement and loss of definition of the margins of the branch, focal hypoechoic areas, or diffuse loss of echogenicity and hyperechoic foci in horses with chronic desmitis are the most frequent ultrasonographic findings.[23] Radiographically the ipsilateral splint bone may appear deviated abaxially, and in horses with chronic lameness, adhesions may develop between the splint bone and suspensory branch, causing fracture during fast exercise. For the latter reason, radiographic monitoring of the splint bones is suggested in horses with chronic desmitis. The insertion of the branch on the PSB must be assessed by ultrasonography for lesions affecting the branch insertion on the bone.

The treatment in horses with acute desmitis includes rest, anti-inflammatory drugs (phenylbutazone 2.2 mg/kg), or local application of dimethylsulfoxide and poultices. Intra-articular injection of corticosteroids may be beneficial when the condition is associated with metacarpophalangeal/metatarsophalangeal joint effusion. In horses with chronic or severe desmitis, rest and local application of mild blisters may help. Fast training must be avoided when possible, and corrective shoeing must be provided. Alternative training programs, especially swimming physiotherapy, are indicated and can allow an acceptable level of exercise without worsening the lesion.

Metatarsophalangeal Joint

The metatarsophalangeal joint represents a major source of hindlimb lameness in STBs.[24] Lameness of the metatarsophalangeal joint and specifically the plantar aspect is frequently subtle, and diagnosis can be challenging. Stress or non-adaptive remodeling of the plantar aspect of the metatarsophalangeal joint, proximal plantar fragmentation of the proximal phalanx, and non-union of the lateral eminence of the proximal phalanx represent the most common conditions. Traumatic osteochondrosis of the distal plantar metatarsal condyles and mineralization of the distal sesamoidean ligaments are observed rarely.

Subchondral stress remodeling of MtIII has been described recently and represents a scintigraphic finding, with a corresponding radiographic pattern not easily identifiable (Fig. 109-7).[22] Proximal plantar fragmentation of the proximal phalanx has been reported by several authors in radiographic surveys of young STBs and is commonly seen (see Fig. 110-2).[25,26] Plantar fragments from the proximal phalanx rarely are associated with lameness at a trot in hand. Trainers' complaints include gait disturbances during fast exercise, especially in turns, and the tendency for the horse to be on one shaft. Intra-articular analgesia of the metatarsophalangeal joint can alleviate lameness, but a fast exercise test is required and owners must be aware of the potential consequences of this procedure. For this reason, when the clinical pattern indicates pain arising from the metatarsophalangeal joint, a radiographic examination including the oblique projections (dorsoproximolateral-plantarodistomedial oblique and dorsoproximomedial-plantarodistolateral oblique) is required. In horses with plantar process osteochondritis dissecans fragments, arthroscopic surgical removal of fragments is indicated. The prognosis after surgical treatment is good. In horses with stress remodeling of the distal MtIII, rest or reduced training, intra-articular injections of PSGAGs, or low doses of corticosteroids and hyaluronan, are recommended.

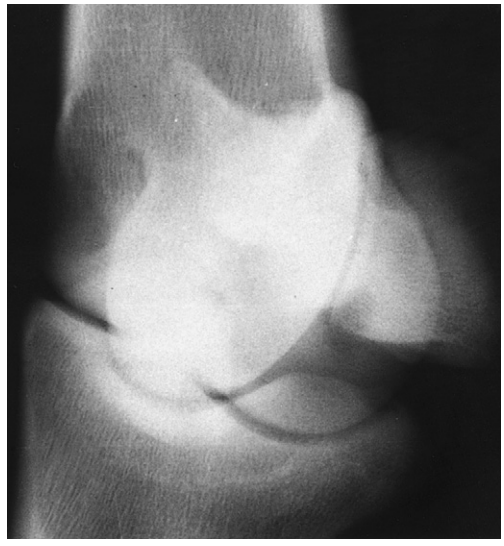

Fig. 110-7 Radiolucent defect in the distal, plantarolateral aspect of the third metatarsal bone is visible on the dorsal 45° lateral 45° proximal-palmarodistal oblique radiographic view, the result of stress or non-adaptive remodeling.

Superficial Digital Flexor Tendonitis

The incidence and morbidity of tendonitis in STBs is lower compared with TBs, but tendonitis of the superficial digital flexor tendon (SDFT) represents the main indication for ultrasonographic examination of the distal limbs in STB racehorses in Europe.

In my experience, most of the lesions are located in the middle and distal thirds of the tendon. A core lesion located in the palmarolateral border of the tendon during ultrasonography characterizes more than 30% of the lesions. More rarely central core lesions are seen. Horses with chronic lesions have the typical pattern of diffuse tendonitis. Therapy includes rest, corrective shoeing, local application of anti-inflammatory ointments, poultices, dimethylsulfoxide, paints, or blisters, tendon splitting, desmotomy of the accessory ligament of the SDFT, and desmotomy of the palmar annular ligament. Intralesional injections of hyaluronan or PSGAGs have been reported. My treatment of choice in horses with acute lameness includes corrective shoeing (moderate lowering of the heels, correcting lateral-to-medial balance, and using wide-based shoes without pads, especially rubber pads), local application of dimethylsulfoxide, cold water therapy twice a day, poultice application, and walking exercise for 2 to 4 weeks. The initial treatment is followed by the local application of an iodide blister, and the horse is given an additional 4 to 8 weeks of walking exercise. The horse usually is able then to resume jogging unless lameness is present. Ultrasonographic examination is performed 12 weeks later.

In horses with recurrent tendonitis, our treatment of choice includes desmotomy of the accessory ligament of the SDFT (superior check desmotomy). Desmotomy of the palmar annular ligament also is performed when substantial tendonitis involving the distal aspect of the tendon or digital flexor tendon sheath effusion is present. After surgery, horses are given 2 weeks of box stall rest, followed by 8 weeks of walking exercise.

Tendonitis of the SDFT within the pastern region is less common but more difficult to manage than tendonitis in the metacarpal region. Lameness is more pronounced, and recurrence of clinical signs is common. With ultrasonography lesions can be detected at the tendon insertion over the lateral or medial aspect of the middle phalanx. Treatment includes

rest, local and systemic anti-inflammatory drugs, blisters, corrective shoeing, and alternative training, particularly swimming. Monitoring of the healing process is important to prevent recurrence.

Osteochondrosis of the Tarsocrural Joint

Joint effusion represents the most common feature of tarsocrural osteochondrosis, a condition that has been reported with a prevalence ranging between 4% and 20% in several surveys.[25-33] Tarsocrural osteochondrosis frequently is diagnosed in yearlings, and breeders are particularly concerned because the condition may lower the yearling's price at the autumn sales. For this reason, tarsocrural effusion in yearlings now represents a frequent indication for radiographic examination and preventative arthroscopy.

Lameness associated with osteochondrosis is rare, but selected lesions may cause gait disturbances. In horses with effusion and osteochondral fragmentation, it is important to rule out other causes of lameness. Lesions affecting the lateral trochlear ridge of the talus and medial malleolus of the tibia are more likely to cause lameness and synovial effusion than are lesions of the cranial aspect of the distal intermediate ridge of the tibia. Focal areas of radiolucency or loss of radiopacity on the medial malleolus may be associated with osteochondral fragmentation and may represent an indication for diagnostic arthroscopy.[34] In young horses with effusion, arthroscopic removal of fragment(s) represents the best option. Prognosis after arthroscopic treatment of tarsocrural osteochondrosis is good, but synovial effusion may persist especially when osteochondrosis involves the lateral trochlear ridge of the talus and the medial malleolus.[35,36] Recent studies performed in Europe found no significant differences in the racing performance and longevity of STB trotters with or without tarsocrural osteochondrosis dissecans.[1,32,37]

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THE AUSTRALASIAN STANDARDBRED

• Chris Whitton

DESCRIPTION OF THE SPORT

STB horses have been racing in Australia and New Zealand for more than 130 years. In both countries the sport does not enjoy the same high profile as TB racing, and prize money is generally lower, which attracts fewer professional trainers than TB racing. Owner/trainers are common, with most stables having fewer than 10 horses. Horses can begin racing at 2 years of age and generally race until 8 or 9 years of age.

Most races are for pacers, with trotting races being much less common. In Australia races range in distance from 1600 to 2400 m, and most pacing races are from a mobile start, with an occasional standing start. All trotting races are from a standing start. In New Zealand, races tend to be longer, up to 3200 m, and standing starts are more common.

Regional meetings carry prize money of $A2000 (Australian dollars) to $A4000 per race, whereas metropolitan meetings offer $A15,000 per race. The 15 most valuable pacing races in Australia and New Zealand make up what is called the Grand Circuit. Prize money for Grand Circuit races ranges from $A100,000 to $A500,000 for the Interdominion Pacing Championship Final. Prize money for trotting races is substantially less than that for pacers. The Interdominion trotter's Championship Final carries a $A200,000 prize.

An increased number of stakes programs for young horses have become available in recent years. The Australasian Breeders Crown is held over all states of Australia and both islands of New Zealand for 2-, 3-, and 4-year-old pacers and trotters. Australian Pacing Gold is a program for yearlings sold at the sales of the same name and also has feature races for 2-, 3-, and 4-year-olds. The value of these programs puts great pressure on trainers and clinicians to persist with young horses that may benefit from rest.

Race speeds generally are expressed as mile rates, which is the time taken to travel 1 mile averaged over the whole race. Tight tracks result in slightly slower rates than large tracks. Typical mile rates for pacers in 1600-m races are 1:56 to 1:58. For 2400-m races mile rates of 2 minutes are typical.

TRACK SURFACE OR TRAINING SURFACE AND LAMENESS

Most of the racetracks are 700 to 1000 m in circumference, and races are run in a counterclockwise direction, except for the north island of New Zealand where races are run clockwise. Track surfaces vary but usually consist of sand or fine gravel. Banking of turns tends to be modest, although a trend to greater degrees of banking and reduced injury rates has been demonstrated. Many trainers have their own homemade training track that is often just graded dirt or sand and not always well maintained, and recurrent foot bruising can be a major problem. Banking on these tracks tends to be minimal. In coastal areas, training on the beach is popular and considered beneficial for horses with injuries.

TRAINING METHODS

A typical training program involves a period of jogging exercise, generally of about 6 weeks, and these sessions generally last 35 to 40 minutes. Higher-speed work in hobbles (pace work) is introduced 2 to 3 times a week. A typical workout is two intervals of half to three-quarter pace over 1½ miles. The speed of these workouts is steadily increased over 4 to 6 weeks until speeds approaching those of race speed are achieved. A horse that is racing generally is hobbled twice a week and jogged on the other days. Training in New Zealand is similar, with the major difference being the tendency to house horses in paddocks rather than in stables.

THE TEN MOST COMMON LAMENESS CONDITIONS

The following are the 10 most common lameness conditions:

1. Subsolar bruising
2. Foot abscess
3. Osteoarthritis of the metacarpophalangeal or metatarsophalangeal joint
4. Suspensory desmitis
5. Osteoarthritis of the distal tarsal joints
6. Superficial digital flexor tendonitis
7. Fracture of the distal phalanx
8. Sagittal fracture of the proximal phalanx
9. Carpal joint disease
10. Osteochondrosis of the tarsocrural joint

LAMENESS EXAMINATION

Experience in examining STBs for lameness is essential because of their awkward gait, even when trotting. An extremely high incidence of lameness occurs in horses in full work, and often several limbs are affected.

Examination at Rest (Preferably in a Stable)

The clinician should observe the horse standing for any obvious swelling, abnormal weight bearing, and areas of hair loss on the proximal limbs associated with the position of the hobbles. The clinician should palpate the neck and back to detect any areas of pain; palpate the supraspinous ligament,

dorsal spinous processes, and dorsal sacroiliac ligament, along with the longissimus dorsi muscles; and then examine each limb in turn. Palpation is performed with the limb bearing weight and not bearing weight. The veterinarian should palpate the digital pulses on the distal pastern or over the abaxial surface of the proximal sesamoid bones; observe each joint and palpate for swelling or effusion. Palpate the tendons and ligaments for heat, pain, and swelling. Particular attention should be paid to all levels of the SLs of the forelimbs and hindlimbs. The clinician should take time to examine the hind fetlock joints for effusion; flex the joints firmly to detect pain and apply hoof testers to each foot.

Trotting in a Straight Line

The horse should be trotted on a firm flat surface. Many horses pace initially, which makes the diagnosis of subtle lameness difficult. Persistence is important, because most horses will trot after 2 or 3 runs up and down. The horse should be observed trotting away from and toward the observer and should be observed from the side.

Flexion Tests

Flexion tests have limitations but are a useful addition to the lameness examination. It is important to perform hindlimb fetlock flexion tests separately from proximal limb flexion tests because hindlimb fetlock problems are common.

DIAGNOSTIC ANALGESIA

Nerve blocks generally are required where no cause of lameness is obvious or the significance of a clinical finding is not clear. Where no localizing clinical signs are apparent, the clinician uses a standard approach: in the forelimb a pastern ring block is performed. The veterinarian should avoid nerve blocks at the level of the PSBs, because distinguishing between PSB pain and foot pain is difficult. This is followed by a low four-point block and then a subcarpal block and intra-articular blocks of the middle carpal and then the antebrachiocarpal joints. Should the horse fail to respond to these blocks, median and ulnar nerve blocks are performed to rule out the lower limb as a source of pain. Rarely are blocks of the elbow joint, intertubercular (bicipital) bursa, or shoulder joint required.

A similar sequence of blocks is used in the hindlimb. A single lateral plantar metatarsal nerve block may be performed if plantar condylar subchondral bone pain is suspected. A subtarsal block is followed by an intra-articular block of the tarsometatarsal joint and then the centrodistal joint. The tarsocrural joint is seldom blocked because swelling generally is associated with intra-articular pathological conditions. Tibial and fibular blocks are performed to rule out the lower limb as the source of pain. Occasionally an intra-articular stifle block is required, in which case all compartments should be blocked at one time.

IMAGING CONSIDERATIONS

Radiography remains an important imaging technique. Oblique views of the distal phalanx are important when a fracture is suspected, because some may be missed on dorsopalmar views. A flexed lateromedial view of the metacarpophalangeal joint should be obtained to highlight the sagittal ridge of the distal aspect of McIII and the dorsal surfaces of the PSBs. Proximodistal oblique views of the hind fetlocks are obtained to demonstrate proximal plantar fragments of the proximal phalanx and the lateral condyle of MtIII. Carpal views should always include a skyline of the third carpal bone.

Ultrasonographic examination of the flexor tendons and SLs commonly is required. SDFT lesions are often peripheral rather than core lesions. Lesions also may involve the distal third of the metacarpal region or the proximal metatarsal region, areas that are examined less commonly by ultrasonography and may be more difficult to assess. Cross-sectional area measurements comparing affected and non-affected limbs are essential when assessing subtle lesions. When examining the SLs, it is important to assess the full length, because lesions can affect the origin, body, and branches.

PROCEEDING WITHOUT A DIAGNOSIS

Intra-articular corticosteroids often are used to assist in diagnosing subtle hindlimb lameness. Bilateral intra-articular injection of the tarsometatarsal and centrodistal joints may be used in horses in which hindlimb lameness is too subtle for accurate assessment by nerve blocks. Triamcinolone acetonide is the most commonly used intra-articular corticosteroid because of its relatively short and predictable detection time, combined with a long duration of action. Treatment of metatarsophalangeal joints also is often performed, because lameness can be subtle. The clinical relevance of plantar proximal fragments of the proximal phalanx is often questionable, and intra-articular therapy may be the only method of confirming that metatarsophalangeal joint pain exists.

SHOEING CONSIDERATIONS

Most horses are shod and trimmed by the trainer or owner. The quality therefore varies extremely. Steel rim shoes generally are used, with trailers on the hind feet being universal. These shoes are thin and provide little protection for the sole. An occasional horse is trained and raced barefoot. Wider web aluminum shoes, similar to those used on TB racehorses, are available, but the use of these shoes is less common. These shoes generally have steel inserts to improve grip. Glue-on shoes rarely are used. In an attempt to promote increased length of stride, toes are often left overlong. The combination of overlong toes and the lack of sole protection predisposes horses to subsolar bruising.

DIAGNOSIS AND MANAGEMENT OF LAMENESS

Subsolar Bruising

The diagnosis of subsolar bruising is based on pain with the application of hoof testers over the sole, either localized or generalized, and increased lameness after concussion of the foot. Lameness is localized to the foot with a pastern ring block, and radiography is performed to rule out a fracture of the distal phalanx. Chronic bruising may result in lysis and modeling of the margins of the distal phalanx, but these changes do not necessarily mean that the bruising is active. Hemorrhage within the horn of the sole may or may not be evident. Horses with acute lameness are treated with rest and NSAIDs. Careful attention should be paid to foot balance and wide web aluminum shoes should be fitted.

Fracture of the Distal Phalanx

STBs with a sudden onset of forelimb lameness after racing or fast work but with no localizing signs should be suspected of having a fracture of the distal phalanx. Application of hoof testers usually elicits pain, but this is not always consistent. Oblique radiographic views of the foot often are required to assess the fracture properly. It is important to determine whether the fracture enters the distal interphalangeal joint.

Most fractures are intra-articular. In horses that race counterclockwise, left forelimb fractures are generally of the lateral palmar process and right forelimb fractures are generally of the medial palmar process. Horses with non-articular fractures are treated with a bar shoe with quarter clips or a rim shoe and 12 months of rest. Horses with articular fractures may be treated in the same manner or by internal fixation with a single 4.5-mm lag screw along with a bar shoe with quarter clips. Evidence on the success of these treatments is limited, with the choice depending on individual preference.

Sagittal Fracture of the Proximal Phalanx

The clinical presentation of horses with sagittal fractures of the proximal phalanx depends on fracture configuration. Short, incomplete fractures may be associated with chronic lameness that is localized to the fetlock joint with nerve blocks. Longer fractures often cause acute onset of lameness. Swelling may be present, and the horse often has pain on palpation of the dorsal aspect of the proximal phalanx. Nerve blocks are contraindicated because of the risk of progression of the fracture. Radiography confirms the diagnosis. Horses with non-displaced fractures may be treated with external coaptation. Internal fixation with lag screws is recommended for horses with complete and displaced fractures. Horses with short, incomplete fractures may heal with rest. Lag screw fixation has been recommended for fractures that fail to heal.

Osteoarthritis of the Fetlock Joint

Osteoarthritis of the metacarpophalangeal or metatarsophalangeal (fetlock) joint may or may not be associated with joint effusion and swelling. Usually the horse shows pain on passive flexion and a positive response to a fetlock flexion test. Lameness should be improved with a low four-point block or intra-articular analgesia. If lameness is recent, no radiographic abnormalities will be apparent, but more chronic lameness is associated with modeling changes on the dorsal aspects of the proximal phalanx and McIII/MtIII. Modeling also may be observed on the articular margins of the PSBs and the palmar/plantar aspect of the proximal phalanx. In advanced osteoarthritis, subchondral lysis or cystic lesions may be observed in the palmar/plantar aspect of the condyle of McIII/MtIII. This is most common in the lateral condyle of MtIII and is best observed on a proximodistal oblique view. Scintigraphy may be required for horses with few radiographic changes and demonstrates increased radiopharmaceutical uptake in the subchondral bone.

Proliferative Synovitis

Proliferative synovitis occasionally causes lameness in STBs. Lameness generally is localized to the metacarpophalangeal joint by either perineural or intra-articular analgesia. Abnormal concavity proximal to the sagittal ridge of McIII is observed on the dorsal and occasionally the palmar aspects on lateromedial radiographs. Ultrasonography demonstrates enlargement of the synovial pad medial or lateral to the sagittal ridge on the dorsal aspect, which must be differentiated from the joint capsule. Intra-articular corticosteroids may be used, but the results often are disappointing. Surgical excision of the synovial pad via arthroscopy is usually effective in resolving the lameness. The prognosis is poorer in horses with modeling of the palmar aspect of McIII, because this usually reflects a more advanced, chronic condition.

Axial, Articular (Type 1) Osteochondral Fragments of the Proximal Plantar Aspect of the Proximal Phalanx

Proximal plantar fragments of the proximal phalanx are best observed on proximodistal oblique radiographs of the metatarsophalangeal joints. These views should be included in the workup of horses with low-grade hindlimb lameness or a history of not running straight. Lameness is generally mild or not present at low speeds. Osteochondral fragments of the proximal plantar aspect of the proximal phalanx are most common on the medial aspect but can occur laterally or biaxially. These fragments are present by 1 year of age and are thought by some to be traumatic in origin. Treatment involves arthroscopic removal, and treated horses can be returned to training within 6 weeks. Intra-articularly administered corticosteroids are sometimes used to determine the significance of these lesions, because not all are associated with poor performance.

Superficial Digital Flexor Tendonitis

Tendon injuries are easy to diagnose when the mid-metacarpal or mid-metatarsal area is involved. Swelling and pain on palpation are indications for ultrasonographic examination to confirm the diagnosis and differentiate from peritendonous inflammation. Horses with moderate to severe injuries are treated with rest and anti-inflammatory therapy until the swelling is reduced, followed by a controlled exercise program. Full work should not be re-introduced until 12 months after injury. Less severely injured horses may be kept in work, provided the exercise level is reduced and the tendon is monitored by ultrasonography. Such management is more successful with hindlimb injuries. Tendon injuries at the level of the digital flexor tendon sheath may result in secondary palmar annular ligament (PAL) constriction and tenosynovitis. Lameness and tendon sheath effusion are observed when the horse is returned to training. Provided healing of the tendon injury is adequate, the PAL can be sectioned and the horse rapidly returned to training to prevent the formation of adhesions.

Suspensory Desmitis

In most horses with suspensory desmitis, swelling and pain on palpation of the affected area of the SL is obvious, but lesions confined to the origin may be more difficult to diagnose, and subcarpal/subtarsal nerve blocks are required for diagnosis. Ultrasonographic examination confirms desmitis based on increased cross-sectional area and areas of decreased echogenicity. Radiography is used to assess the palmar/plantar aspect of McIII/MtIII and the PSBs at the proximal and distal attachments of the SL. The ideal treatment involves an initial period of rest and anti-inflammatory treatment to allow the inflammation to resolve, followed by a period of controlled exercise. Horses should not return to full work for 12 months. In practice this is not always possible, and many horses can be managed by reducing the work load for shorter periods, treating with anti-inflammatory drugs, and monitoring the response with ultrasonography.

Carpal Joint Disease

Intra-articular fractures of the carpal bones generally involve the middle carpal joint. Fractures involving the antebrachiocarpal joint are less common. The horse may be performing poorly or have mild lameness. More severe lameness is associated with slab fractures or severe joint injury. Joint swelling and pain on flexion are common. Radiographs should always include a skyline view of the third carpal bone. Often lameness may be localized to the middle carpal joint with intra-articular analgesia, and only sclerosis with or without focal lytic areas of the third carpal bone are observed. Treatment options include arthroscopic surgery to remove diseased bone and cartilage, intra-articularly administered corticosteroids, and rest.

Osteoarthritis of the Distal Tarsal Joints

Confirmation of the distal tarsal joints as the source of lameness involves using intra-articular analgesia or intra-articular medication in horses with more subtle lameness. Although

radiographs are useful for determining the extent of bony changes, they are not always diagnostic. Treatment involves intra-articular injection of long-acting corticosteroids. Triamcinolone acetonide is the most commonly used because of its reliable excretion times and relatively long duration of action.

Tarsocrural Osteochondrosis

The most common osteochondrosis lesion affecting the tarsocrural joint of STB horses involves the distal intermediate ridge of the tibia. Most horses have effusion of the tarsocrural joint, and lameness is absent or subtle. Radiographs demonstrate fragmentation of the intermediate ridge. The fragments are removed arthroscopically.

Stifle Disease

Lameness localized to the stifle is rare in the STB racehorse. These horses appear to be able to race successfully with radiographic evidence of osteoarthritis of the femorotibial joints. Direct trauma to the stifle may result in soft tissue and bony injuries as in other types of horses. Trainers often are concerned that a horse may be locking its stifles, but this generally resolves with increased fitness, and sectioning of the medial patellar ligament is rarely necessary.

CHAPTER • 111

The Racing Quarter Horse

Robert D. Lewis

HISTORY AND DESCRIPTION OF THE SPORT

In the early 1600s, horses imported to Virginia from England were crossed with local horses of Spanish ancestry to produce a compact and heavily muscled horse that could run short distances at incredible speeds. Colonists referred to these horses as Quarter Pathers, and they were the forerunner of what are now known as American Quarter Horses. Early races were generally match races between two horses running no more than a quarter of a mile. These sprint races were the earliest known examples of Quarter Horse (QH) racing in the United States. The first QH races were recorded in Enrico County, Virginia, in 1674.

Racing popularity grew as the breed grew, and racing spread west as pioneers and early settlers moved in that direction. Organized QH racing started in Tucson, Arizona, with the first track devoted to QH racing being Rillito Park, built in Tucson in 1943. Today QH racing is conducted at more than 100 racetracks throughout North America, with total purses reaching $53 million. The goal of many in this industry is to own, saddle, or ride the winner of the $2 million All American Futurity, run each year on Labor Day at Ruidoso Downs in New Mexico. The racing QH today is faster than ever, some having been clocked at speeds exceeding 50 mph, earning them the title America's Fastest Athlete.

Racing QHs traditionally are broken to ride as yearlings in the fall, and breaking commonly is not delayed beyond the year end, with the exception of delays from previous injury or illness. Training a racing QH is quicker than training a Thoroughbred (TB), and most race as early as 2-year- olds, and although some jurisdictions prohibit racing of 2-year-olds before a certain date (e.g., March 1 or actual second birthday of the horse), many regions commence racing early in the year. In general, QHs are raced younger than TBs.

Futurities are races for 2-year-olds for which a horse must be nominated, with regular payments. Scheduling of these races largely dictates the planning of races for 2-year-old QHs. Derbies are managed similarly for 3-year-old horses. Similar races also exist for older horses. Preliminary trial races usually are held to determine qualifiers for the final race, which is run about 2 weeks later.

Purses for futurities and derbies vary, but they are usually attractive and drive the racing QH industry. Owners strive to acquire horses that are talented enough to be competitive in these races.

TRAINING THE RACING QUARTER HORSE

Actual training methods vary widely, but the number of gallops and amount of work performed before the first race and subsequently between races is substantially less than for TBs. Many older racing QHs exercise daily on a mechanical walker and may have one or two easy gallops per week between races.

LAMENESS RELATED TO TRACK SURFACE

QHs race at many racetracks throughout the United States, with some racing conducted on track surfaces devoted primarily to QHs and other racing being conducted on track surfaces also used for TB racing. Some tracks conduct separate racing meets for TBs and QHs, whereas others conduct mixed race meets. Characteristics of track surfaces vary. In my experience, QH trainers prefer a firmer, faster surface than do TB trainers. Racetrack management often requests that maintenance crews produce a firm surface during QH race meets. Uniformity across the entire width of the racing surface is paramount.

Most racing QH trainers agree that deeper, more yielding surfaces result in more soft tissue injuries, such as suspensory desmitis and flexor tendonitis, and a higher incidence of hindlimb lameness and what is typically described as muscle soreness in the hindquarters and loin muscles. Harder surfaces are considered to produce more lameness attributed to bones and joints of the forelimb, and these reflect most of the lamenesses diagnosed in racing QHs.

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CONFORMATION RELATING TO LAMENESS

Many successful racing QHs have obvious flaws in conformation, but good conformation is at least as important, if not more so, than in other breeds of racehorses. Racing QHs do not have the option of being shipped to Europe to race on grass courses, which is a common practice with TBs with less than desirable conformation.

Undesirable conformation flaws in forelimbs include calf knees, upright pasterns, and unusually small front feet, especially because of the hard racing surfaces. Excessively straight stifles and sickle-hocked or excessively cow-hocked conformations should be avoided. It is critical that horses break sharply from the starting gate, and horses with hindlimb pain for any reason are likely to fail to break competitively.

LAMENESS EXAMINATION

Examination of racing QHs is done largely in the same fashion as for other breeds of racehorses. Although not always entirely achievable at many racetracks, observing the horse at a trot on a hard surface is the most productive means of detecting lameness. Jogging the horse in a circle, in both directions, is useful for evaluating hindlimb pain.

Variations do exist in interpreting results of an examination. Normal TB racehorses often show some pain on fetlock flexion (lower limb flexion), but pain on fetlock flexion in a QH is of greater clinical significance. Many TBs exhibit positive responses to the Churchill test and to flexion of the hocks for 90 to 120 seconds. This may be viewed as normal, but in a racing QH this response is important.

If obvious lameness is noted at a trot in hand, the affected limb is evaluated for heat or swelling, and the character and intensity of digital pulses are assessed.

In a forelimb the fetlock and the carpus are flexed separately to detect pain, and comparison is made with the contralateral limb. Firm digital palpation of the lower limb is performed to detect pain, including deep palpation of the fetlock, suspensory ligaments (SLs), flexor tendons, and both carpal joints. Palpation of the dorsum of the third metacarpal bone (McIII) is routine, especially in 2-year-olds, to detect pain. With the forelimb pulled craniad, resting on the clinician's thigh, deep digital pressure is applied just proximal to the dorsal aspect of the coronary band, to detect pain caused by synovitis of the distal interphalangeal joint. Hoof tester examination is performed routinely on any horse observed to be lame, but results must be assessed with care, because many horses respond to pressure with hoof testers, especially those training and racing on firm surfaces. Horses with significant hoof pain usually have a prominent increase in the digital pulse amplitude. I look elsewhere if the digital pulse amplitude is not increased after exercise.

If no abnormalities are detected from the carpus down, the elbow is assessed by maximal forced flexion. This quick test yields a positive response in some but not all horses with elbow pain. I detect pain in the shoulder joint by picking up the lower limb and allowing the horse to relax with the carpus partially flexed, applying deep digital pressure in the depression between the cranial and caudal eminences of the greater tubercle of the humerus, and then comparing the response in the contralateral limb. Horses with pain in the shoulder joint often try to withdraw from this pressure.

For hindlimb lameness, palpation for heat and character of the digital pulse is done as for the forelimb. Fetlock flexion, hoof tester examination, and the Churchill test for pain in the distal joints of the tarsus are performed routinely. If pain in the tarsus is suspected, flexion of the limb for 90 to 120 seconds, followed by observing the horse at a trot often yields valuable information. I assess the stifle joint by holding the hindlimb partially flexed with one hand, and exerting deep pressure over the medial femorotibial joint with the thumb, followed by similar pressure over the lateral femorotibial and femoropatellar joints. In my experience, effusion usually is associated with femoropatellar joint pain, but that is not necessarily the case with femorotibial joint pain. The medial femorotibial joint most often is affected in the racing QH. Soreness in the loin and gluteal muscles occurs as in other racehorses, and palpation of these muscle groups is routine.

At times it is necessary to use perineural and or intra-articular analgesia to localize the source of pain causing lameness. Assessing the horse jogging under tack with a rider up can be helpful. Extended examinations at the racetrack are the exception rather than the rule, and horses with more difficult lameness are referred for further investigation.

THE TEN MOST COMMON LAMENESS CONDITIONS

The following are the 10 most commonly diagnosed lamenesses:

1. Bucked shins
2. Desmitis of the interosseous ligaments (splints)
3. Synovitis of the metacarpophalangeal and distal interphalangeal joints
4. Carpal synovitis
5. Distal hock joint pain
6. Stifle pain
7. Foot soreness
8. Osteochondral fractures of the carpus
9. Osteochondral fractures of the metacarpophalangeal joint
10. Osteochondrosis

IMAGING CONSIDERATIONS

Radiography is the most common imaging modality and is particularly invaluable for assessing carpal and fetlock lameness. The incidence of flexor tendonitis and suspensory desmitis is low, but diagnostic ultrasonography is useful in selected horses. I identify pain in the proximal SL in young horses in training that have just begun speed work. In most horses structural abnormalities cannot be detected with ultrasonography, and the problem resolves with 30 days of rest and slow resumption of training. Nuclear scintigraphy is used less than in TB racehorses because the incidence of stress fractures is considerably less.

SHOEING

The two most commonly observed errors in shoeing are the long toe, low, underrun heel configuration, and improper medial to lateral hoof balance. QHs do not race on turf. Many trainers break horses with flat plates, and later horses train in a level grip type of shoe, but by the time they begin racing, nearly all horses have toe grabs. Aluminum shoes are the norm. Full pads and rim pads are used commonly to try to avoid foot soreness.

In my opinion, training or racing a QH with chronic soreness anywhere in the limb inherently results in increased trauma to the carpus. Although many of the orthopedic conditions that create soreness (such as bucked shins and metacarpophalangeal or distal interphalangeal joint synovitis) cannot be avoided totally, many conditions of the foot can be avoided with careful attention to good shoeing.

DIAGNOSIS AND MANAGEMENT OF SPECIFIC LAMENESS

Although nothing is unique about the methodology in treating lameness in the racing QH, close attention must be paid to minor sources of pain. For horses to perform competitively, they need to be sound. QHs race at tremendous speeds for short distances and must break sharply and competitively to race successfully. One of the earliest indications of developing lameness is refusing to break from the starting gates properly.

Rules of racing in most jurisdictions make no distinction between breeds relative to permitted therapeutic medications, doses, and times of administration. The use of intra-articular medication is more prevalent in the day-to-day management of the racing QH than in other racehorses. The incidence of synovitis is high, and several joints often are treated simultaneously.

In my experience, lameness and soreness in the forelimbs are identified easily, but detecting subtle lameness in the hindlimb often is more challenging. Yet given the nature of the way these horses race and the tremendous speed required at the start of a race, hindlimb pain can be an insidious cause of poor performance.

Bucked Shins

Bucked shins are common, and they usually occur in 2-year-olds. In some horses clinical signs develop as speed work begins, before the first official race, but more often signs appear after racing, usually by the third official race. Occasionally, bucked shins occur later in a horse's racing career. Lameness varies in degree, and if severe, radiographic examination of McIII is recommended. Focal or widespread loss of bone opacity in the outer 50% of the dorsal cortex of McIII may occur with multiple parallel cortical stress fractures in more lame horses. Vertical fractures also occur; these appear as a vertical shear line between original cortical bone and newly developed bone. Stress fractures are common and are best managed if detected early.

Treatment of QHs with bucked shins varies. Managing horses with early bucked shins with continued but reduced training regimens has gained acceptance. With more severe signs, using counterirritants and mild blistering agents is popular. Other treatments include injections of corticosteroids (often diluted in saline or a local anesthetic solution), flooding the subcutaneous tissues over the dorsal aspect of McIII, thermocautery, and a percutaneous technique that produces vertical transection of the periosteum over the dorsal aspect of McIII using a 14-gauge needle (periosteal scratching). Periosteal scratching is used in horses with no radiographically apparent abnormalities that have experienced recurrent lameness, despite rest and a period of reduced training. The technique is performed in a standing horse, using appropriate sedation and local analgesia, flooding the dorsal metacarpal region with local anesthetic solution and 2 mg of dexamethasone. Lacerations in the periosteum from proximal McIII to the origin of the metacarpophalangeal joint capsule are created in three locations, dorsomedially, dorsally, and lateral to the common digital extensor tendon. This provides rapid relief from periosteal pain and in my experience allows a more rapid return to reduced training. The short-term relief of pain likely can be attributed to the corticosteroid used, but similar short-acting corticosteroids used in the past by simple subcutaneous injection did not give extended relief.

Full-thickness dorsal cortical stress fractures usually are detected after a race. Two-year-olds with acute injuries heal without surgical intervention if given immediate, sufficient rest for 90 to 120 days, followed by extended and slow return to full training. Follow-up radiographic examination is important, and if evidence of healing does not occur within 60 days, then surgical cortical fenestration with lag screw compression is recommended. If the fracture is unusually large or shows evidence of an exit line dorsally, surgery usually is recommended initially. Surgery also is recommended for horses with acute fractures if they are older than 2 years of age.

Exostoses of the Second and Fourth Metacarpal Bones

Exostoses of the second (McII) and fourth (McIV) metacarpal bones (splints) are common, and although they rarely produce profound lameness, they do cause acute discomfort. Exostoses usually involve the proximal 50% of McII. A firm bony swelling develops rapidly, with heat and pain on palpation. Swelling usually persists, although once the acute soreness dissipates, performance is unaffected. Distally located splints or those involving McIV are more troublesome. Radiography should be performed to eliminate the presence of a fracture.

Most horses respond to rest for 3 to 4 weeks. Treatment with counterirritant paints and mild blisters and therapeutic thermocautery is popular. If McII or McIV is fractured, surgical removal of the distal portion of the bone is necessary, and post-operative prognosis is excellent.

Acute Synovitis of the Metacarpophalangeal Joint

Acute synovitis of the metacarpophalangeal joint, characterized by heat, effusion, and pain on flexion, is fairly common in young QHs. Acute synovitis is distinguished from chronic fetlock disease (osteoarthritis) by the ability to reduce swelling in the joint to a clinically normal appearance by cessation of work, ice or cold therapy, non-steroidal anti-inflammatory drugs (NSAIDs), poultices, or a combination of these treatments. Usually no detectable radiographic abnormalities are apparent. The condition is usually bilateral and often is referred to as green osselets.

Although rest is desirable, racing schedules often create pressure on the trainer and veterinarian to manage these horses medically while the horse continues to train. Intra-articular therapy with hyaluronan, with or without corticosteroids, is common. The use of intravenously administered hyaluronan or intramuscularly administered polysulfated glycosaminoglycans (PSGAGs) is widespread.

Acute Synovitis of the Distal Interphalangeal Joint in the Forelimb

Acute synovitis of the distal interphalangeal joint in a forelimb is common, particularly when horses are training or racing on firm surfaces. The condition is usually bilateral, and affected horses move like horses with sore feet. Increased amplitude of digital pulses in both forelimbs often is detected, so a diagnosis of bruised soles often is made initially. Fetlock flexion often produces pain, but careful examination reveals that this pain is not originating in the metacarpophalangeal joint. Digital pressure just proximal to the dorsal aspect of the coronary band often elicits a painful response, and effusion may be detected by soft swelling in this area.

Radiographic examination usually reveals no abnormality, although enthesophyte formation on the extensor process of the distal phalanx often is seen in horses that have been racing with recurrent synovitis.

Treatment of this condition varies, with systemic NSAIDs; intra-articularly administered hyaluronan, with or without corticosteroids; intravenously administered hyaluronan; and intramuscularly administered PSGAGs all being used widely. Ice or cold therapy is routine, and most trainers tend to treat horses like those with bruised soles, using foot soaks and packing the bottom of the foot with various poultices or other medicaments. Prognosis is favorable.

Synovitis of the Carpus

Acute synovitis of the carpus is seen commonly and is often unilateral. Clinical signs may develop during training but more often develop after a horse commences racing. If the condition persists despite medical management, it often becomes bilateral.

The horse tends to travel wide with the forelimbs slightly abducted and somewhat stiff legged at the trot, especially on a hard surface. Localized heat and effusion are often present in both the antebrachiocarpal and middle carpal joints. Maximal flexion of the carpus sometimes elicits a painful response, but I rely more heavily on pain elicited by deep digital palpation with the joints flexed.

Medical therapy as described for synovitis of the metacarpophalangeal and distal interphalangeal joints is usually successful. In my experience, horses that are refractory to treatment or have recurrent clinical signs often develop osteoarthritis, and bone failure may result in osteochondral fragmentation (see later discussion). Extended rest always should be considered as a management option in horses with acute synovitis of the carpus.

Distal Hock Joint Pain

Pain in the centrodistal and tarsometatarsal joints is common and can have a profound effect on performance, resulting in failure to break sharply from the starting gates. The Churchill test and hock flexion test are useful diagnostic aids.

The condition is often bilateral, and affected horses often travel narrow behind, with the limbs adducted slightly. Many horses with painful hocks begin to interfere, striking the opposite limb from the pastern up to the tarsus. The condition may develop at any point during a horse's career and, if chronic, radiographic evidence of osteoarthritis is often apparent.

Treatment comprises intra-articularly administered hyaluronan or corticosteroids, with or without NSAIDs, intravenously administered hyaluronan, or intramuscularly administered PSGAG. In the absence of obvious radiographic evidence of osteoarthritis, most horses can be managed successfully, although repeated treatment often is required.

Stifle Pain

Lameness originating in the stifle is not uncommon and may cause signs similar to distal hock joint pain. In some horses performance is reduced without overt lameness. I find deep digital palpation of a partially flexed joint useful for identifying pain, but intra-articular analgesia is often required to confirm the source. The medial femorotibial joint is affected most often. In a young horse radiographic examination should be performed to rule out osteochondrosis; in older horses radiographic examination is useful to identify evidence of previous injury.

I prefer intra-articular treatment with hyaluronan and methylprednisolone acetate and consider that any horse showing pain on deep palpation should be treated. If lameness is more severe or a horse fails to respond to treatment, arthroscopic evaluation of the joint is indicated.

Foot Soreness

The incidence of sore feet varies from track to track, depending on the track surface, and is usually from bruising. Most trainers pack the soles with products such as ichthammol under a foot bandage and walk the horse for several days. If lameness persists, radiographic examination is warranted to determine the presence of pre-existing disease or a fracture of the distal phalanx. Solar margin fractures sometimes occur. A fracture of a palmar process of the distal phalanx usually causes a more severe, acute lameness. Subsolar abscesses are common, and routine extensive hydrotherapy resulting in soft soles may be a predisposing factor. Navicular disease occurs uncommonly.

Osteochondral Fractures of the Carpus

Chip fractures are common especially in the faster horses, particularly in 2-year-old horses that compete in futurities. Extent of injuries varies widely from microfractures of the subchondral bone to failure of bone integrity resulting in fragmentation. Major fractures, particularly of the third carpal bone, occur more commonly than in TB racehorses. Osteochondral fragmentation is usually bilateral and often involves the antebrachiocarpal and middle carpal joints.

The horse usually has a history of previous carpal pain, and the radiographic changes suggest accumulated trauma over time resulting in bone failure, emphasizing the need for careful management of acute carpal synovitis.

Arthroscopic surgery for removal of osteochondral fragments is common, particularly for more successful racehorses. Some good horses undergo arthroscopic surgery several times during a career. Lesser horses often are managed conservatively, with extensive intra-articular medication.

Osteochondral Fractures of the Metacarpophalangeal Joint

Fracture of the dorsomedial aspect of the proximal phalanx occurs most commonly; fracture of the dorsolateral aspect is less common. Fractures of the proximal sesamoid bones most commonly affect the medial sesamoid bone, but the incidence is less than in TBs. In my experience, condylar fractures of McIII do not occur. Subchondral bone trauma in the palmarodistal aspect of McIII is seen in older horses that have raced extensively.

Osteochondrosis

Osteochondrosis occurs in many joints in QHs and is usually clinically evident before a horse starts training. Osteochondrosis can be career limiting, but some horses are treated surgically and are suitable for racing. Mild lesions may not cause obvious lameness until a horse is racing, and some affected horses race successfully without apparent clinical problems.

Osteochondrosis of the stifle and hock usually is diagnosed before training begins. In my practice, osteochondrosis of the fetlock is the most common form in racing QHs. Osteochondral fragmentation involving the sagittal ridge of McIII or the third metatarsal bone is most common. Fragmentation of the proximopalmar (plantar) aspect of the proximal phalanx also occurs, especially in hindlimbs. Osseous cyst-like lesions occur occasionally in the distal aspect of McIII.

CHAPTER • 112

Lameness of the Arabian Racehorse

Mark C. Rick

HISTORY OF RACING

The Arabian racehorse originates directly from the Thoroughbred (TB) foundation sires of all light- or hot-blooded horses. In the seventeenth century, these TB sires—the Darley Arabian, Godolphin Barb, and Byerly Turk—were imported to England and bred to the Queen's mares. The Arabian was used originally as a war horse, and although the true beginnings of the Arabian horse are under a shroud of mystery and legend, the consensus is that the Middle Eastern desert Bedouin tribes played a large role in the breeding and early development of the breed.

Arabian racing in North America and around the globe is less popular, and the number of races is fewer and the amount of prize money is less compared with TB, Standardbred, and Quarter Horse racing. Throughout the Middle East and Europe, Arabian racing and performance are more deeply rooted than in North America. The popularity of Arabian racing has grown enormously in the United Kingdom in the last 15 to 20 years, with a growing number of professional trainers and jockeys and a progressive increase in prize money, in part because of the high Middle Eastern sponsorship.

The Arabian Horse Registry of America, founded in 1908, includes many types and uses. Known for stamina, speed, and elegance, Arabian horses often were bred and raised for showing in halter and performance classes. In the latter part of the twentieth century Arabian horse popularity and breeding selection shifted to criteria based more on aesthetics than athleticism.

The Arabian racehorse lineage reflects more athleticism than is found in Arabian show horses. Consistent winners often are more heavily muscled and have stronger hindquarters with a more sloping croup and a lower head and neck carriage than a typical Arabian show horse. Recent influx of new breeding lines has given rise to concern and controversy over the purity of the lineage and the possible infusion of impure Arabian blood. Certain new stallions appear to be much taller and longer, with a body type similar to the modern day TB racehorse. Constant vigilance and careful documentation of lineage is required to preserve the pure Arabian racehorse breed.

Arabian racehorses race on the same surfaces, dirt and turf, as TB racehorses. In North America selected meets are held from California to Delaware, Florida to Michigan, Colorado, Texas, and Washington and a few other tracks. Arabian racehorses perform in fair meets, allowance races, claiming races, and futurity nominated stakes races. Racing Arabian horses also compete in the United Kingdom, Poland, France, Russia, and South America and in many Middle Eastern countries. In North America, racing begins on March 1 of the 3-year-old year. Race distances are similar to those for TB races, but the length and configuration of the racecourses vary. Shorter sprint distances, 4½ to 6 furlongs, often are run on the small tracks, whereas the longest race (2 miles) is usually run on a large track. Typically, races are 4½ furlongs to 1¾ miles. A sound racehorse may compete as often as every 7 to 10 days, but most are given 2 weeks between races. Because relatively few Arabian racehorses are raced, lack of entries may mandate racing whenever enough horses are entered to meet race conditions rather than when trainers and owners prefer. Racing in the United Kingdom starts in late April. Until 2001, horses did not race until 4 years of age, but in 2001 a restricted number of races for horses 3 years of age were introduced. These are high-value races and also attract horses trained in France and other European countries. Races range from 5 furlongs to 3 miles.

TEN MOST COMMON RACING-RELATED LAMENESS CONDITIONS

The following are the 10 most common racing-related lameness conditions in the Arabian racehorse:

1. Dorsal third metacarpal bone (McIII) disease
2. Superficial digital flexor tendonitis
3. Suspensory desmitis
4. Stifle lameness
5. Tarsocrural osteochondrosis and distal hock joint pain
6. Back pain
7. Proximal sesamoid bone (PSB) fractures
8. Metacarpophalangeal joint lameness
9. Carpal osteochondral fragmentation
10. Lameness of the foot

Dorsal Third Metacarpal Bone Disease

Many racehorses trained intensely at speed at a young age experience the sore-shin or bucked-shin complex. Although intense training may not begin until the 3-year-old year and the Arabian racehorse is smaller in stature and weight than its TB counterpart, bucked shins remain a major cause of lameness requiring reduction in training intensity. Trainers are well aware of this problem and often can diagnose it accurately based on clinical findings and the observation of a sore horse, traveling short. A veterinarian usually confirms the diagnosis clinically, but in some horses radiography and occasionally scintigraphy are necessary. With advanced dorsal cortical pain. typical dorsal cortical periostitis or a dorsal cortical fracture is seen radiographically (see Chapter 104). With periostitis comes intense, diffuse increased radiopharmaceutical uptake (IRU), whereas focal IRU is seen in horses with a dorsal cortical fracture (see Chapter 19). However, scintigraphy is used more commonly to diagnose stress-related bone injury and stress fracture of other long bones in Arabian racehorses. Conservative management is preferred with rest, reduction in strenuous training, or return to the layup farm. Pin firing and blistering are not used routinely. Dorsal cortical fractures are rare, but if present, I prefer surgical management using osteostixis (dorsal cortical drilling) or insertion of a bone screw placed in lag fashion in the dorsal cortex. The prognosis is good.

Superficial Digital Flexor Tendonitis

Superficial digital flexor tendonitis (bowed tendon) is common and occurs from a combination of training overload and fatigue. Occasionally, horses run uphill at the end of a

race, and superficial digital flexor tendonitis occurs commonly under this condition. Sudden changes in track surfaces or training conditions are associated with an increased incidence of tendonitis. Severe tendonitis usually is recognized by the trainer, because swelling and pain are present during palpation. More subtle swelling and pain are detected during careful palpation by a veterinarian. Thorough ultrasonographic examination is imperative to confirm the diagnosis. Careful assessment requires proper patient preparation, sedation, clipping and cleaning the leg, and use of a high-quality ultrasound machine and a 7.5 (or greater) MHz linear transducer. Cross-sectional area (CSA) of the tendon and lesion, fiber alignment, and echogenicity of the lesion are evaluated, and any associated pathological conditions such as palmar annular ligament constriction, carpal tenosynovitis, or other soft tissue damage are assessed. Swelling is often mild if horses have been given local and systemic anti-inflammatory therapy and rest. A slight increase in CSA measurement may be the only indication of superficial digital flexor tendonitis, and comparison with the contralateral superficial digital flexor tendon (SDFT) is mandatory. Initial management includes rest, local ice and bandage application, and administration of systemic non-steroidal anti-inflammatory drugs (NSAIDs). Horses with mild or moderate superficial digital flexor tendonitis often are sent to a layup or rehabilitation farm for 3 to 6 months. Follow-up examinations are performed at 2-month intervals to determine quality of the healing and the appropriate time to return the horse to race training. A slow return to training includes progressive walking, jogging, cantering, speed work (breeze), and then racing. Time span and progression depend on maintaining an acceptable ultrasonographic appearance during each incremental increase in stress or exercise level. Tendon splitting and desmotomy of the accessory ligament of the SDFT used separately or concomitantly are successful in horses with with moderate or severe tendonitis. Intra-lesional injections of β-aminoproprionitrile fumarate (Bapten; no longer commercially available) have been used successfully. Prognosis varies with the severity of the injury and the stage of racing when the injury occurred. Because many Arabian horses race as older horses, even stallions and mares, providing up to a year or more of rest is not uncommon, assuming the lesion heals, before returning the horse to race training.

Suspensory Desmitis

Forelimb suspensory desmitis is an intermittent problem of many Arabian racehorses early in training. Suspensory desmitis is not considered to be as debilitating or career limiting as it is for TB racehorses. A trainer often complains that the horse is sore, but overt lameness is not present. The differential diagnosis includes bucked shins, superficial digital flexor tendonitis, and metacarpophalangeal joint and carpal lameness. Careful palpation reveals pain and enlargement of the suspensory ligament. Although ultrasonographic examination should be performed to confirm and grade desmitis, this pattern of subtle inflammation and soreness often precedes lesions detectable by ultrasonography. Scintigraphy may be useful but is seldom recommended. Suspensory desmitis is not severe, and most often the finding of body soreness in response to increased training intensity is the only apparent clinical sign. Traumatic disruption of the suspensory apparatus is rare. Horses with suspensory desmitis usually are kept at the track because they do not require or benefit from extensive time off. I recommend 1 to 2 weeks of rest or simply a decrease in training intensity to allow for tissue adaptation. The prognosis for horses with early suspensory desmitis is good if the condition is recognized early and horses are given a period of much reduced work intensity and slow rehabilitation.

Stifle Lameness

The most common source of hindlimb soreness is the stifle region. Early in training a horse may become sore and stiff, usually bilaterally. Intermittent upward fixation of the patella is common as in other young sport horses. Stifle soreness is common in young horses shod with flat shoes and training on a soft track. Soft tissues around the stifle become inflamed. Clinical signs include a shortened stride and an unwillingness to extend the stride behind, or actual upward fixation of the patella, with characteristic stifle and hock extension and toe drag. With early detection, horses with stifle region lameness are assumed to have a soft tissue problem and are treated with decreased training and NSAIDs. Occasionally, an internal blister is injected around the patellar ligaments, especially if evidence of upward fixation of the patella exists. If effusion of the femoropatellar joint accompanies the upward fixation of the patella, radiographs should be obtained. Results are usually negative, but some horses have underlying osteochondritis dissecans of the lateral trochlear ridge of the femur. Surgical debridement is recommended, especially if a flap-like lesion exists. Osteochondritic lesions usually are detected early in race training if they are clinically important. Rarely, subchondral bone cysts of the medial femoral condyle are seen. Horses with subchondral bone cysts are treated by rest, injection with corticosteroids, or surgery. If radiographs reveal evidence of osteoarthritis, such as enlargement of the medial tibial plateau, or if ultrasonographic examination reveals flattening, wrinkling, or other change of the medial meniscus, then the prognosis for racing is diminished. If discovered early in training, horses with osteochondrosis and subchondral bone cysts are best managed with arthroscopic surgery. The prognosis for a horse with a sore stifle, ligament laxity, and intermittent upward fixation of the patella is good, assuming a favorable response to alterations in training regimen. Lameness in Arabian racehorses with sore stifles appears similar to that seen in young TBs with tibial stress fractures, but the origin of pain is different. The prognosis for horses with osteochondrosis varies but is poorer if evidence of osteoarthritis exists.

Tarsocrural Osteochondrosis and Distal Hock Joint Pain

Tarsocrural osteochondrosis is an occasional cause of hindlimb lameness. If bog spavin is recognized when the horse is a weanling or yearling, arthroscopic surgical removal of osteochondritic fragments usually is performed then. However, horses may arrive at the racetrack or training stable with mild tarsocrural effusion. If lameness is observed, if a horse has a positive response to upper limb flexion, or if moderate effusion is persistent, then radiographs should be obtained. If osteochondritic fragments are found, I recommend arthroscopic surgery and a short (2- to 3-month) period of rest before training resumes.

Distal hock joint pain occurs in the Arabian racehorse and is seen most commonly after changes in track surfaces. These horses often do not push off or propel themselves well behind, may refuse to grab the bit or bow the neck, and use the front end to pull ahead, a gait that may lead to secondary forelimb lameness. Clinical signs often are lacking, and an upper limb flexion test may be only mildly positive. Radiographs are often negative, but scintigraphic examination reveals IRU in the distal hock bones.

Back Pain

Primary hindlimb lameness causes secondary back pain in most Arabian horses, particularly those with primary lameness of the stifle and hock joints. Often back pain resolves after management of the primary hindlimb lameness. However, treatment of back pain concomitantly allows earlier resolution of both problems. Exercise riders or jockeys may suspect back pain and

often report a sensitivity or soreness over the top line. Horses usually show pain on palpation or when pressure is applied along the back. The back is palpated carefully, and pressure should be applied uniformly and gently. Thermography has been of some value in horses with back pain resulting from a poorly fitting saddle. The saddle can be evaluated thermographically and compared with any warm spots on the horse's back. Nuclear scintigraphy may reveal IRU in the summits of the dorsal spinous processes. Radiographic examination may confirm overriding of the dorsal spinous processes. However, radiography and scintigraphy are often negative, and back pain is assumed to originate from soft tissues. Nonetheless, if back pain is severe, I suspect a bony source of pain. The back can be evaluated by ultrasonography, dorsally or rectally, for myositis, nerve root impingement or enlargement, and osteophytes associated with the vertebral articulations.

Proximal Sesamoid Bone Fractures

PSB fractures do occur in Arabian racehorses but are less common than in TBs. Clinical signs, management, and prognosis are similar to the TB racehorse (see Chapter 37). Other fractures, such as mid-sagittal fractures of the proximal phalanx and condylar fractures of the distal aspect of McIII, are rare in North America. However, in the Middle East both medial and lateral condylar fractures of McIII occur.

Metacarpophalangeal Joint Lameness and Carpal Osteochondral Fragmentation

Osteochondral fragmentation or chip fractures of the carpal and metacarpophalangeal joints occur in Arabian racehorses but less frequently than in TBs and Quarter Horses. Smaller body size, a more gradual training regimen, and racing older horses likely account for the difference in incidence. Early signs of arthrosis without chip fracture resolve quickly, with minor interruption in race training. The diagnosis of fetlock or carpal osteochondral fragments is straightforward. Arthroscopic surgery to remove osteochondral fragments is well accepted and successful. The prognosis depends on location, size, duration, previous treatment, and amount of associated cartilage damage. Horses with acute osteochondral fragments with only mild cartilage damage have a good prognosis. The decision for surgery often is based on economic factors.

Proliferative synovitis (villonodular synovitis) and associated fragmentation of the dorsal, proximal aspect of the proximal phalanx occurs in the young Arabian racehorse. Horses have characteristic signs of effusion and a noticeable dorsal swelling. Dorsal swelling can be insidious and go unrecognized early in the disease process. Plain radiographs often reveal soft tissue swelling on the dorsal, distal aspect of McIII and osteochondral fragments of the proximal phalanx. Radiolucent changes of McIII are seen in horses with severe proliferation. Ultrasonographic examination usually reveals enlargement of the dorsal synovial pad. I recommend arthroscopic evaluation, removal of osteochondral fragments, and debridement of the synovial pad with a 5.2- or 3.4-mm suction punch (Dyonics; Andover, MA).

In older Arabian racehorses, chronic osteoarthritis of the metacarpophalangeal joint is recognized. The trainer complains of poor performance or racing below previous levels. The metacarpophalangeal joint is enlarged from effusion or fibrosis and is warm. Horses usually respond positively to lower limb or fetlock flexion tests. Comprehensive radiographic examination should be performed. Radiographic evidence of osteoarthritis, such as marginal osteophytes of the PSBs and joint space narrowing, enthesophytes at capsular attachments, and soft tissue thickening often are seen. Osteoarthritis of the metacarpophalangeal joint is seen frequently without osteochondral fragments and appears to be related to chronic wear and tear. In some horses osteoarthritis can be managed by judicious use of intra-articular medication, but the prognosis for return to previous performance levels is guarded.

Lameness of the Foot

Although Arabian horses are alleged to have solid foot structure, they do get sore feet. Long-toe, low-heel conformation is not common. Arabian horses are protected from some of the lameness conditions of the feet simply because of small body size and weight. Sore feet develop after a fast workout or race on a hard, packed racetrack. Trainers often recognize signs, and the condition is managed using ice baths, NSAIDs, and 3 to 4 days of rest.

PROCEEDING WITHOUT A DIAGNOSIS

Occasionally, lameness is suspected but cannot be pinpointed. Horses with such lameness are characterized by a drop in performance, increase in race times, a subtle gait change, refusing to switch leads, a drop in class, or acting sore, but no clinical signs are observable. In this situation, I usually recommend a whole body scintigraphic examination, but correlating findings with clinical signs is often difficult. Comprehensive evaluation for poor performance considers not only a musculoskeletal problem but also cardiovascular and muscle abnormalities. Arabian horses may be more fragile and highly strung than other racehorses, and some trainers attribute poor performance to this portion of the horse's personality. Under certain circumstances the Arabian racehorse may not endure hard training or racing.

CHAPTER • 113

National Hunt Racehorse, Point to Point Horse, and Timber Racing Horse

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DESCRIPTION OF THE SPORT

For as long as horses have been domesticated and ridden, they have been raced. The oldest record of racing in Britain shows that the Romans used to race their horses in Chester. Subsequently, little is known about any organized horse racing during the Middle Ages, but by about 1150 racing had become established at Smithfield, a horse market, where horses were tried and sometimes raced before sale. By the early part of the sixteenth century, racing had returned to Chester, where the prize for the winner in 1511 was a silver bell.

All of these races were on turf with no obstacles to negotiate. At about the same time that horses were competing for the Chester bell, fox hunting (rather than hunting deer or wild boar) started to become established and rapidly increased in popularity. One reason for this may have been the changing agricultural landscape as more and more land was enclosed, providing natural obstacles for those following the hunt to jump. Inevitably, rivalry developed between those who regularly followed fox hunts across country as to who had the fastest horse, and a new sport was born, known as steeplechasing. The origin of the name is simple. Because no courses were defined over which the races could take place, the participants had to race from one church to another, using the high church steeple toward which they were heading as a landmark. The riders could choose their own route and had to jump a variety of fences such as hedges, banks, walls, timber fences, and brooks during the course of the race. The first steeplechase of this type was held in Buttevant in Ireland in 1752, when two neighbors raced between Buttevant church and the St. Leger church, a distance of 4½ miles (7.2 km).

After a time this new sport was formalized, with specially constructed courses, which allowed more participants to take part and more spectators to watch. The first such organized race meeting in Britain was held at St. Albans in 1830. The Grand National was first staged in 1839, the National Hunt Steeplechase followed at Market Harborough in 1859, and the first meeting at Cheltenham (Prestbury Park), arguably the best known modern jump racing venue, was in 1898.

As the sport developed, regulating it became important, but the Jockey Club, which had been regulating flat racing since the mid-eighteenth century, regarded the new sport with suspicion. Accordingly, a separate National Hunt Committee was established in 1866 and continued to run jump racing until 1969, when it and the Jockey Club merged to bring all racing, on the flat and over jumps, in Britain under one governing body. The National Steeplechase Association oversees racing over fences in the United States.

Modern National Hunt racing now consists of several categories, all run on turf on clockwise and counterclockwise courses. Horses run under similar rules in the United Kingdom, Ireland, and France. Elsewhere in Europe, racing over fences takes place but on a much lower scale. Most races are hurdle races or steeplechases, but some races run over more natural obstacles remain, some over exclusively timber fences and some, called National Hunt flat races, that are run without obstacles.

Apart from the fences, three major differences exist between National Hunt and flat racing, which are important in the epidemiology of the injuries that may occur in the different sports. These differences are the race distance, the age of the horses, and the weights of the riders. All National Hunt races are more than a minimum of 2 miles (3.2 km) compared with the minimum distance of 5 furlongs (1 km) on the flat, and the horses are at least 3 years old. Forty percent of National Hunt Flat races involve horses of 4 years of age, a slightly higher percentage are 5 years of age, and less than 20% are 6 years of age or older. Most horses competing in flat races are 2 or 3 years old, although for horses over the age of 10 to race in National Hunt races is not unusual, especially in steeplechases. Finally, National Hunt horses carry between 133 and 175 lb (60 to 79 kg), considerably more weight than flat horses.

Hurdle races are held over fences smaller than those encountered in steeplechasing. In Britain, most hurdles are based on the simple portable fences used to create temporary pens for sheep. They are usually 72 inches (1.83 m) wide and must be not less than 42 inches (107 cm) from top to bottom bar and constructed of ash or occasionally oak. Several hurdle sections are placed end to end to produce an obstacle that must be at least 30 feet (9 m) wide. Each hurdle section consists of two uprights with pointed legs that are driven into the ground and five horizontal rails, between which is interwoven birch or another suitable material. Gorse, which is durable but has sharp thorns, is not permitted. The hurdles must be driven into the ground at an angle of 62° so that the top bar is set back 20 inches (51 cm) from the vertical, and the effective height of the hurdle is 37 inches (94 cm). All of the exposed timber parts must be padded with a minimum of ½ inch (1.3 cm) of high-density polyethylene or closed cell foam rubber (Fig. 113-1).

Timber hurdles have the advantage that if a horse misjudges the fence and does not jump it cleanly, the hurdle gives way on impact. Old style hurdles were not padded as well as the modern versions and occasionally led to lacerations on the dorsal aspects of the hindlimbs, which could be extensive with degloving injuries of much of the metatarsal region. These injuries have been virtually abolished by the new padding.

In countries other than Britain and Ireland, timber hurdles are replaced by fences that look like small versions of steeplechase fences. Such fences also are seen on a small number of racecourses in Britain, and proponents of these types of fences argue that they provide a better introduction to racing over obstacles for horses that ultimately are intended to be steeplechasers. This may be true, but only 35% of horses that run over hurdles convert to steeplechasing, indicating that hurdle

Fig. 113-1 A hurdle race. The jump of the leading horse, which has almost run through the hurdle, is awkward, the hindlimbs are positioned asymmetrically, and the horse's back is hollow.

racing has become a specialty sport that draws many of its participants from horses that have raced previously on the flat.

Steeplechases are run from 2 to 4½ miles (3 to 7 km) over fences which, with the exception of those at Aintree over which the Grand National is held, have a standard construction. The course must have at least six fences per mile, one of which must be an open ditch, with the other plain fences. The plain fences must be a minimum of 54 inches high (1.37 m) and constructed of birch, or birch and spruce, in a frame. The use of gorse is not permitted. The base of the fence must be 72 inches (1.83 m) from front to back, with the thickness of the fence at its top not less than 18 inches (46 cm) (Fig. 113-2, *A*). Plain fences usually have a guard rail on the face of the fence that usually is padded with the same material as the hurdles. An open ditch has similar overall dimensions, but the ditch in front of the fence, which may or may not be dug out, must be at least 72 inches (183 cm) from front to back and be delineated by a takeoff board that is up to 24 inches high (146 cm). Designers of racecourses also, if they wish, may include a water jump in steeplechases. These consist of a smaller fence, up to 36 inches (91 cm) high with a 108-inch (2.74-m) wide water ditch, that must be 3 inches (7.6 cm) deep, on the landing side of the fence.

Point to point races (Fig. 113-2, *B*), named because originally they were run from one point to another, represent the amateur branch of steeplechasing and are restricted to horses that have qualified to race by hunting with a registered pack of hounds. Races for such horses also take place on licensed racecourses and are known as Hunters' Steeplechases.

Some races, notably in France, at Punchestown in Ireland, and at Cheltenham in Britain, are run over more natural obstacles, including banks and growing hedges. Timber races are held over upright (United States) or sloping (Britain) post and rail fences (Fig. 113-3). To make the obstacles less dangerous, the top rails may be sawn through so that they will knock down if they are hit hard.

Finally, National Hunt flat races are staged for horses that have not run previously on the flat and are at least 4 years old. The races are intended to teach horses to acclimatize to the environment of a racecourse and the rigors of a race, without the additional hazard of obstacles. They also provide a way of demonstrating a horse's ability, so that it can be sold. Colloquially, National Hunt flat races are known as bumpers, because originally they were restricted to amateur riders, and the combination of inexperienced riders and horses led to their pejorative nickname.

NATIONAL HUNT HORSES

British and Irish National Hunt horses may be Thoroughbreds, which are registered in the General Stud Book, or non-Thoroughbreds that are in the Non-Thoroughbred register. Many top-quality French jumping horses are of the Selle Français breed. Horses are started in jump racing by one of two routes. They are raced on the flat at 2 or 3 years of age before moving on to hurdling and possibly to steeplechasing, or they are bred specifically for National Hunt racing. Red Rum, who won the Grand National on three occasions, is an example of a horse that started racing in flat races as a 2-year-old. In general, however, horses that graduate from the flat tend to be restricted to hurdle races, and this is why only 35% of horses that race over hurdles go on to race in steeplechases.

Steeplechasers, however, tend to be bred for that particular type of racing and are usually bigger framed Thoroughbreds compared with flat racehorses. Breeding steeplechasers is less straightforward than breeding for flat races, because most steeplechasers are geldings, meaning that it is unlikely that males can be chosen based on racecourse performance. Finding performance-tested mares also is difficult, because the average age of steeplechasers is the oldest of all racing categories, and by the time a mare has proved her ability, she may be past her breeding prime. Therefore most stallions that are popular as sires of steeplechasers are horses that have shown stamina on the flat and then prove to sire successful progeny. Many mares

Fig. 113-2 **A,** A steeplechase race in France. **B,** A Point to Point race. The fences are smaller than in steeplechase races, and the amateur jockeys tend to be less well positioned.

that are used to breed steeplechasers are chosen because of pedigree rather than performance.

Once foaled, many horses destined for steeplechasing are left unbroken until 3 or 4 years of age, when they are often sold as National Hunt stores intended to start a racing career at 4 or 5 years of age. This traditional system has been used for many years and could be said to have stood the test of time. However, recent preliminary research suggests that horses benefit from an early introduction to regular exercise and from early racecourse experience, and this may reduce the risks of injury.

TRAINING NATIONAL HUNT HORSES

At its simplest, training involves conditioning the cardiovascular, respiratory, and musculoskeletal systems of horses to tolerate maximal exercise. The skill of the trainer is to exert the horse sufficiently to achieve this while avoiding physical injury and without inducing an aversion to hard work. Human athletes, being motivated to succeed, tolerate extreme discomfort during training to achieve their goals. Horses have to be encouraged to exercise and never to anticipate that the result of exercise will be discomfort or pain.

Horses that move to National Hunt racing from flat racing receive the basic conditioning as yearlings and young 2-year-olds. Store horses (horses bred specifically for National Hunt racing), however, may do little regular exercise until they are virtually skeletally mature at 4 years. Because they are older, thinking that they require less time to adapt to exercise is tempting, whereas the reverse may be true. It is therefore essential that early preparation is graduated gently and that early signs of failure to adapt, such as sore shins or joint effu-

Fig. 113-3 A timber race. The horse is jumping well, and the rider is in good balance.

sions, are noted and training intensity adjusted. If clinical signs go unrecognized or ignored, more serious skeletal defects may develop, such as stress fractures of the tibia, humerus, or pelvis, which may precede catastrophic fractures on the gallops or racecourse. Traditionally, store horses spent at least 6 weeks walking and trotting on quiet roads and tracks before they commenced any faster work. This initial slow preparation has now been abandoned by many trainers, partly because of the difficulty of finding a suitable safe, quiet environment and partly because of the economic pressure to see the horse on the racecourse.

Most trainers of National Hunt horses now use a simple adaptation of interval training over distances of about 1000 m, almost invariably up an incline that may be steep. An average morning workout would be an initial slow warm-up, followed by two brisk canters up the incline on an easy morning, alternating with three faster ascents on a work morning.

One of the most important aspects of training National Hunt horses is teaching them to jump appropriately. Hurdle races are conducted at a fast pace, and some trainers believe that horses that jump the obstacles without touching them and with the same action as a show jumper, use energy unnecessarily and concede ground to rivals who jump low and flat. This is possible because the timber hurdles give way if the horse hits them, although the ease with which they do this depends on the ground into which the legs of the hurdles are driven. Once horses have acquired this style of jumping, some trainers argue that the horse finds it difficult to jump the larger, more solid, steeplechase fences. This accounts for the relatively low number of horses that make the transition from hurdling to steeplechasing and the demand in Britain by some trainers for a brush hurdle that, although relatively small, has to be jumped with care. Specialist steeplechasers are encouraged to jump much as horses intended for other disciplines that involve jumping, and they jump low poles and logs, with and without a rider, before progressing to larger obstacles. However, the amount of training carried out over fences by steeplechasers is proportionately much less in the United Kingdom and Ireland than for event horses or show jumpers.

RACING NATIONAL HUNT HORSES

Jump racing developed as a winter sport, probably originally because of the connection with fox hunting, which also is conducted during the winter months. However, jump racing is now held in Britain throughout the year, although those courses that hold summer jump meetings are required to ensure by artificial irrigation that the ground conditions are kept no worse than good to firm. This is because epidemiological studies have shown that firm ground conditions are more likely to be associated with serious injuries. The reason for such a relationship is complex. It is probably related to the speed at which the horses travel, but other complex factors influence the interaction of the horse's foot with the ground under various conditions, some of which are related to ground hardness, and these require further research and elucidation. Jump racing remains seasonal, however, because the major races take place between November and March, and many horses spend a few weeks turned out during the summer. Seasonal racing influences injury management, because if a horse sustains a significant injury in, for example, late February, the owner or trainer applies pressure for the horse to be ready for the next season, that is, to resume training by October of the same year.

Horses are trained by individual trainers spread throughout the United Kingdom, Ireland, and France who may travel long distances to compete. Therefore any single veterinary surgeon usually does not deal with more than 4 or 5 trainers and their horses. At race meetings the horses are subjected to pre-race veterinary inspections, and each race is monitored carefully by veterinary surgeons driving on the outside of the track alongside the race and a veterinarian observing the entire race from an appropriate vantage point.

Point to point races are held between January and June. Point to point racing is an amateur sport, raced over obstacles that are smaller and softer versions of the steeplechase fences on licensed racecourses. Some horses that perform well in Point to Points successfully graduate to steeplechasing, and this route to steeplechasing is chosen by some owners in preference to hurdle racing, possibly after one or more National

Fig. 113-4 A faller at a Point to Point race. Note the extreme position of the hindlimbs.

Hunt flat races. While National Hunt horses run on average between four and five times per year, Point to Point horses may run more frequently during the season, because the race meetings are usually held at weekends. However, the average number of starts per Point to Point horse in 2000 was only three.

Because of the two distinct sources of horses that enter National Hunt racing, a wide variety of injuries are seen, ranging from injuries related to beginning training to degenerative injuries associated with overuse. In addition to the injuries sustained while the horse is in training, a National Hunt horse is more prone to injury after a fall than is a flat racehorse (Fig. 113-4). It is also important to be aware that horses that are skeletally mature when they begin training (4- to 5-year-old store horses) still suffer from the same pathophysiological processes that lead to stress fractures, albeit in different sites from the 2- or 3-year-old Thoroughbred. Because National Hunt racing continues throughout the year, the going under foot (footing) can vary and both extremes of soft and firm going place the National Hunt horse under extra stress from injury.

A substantially higher death rate occurs in National Hunt racing compared with flat racing. In a retrospective analysis of data from all starts between January 1990 and December 1999, 2015 deaths were recorded on racecourses from 719,099 starts.[1] The death rate per 1000 starts was substantially higher in steeplechasers (6.7; 34.5% of the total) and hurdlers (4.0; 43.4% of the total) compared with flat racehorses (0.9; 18.8% of the total). Spinal injuries occur much more frequently in hurdlers (19% of all hurdle deaths) and steeplechasers (23% of steeplechase deaths) compared with flat racehorses (1% of flat racehorse deaths). Tendon breakdown injuries resulting in humane destruction at the racecourse were also substantially higher in hurdlers (20% of hurdler deaths) and steeplechasers (14% of all steeplechase deaths) compared with flat racehorses (8% of flat racehorse deaths). Risk of mortality was associated with a number of variables. With steeplechase horses a higher risk occurred in horses that started steeplechase racing at 8 years of age or older. The weight carried was also influential, with horses carrying more than 70 kg minimum weight being more at risk. Races longer than 4 miles had a higher risk than shorter races. Heavy going, resulting generally in slower speeds, reduced the risk. Good to firm or hard going increased the risk of mortality in hurdlers and steeplechasers.

TIMBER RACING

Timber racing is considerably more popular in the United States than in the United Kingdom and is more structured. Novice or stakes horses compete only against each other, with greater prize money for stakes races, the most valuable being the Maryland Hunt Cup. The most prestigious race in United Kingdom is the Marlborough Hunt Cup. Both of these races are open only to amateur jockeys. Timber racing horses often have raced previously on the flat or over hurdles, are usually 6 to 12 years of age, and may have injuries from earlier racing.

TRACK SURFACE OR TRAINING SURFACE AND LAMENESS

The surfaces and terrain over which horses train vary extremely because the trainers are dispersed widely geographically. Much of the work is done on grass, but fast work often is done on all-weather purpose built gallops. Many horses hack up to a mile to and from the gallops, ensuring good warm-up and warm-down. However, the standard of maintenance of the gallops varies. Poor gallops with an inconsistent surface varying from soft to deep may increase the risk of tendon injuries or predispose horses to stumbling and accidents such as third carpal bone fractures. Many trainers are based in areas of chalk downland (natural rolling hills with a chalk subsoil), which drains well, but the large number of flints (sharp stones) in the soil may result in a high incidence of bruised soles or sole punctures unless the horses have well-conformed feet. The steepness of the terrain over which the horses do fast work may influence injury. An increased number of pelvic fractures was noted after a new gallop was laid, the last section of which was up a steep incline (R.v.P.).

Fig. 113-5 Horse No. 12 is about to be brought down by a fallen horse and jockey.

The influence of falls on the nature of injuries is substantial. Most falls occur on landing over a fence. Falls may result from the horse or jockey making a jumping error, from interference by another horse still in the race, or from a loose horse that had previously unseated its rider. The fall of one horse may result in the fall of one or more other horses (Fig. 113-5). Thus injuries may result from the fall and impact with other horses. Falls may result in fatal fractures of the cervical or thoracolumbar vertebrae. Rib fractures usually result from a fall and may cause severe lameness and or respiratory signs. Other fractures seen commonly, usually resulting from a fall, include scapular, radial, and humeral fractures; fractures of the accessory carpal bone; and fractures of the lateral malleolus of the tibia. Major muscle ruptures, especially in the hindlimbs (e.g., semimembranosus, quadriceps, or adductor) also usually result from a fall. Rupture of fibularis tertius may occur if the horse falls with forced hyperextension of the hock.

A significant statistical correlation exists between certain factors and the incidence of injury on racetracks:

- Increased firmness of the going results in an increased injury rate in all forms of National Hunt racing.
- Increased incidence of injury on firm ground is further increased by increasing the length of the race.
- In hurdle races run over more than 2½ miles with only a few obstacles (6 to 8), the casualty rate increases compared with the same distance with nine or more fences.
- Races ridden by amateur riders carry a higher risk of serious injuries.

Flexor tendon lacerations frequently are sustained as horses race over fences and generally occur on the palmar aspect of the metacarpal region, proximal to the proximal sesamoid bones (PSBs). It is important to recognize that the site of a skin laceration may not coincide with the site of a tendon laceration.

Some important injuries occur more commonly during racing than training. Luxation of the superficial digital flexor tendon (SDFT) from the tuber calcanei sometimes occurs. Rupture of the musculotendonous junction of the superficial digital flexor muscle is an unusual injury, but is an important injury in steeplechasers. Superficial digital flexor tendonitis is common in National Hunt horses, and recurrent injuries may result in complete rupture of the tendon.

CONFORMATION AND LAMENESS

With an increasing proportion of National Hunt horses starting training earlier, and running first on the flat at 3 years of age and then over hurdles at 4 years of age, the trend has been toward using smaller, lighter-framed horses. Although such horses are not necessarily more prone to injury, they seem less able to cope with deep, holding going often encountered in the winter months compared with the more traditionally bred rangy National Hunt store horses, which are often rather late maturing.

For a National Hunt steeplechaser or hurdler to race until 10 years of age is not uncommon, so the racing career is considerably longer than for a European flat racehorse. Horses should be well balanced and proportioned, with good feet and adequate bone for body size and weight. Horses with back-at-the-knee conformation, carpus valgus, offset knees, or substantial toed-in or toed-out conformation particularly maybe predisposed to forelimb problems. A long back may be associated with an increased risk of back problems. A horse with straight hocks or long, sloping hind pasterns may have an increased risk of hindlimb lameness.

THE TEN MOST COMMON CAUSES OF LAMENESS

The following are the 10 most common lameness conditions of hurdlers, steeplechasers, and Point to Point horses:

1. SDFT injuries
2. Suspensory ligament (SL) injuries
3. Lameness associated with the carpus
4. Lameness localized to the hocks
5. Lameness associated with the pelvis

6. Lameness localized to the feet
7. Fractures of the third metacarpal (McIII) and third metatarsal (MtIII) bones
8. Lameness localized to the metacarpophalangeal joints
9. Traumatic injuries, particularly of the back and neck, after falls
10. Back problems

The following are the 10 most common lameness conditions of timber racing horses:

1. Superficial digital flexor tendonitis
2. SL desmitis
3. Osteoarthritis of the distal hock joints
4. Distal sesamoidean desmitis
5. Osteoarthritis of the metacarpophalangeal joint
6. Soft tissue trauma of the stifle
7. Interference injuries during racing
8. Timber shins
9. Fracture of the patella
10. Fracture of the accessory carpal bone

LAMENESS EXAMINATION

Diagnosis of lameness starts with a full history, which should include stage of training, because, for example, a 6-year-old store horse starting training is as susceptible to stress fractures as an immature athlete. The clinician should establish whether the horse has run recently. Is the lameness of recent onset, or is it a chronic problem that has been getting progressively worse? Some trainers request advice as soon as lameness is recognized, whereas others may restrict the horse to light work and seek veterinary advice only if lameness persists. Some trainers treat a lame horse with phenylbutazone. Many trainers are happy for horses to come out rather short, shuffly, and stiff and warm up to move more freely; however, inevitably more overt lameness usually supervenes or a back problem develops secondarily.

It is important to determine if the horse has had any time off recently with the present trainer or a previous trainer that may suggest a previous injury. There may be a history of warmth associated with the palmar metacarpal region which, if the horse has been subsequently rested, may not be obvious on clinical examination. Does the horse have any history of trauma? Jumping history is also important: did the injury occur while the horse was schooling over fences or in a race? Did the horse fall or collide with another horse (Fig. 113-6)? Does the lameness improve with rest or with work?

If the horse had a history of a fall, it is important to establish how the horse fell. Did the horse turn a full somersault and land on its pelvis and develop lameness thereafter? A pelvic injury should be suspected. Did the horse fall at the end of a 3-mile race on heavy going and lay winded? Information from the racecourse veterinarian may be particularly useful.

If a horse has a history of poor jumping performance, trying to establish if the horse has ever been a good jumper over hurdles or fences is worthwhile. Many of these horses are lame. The veterinarian needs to find out how the horse is jumping. Does the horse stand off the fences or jump flat? If a horse does not want to take off, it may have a hindlimb problem. If the horse jumps flat, it may have a back problem. If the horse is reluctant to land, it may have a forelimb problem.

If a horse is presented for evaluation for poor performance, it is important to try to assess the orthopedic component. About 50% of horses with a complaint of poor performance are lame. When did the horse last race and over what ground conditions? Did the jockey make any comments when he dismounted? Has the horse coughed? Is rectal temperature routinely monitored? Routine hematological examination and measurement of fibrinogen, comparing results with a baseline for that horse, are useful for detecting systemic abnormalities. Endoscopic examination of the upper airways and trachea, combined with a tracheal wash, are useful screening tools to eliminate a respiratory component to the problem.

Fig. 113-6 A fall at The Chair at the Grand National. The horse pitches steeply and lands on its neck.

The clinical examination does not differ from a routine lameness evaluation of any other type of horse, but because many horses have chronic problems with which they have been living until more obvious lameness supervened, the entire horse should be assessed, not just the postulated lame limb. In view of the high incidence of superficial digital flexor tendonitis and suspensory desmitis, particular attention should be paid to the palmar metacarpal soft tissues. If the horse has a history of a fall while jumping, particular attention should be paid to the neck, back, and pelvic regions.

A thorough clinical examination may reveal palpable abnormalities in the palmar metacarpal region, evidence of effusion, or pain on flexion of a particular joint. Skeletal pelvic asymmetry or muscle wastage over the quarters also may be evident. Back and pelvic reflexes and the tone of the back and pelvic muscles should be assessed. Is any evidence of guarding apparent? Abnormal shoe wear may give clues about which limb or limbs are lame, which is otherwise not always easy to determine in a horse moving short because of pain in several limbs.

Dynamic examination at the walk and trot in a straight line, followed by flexion tests, should be followed by examination on firm and soft going on the lunge at the trot and canter. The horse should be turned tightly to the left and to the right. If a history of a fall exists, a complete neurological examination should be performed.

If a horse is lame after a recent fall, the investigative approach depends on the degree of lameness and the rate of improvement. A horse with a pelvic injury is usually very lame initially, although lameness associated with an ilial wing fracture usually improves substantially within 24 hours. Lameness associated with an ilial shaft fracture is usually persistent, and the horse remains extremely lame. These horses should be cross-tied, assuming the horse's temperament is suitable. Some ilial wing fractures can be detected with ultrasonography, but the diagnosis of others requires nuclear scintigraphy. Nuclear scintigraphy may give false-negative results if done before 5 to 7 days after injury. If a horse shows only mild to moderate lameness after a recent fall, the horse generally is allowed rest for 7 to 10 days and is then re-assessed, and only if lameness persists is further investigation carried out.

DIAGNOSTIC ANALGESIA

If no obvious cause of lameness is apparent, then diagnostic analgesia is performed, but no particular differences in approach exist for this type of horse. However, if clinical signs suggest an intra-articular problem, such as synovial effusion and pain on passive manipulation of a joint, then intra-articular analgesia of the suspect joint may be performed first. If intra-articular analgesia is carried out, clipping a small area is preferred, but some trainers are reluctant to allow this, and provided that the hair coat is not excessively long, a timed 5-minute surgical scrub is performed before injection.

If a fracture is suspected based on the history or clinical signs, nerve blocks are not performed. A horse that pulled up lame on the gallops or finished work and then became severely lame while walking home may have a stress fracture. Stress fractures of McIII and MtIII and the tibia are common. If a stress fracture is suspected, the horse is examined radiographically or scintigraphically.

If soft tissue swelling is identified clinically, then ultrasonography is performed routinely as the next diagnostic step.

IMAGING CONSIDERATIONS

Radiography is performed routinely using standard radiographic projections. Special views at different angles may be required for demonstrating specific lesions, based on the preliminary examination. Some stress fractures are difficult to identify radiographically, and if a fracture is suspected on clinical grounds, radiographic examination is repeated 10 to 14 days later.

If the clinician suspects an abnormality of the palmar metacarpal soft tissues, an ultrasonographic examination should be performed. In view of the high incidence of bilateral lesions, both limbs should be examined routinely. A systematic approach is essential, focusing on the SDFT, deep digital flexor tendon, and SL in turn. It is almost invariably necessary to clip the metacarpal and metatarsal regions to achieve satisfactory image quality, because most National Hunt horses have thick guard hairs. However, satisfactory images of the pelvis usually can be obtained after washing with chlorhexidine for about 10 minutes, soaking with alcohol, washing off (to avoid alcohol-induced damage to the transducer), and liberal application of coupling gel.

Transverse and longitudinal images of the metacarpal and metatarsal regions are required to gain a full appreciation of the severity of injury. An injury index giving a quantitative assessment of damage can help in communication with trainers and may help to convince them of the significance of an injury associated with only mild clinical signs. Each structure should be evaluated sequentially from proximally to distally at predetermined measured intervals (4 or 5 cm) distal to the accessory carpal bone or examining each zone (see Chapter 17). Cross-sectional area (CSA) or circumferential measurements are made at comparable sites in each limb. Measurement of the size of damaged fibers is also useful. One author (R.v.P.) describes the severity of a core lesion by multiplying the percentage of CSA of the tendon damaged by the length of the lesion and by the percentage of the fibers damaged within the injury (assessed visually). For example, a core lesion that occupies 10% of the CSA of the tendon and extends 10 cm proximodistally and is assessed visually as having 75% of the fibers within the core lesion damaged has an index of $10 \times 10 \times 0.75 = 75$. A similar lesion occupying 30% of the CSA has an injury index of $30 \times 10 \times 0.75 = 225$. All images should be recorded routinely for future comparisons.

Nuclear scintigraphy is indicated as a screening tool when a lame horse is presented after a bad fall, particularly when a moderate to severe lameness has persisted for more than 3 or 4 days. Scintigraphy also may be indicated if severe, sudden-onset lameness occurs with no localizing clinical signs to rule out fracture before a dynamic workup is contemplated. Scintigraphy often is more rewarding in horses with acute injuries than those with chronic lameness. Scintigraphy usually can be targeted to specific areas such as the pelvis or both hindlimbs unless the horse has lameness involving several limbs. During evaluation of the pelvis, radioactive urine in the bladder can confound interpretation. The use of furosemide may help, but this drug may make the horse fidgety, and catheterization of the bladder, followed by flushing with warm water, may be preferable.

PROCEEDING WITHOUT A DIAGNOSIS

National Hunt horses have a longer career than flat racehorses, and less prize money is available to be won. Therefore the pressure to get a horse sound quickly often is less, and many trainers accept resting the horse if a diagnosis cannot be achieved by the techniques described previously. However, an attempt to reach a diagnosis always should be made.

SHOEING CONSIDERATIONS AND LAMENESS

Most National Hunt horses train and run in light steel shoes with one toe clip in front and two side clips on hind shoes. Some trainers change to a hind shoe with a single toe clip for

racing. Good shoeing is essential. Given the high incidence of foot problems causing lameness, a good relationship with a skilled farrier is invaluable. Poor foot conformation, especially low collapsed heels, may predispose the horse to superficial digital flexor tendonitis. Bar shoes combined with a rolled toe often are used for horses with collapsed heels. If a foot is conformed poorly, the risk exists of shoes being pulled off repeatedly in training, particularly if the branches of the shoe are set too far medially or laterally.

DIAGNOSIS AND MANAGEMENT OF LAMENESS IN STEEPLECHASERS, HURDLERS, AND POINT TO POINT HORSES

Superficial Digital Flexor Tendonitis

An epidemiological study demonstrated an 86 % incidence of SDFT injuries in National Hunt horses.[2] Although most of the injuries occur in the forelimbs, hindlimb injuries also occur. Many trainers routinely assess forelimb SDFTs daily, and some are adept at detecting relatively subtle lesions. Veterinary involvement may enhance these skills. Many, but not all, trainers are keen to have ultrasonographic assessment of suspected tendon injuries. Some trainers tend to ignore suspicious injuries at the end of a season, contrary to veterinary advice. This may be one reason why a peak incidence of tendon injury tends to occur at the beginning of the season when horses start galloping after a short summer break. Other horses may have been kept in training late in the season to get an extra race and may have suffered injury, which may or may not have been manifest clinically. A second peak incidence of injuries tends to occur at the end of the season, perhaps related to races run on faster going. Superficial digital flexor tendonitis is more common in steeplechasers than in hurdlers, but this may reflect the older population of the horses rather than the type of racing itself. The degree of lameness at the time of acute injury may reflect the severity of tendon damage.

Horses with acute lesions are managed with aggressive anti-inflammatory treatment for 5 days, including systemic non-steroidal anti-inflammatory drugs (e.g., phenylbutazone and eltenac), with or without a single dose of corticosteroids (e.g., dexamethasone), and cold hosing several times daily. Cold water bandages or cold kaolin is applied to the limbs. Some thin-skinned horses are prone to blistering, so any bandaging must be done with care. Other popular proprietary poultices, such as Animalintex (Robinson Animal Health Care, Chesterfield, UK) and a variety of clay-based preparations, may irritate small unnoticed wounds and are therefore avoided.

A first ultrasonographic examination is performed about 7 days after the injury is first recognized. Horses with central core lesions may be treated by decompression, using needle fenestration or a fixed blade. This is performed with the horse sedated and using regional analgesia. Follow-up ultrasonographic examinations frequently are performed 3 to 4 weeks after injury, because the preliminary examination may underestimate the degree of damage because of ongoing enzymatic degradation. This gives a baseline scan for the injury.

Many differing views are given on the best management of superficial digital flexor tendonitis in National Hunt horses. Adequately rehabilitating a tendon that was injured late in a season so that the horse can race the following season, without a disproportionately high risk of re-injury, is difficult. Firing remains a popular treatment, and because many owners and trainers are prepared to rest a horse for 12 months after firing, when they would not give such a long convalescent period if rest alone was recommended, this treatment still is carried out widely. Nonetheless, the re-injury rate remains high, so many alternatives have been tried with various success. These alternatives range from at least 9 months of field rest, intralesional injection of hyaluronan, polysulfated glycosaminoglycan, a phenol derivative, β-aminoproprionitrile fumarate or growth factors, combined with a variety of exercise regimens. Intralesional β-aminoproprionitrile fumarate combined with a strictly controlled exercise program has produced good results in selected horses (R.v.P. and S.J.D.); however, the time for return to racing often is prolonged, in part dictated by the time of injury and the seasonal nature of racing. The average time to return to racing was 17 months. The owner or trainer must be committed to at least 3 to 4 months of walking exercise after treatment, and some horses are unsuited to this temperamentally, unless a horse walker is available. Regardless of the methods of management, a slow, gradual rehabilitation before resuming ridden work seems beneficial. Ideally the horse should be walking on a horse walker daily for 3 months before resuming ridden exercise. The horse can be turned out during this 3 month period. A cage horse walker in which the horse is free and in which the speed can be set manually to encourage the horse to walk briskly is best.

Serial ultrasonographic examinations are particularly useful as exercise is resumed, about 1 month after resuming walking, then again before cantering commences, and a third time before fast work commences. Commonly as work intensity increases, small hypoechoic areas develop, especially in areas in which the fiber pattern is not parallel. In some horses these lesions disappear, provided the horse is maintained at the same exercise level, whereas in others a core lesion redevelops, and the trainer should be warned accordingly.

The prognosis is related partly to the severity of injury.[3] In a follow-up study of 73 National Hunt and Point to Point racehorses, lesions were graded by ultrasonography as mild (<50% CSA or <100 mm in length), moderate (50% to 75% CSA or 110 to 160 mm in length), or severe (>75% CSA or >160 mm in length). All mildly affected horses returned to training and 63% raced. Fifty percent of horses with a moderate lesion resumed training and 23% raced, but only 30% of horses with severe lesions resumed training, despite up to 6 months longer convalescence, and 23% raced. The mean re-injury rate of those resuming work was 40% in the period of follow-up (9 to 30 months).

The type of racing in which the horse is involved may influence the prognosis for return to racing after superficial digital flexor tendonitis. Fifty-one (73%) of 70 hurdlers raced five or more times after desmotomy of the accessory ligament of the SDFT for treatment of tendonitis compared with 39 (58%) of 67 of steeplechasers.[4]

Cellulitis, skin necrosis, and necrosis of the underlying SDFT are poorly understood conditions that have been recognized in National Hunt horses (S.J.D.) and flat race horses that have run over long distances (>1½ miles) (R.v.P.). Many horses run in boots or exercise bandages, which are removed after racing. Whether exercise bandages would be on long enough to cause pressure necrosis is debatable. The typical history is that a proprietary clay-like substance (Kaolin, Ice-Tite) or commercial poultice (e.g,. Animalintex) is applied to the metacarpal regions after racing, with or without overlying bandages. The applied substance is removed the following day. Clinical signs may be apparent within 24 to 72 hours, with the development of peritendonous edema and serum ooze, progressing to skin slough and slough of deeper tissues, which may take several weeks. The degree of damage varies between horses, and one or both limbs may be affected. One author (S.J.D.) has seen this unassociated with superficial digital flexor tendonitis, whereas another (R.v.P.) often has seen concurrent superficial digital flexor tendonitis. The prognosis depends on the depth of the lesions. One author (R.v.P.) recommends that horses should be thoroughly cooled after

racing before anything is applied topically to the limbs to minimize the risks of these complications. If a SDFT injury is suspected, then the advice of the course veterinarian should be sought.

Suspensory Desmitis

Suspensory desmitis is a major problem in National Hunt horses, especially steeplechasers and Point to Point racehorses. Injuries of the body or branches occur in forelimbs and hindlimbs, sometimes with a fracture of the distal third of the second (McII/MtII) or fourth (McIV/MtIV) metacarpal or metatarsal bones, apical or abaxial sesamoid fractures, or sesamoiditis. Proximal suspensory desmitis does occur in forelimbs and hindlimbs and is recognized more commonly in hindlimbs, but the true incidence may be underestimated.

Body and branch injuries of the SL often go unrecognized until the ligament is grossly enlarged; possibly some of these are cumulative injuries rather than single-event injuries. More careful monitoring of the SLs by regular palpation with the limb not bearing weight may lead to earlier detection of important lesions. The degree of swelling sometimes makes it difficult to palpate accurately the distal end of McII and McIV.

Assessment of structural damage is done by ultrasonography in a way similar to that described for SDFT lesions. Lesions are assessed acutely (up to 7 days after injury and 4 to 6 weeks later) to determine the baseline degree of injury. Some of the swelling contributing to the apparent swelling of a SL branch is often a periligamentous reaction. Radiographic examination is necessary to evaluate McII/MtII or McIV/MtIV and the PSBs.

Body and branch injuries are associated with a prolonged convalescence irrespective of the way in which they are managed, and returning to racing often takes longer than for a horse with superficial digital flexor tendonitis. The rate of recurrent injury is high. Surgical removal of fractures of McII or McIV (MtII or MtIV) has little bearing on the horse's final outcome.

Conservative management of rest alone, splitting the SL, intralesional injections of β-aminoproprionitrile fumarate, and pin firing have been used with no significant differences in outcome.

Fractures of the Third Metacarpal and Metatarsal Bones

The most common long bone fractures in National Hunt horses are condylar fractures of the McIII or MtIII bones. Such fractures often are recognized in hindlimbs and frequently involve the medial condyle and may spiral proximally. Full evaluation of the fracture may require multiple oblique radiographic views of MtIII. Such fractures are more common in hurdlers than in steeplechasers. With prompt surgical treatment by internal fixation the prognosis is good. Compound, comminuted fractures of the distal aspect of McIII and MtIII may occur during racing and may be associated with luxation of the fetlock. These horses have a guarded prognosis for return to racing.

Incomplete palmar cortical fatigue fractures of McIII occur commonly, especially in horses older than 4 years of age entering National Hunt or Point to Point training for the first time. Pre-existing sclerosis in the proximomedial aspect of McIII detected when lameness is first recognized in some horses suggests that abnormal bone modeling has existed for some time, subclinically or without clinical signs being recognized. Lesions are often bilateral. Lameness is characterized by the horse becoming lamer the further it trots and then improving if walked a few steps. Usually no localizing clinical signs are apparent, and diagnosis depends on localizing pain to the proximal palmar metacarpal region, and identifying radiographic lesions or scintigraphic evidence of increased bone turnover, in the absence of ultrasonographic abnormalities of the proximal aspect of the SL. Treatment of 3 months of rest with a graduated return to work is usually successful, and recurrent injury is unusual.

Less commonly transverse stress fractures of the distal metaphyseal region of McIII cause acute-onset lameness. No localizing signs may be apparent, but lameness is alleviated by a four-point nerve block of the palmar and palmar metacarpal nerves. Usually pre-existing callus formation is evident on the distal metaphyseal region of McIII on the dorsal or palmar aspects.

Lameness Associated with the Hock

The degree of lameness caused by hock pain varies, and lameness is more commonly unilateral, unlike bilateral lameness usually seen in the flat racehorse. The most common cause is osteoarthritis of the distal hock joints, although traumatic injuries also occur. The horse may adduct the lower limb as the leg is brought forward at the walk and trot. The lameness often worsens after flexion and when the lame limb is on the inside on the lunge. More subtle signs of poor jumping or back pain may herald a problem originating in the hocks.

Lameness most often is alleviated by intra-articular analgesia of the tarsometatarsal joint. It is rare to have to inject the centrodistal joint as well, despite the variable communication between the joints and the fact that osteoarthritic changes seen radiographically often affect both joints. Radiography of the hocks should include four standard views, because radiographic changes may be visible only on one projection. Abnormalities range from mild peri-articular osteophyte formation to osteolysis, with narrowing of the joint spaces, with or without peri-articular new bone. Occasionally, young horses in the first year in training have significant radiographic changes in one limb. The damage was probably present before onset of training and may be associated with collapse of the distal tarsal bones or a traumatic incident earlier in the horse's life.

Care should be taken when interpreting scintigraphic images of the hocks in National Hunt horses. Often areas of increased radiopharmaceutical uptake (IRU) are seen but do not appear to correlate with clinical signs of lameness (A.N.).

Treatment of horses with osteoarthritis of the distal tarsal joints usually consists of intra-articular medication of the tarsometatarsal joint with combinations of hyaluronan and corticosteroids. Consideration should be given to chemical fusion of painful distal hock joints in young National Hunt horses.

Traumatic injuries involving the hock are common. Injuries frequently sustained after a fall include fracture of the lateral malleolus of the tibia and tearing of the attachments of the collateral ligaments. The lameness associated with lateral malleolar fractures can be mild, and lesions may be missed if radiography is not carried out. Ultrasonography may be more useful than radiography for the early diagnosis of collateral ligament injury. Follow-up radiography some weeks later may reveal some entheseous new bone formation. Horses with these injuries have a good prognosis for a return to racing, although improvement in lameness may be slow. Some debate exists about the optimal management of horses with lateral malleolar fractures: conservative or surgical. Both treatments carry favorable prognoses. Surgical removal is usually easiest by making an incision directly over the fracture fragment(s) rather than by attempting arthroscopic removal.

Lameness Associated with the Pelvis

Lameness associated with the pelvis in the National Hunt horse is more likely to result from a traumatic incident than a stress fracture. Falls or poor landings while jumping (see Fig. 113-4) may lead to subluxation of the sacroiliac joint or pelvic fractures. However, ilial stress fractures also occur, especially

in horses entering training at 5 years of age or older. A strong correlation between pelvic lameness and working horses on loose all-weather surfaces, especially an unmanaged wood chip surfaces and particularly uphill, has been noted (A.S. and R.v.P.).

Clinical signs can vary from severe lameness with obvious crepitus to mild stiffness. It is important to examine the pelvis per rectum when pelvic damage is suspected. A horse with an ilial fracture sustained during a fall may be lame initially and improve rapidly within 48 hours. If an ilial wing fracture is non-displaced, the horse could return to cantering exercise before lameness recurs. Muscle spasm may result in significant asymmetry of the tubera coxae in the acute stage, but this usually also resolves within 24 hours. Severe lameness associated with an ilial shaft or acetabular fracture is invariably persistent. Ilial stress fractures may cause only mild clinical signs, and sometimes the most important abnormality is shortening of the contralateral forelimb stride as the horse moves off from standing still. This gait abnormality rapidly resolves.

Fractures of the ilial wing involving the dorsal surface often can be diagnosed by ultrasonography, but incomplete stress fractures involving the ventral aspect cannot be seen.

Nuclear scintigraphy is invaluable for assessing pelvic pain. With sacroiliac disease IRU in the sacroiliac joint contralateral to the lame limb occurs, in one contributor's (A.N.) experience. Assessment of pelvic fractures should be delayed for at least 5 to 7 days after injury to avoid false-negative results. If scintigraphy indicates IRU in the region of the coxofemoral joint, obtaining multiple oblique scintigraphic views is worthwhile to try to assess whether the fracture involves the joint. Other scintigraphic changes seen include IRU at the greater and third trochanters of the femur associated with damage to the insertions of the deep and middle gluteal tendons and the superficial gluteal insertion, respectively.

Horses with non-displaced ilial wing fractures have a good prognosis, whereas the prognosis for those with fractures of the ilial shaft or ilial fractures involving the acetabulum is poor.

Back Pain

Back pain in the National Hunt horse may be primary or may develop secondary to chronic lameness in one or more limbs and is being recognized with increasing frequency. Nuisance problems include the development of seromas underneath the saddle, which are probably attributable to an ill-fitting saddle and poor riding. Most trainers regularly use a physiotherapist, who may be the first person asked to look at a horse that is not right. Only if the horse fails to respond to one or two treatments, or if the physiotherapist recognizes obvious lameness, is veterinary advice sought. A good working relationship with the physiotherapist is valuable, because without a doubt the rehabilitation and long-term management of horses with chronic back pain can be helped by regular physiotherapy treatment. However, the veterinarian should have responsibility for both the diagnosis and the development of the treatment program.

Knowledge of back problems in National Hunt horses has increased greatly with more routine use of nuclear scintigraphy. Thorough investigation of the back is warranted in horses with chronic back pain, a history of jumping awkwardly (see Fig. 113-1), or after bad falls. Physical examination of the back often can be unrewarding concerning specific diagnosis, because the examination may only reveal stiffness and pain on palpation. Underlying problems such as active kissing spines or osteoarthritis of the dorsal articular facets of the vertebrae are common in the National Hunt horse and can be ruled in or out using scintigraphy and radiography. In a horse with an acutely sore back after a fall, scintigraphy can be used to determine if any bony damage occurred. The clinician should bear in mind that falls may involve more than one horse, and a fallen horse may get shunted (pushed) by one behind. Serious vertebral fractures can produce nothing more than severe stiffness and guarding in some horses after a fall, if the spinal cord itself is not compromised. For example, scintigraphy may reveal intense focal IRU suggesting a fracture in the region of the second or third lumbar vertebra. Radiography may demonstrate an obvious change in orientation of the spinous processes at this level, without being able to demonstrate a fracture. Lateral and dorsal scintigraphic images are useful to identify fractures of the transverse processes of the lumbar vertebrae.

Ultrasonographic examination is also useful for identifying some soft tissue injuries such as desmitis of the supraspinous or dorsal sacroiliac ligaments.

Horses with traumatic injuries often respond well to prolonged rest combined with physiotherapy. Horses with kissing spines often improve after local infiltration with corticosteroids.

Successful management of chronic back pain needs a broader approach. Many National Hunt horses have an inadequate jumping education and poor technique (see Fig. 113-1). Traditionally in the United Kingdom and Ireland, horses learn to jump by loose schooling over small fences; horses are schooled ridden over two or three small fences, once or twice when they are fresh. In France horses often are trained to jump out of deep going, when they are tired, providing a better grounding for racing conditions. The standard of riding of the work riders is often only moderate, and employing some staff with a background of working with event horses or show jumpers may be beneficial. Time should be spent teaching the horse to jump properly. Often more time is available in the summer break period to devote to such problems.

Additional work from the field, or keeping the horse in work, or bringing the horse in early should be advised. The horse should be encouraged to work in a round outline by exercising in draw reins to improve the development of the epaxial muscles. The horse can be given a warm-up period on a horse walker before being ridden. Use of a well-fitting hunting saddle rather than a racing saddle should be encouraged. After exercise the horse may benefit from being led home from the gallops rather than ridden.

Neck Lesions

Neck trauma usually results from a fall (see Fig. 113-6) and is more common in steeplechasers and Point to Point racehorses than in hurdlers. Neck trauma may result in ataxia or a stiff neck, with or without forelimb lameness, and can cause death. Ataxia may be transient or persistent. Radiographic examination should be performed if ataxia or neck pain and stiffness persist for more than a few days. Fractures in the cranial or mid-neck regions are most common, but occasionally fractures occur caudally, which may be difficult to assess without a fixed, high-output x-ray machine. Nuclear scintigraphy can be helpful in localizing such fractures, but false-negative results in the caudal cervical spine are possible. Horses with persistent ataxia have a guarded prognosis. Most horses with a fracture unassociated with ataxia can return to racing despite residual neck stiffness.

Lameness Associated with the Front Feet

Lameness associated with the front feet may vary from a shortened gait to an obvious lameness. The most common causes of foot lameness include solar bruising, corns, and subsolar abscesses. Other commonly recognized causes of foot lameness in the National Hunt horse include pedal osteitis, palmar heel pain, and fractures of the distal phalanx. Palmar heel pain may be caused by bruising of the heel bulbs or may be caused by deeper pain associated with the deep digital flexor tendon or navicular bone, with a much poorer progno-

sis. Horses that train on downland are particularly at risk to bruising the feet or penetrating injuries of the sole caused by flints. The latter may result in infectious osteitis of the distal phalanx. Protective aluminium pads may prevent solar penetrations but do not prevent bruising, because the pad is distorted and puts pressure on the sole if a horse stands on a flint.

In horses with chronic lameness associated with foot pain, local analgesia, radiography, and ultrasonography sometimes can be unrewarding or give equivocal results. Nuclear scintigraphy using soft tissue and bone-phase images is sometimes helpful.

Lameness Associated with the Carpus

Synovitis and osteoarthritis of the middle carpal joint are common and are treated by intra-articular injection with hyaluronan, with or without triamcinolone. A horse with synovitis may be treated more conservatively than a flat racehorse when the pressure is great to maintain the horse in training if at all possible. With a young National Hunt horse with a career of several years ahead, restricting the horse to walking exercise until clinical signs have resolved fully often is more prudent.

Chip fractures of the dorsal border of any of the carpal bones are common and often are associated with pre-existing osteoarthritis. Treatment is by surgical removal of the fragment(s), and prognosis depends on the degree of osteoarthritis; most horses are able to return successfully to racing. Slab fractures of the third carpal bone are also common but are not always associated with obvious clinical signs. Lameness is often mild and associated with only slight effusion of the middle carpal joint.

Accessory carpal bone fractures usually result from a fall and are common in National Hunt horses. Most fractures are longitudinal and occur midway between the dorsal and palmar borders of the bone, but less commonly articular chip fractures occur on the proximal or distal dorsal articular margin. The latter must be removed surgically or otherwise severe osteoarthritis ensues. Horses with the more common longitudinal fractures do not require treatment other than prolonged rest. Some fractures heal only by fibrous union, but the prognosis for return to racing is good. Some horses have effusion in the carpal sheath when work is resumed, which responds well to the intrathecal administration of triamcinolone and for which retinacular release is seldom necessary.

Lameness Associated with the Metacarpophalangeal Joint

Most lameness associated with the metacarpophalangeal (fetlock) joint is degenerative. Fetlock lameness is often present bilaterally with a shortened forelimb gait, warmth around the joint, and reduction of range of motion. Flexion often exacerbates lameness. Lameness often is improved by intra-articular analgesia of the metacarpophalangeal joint. Radiography may reveal peri-articular osteophytes or a small fragment on the dorsoproximal border of the proximal phalanx. If the proximal sesamoid bones and SL attachments are involved in sesamoiditis, a low palmar or four-point nerve block is required to alleviate lameness. New bone formation and osteolysis may occur on the abaxial surface of the PSBs at the attachments of the SL. This form of sesamoiditis more commonly is associated with damage to the attachments of the SL onto the PSB rather than a primary sesamoid bone disease and is seen much more frequently in National Hunt horses than other types of horses. The condition is related to the stress to which the palmar metacarpal soft tissues are subjected during jumping and galloping.

Treatment options include the following:

1. Injection of the fetlock joint with hyaluronan, with or without corticosteroids
2. Arthroscopy and removal of any free fragments
3. Blistering the palmar aspect of the fetlock to treat sesamoiditis

The prognosis for lameness affecting the fetlock is more guarded in the National Hunt horse than in the flat racing horse but occurs much less frequently.

Other Injuries

National Hunt horses seem particularly to lacerations involving the distal palmar (plantar) aspect of the metacarpal (metatarsal) region, which may involve the SDFT or the digital flexor tendon sheath (DFTS), usually resulting from the horse being struck or from penetration by a sharp stone. Such injuries should be treated early and aggressively, especially if the DFTS is involved. With early lavage of the DFTS using an arthroscope, combined with intravenous broad-spectrum antimicrobial treatment for 5 to 7 days, the prognosis is usually excellent unless significant damage of the SDFT has occurred.

A high number of complex injuries to the palmar (plantar) soft tissues of the pastern have been seen in National Hunt horses as sequels to previous superficial digital flexor tendonitis in the metacarpal region or as primary injuries. Although injury to one or both branches of the SDFT may occur alone, simultaneous injuries of the oblique and straight sesamoidean ligaments are not uncommon. The prognosis for such injuries generally is guarded.

DIAGNOSIS AND MANAGEMENT OF LAMENESS IN TIMBER RACING HORSES

Lameness falls into three groups: acute injury or breakdown after racing, chronic wear injury caused by the horse's age and length of competitive career, and traumatic injuries caused by interference from another horse or hitting a fence.

Eighty-five percent of acute injuries after racing are caused by superficial digital flexor tendonitis and SL desmitis. Many timber horses have had previous SDFT injuries from earlier flat racing. The long courses (up to 4 miles), variable turf quality and terrain, fatigue, and weather conditions predispose horses to injury. Injuries range from small tears to catastrophic breakdowns that may necessitate humane destruction. Regular clinical and ultrasonographic monitoring is useful for detecting subtle changes and for making recommendations about running on specific footing (ground) conditions (K.K.).

Once recognized, osteoarthritis of the metacarpophalangeal and the distal hock joints requires management, because increased lameness may be induced by the increased work leading up to a race. Keeping horses as comfortable as possible throughout training is preferable, which may necessitate intra-articular medication 2 to 3 times during a season, using hyaluronan for the metacarpophalangeal joints and hyaluronan and corticosteroids for the distal hock joints (K.K.). Orally administered glucosamine and chondroitin sulfate are used commonly. It is important to keep the horse well shod and to use cold hydrotherapy after strenuous exercise.

Traumatic injuries sustained during racing are common, with the hindlimbs particularly vulnerable because of the horse hitting fences at speed. Damage to the quadriceps muscles, peri-articular soft tissues of the stifle, and stifle joint are common, although determination of the severity of injury is usually easiest several hours after a race rather than immediately. Radiographic examination is indicated to rule out a patellar fracture, which requires surgical management. Bruising of the patellar ligaments is common, with or without effusion of the femoropatellar joint, and is treated by drainage of excess synovial fluid and injection of hyaluronan (4 to 6 ml) combined with controlled exercise and non-steroidal anti-inflammatory drugs.

Timber shin describes firm enlargement of the dorsal aspect of the metatarsal region because of chronic bruising of the long digital extensor tendon, with fibrosis of the tendon and peritendonous soft tissues. Timber shin is unsightly and is not associated with long-term lameness, but recent trauma results in lameness from severe swelling of the leg, with pain on protraction.

Injuries sustained by interference from another horse, for example, traumatic heel bulb laceration or strike injuries on the palmar or plantar aspect of the fetlock, are common (see page 939). Injuries also can result from falls, for example, fracture of the accessory carpal bone, or from a galloping horse treading on a faller.

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CHAPTER • 114

The Finnish Horse and Other Scandinavian Cold-Blooded Trotters

Kristiina Ertola and Jukka Houttu

HISTORY OF THE BREEDS

The Finnhorse is the only original horse breed in Finland. Sweden and Norway also have similar cold-blooded breeds. The Finnhorse has been known for about 1000 years and originally was used for farm and forest work and in the army. The Stud Book of the Finnhorse was founded in 1907 for Draft and carriage horses but is now divided into four parts for racing trotters, riding horses, draft horses, and small pony-type horses. Seventy-five percent of the registered Finnhorses are trotters. The Finnhorse is heavy and well muscled, with a short neck and usually a heavy head. The average height is 155 to 160 cm, but pony-sized Finnhorses exist. Swedish and Norwegian cold-blooded horses are slightly lighter and smaller than Finnhorses, with an average height of 150 to 160 cm.

Trotting competitions are the main use of Finnhorses. In Scandinavia all harness races are for trotters; there are no pacing races. The first records of trotting races are from 1817, when races were arranged on the ice of the river Aura in Turku. The first races with official timing took place 1862 in Viipuri. In the early years the trotting races were arranged by the state to support and develop horse breeding in Finland. In the first races the distance was 2138 m, and they were on a straight track. The horses raced individually, and prizes were given according to the times. The first official Finnish record was from 1865 by the mare Brita. The average time for 1000 m and was 1.51.3 (1 minute 51.3 seconds, equivalent to a mile in 2 minutes 59 seconds). Today the record is held by Viesker and is 1.19.9 (2:08.5). Betting was first introduced 1928 but developed slowly until the 1960s, after which rapid growth has followed.

In Sweden, racing also was started in the early nineteenth century on roads and icetracks. The first permanent tracks were built in the early twentieth century. The first official record in Sweden was in 1829, 1.37.6 (2:37), held by a Norwegian horse, Sleipner Varg. Today the record is 18.6 (2:07) by a Norwegian stallion, Spikeld. When racing became more popular, Swedish and Norwegian horsemen started trading horses across the border, and the Swedish and Norwegian breeds began to merge together. Today the breeds are genetically alike, and they now are considered to be the same breed. Sweden and Norway have a close collaboration in breeding and racing, and their horses race in the same races.

Today the races are held at modern dirt tracks, and in the same events there are separate races for Finnhorses and Standardbreds (STBs). The Finnhorses are allowed to race first at the age of 3 years, but not uncommonly they start racing as late as 5 or 6 years old. They are allowed to race until 16 years of age, and a Finnhorse usually is considered to be best between 7 and 10 years of age. Some stakes races are held for 4- and 5-year-olds, but the main events are for older horses. The biggest event for Finnhorses is the Kuninkuusravit, The Royal Trot, which is held annually in July and August at a different track in Finland each year. The race is one of the major sporting events in Finland, attracting about 50,000 people. Separate races are held for mares and stallions; geldings are excluded. To be allowed to race in the Kuninkuusravit, the horse has to be approved for public breeding and has to be entered in the Stud Book, which requires inspection and approval by a special board. The horses race 3 times in 2 days over distances of 1609 m, 2100 m, and 3100 m, and the final result is based on the total time for all three races. To win the royal title is the biggest honor a Finnhorse can ever get, and the best stallions have won the title 5 times (Vieteri, Vekseli, and Viesker), and the best mares, 4 times (Suhina and Valomerkki). This is a good proof of stamina and endurance, which are typical for the Finnhorse at its best.

In Sweden and Norway horses usually start racing at 3 years of age and are allowed to race until the age of 16. Stakes

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In Sweden and Norway horses usually start racing at 3 years of age and are allowed to race until the age of 16. Stakes

races are open to Swedish and Norwegian horses, and big races are held for 3- and 4-year-olds and for older horses. Finnish horses are not allowed in these races, and only a few races are held in which all Scandinavian horses race together.

TRAINING A COLD-BLOODED TROTTER

Traditionally the horses have been bred and trained by farmers, and many remain home bred and trained. There are no sales for Finnhorses, Swedish, or Norwegian trotters. Most horses are broken at 2 years of age, spend the next summer at pasture, and then start more serious training in the winter at 3 years of age, often with a summer break. Farm and forest work has been part of the training to build up strength. Speed work is not done at all at this stage, and traditionally some trainers have never used speed work. Horses started racing at 5 to 6 years of age and raced themselves to condition. However, many professional trainers now train cold-blooded horses, and the horses are better prepared for the races and start racing younger. The race times for the beginners are faster, which has caused problems for many old-school trainers. Many Finnhorses are not natural trotters, and building them up to speed takes time and training. If fast speeds are demanded too early, gait abnormalities develop at higher speeds, and horses lose complete control of the trotting action while appearing sound at a slower trot.

Training cold-blooded trotters takes much more time than for STBs, making training more expensive and leading to problems in getting new owners and trainers. In all Scandinavian countries, fewer cold-blood races are held than STB races, and they are concentrated in the north. Nonetheless, cold-blood racing is supported by the central racing organizations of each country and continues to thrive.

RACETRACKS AND WEATHER CONDITIONS

The racetracks in Scandinavia are 1000-m oval dirt tracks, and the horses race counterclockwise. The climate is cold in the winter, and the tracks freeze and get covered by snow (Fig. 114-1). In winter temporary tracks also are made on ice for smaller races with no betting. The variable climate causes track problems. In late autumn when the track is frozen but not yet covered by snow, the surface can be treacherous. Snowfall makes the tracks smooth but not hard, and provided the horse is shod properly, the track is not slippery. In the springtime, when the snow begins to melt, the track conditions vary throughout the day, being hard in the morning after a night frost, becoming good for a few hours when the frost melts, and then becoming wet and soft the rest of the day. Keeping the track in good condition requires much skill.

Because of the long winter, much of the training has to be done on snowy or icy surfaces. Snow is a good surface on which to train a horse, but special shoeing with studs is essential so that the horses do not slip. In the winter many trainers do much of the speed work in snow, which can be deep. The training speeds can be lower because of the resistance, but the training effect is equal or even better than when the training is done on the track. Snow provides a soft and smooth surface, acting as a shock absorber. Many horses with lameness problems race better in winter because of the training in snow. Horses also are trained on the ice of lakes, which is also a good surface. The surface of the ice gives slightly, and shoes with studs can get a good grip. The worst training surface is ground frozen hard like asphalt, creating many lameness problems.

SHOEING CONSIDERATIONS

In the winter (November to April) special requirements for shoeing are needed to prevent the horses from slipping on ice. Studs, 5- to 15-mm in length with sharp tips that offer a good grip of the ice or snow, are screwed into the shoes. Four to 15 studs are used per shoe; more studs usually are used in the hind shoes. It is important to have enough studs to avoid slipping slightly at every step, causing joint and muscle soreness. A risk of interference injuries exists, and some horses with poor action cannot race in the winter. Forelimb heel

Fig. 114-1 Winter racing. (Courtesy Olavi Ilmonen, Lahti, Finland.)

injuries are common if a horse breaks stride. Elbow boots may be necessary to protect the elbows from stud-induced trauma. Some lameness problems get worse during the winter, because traction by the studs stops the feet abruptly. Hock-related lameness is often worse during the winter, and tendon and suspensory ligament (SL) problems increase.

The shoeing of racing Finnhorses is otherwise much like that in STBs, but many Finnhorses are not natural trotters and tend to gallop or pace, so many trainers use special shoeing to help the horse to balance. Young horses often need heavy front shoes with toe weights and bell (overreach) boots when they are learning to trot. The total weight per foot can be up to 500 to 800 g, and this predisposes the horses to lameness, particularly tendon and SL injuries. On the hind feet Finnhorses usually wear normal STB shoes. The lateral branch of the shoe is often longer and bent slightly outward to make the hind action wider and therefore avoid interference between the forelimbs and hindlimbs. Although many STBs race unshod, this is rare in Finnhorses.

CONFORMATION AND LAMENESS

The Finnhorse is a heavy, draft-type horse compared with other racehorses. The forehand is heavier than the rear of the horse and is combined with a heavy head and a thick and short neck, causing much stress to the forelimbs. Finnhorses often stand back at the knee, and this sometimes is combined with lateral deviation of the carpus (offset knees). Forelimb tendon, SL, and carpal injuries are not uncommon. The rear end usually is conformed better in Finnhorses than the front end. The most common faults are sickle hocks and cow hocks, which can cause curbs or other lameness problems in the tarsal area. Fetlock conformation is often good, and most Finnhorses have big hooves with good-quality horn.

LAMENESS EXAMINATION

Many Finnhorses complete a racing career without serious lameness problems and require substantially less veterinary treatment than STBs, perhaps because of slower racing speeds. The lameness examination for a Finnish horse is similar to that for a STB racehorse, and similar problems occur. Clinical history is particularly important, and the following questions should be asked:

1. How long has the horse been in training, and how has it been trained? What is the duration of lameness, and has the horse ever trotted sound at high speed? A Finnhorse often develops slowly and has to be taught to trot. Therefore many action problems in young horses are not caused by lameness but by lack of coordination. Lack of strength or condition may predispose horses to lameness.
2. Does the horse trot straight? Is the horse hanging on a line? Is the horse hanging on a shaft? Does the horse need sidepoles or headpoles? A great many lameness problems only show at higher speeds, especially in the early stages of the problem. This is particularly true with hindlimb lameness. Usually in right hindlimb lameness the horse's rear end turns to the left shaft, and the horse hangs on the right line. Correspondingly problems in the left hindlimb cause hanging on the left line and turning the rear end to the right shaft. In forelimb lameness this is less obvious. A horse with bilateral lameness may move straight.
3. Does the horse trot better in turns or straightaways? In which direction are the turns better? In a counterclockwise direction horses with right hindlimb lameness are usually worse in the turns. Horses with left hindlimb lameness are worse in the straightaways and usually are worst in the turns in a clockwise direction.
4. Is the horse better on soft or hard surfaces? Sidebones often cause lameness on hard surfaces, as do many other foot problems.
5. Is the horse worst when it starts to trot? Does the horse warm into or out of the lameness problem? In young horses (4- to 5-year-olds) bilateral hind fetlock pain is a common cause of lameness, resulting in a short hindlimb stride when first trotting. The lameness usually improves if the horse is jogged for about 1000 to 3000 m.
6. Do the studs in the shoes affect the lameness in the winter? Many horses with carpal or tarsal lameness are worse with studs.

The veterinarian should assess the type and use of the horse, its conformation, muscular development, and general condition and should systematically palpate all the limbs, because concurrent lameness in several limbs often occurs.

Many horses are trained with sleighs in the winter and are harnessed with collars. If the collar is not fitted properly, it can cause shoulder musculature soreness. Soreness of the shoulder or scapular musculature also can point to other problems in the forelimbs, especially when the lameness is bilateral. Inflammation of the carpal sheath is common because of the heavy front of Finnhorses and the conformation of the carpi and invariably is associated with palpable distention. Proximal suspensory desmitis (PSD) sometimes occurs, and pain may be induced by palpation of the proximal metacarpal region.

The second (McII) and fourth (McIV) metacarpal bones in Finnhorses are sometimes thick and prominent, but this is not usually significant (Fig. 114-2), although thick metacarpal bones are sometimes seen in horses with PSD or tenosynovitis of the carpal sheath. Ossification of the cartilages of the foot

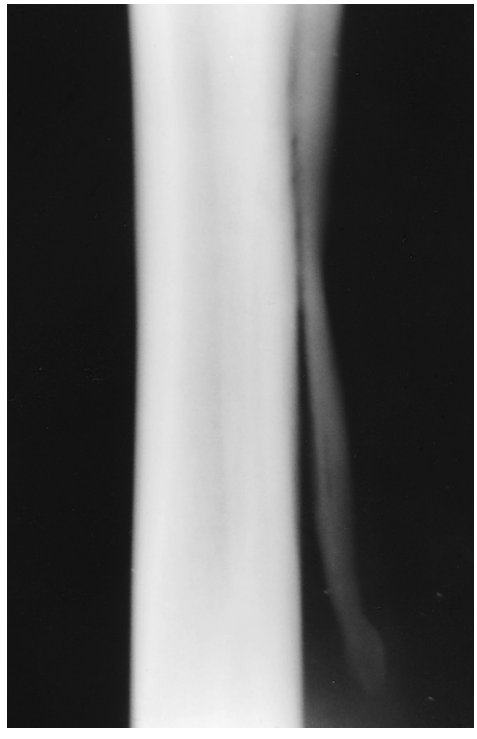


Fig. 114-2 Dorsomedial-plantarolateral oblique radiographic view of the metacarpal region. The splint bone is enlarged, which usually has no clinical significance.

usually can be felt by thickening and loss of elasticity in the region of the coronet, but pain is seldom evident in palpation.

In the hindlimbs special emphasis needs to be given to palpation of the tarsus. Tarsal pain is common in Finnhorses with poor conformation. Curbs are common in younger horses (3 to 6 years of age), and distal hock joint pain is common in older horses. The Churchill test works well in Finnhorses. Stifle lameness is not common in Finnhorses, and upward fixation of the patella is rare. Osteochondrosis of the lateral trochlear ridge of the femur occurs occasionally in foals and young horses, sometimes with large defects that respond poorly to arthroscopic debridement.

The gait and response to flexion tests can be evaluated properly only when the horse is calm and well controlled by the handler. It is essential that the horse trots straight and not too fast. Most horses are too eager and excited and need to be sedated or tranquilized mildly for flexion tests. Acepromazine (0.01 to 0.025 mg/kg) or romifidine (0.01 to 0.03 mg/kg) are suitable. Sedation is especially important for Finnhorse stallions, which are often difficult to handle, and sometimes two leaders are necessary to control the horse and keep it trotting straight. Horses used for riding can be examined in a circle on the lunge, but trotters are seldom taught to lunge.

When doing flexion tests, it is essential for the veterinarian always to examine all limbs and not only those that are suspected to be lame. The tests always should be done in a similar manner and order. When doing flexion tests, the clinician should note all reactions. We do not think false-positive reactions exist, only reactions of different grades. The clinician needs to collect all the information and decide which reactions are significant. Proximal and distal limb flexion tests are done separately in the forelimbs and hindlimbs, using a force of approximately 40 kPa for 60 seconds.

In general, all the trotting Finnhorses also are examined at the track at high speeds. Many action problems, especially in the young horses, show only at high speeds. The horse is harnessed in a higher sulky than normal, so the driver of the vehicle does not obscure the horse. The veterinarian drives a car behind or beside the horse at the racetrack. Examining the horse from the car is more helpful than driving the horse, because the clinician can be some distance away from the horse to see the action more clearly, and abnormalities can be assessed more easily.

Diagnostic analgesia is used as in STBs. The clinician must keep in mind that high-speed lameness seldom can be resolved totally by analgesic techniques. Every block changes the horse's action, and when the veterinarian does several blocks on the same horse, assessing the meaning of each block is difficult. When examining a horse with high-speed lameness, the clinician must believe what vision, touch, and experience indicate.

IMAGING CONSIDERATIONS

In Finnhorse trotters, radiographic changes are found much less commonly than in STBs. Carpal chip fractures are rare in Finnhorses, and sclerosis of the third carpal bone is unusual. Osteochondrosis is also rare in the Finnhorse, though the prevalence has been increasing slightly in the past decade. Because osteochondrosis is unusual, young Finnhorses are not radiographed routinely if clinical signs are absent. Controlling osteochondrosis in Finnhorses is attempted by radiographing all stallions that are proposed for public breeding. Stallions with osteochondrosis generally are not accepted. Ossification of the cartilages of the foot is a common radiographic finding in Finnhorses. Navicular disease is a rare diagnosis. Enlargement of McII and McIV is common and often does not cause clinical signs. Flake-like fragments adjacent to McII usually result from interference.

Ultrasonography is invaluable for assessing soft tissue injuries and may be more useful than radiography in examining the splint bones, because splint-bone problems often are associated with carpal canal syndrome or PSD.

UNDIAGNOSED LAMENESS

Some young trotting Finnhorses trot soundly at slow speeds and show no signs of lameness during a lameness examination, but when asked for speed, the horses lose the action completely. Loss of rhythm causes this; the horse simply cannot trot at higher speeds. This often happens to talented horses that have early speed with little training but do not have the strength to maintain it. The training regimen must be aimed at building strength, especially in the hindlimbs. Horses are trotted in 1- to 3-minute intervals with resistance with a special resistance cart, in deep snow, or uphill. Training takes a long time, sometimes up to a year, until these horses learn to trot again, and some horses never recover. Because the main racing events are for aged horses, time is not as big a factor for Finnhorses as for STBs. It is quite possible for a horse to recover completely and become a top-class racehorse at an older age.

THE TEN MOST COMMON CAUSES OF LAMENESS

The following are the 10 most common causes of lameness:

1. Carpal lameness
2. Tarsal lameness
3. Superficial digital flexor tendonitis
4. Suspensory desmitis
5. Curb
6. Ossification of the cartilages of the foot
7. Splints
8. Metatarsophalangeal joint pain
9. Tenosynovitis of the digital flexor tendon sheath
10. Loss of rhythm

LAMENESS CONDITIONS TYPICAL OF FINNHORSES

Carpal synovitis is common in Finnhorses, and signs are similar to those seen in STBs. In the early stages a horse warms out of lameness quickly. Often horses with carpal problems begin to roll over and may try to pace or gallop instead of trotting. The horse may trot if the shoes are weighted more, but this may accentuate the primary problem. Without treatment the lameness deteriorates, and the horse begins to hang on a line and gradually becomes overtly lame. Radiographic changes rarely are seen, probably because of the slower racing speeds compared with those of Thoroughbreds and STBs. Treatment consists of intra-articular medication with hyaluronan, polysulfated glycosaminoglycans or corticosteroids, alone or in combination, together with rest for several weeks.

Carpal canal syndrome is also common in Finnhorses, sometimes simultaneously with carpitis, sometimes on its own. Ultrasonography is useful for confirming the diagnosis. The treatment is usually rest, combined with intrathecal administration of hyaluronan or corticosteroids. Some horses do not respond to conservative therapy, and surgical treatment using tenoscopy may be necessary.

Tarsal lameness is common in Finnhorses and is the most frequent cause of hindlimb lameness, usually because of distal hock joint pain. Mild lameness in horses is not noticed easily at slow speeds, but with increasing speed the horse begins to

drag the affected limb, and the rear end moves to the opposite shaft. The horse usually hangs on the line on the side of the affected limb. Radiographic evidence of osteoarthritis may or may not be apparent. Intra-articular treatments with corticosteroids, hyaluronan, or polysulfated glycosaminoglycans are used widely. In some horses cunean tenectomy can be useful.

Superficial digital flexor tendonitis and suspensory desmitis are common. Superficial digital flexor tendonitis usually occurs in forelimbs, and poor conformation or overweighting the shoes may be predisposing factors. In the hindlimb, tendon injuries are usually traumatic. Suspensory desmitis occurs more frequently in forelimbs than hindlimbs, probably because of the heavy front conformation. Suspensory branch injuries are most common.

Finnhorses cope better with chronic suspensory desmitis than do STBs because of the lower racing speeds, longer rest periods, the horses being able to race at an older age, and thus better opportunities to heal. Suspensory desmitis is sometimes associated with splint bone fractures or exostoses.

Treatment of Finnhorses with superficial digital flexor tendonitis and suspensory desmitis is most often conservative, with long rest periods. Tendon splitting or desmotomy of the accessory ligament of the superficial digital flexor tendon sometimes is used for tendonitis. Hot firing has been a traditional treatment for tendonitis and desmitis for many decades, and some veterinarians still use it in some horses, though with mixed success.

Curbs (soft tissue enlargement in the proximal plantar metatarsal region) often are seen in young horses (3 to 5 years old) in early training, especially those with sickle hocks (see Chapter 79). Curbs seldom cause spontaneous lameness. Usually the signs show first at higher speeds, when the cranial phase of the stride in the affected limb is shortened, and the horse begins to drag the limb. Most veterinarians treat curbs symptomatically, without ultrasonographic examination to determine the structure involved. Treatments include rest, corticosteroid or dimethylsulfoxide injections, or pin firing. Cryotherapy is not used as widely as pin firing. The prognosis with all treatments is very good.

Ossification of the cartilages of the foot (sidebones) is a common finding in the front feet of heavy horses, occurring in 80% of Finnhorses. The condition is more common in females than in males and positively correlates with the size of the horse. The grade of the ossification is usually mild, but extensive ossification sometimes occurs. The cause is unclear, but a heritable component exists. The cartilages start ossifying at an early age, unassociated with training of the horse. Other contributory factors may include hoof conformation, shoeing, and concussion.

The clinical significance of sidebones is questionable. Most horses with mild or moderate ossification show no clinical signs. Ossification is detected only by radiography. With extensive ossification the cartilages are palpable proximal to the coronary band. Palpation does not induce pain. In general, horses with large sidebones show some clinical signs, especially on hard ground. Lameness at slow speeds is rare, but at high speeds with increased concussion the forelimb stride is shortened and the horse breaks to pace or gallop.

The diagnosis is confirmed radiographically, using dorsopalmar views. Osteoarthritis of the distal interphalangeal joint and navicular disease may cause similar clinical signs and may be present concurrently with sidebones.

The treatment options are limited, because the condition persists. Shoeing with egg bar shoes, often combined with thick pads, is helpful in some horses. Most horses with sidebones can race with moderate to good success when shod properly and raced only on good surfaces. Because of the hereditary background all Finnhorse stallions that are used for public breeding have to be radiographed for sidebones before being accepted.

Splints are fairly common in the forelimbs of young, growing horses and horses are usually treated conservatively with rest. Most splints resolve spontaneously, but local corticosteroid injections or cryotherapy sometimes is used. Interference injury may result in a flake of bone detached from McII. These fragments do not heal with rest and respond poorly to corticosteroid injections or cryotherapy. Surgical removal is usually necessary.

Chronic tenosynovitis of the digital flexor tendon sheath is a common cause of lameness in aged cold-blooded trotters and usually develops gradually, with mild or moderate swelling and no lameness for long periods. In time the sheath becomes fibrotic, and masses and adhesions develop. At this stage constriction by the palmar annular ligament may occur. Many horses can perform well despite chronic tenosynovitis, especially in hindlimbs. Intrathecal administration of hyaluronan, with or without corticosteroids, is used for horses with acute tenosynovitis. Frequent corticosteroid injections often seem to increase tenosynovial masses. Tenoscopy and transection of the palmar annular ligament is used if the condition is advanced. Cold-blooded horses with chronic tenosynovitis seem to respond poorer to treatment than do STBs with similar lesions. This may be because Finnhorses are often older and of greater weight.

In young Finnhorses, bilateral hindlimb fetlock pain is fairly common and causes a short and stiff hindlimb stride when they start to trot, which improves with exercise early in the condition. When both hind fetlocks are blocked simultaneously, the action changes completely. Pain is considered to be associated with subchondral bone remodeling of the distal aspect of the third metatarsal bone.

A variety of treatments have been used, including joint lavage and intra-articular treatment with hyaluronan or corticosteroids, often combined with NSAIDs. If lameness is severe, surgical drilling of the subchondral bone is performed. Horses remain lame for several months, but the long-term prognosis is reasonable.

Osteochondrosis is rare in Finnhorses, but when it does occur, osteochondrosis most often is seen in the distal intermediate ridge of the tibia and causes effusion of the tarsocrural joint. Treatment is removal of the fragment(s) by arthroscopy.

SECTION • 3
Non-Racing Sport Horses

CHAPTER • 115

Prepurchase Examination of the Performance Horse

Richard D. Mitchell and Sue J. Dyson

The purchase or prepurchase examination is a much discussed and sometimes feared subject for equine practitioners. Careless conduct and poor documentation can leave veterinarians wishing they had not agreed to perform the examination, whereas forethought and good planning can lead to a rewarding experience for the practitioner and the client. In the United States the examination is referred to as the *purchase examination*, because in many cases the deal already has been completed, whereas in Europe the examination usually is referred to as *the prepurchase examination*, because generally the purchaser has agreed to buy the horse subject to a satisfactory veterinary examination. However, in some countries (e.g., Holland), where many horses are sold through professional dealers, a horse may be purchased by the dealer and then examined by a veterinarian on behalf of the dealer before resale within a few days.

GOALS OF THE EXAMINATION

When requested to evaluate a horse for purchase, the veterinarian should keep several goals in mind. First, the examination should be an objective assessment of the horse's physical condition. Second, the examination should be a fact-finding mission to aid the purchaser in his or her decision to make a purchase. This may involve the veterinarian in making some predictions based on experience and probability, but care must be taken to be factual and objective. Last, the prepurchase examination can serve as a formal introduction to a horse for which the practitioner may provide long-term care. Such relationships may affect the veterinarian's decision-making process relative to a client's needs.

It is helpful to have knowledge of the disciplines in which the horse has been and will be involved. Various equine sports place differing demands on the horse, and the clinician should be aware of sometimes subtle, yet important, differences. Some physical characteristics or conditions may be acceptable for certain levels of performance but not acceptable for others. For example, a previous strain of a superficial digital flexor tendon (SDFT) may be an acceptable risk for a show hunter but may carry a high risk for a racehorse. Veterinarians should avoid performing prepurchase examinations on horses that will be involved in disciplines with which they are not familiar.

The veterinarian needs to discuss the goals of the examination and horse ownership with the prospective purchaser. Understanding the client, the trainer, and what is expected of the horse will help the veterinarian in assessing the horse's potential suitability. Passing or failing the horse is not the veterinarian's job, but it is his or her role to advise on how existing conditions may affect the future use of the horse. It is therefore self-evident that prepurchase examinations should not be performed by recent graduates, who may be fully competent in performing clinical examinations, but generally do not have the experience of how to interpret the findings of the examinations and are not in a position to evaluate risk. The prospective purchaser requires advice about the risks of proceeding with the purchase. Prior knowledge of clients, their expectations of the horse, and their attitude toward risk make offering such advice easier, compared with clients about whom clinicians know little.

A veterinarian must be open-minded and should consider themselves as a facilitator for the sales contract: the vendor wishes to sell the horse, the purchaser may have searched for a long time to find a suitable horse, and the veterinarian's role is to enable the transaction to take place if such is reasonable. Nonetheless, the veterinarian must be streetwise and recognize that a minority of unscrupulous vendors may try to misrepresent a horse. Caveat emptor. Veterinarians also should be aware that prospective purchasers often are keen to buy horses and in their enthusiasm may wish to overlook any problems that are identified during the prepurchase examination. A veterinarian who believes that the risks of buying a horse are too high is responsible for trying to dissuade the purchaser from proceeding further. If the purchaser ignores the advice, it is essential that the veterinarian has documented adequately his or her observations and advice. Purchasers can have remarkably selective memories when things start to go wrong.

The scope of the examination can range from a comprehensive clinical examination of the horse, using basic powers of observation, to a complex investigation using advanced techniques such as radiography, ultrasonography, endoscopy, thermography, and scintigraphy. The wishes of the buyer are important in determining the extent and depth of the examination, which also must be dictated to some extent by the value of the horse and its future athletic expectations. Some consideration to cost should be given, however, but not at the cost of the quality of the examination. The veterinarian should allow some latitude for deciding what is needed to answer questions posed by the clinical examination.

CONTRACT

A veterinarian enters a business arrangement with a purchaser when he or she agrees to perform a prepurchase examination. It is imperative for the veterinarian to understand the buyer's intentions for the horse and the expectations of the proposed examination. The terms, details, and costs of the examination should be discussed at the time of the initial request. The extent and depth of the examination and its limitations should be emphasized. This is straightforward when the veterinarian is dealing directly with the purchaser, assuming that the purchaser has knowledge of horses. The terms of

agreement become more difficult when a veterinarian is speaking to an agent for the owner or to the prospective rider of the horse, when the actual purchaser has no knowledge of horses. Such persons may have expectations of the horse as if it was a mechanical object like a car.

Purchaser's Reservations

The veterinarian is responsible for the following:

1. Establishing if the purchaser has any reservations about the horse
2. Determining whether the horse has been in regular work up until the examination
3. Warning the client of the hazards of performing an examination on a horse that has not been in regular work; previous lameness or back problems may be inapparent until the horse is in regular work. It may be helpful to suggest that the purchaser check the horse's official competition record to determine whether any unexpected breaks from competition have occurred.

Purchase for Resale

If the horse is to be purchased for resale, this should be noted. The purchaser should be warned that the clinician's interpretation of the findings may not be identical to that of another veterinary surgeon. The examining veterinarian may regard the horse as a reasonable risk for purchase, but this is not a guarantee that others have the same opinion. All observations should be well documented so that comparisons can be made should questions arise at a subsequent resale examination. Such notes may well help save a sale in the future and save face for the initial veterinarian.

Insuring the Horse

It is important to establish if the horse will be insured for loss of use for a specific athletic activity or for veterinary fees. The purchaser should be advised that the examining veterinarian may consider the horse a reasonable risk for purchase but that does not necessarily equate with the horse being a normal insurance risk. The veterinarian may consider that a small, well-rounded osseous opacity on the dorsal aspect of the distal interphalangeal joint is unlikely to be of clinical significance, but for an insurance company to place an exclusion on problems related to the joint would not be unreasonable. The veterinarian should advise the purchaser that if such problems arise, the purchaser should communicate with the insurance company before completing the purchase transaction.

Blood Tests and Limitations

The client should be informed clearly that the results presented are good for the day of examination, but predictions about future soundness and suitability are impossible. The limitations of analysis of blood for drugs must be detailed, bearing in mind the difficulties of detection of many drugs administered by the intra-articular route. The client should be warned that several days may elapse before the results of blood tests are known and that the purchase transaction should not be completed until the results are known. The veterinarian should discuss with the client how the findings will be transmitted and what kind of report will be issued.

Conflicts of Interest

Any potential conflicts of interest for the veterinarian must be disclosed to the buyer. Previous dealings with the vendor, although the vendor may not be a current client, could be perceived as a conflict of interest. In the horse world today, not to have such conflicts arise is difficult, but such conflicts should be acknowledged, and the buyer should be given the option of having someone else perform the examination.

COMMUNICATION WITH THE VENDOR

The vendor must understand clearly what facilities are required for the examination and should be advised that the horse should be stabled before the examination and not worked earlier in the day. If the vendor is unable to be present at the examination, the veterinarian should establish the answers to a number of important questions in advance:

1. How long has the horse been in the owner's possession?
2. Is the horse in regular work?
3. Has the horse had any previous lameness?
4. Has the horse had any previous medical problem?
5. Is the horse receiving any medication, or has it done so in the last 8 weeks?
6. Does the horse have any vices (crib-biting, wind sucking, box walking), or does it bite or kick?
7. Has the horse been seen to shake its head?
8. Has the horse had previous surgery?
9. Does the horse normally live in or out?
10. Does the horse have dry or soaked hay or a haylage preparation?
11. When was the horse last trimmed or shod?

Ideally vendors should sign a copy of their responses to these questions (Fig. 115-1).

When the examination actually is performed, ideally all involved parties or their agents should be present. This provides an environment in which the veterinarian can ask pertinent questions of the buyer and seller regarding the horse's history and future use and can assure the buyer a complete examination was performed. Problems that arise during the examination can be discussed with the purchaser.

The veterinarian should not compromise the standard of the examination because of physical conditions. If modifying the procedure of the usual examination technique is necessary, such should be noted in the subsequent report, and the purchaser should be advised accordingly. The limitations of the conclusions drawn from the examination should be documented. If an uncooperative vendor or agent makes conducting the usual examination difficult, the veterinarian may choose not to continue the examination to protect the interests of the buyer and personal interests.

EXAMINATION AT A DISTANCE

A client may request the veterinarian to examine a valuable competition horse that is a long distance away or possibly in a foreign country. A number of alternative strategies can be applied. It may be prudent to first have the horse undergo radiographic examination and proceed with visiting the horse only if these radiographs are considered acceptable. The veterinarian is of course relying on the honesty of everyone concerned that the radiographs provided are current images of the horse in question. It is critical that the veterinarian provide clear guidelines of the views required and be prepared not to compromise on quality. Alternatively, the veterinarian can travel and examine the horse and be present for the radiographic assessment. However, this may mean that the clinical examination is compromised by inadequate riding facilities at a veterinary clinic or that substantial time is spent traveling between the site where the horse is examined and the veterinary clinic where ancillary tests are performed. However, clinic facilities may be required anyway for ultrasonography or endoscopy.

A client may inform the veterinarian of an intended purchase of a horse from a foreign country and that he or she has been recommended to employ a specific clinician from that country to carry out the prepurchase examination. The client

Text continued on page 958

BEVA Prepurchase Examination Worksheet

Purchaser's name and address .....

.....

Telephone no. .....

Agent's name and address .....

.....

Telephone no. .....

Vendor's name and address .....

.....

Telephone no. .....

Intended use of horse ..... Stated age ..... Stated height .....
Purchaser's reservations .....

Is horse to be insured? ☐ YES *Permanent incapacity/All risks of mortality* ☐ NO

Name ..... Breed .....

Sex ☐ *Gelding* ☐ *Mare* ☐ *Entire*

Colour .....

Size ..... *Estimate app./Measurement/By documentation*

Duration of current ownership? .....
In current work? ☐ YES ☐ NO .....
Was horse stabled prior to examination? ☐ YES ☐ NO .....
Receiving medication or received medication in last 4 weeks?
☐ YES ☐ NO .....
Previous lameness? ☐ YES ☐ NO .....
Previous medical problems? ☐ YES ☐ NO .....
Previous surgery? ☐ YES ☐ NO .....
Vices? ☐ YES ☐ NO ☐ *Cribbing* ☐ *Windsucking* ☐ *Weaving* .....
Behavioural abnormalities? ☐ *Head shaking* ☐ *Box walking* ☐ *Biting* ☐ *Other* .....
Bedding? ☐ *Straw* ☐ *Shavings* ☐ *Paper* ☐ *Other* ☐ *Lives out* .....
Food? ☐ *Dry hay* ☐ *Soaked hay* ☐ *Haylage/silage* .....
Husbandry? ☐ *Stabled* ☐ *At grass* ☐ *In & out* .....
When was horse last shod? .....

Vendor's declaration: To the best of my knowledge the answers to the above questions are correct.

Signature of vendor/vendor's agent: .....

Continued

Fig. 115-1 British Equine Veterinary Association (BEVA) prepurchase examination worksheet. (Courtesy British Equine Veterinary Association.)

Examined at .....

.....

On .....

At .....AM/PM

Present .....

.....

.....

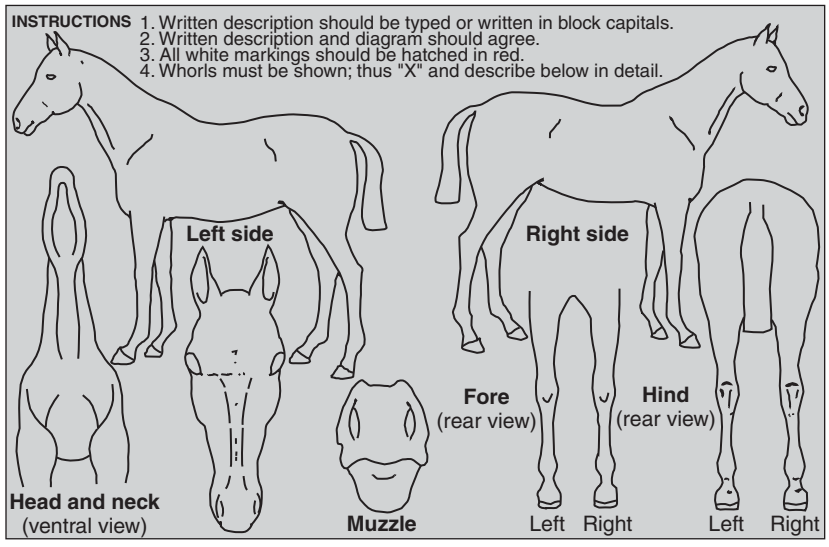
.....

Weather .....

.....

.....

.....



Identification

Head .....

.....

Neck .....

.....

Limbs: LF .....

RF .....

LH .....

RH .....

Body .....

.....

Acquired marks/Brands/Freeze brands ref loss of use .....

Microchip scanned ☐ Not scanned ☐ Present? Yes/No No. .....

Stage 1–Preliminary Examination (tick if normal; note abnormalities)

Bodily condition: Overweight ☐ Good ☐ Lean ☐ Poor ☐

Stance, attitude, and demeanour .....

Head Ears .....

Eyes (including opthalmoscopic examination) .....

Nose .....

Gums .....

Mandible .....

Other .....

Age and teeth Wolf teeth Yes/No Location .....

Incisor	Permanent	Infundibulum	Dental Star	Enamel Spot	Shape
1					
2					
3					

Fig. 115-1—cont'd British Equine Veterinary Association prepurchase examination worksheet. *LF,* Left forelimb; *RF,* right forelimb; *LH,* left hindlimb; *RF,* right hindlimb. (Courtesy British Equine Veterinary Association.)

Hook incisor 3—Left ..... Hook incisor 3—Right .....

Galvayne's groove Left..... Galvayne's groove Right .....

Angle .....

Abnormal wear .....

Molars .....

Approximate age ..... Range ..... Documented age .....

Integument

Surgical scar Laryngoplasty ☐ Ventriculectomy ☐ Laparotomy ☐ Medial patellar desmotomy ☐ Neurectomy ☐

Other ☐

Other acquired scars .....

Sarcoids Yes/No (note location) .....

Melanomata Yes/No (note location) .....

Other .....

Faeces, urination .....

Respiratory system Spontaneous cough Yes/No Cough reflex Yes/No

Auscultation of thorax .....

Cardiovascular system .....

Urogenital system .....

Nervous system .....

Conformation

LF .....

RF .....

LH .....

RH .....

Body .....

Hindquarters

Symmetrically muscled Yes/No Tubera sacrale symmetrical Yes/No

Musculoskeletal system

LF .....

RF .....

LH .....

RH .....

Back .....

Bulb of heel sensation .....

Feet Horn quality Good ☐ Poor ☐

Foot pastern axis Straight ☐ Broken back ☐ Broken forward ☐

Hoof testers .....

Trimming & shoeing .....

Symmetry .....

Continued

Fig. 115-1—cont'd British Equine Veterinary Association prepurchase examination worksheet. *LF,* Left forelimb; *RF,* right forelimb; *LH,* left hindlimb; *RF,* right hindlimb. (Courtesy British Equine Veterinary Association.)

Stage 2–Trotting up (tick if normal/note abnormalities)

Walk .....
Trot .....
Circle .....
Reverse .....

Flexion test Yes/No
LF ☐ – ☐ + ...
RF ☐ – ☐ + ...
LH ☐ – ☐ + ...
RH ☐ – ☐ + ...

Lunged on soft Yes/No Left rein .....
Right rein .....

Lunged on hard Yes/No Left rein .....
Right rein .....

Stage 3–Strenuous Exercise (tick if normal/note abnormalities)

Ridden/lunged Inside/outside State going .....
Trot .....
Canter .....
Gallop .....

Respiratory noise No/Yes < Inspiratory / Expiratory
Nasal discharge Yes/No
Cough Yes/No

Auscultation .....
Recovery .....

Stage 4–Rest

Observations .....

Crib biting Yes/No

Stage 5–Second Trot and Foot Examination (tick if normal/note abnormalities)

Walk .....
Trot .....
Circle .....
Reverse .....

Flexion test Yes/No Lunged on firm surface Yes/No

LF .....
RF .....
LH .....
RH .....

Shoes removed Yes/No

Fig. 115-1—cont'd British Equine Veterinary Association prepurchase examination worksheet. *LF,* Left forelimb; *RF,* right forelimb; *LH,* left hindlimb; *RF,* right hindlimb. (Courtesy British Equine Veterinary Association.)

General observations .....

Blood collected Yes/No Analysed/stored

☐ NSAID only ☐ Full screen ☐ Health profile

Specialised Techniques
Radiography

Area Examined	Views (circle which used)	Comments
LF Foot	LM, DPr-PaDiO, PaPr-PaDiO	.....
RF Foot	LM, DPr-PaDiO, PaPr-PaDiO	.....
L Mc/P Joint	LM, DL-PaMO, DM-PaLO, DPa	.....
R Mc/P Joint	LM, DL-PaMO, DM-PaLO, DPa	.....
L Carpus	LM, DL-PaMO, DM-PaLO, DPa	.....
R Carpus	LM, DL-PaMO, DM-PaLO, DPa	.....
L Hock	LM, DL-PIMO, PIL-DMO, DPL	.....
R Hock	LM, DL-PIMO, PIL-DMO, DPL	.....
Other	.....	

Ultrasonography

Yes/No Area examined & comments .....

.....

Endoscopic examination of URT ☐ Yes ☐ No .....

.....

Other special techniques

.....

.....

Signed ..... Date ..... Time .....

Record of discussion ☐ In person ☐ Telephone Date ..... Time .....

.....

.....

.....

.....

Suitable for purchase ☐ Yes ☐ No ☐ Deferred: reason .....
Certificate issued (Date) .....
Signed .....

Fig. 115-1—cont'd British Equine Veterinary Association prepurchase examination worksheet. *LF,* Left forelimb; *RF,* right forelimb; *LH,* left hindlimb; *RF,* right hindlimb; *L,* lateral; *M,* medial; *D,* dorsal; *P,* proximal; *Pa,* palmar; *Di,* distal; O, oblique; *Pl,* plantar; *URT,* upper respiratory tract. (Courtesy British Equine Veterinary Association.)

should be warned that the method of carrying out and reporting the examination may differ from what he or she is used to seeing and may have limitations of which he or she is unaware. For example, in some countries in Europe the examination is much more limited and does not encompass assessment of conformation. It is not usual practice to examine the horse being ridden. It is worthwhile developing a group of professional colleagues whose clinical expertise and trustworthiness the veterinarian respects, one of whom can be recommended to the purchaser. The veterinary surgeon performing the examination should be requested to communicate with the client's own veterinarian and to send radiographs for assessment. Discussion between two colleagues, one of whom has examined the horse and the other who knows the client, can result in a highly satisfactory outcome.

CLINICAL EXAMINATION AT REST

Various national bodies have established guidelines for the way in which a prepurchase examination should be carried out and reported, to which the veterinarian obviously should adhere. The legal responsibilities for the veterinary surgeon and the vendor may vary in different countries. For example, in Holland the expectations of the veterinary examination performed on behalf of an amateur purchaser are higher than that for a professional purchaser. In Denmark, if clinically significant radiological abnormalities are discovered soon after purchase, which obviously predate the purchase, the vendor is liable.

The clinical examination should evaluate all organ systems as comprehensively as possible. The examination should be methodical and repeatable. Using a checklist may help. The principle aims of this chapter are to focus on the examination of the musculoskeletal and neurological systems[1,2] and to discuss the interpretation of abnormal findings.

The veterinarian should identify the horse, including name, breed, sex, age, markings, tattoos, freeze marks, brands, and height. In Europe a freeze brand L signifies that the horse has previously been a loss of use insurance case. The horse's identity should be compared with its passport or vaccination certificate.

The horse first should be examined in the stable, with assessment of demeanor, attitude, stance, conformation, and thorough observation and palpation of the head, neck, back, and limbs as described in Chapters 4 to 6. Collection of blood samples may be performed at this stage or when the examination is completed and the veterinarian deems that purchase will probably be recommended. More comprehensive evaluation of the feet, overall conformation, and evaluation of muscle symmetry is best performed outside, where the horse can be viewed better from all angles.

If the horse will be used for show purposes, when the cosmetic appearance of the horse is important, the veterinarian should draw the purchaser's attention to all possible abnormalities, such as a prominent head to the fourth metatarsal bone, that a lay judge may misconstrue as a curb. If the purchaser has expressed reservations about a swelling such as a splint, the veterinarian should be sure to document its size and possible significance. If the purchaser is concerned about the horse's hock conformation, recommending radiographic examination of the hocks is worthwhile, even if the veterinarian considers the conformation to be acceptable and the horse appears sound.

Conformation

Although assessment of conformation as far as it may influence future lameness is considered an important part of the examination in the United States and Great Britain, this is not usual practice, and in Holland, for example, is not included. It is important to recognize that breed differences in conformation exist and that what might be acceptable in one breed does not necessarily apply to others. The significance of conformational abnormalities in part is dictated by the discipline in which the horse is involved.[1]

Most Arabian horses have a short-coupled and slightly lordotic back; thus they are likely to have some degree of impingement of dorsal spinous processes. However, this is unlikely to compromise the horse's show performance. However, a dressage horse with a short back may well develop a clinical problem associated with impinging dorsal spinous processes when working at an advanced level. In such a horse the flexibility of the back at rest and when the horse is moving in hand and ridden must be assessed with great care, paying great attention to the freedom and elasticity of the gaits. Even if the horse is symptom free currently, the purchaser should be advised that problems may occur in the future.

Young Warmblood breeds have a relatively high propensity for intermittent upward fixation or delayed release of the patella, especially those with a straight hindlimb conformation. Although the condition may be manageable in some, in others it can become a chronic problem, albeit a subtle one, resulting in low-grade discomfort and unwillingness to work. The veterinarian should observe the horse carefully as it moves over in the stable, to assess smooth or jerky movement of the patella on the left and right sides, and should check carefully for a surgical scar and the size of the medial patellar ligament, which if enlarged is likely to reflect a previous desmotomy.

Very straight conformation of the hocks or abnormal extension of the hind fetlock joints are predisposing factors for proximal suspensory desmitis and suspensory branch injury. A purchaser should be advised not to proceed with purchase for any form of athletic activity except light pleasure riding. The examination should be terminated at this stage rather than the veterinarian running up a large bill and antagonizing the vendor by wasting more time.

The clinician should pay particular attention to the conformation of the feet and relate this to the type of ground surface on which the horse will have to work. A horse with flat feet and thin soles is not a good candidate for endurance riding. A horse with crumbly hoof walls is predisposed to losing shoes, and this can be disastrous for three-day event horses. Although changing the horse's nutrition and foot management may result in some improvement, accurate prediction of the degree of improvement that may be achieved is difficult. Sheared heels and underrun heels in the forelimbs or hindlimbs may be primary problems or predispose horses to altered ways of moving, causing soreness in the more proximal parts of the limbs. These findings are particularly important in jumping and dressage horses and those used for cutting and reining.

The veterinarian should assess the forelimb conformation carefully from the front and ensure that the foot is positioned symmetrically under the central limb axis of the more proximal parts of the limb. A disproportionately high incidence of distal limb joint problems occurs in horses in which the pastern and foot are set more to the outside, with a tendency for the medial wall to become more upright and the lateral wall flared (Fig. 115-2).

Many Warmblood breeds naturally have much narrower and upright foot conformation than other breeds. This predisposes the horses to develop thrush, and careful stable hygiene and foot cleanliness are necessary to control this problem. Asymmetry of front foot shape and size always should be documented and may reflect previous lameness in the limb with the more contracted foot but also may reflect development of a mild flexural deformity of the distal interphalangeal joint when the horse was a foal, which may be of no long-term significance. The veterinarian should beware if the feet are long and misshapen; this finding may mask underlying conformational problems or asymmetry in foot shape and size. Postponing further assessment of the horse until the feet have been trimmed and shod properly is preferable.

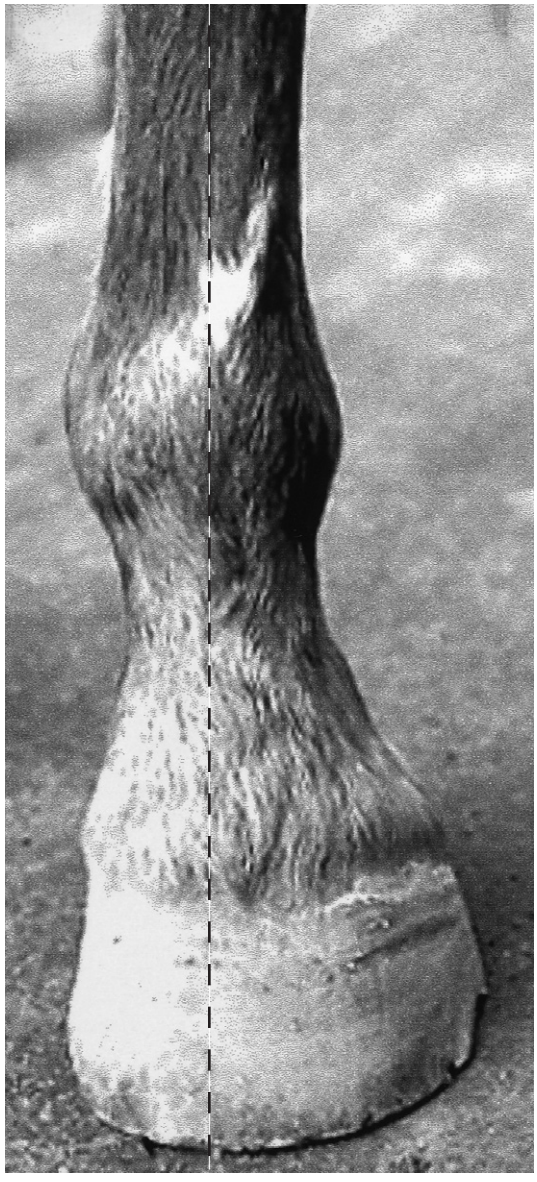


Fig. 115-2 Dorsal view of a forelimb with significant conformational abnormalities. Note the position of the foot and pastern relative to the central axis of the metacarpal region *(dotted line)*. The hoof capsule is distorted, and the foot shape is asymmetrical.

Some conformational abnormalities, such as over at the knee, do not appear to influence a horse's future soundness greatly but must be described. Failure to document observations leaves the veterinarian open to litigation if future problems arise.

Muscle Symmetry

Muscle asymmetry of the hindquarters may reflect previous or current hindlimb lameness, although it is not necessarily associated with future chronic lameness. Muscle asymmetry should alert the veterinarian to pay particular attention to the hindlimb gait in hand, on the lunge, and ridden and to the response to flexion tests. Asymmetry is an indication for flexion tests before and after ridden exercise. The potential significance of muscle asymmetry must be discussed with the purchaser. Slight asymmetry of the tubera sacrale is a common finding and frequently is not of clinical significance. Nonetheless, the purchaser should be informed.

Tendons and Ligaments

Particular attention should be paid to the size, shape, and stiffness of the tendonous and ligamentous structures of the metacarpal and pastern regions, bearing in mind that if the horse has sustained a bilateral tendon injury, both tendons may be enlarged slightly, and the clue to previous damage may be rounding of the margins of the tendons or abnormal stiffness. The significance of a previous injury to the SDFT or suspensory ligament must be assessed in the light of the horse's previous and future career. These injuries carry a high risk of recurrence in racehorses, event horses, high-level show jumpers, and endurance horses. However, the horse may perform satisfactorily as a hunter, a dressage horse, or a pleasure horse or at a low level in more demanding sports. The purchaser should be advised that further information about the repair of the injury may be obtained by ultrasonographic examination. Further information about when the injury was sustained and what the horse has done since then may be helpful. Decision making must be based on the athletic expectations of the horse, all other aspects of the horse's suitability, and whether the horse is being bought for resale. If the horse is a perfect schoolmaster for a junior rider and the price is reasonable, the risk/benefit ratio may be acceptable.

Assessment of Joints

Many horses have mild fetlock joint capsule distention or thickening. The significance of distention must be assessed based on the environmental temperature: if it is cold joint capsules are more likely to be tight than if it is warm. Asymmetry between left and right is of greater significance than bilateral symmetry. The joints should be assessed carefully for any evidence of restricted range of motion or pain on manipulation. The range of motion varies between horses and is in part a reflection of age and work history, but asymmetry between left and right should be viewed with caution. The response to distal limb flexion and the horse's action on the lunge on a hard surface must be assessed carefully. Distention of the antebrachiocarpal or middle carpal joint capsules is rarely an insignificant finding and even if unassociated with detectable lameness should prompt radiographic examination.

During hindlimb assessment, care should be taken to differentiate between pain on flexion of the proximal limb joints and reluctance to stand on the contralateral hindlimb, perhaps associated with sacroiliac pain. Abnormal limb flexion may be present if the horse is a shiverer. A high incidence of this condition occurs among high-performance Warmblood breeds and does not appear to compromise performance. However, an intending purchaser must be warned that the condition may be progressive and may make the horse difficult to trim and shoe.

Flexibility of the neck and back should be assessed, and the presence of any abnormal muscle tension or abnormal reaction to palpation of acupuncture trigger points should be noted. The veterinarian also should pay attention to the presence of sarcoids in a position where they may be abraded by tack. If a sarcoid is identified, the client always must be warned that such lesions may increase in number, but assuming that the client is aware of the risks, a small number of lesions at sites removed from the tack should not mitigate against purchase. The mouth should be examined carefully. Large hooks on the rostral aspect of the first upper cheek teeth are usually an indicator that large hooks also may be present on the caudal aspect of the lower caudal cheek teeth, which may be difficult to manage.

Sensation in the heel region of the forelimbs and the reaction to vigorous application of hoof testers should be assessed carefully to determine if a previous neurectomy may have been performed. The absence of a visible or palpable scar does not preclude previous surgery.

ASSESSMENT OF GAIT

The horse should be examined moving freely at a walk and trot on a firm surface, with particular attention directed to the stride length and lift to the stride relative to the horse's type. The veterinarian should bear in mind that a horse with a bilateral forelimb or hindlimb lameness may not appear overtly lame but merely may have a slightly restricted gait. The veterinarian should beware of the situation encountered particularly in a professional's yard when the horse is encouraged by an assistant to trot excessively quickly, is unduly restrained, or is excessively fresh and trots crookedly. The horse must trot in a regular, relaxed rhythm with freedom of the head and neck; otherwise, lameness may be missed. The veterinarian should pay particular attention to how easily the horse turns when changing direction and the flexibility of the neck and back. If the horse trots in a particularly loose and extravagant way, the veterinarian should bear in mind that the horse may be mildly ataxic. Ataxia may not jeopardize a dressage horse when competing at lower levels, but when finer degrees of muscular strength and coordination are required in advanced dressage, performance may be compromised. The safety of a mildly ataxic horse jumping must be questioned. The veterinarian should watch the horse carefully as it decelerates, when signs of mild ataxia or jerky movement of the patella associated with its delayed release may be apparent. Watch the horse turning in small circles and moving backward, assessing flexibility of the neck and back, limb coordination and placement, and any quivering movement of the tail suggestive of shivering.

Flexion Tests

The interpretation of flexion and extension tests is controversial.[3,4] The force applied, the duration of flexion, the way in which the joints are flexed, and the work history of the horse may all influence the response. Positive results of flexion tests in a horse that does not demonstrate lameness before flexion may not be a cause for termination of the examination, unless other suspicious clinical signs have been identified already. The horse should be evaluated on the lunge and ridden for evidence of lameness. Many positive results on flexion tests are found to change during the course of the exercise examination (perhaps as the horse warms up or loosens up), and these may not have clinical significance. A difference in response between distal limb flexion of the left and right forelimbs may be more important than a symmetrical response. A positive response to carpal flexion or to proximal limb flexion of a hindlimb must be viewed with caution. A veterinarian should aim to perform flexion tests as consistently as possible so that they know the ranges of response anticipated in clinically normal horses.

Lunging and Ridden Exercise

The requirements for lunging and ridden or driven exercise vary between the guidelines for prepurchase examinations in different countries and also are dictated by the type of horse under examination. The methods used for a 3-year-old Thoroughbred racehorse differ from those for a 6-year-old destined for horse trials. Seeing the horse lunged on soft and hard surfaces and ridden is helpful to gain maximum information about any potential lameness problems. The vendor should be encouraged to use any protective boots or bandages that might be used normally. Subtle lameness or restriction in gait because of thoracolumbar or sacroiliac discomfort may not be evident until the horse performs specific movements when it is ridden.[5,6] Small figures of eight may be particularly revealing. Watching the horse do what it specifically is intended to do is good practice. The veterinarian should watch the horse being tacked up and mounted to learn about any cold back behavior or evidence of back pain or other behavioral abnormalities. The horse should be worked reasonably hard relative to its fitness. In many European countries, evaluating a ridden horse is not standard practice, and if an examination is being performed on behalf of the client by a foreign veterinarian, the client should be advised accordingly.

Clinicians who have the prerequisite skill and experience may wish to evaluate horses further by riding or driving them themselves. Feeling the horse in this fashion may answer questions regarding a peculiar gait or way of going and may aid the evaluation of subtle lameness or respiratory noises. Such practice should be done with caution, because legal problems could arise from an unfortunate accident. A signed disclaimer may be helpful in this situation.

After strenuous exercise the horse should be allowed to stand for 15 to 20 minutes before being re-evaluated in hand at the walk and the trot. This is a mandatory part of the examination in Great Britain but is not practiced widely in the rest of Europe. Previously inapparent lameness now may become evident. Any previously questionable response to flexion tests may be re-assessed.

EVALUATION OF IDENTIFIED PROBLEMS

The veterinarian should now have gained enough information either to discontinue the examination after consultation with the purchaser or to make recommendations for further special examinations. If the horse is lame and a potential cause that may resolve is obvious (e.g., nail bind), re-evaluating the horse on a subsequent occasion may be worthwhile, but the veterinarian should try to ensure that the horse has been worked properly for several days before the re-examination.

The veterinarian should always bear in mind that in a mature competition horse, it is relatively unusual not to identify some problems. Taking no risks and advising against purchasing the horse is easy, but that actually may be doing the purchaser, the vendor, and the veterinary profession a disservice. It is important to weigh the risks and describe them to the purchaser as objectively as possible, based on previous experience. At this point having in-depth knowledge of the purchaser, his or her aspirations for the horse, and the attitude to risk and financial ability to take the consequences of risk is most valuable. Further information obtained from radiographic and ultrasonographic examinations may help provide further objective information on which decisions can be based. Decisions also must be related to the horse's recent competition record, its age, and the future expectations for athletic performance. Low-grade hindlimb lameness associated with mild radiographic changes of the distal hock joints might be an acceptable risk for a horse as a schoolmaster for a young rider, but similar abnormalities identified in a 6-year-old about to step up a level in competition must be regarded as potentially more serious. A veterinarian always must bear in mind that minor problems may become major problems with a change of rider, work pattern, and environment.

RECTAL EXAMINATION

Rectal examination is not a routine part of a prepurchase examination and should be performed only with the vendor's consent if indicated clinically or if a mare is to be used later for breeding.

RADIOGRAPHIC EXAMINATION

Radiographic examination is not a standard part of a prepurchase examination. The extent of routine radiographic examination in the United States and some European countries is probably higher than elsewhere. In Holland a strict grading system for evaluation of radiographs is used. The purchaser

must be made aware that the presence of some radiological abnormalities is not necessarily synonymous with future lameness and the absence of radiographic changes does not preclude future lameness.[7] If radiographs are to be obtained for a specific area, then a comprehensive set of radiographs should be ordered to avoid missing lesions apparent on only one view. In many European countries, obtaining only three views in evaluation of the hocks and omitting a dorsomedial-plantarolateral oblique view is common. This action may risk the veterinarian missing lesions such as peri-articular osteophytes, which are present only on the dorsolateral aspect of the joints (Fig. 115-3).

The regions to be examined and the interpretation of findings are dictated by the previous and intended career of the horse and the results of the clinical examination. Examination of the front feet, fetlock, and hock joints is considered routine in many countries. Evaluation of the carpi, hind fetlock and pastern joints, stifles, and dorsal spinous processes of the thoracic region may be considered. It is, however, important not to overinterpret the significance of some radiographic abnormalities, bearing in mind the variability between breeds and the knowledge that even some obvious changes may be clinically insignificant. For example, Warmblood breeds have a tendency to have a greater number of lucent zones along the distal border of the navicular bone than do Thoroughbreds (Fig. 115-4). A relatively high incidence of spur formation on the dorsoproximal aspect of the middle phalanx occurs in Warmbloods (Fig. 115-5), but this rarely is associated with lameness. However, one of the authors (R.D.M.) recognizes a higher incidence of clinically significant osteoarthritis of the proximal interphalangeal joint in Warmblood horses compared with other breeds.

Evidence of abnormal lucent zones in the proximal sesamoid bones (sesamoiditis) (Fig. 115-6) should alert the veterinarian to re-evaluate the branches of the suspensory ligament. An ex-racehorse may have periosteal new bone on the dorsal aspect of one or more carpal bones, but this is unlikely to influence the horse's career as an event horse. A small osteochondral fragment on the dorsal aspect of the distal interphalangeal or metacarpophalangeal joint is often asymptomatic.

A small spur on the dorsoproximal aspect of the third metatarsal bone (MtIII) is a frequent incidental finding. However, such findings also must be described to the purchaser, and the potential future significance must be discussed. Small spurs on the dorsoproximal aspect of MtIII do not necessarily reflect osteoarthritis but may reflect entheseous new bone at the attachment of the fibularis tertius or cranialis tibialis. Despite the fact that these spurs are a common finding, accurately predicting the future behavior is impossible. Some spurs may reflect osteoarthritis and may be progressive (see Fig. 115-3). Small osteochondral fragments at the distal intermediate ridge of the tibia in a 3-year-old that has done little work may be asymptomatic, but if they became unstable with increased work, it might result in distention of the tarsocrural joint capsule, and surgical removal may be indicated. Such a finding in a mature competition horse is usually of no consequence. Complete fusion of the centrodistal joint may be identified

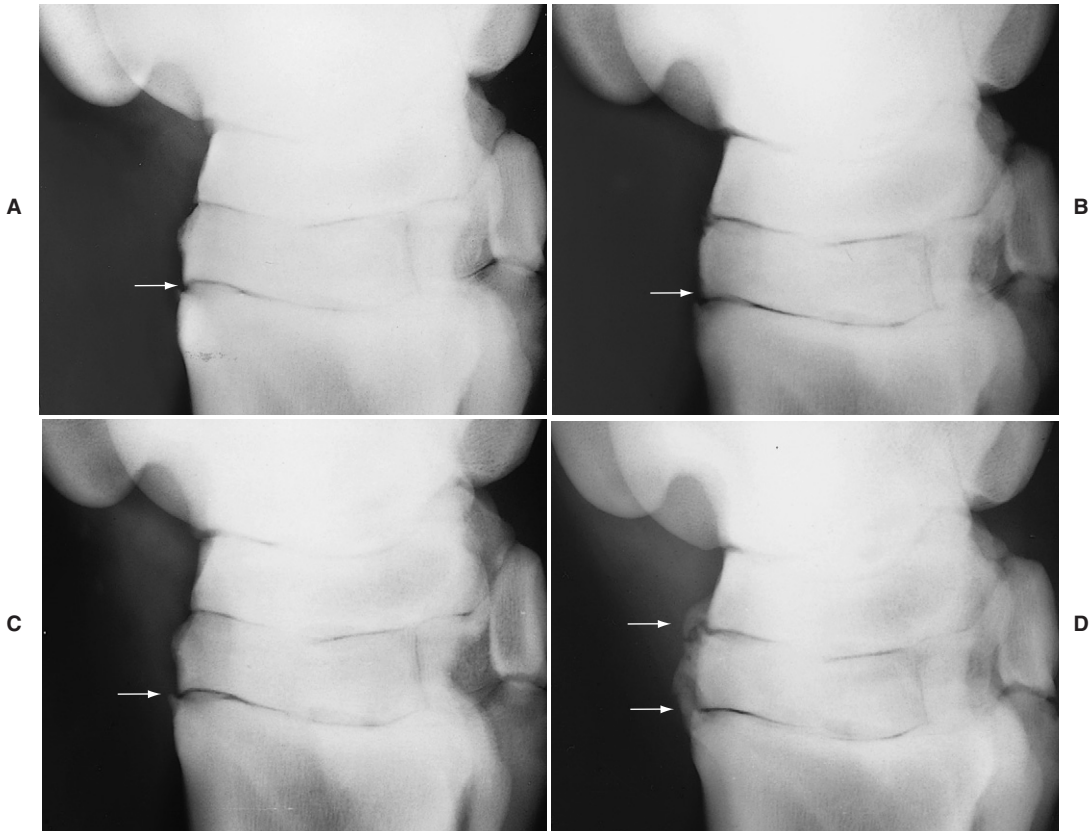


Fig. 115-3 Plantarolateral-dorsomedial oblique radiographic views of the left (**A**) and right (**B**) hocks of 9-a year-old Grand Prix show jumper. There is a small osteophyte on the proximal aspect of the third metatarsal bone *(arrow)*, and subtle modeling of the articular margins of the centrodistal joint in each hock. The horse was clinically sound. **C** and **D,** The same horse 24 months later. At this stage the horse showed right hindlimb lameness that was alleviated by intra-articular analgesia of the tarsometatarsal joint. Considerable peri-articular osteophyte formation *(arrows in **D**)* involves the centrodistal and tarsometatarsal joints of the right hock (**D**). The spur on the dorsoproximal aspect of the left third metatarsal bone was changed little *(arrow)*.

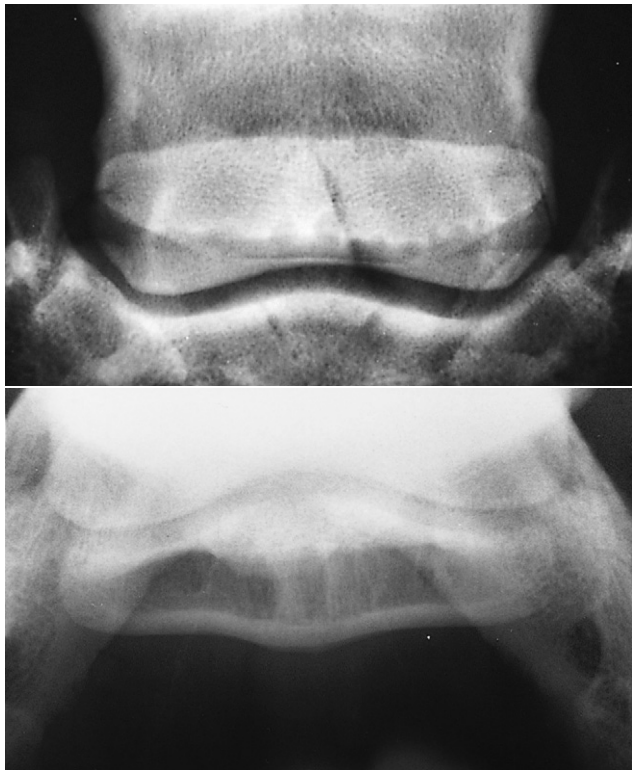


Fig. 115-4 **A,** Dorsoproximal-palmarodistal oblique view of the right front foot of a clinically normal 6-year-old Dutch Warmblood horse with good foot conformation. Note the large size and number of the radiolucent zones along the distal border of the navicular bone. The horse competed successfully for many years with no evidence of foot pain. **B,** Palmaroproximal-palmarodistal oblique view of the same foot. Note the large, oval-shaped lucent zones in the medulla of the navicular bone.

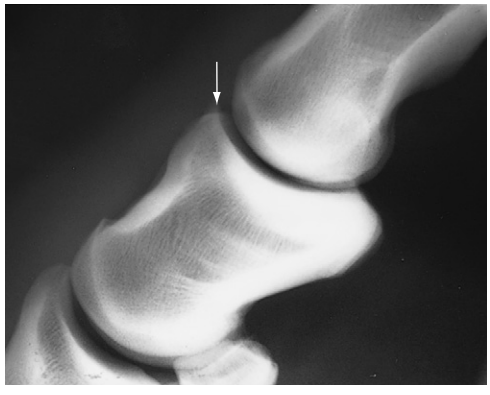


Fig. 115-5 Lateromedial radiographic view of the left front pastern of a clinically normal 7-year-old Belgian Warmblood show jumper. Note the modeling of the dorsoproximal aspect of the middle phalanx *(arrow)*. Such spurs are a common finding in Warmblood breeds and usually are not associated with lameness.

unassociated with any other radiological change of the hock (Fig. 115-7). Some horses may compete successfully with such changes for many years, but occasionally lameness subsequently develops because of pain associated with the talocalcaneal-centroquatral (proximal intertarsal) or tarsometatarsal joints. Well-rounded osseous opacities frequently are identified distal to a proximal sesamoid bone (Fig. 115-8). These are often innocuous, but ultrasonographic evaluation of the distal sesamoidean

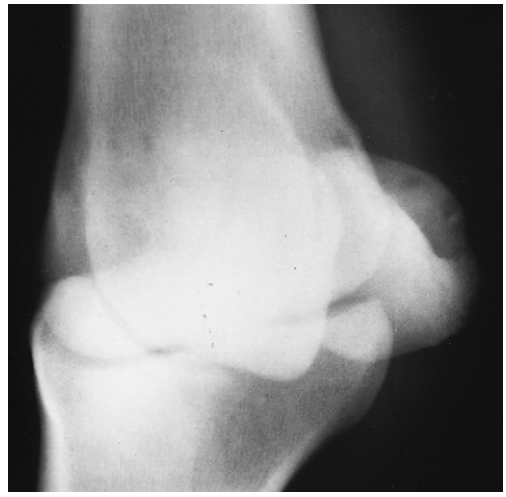


Fig. 115-6 Dorsolateral-palmaromedial oblique view of a 6-year-old Thoroughbred that had raced previously on the flat and was used now for eventing. Note the large lucent zones in the lateral proximal sesamoid bone. The horse had no history of lameness.

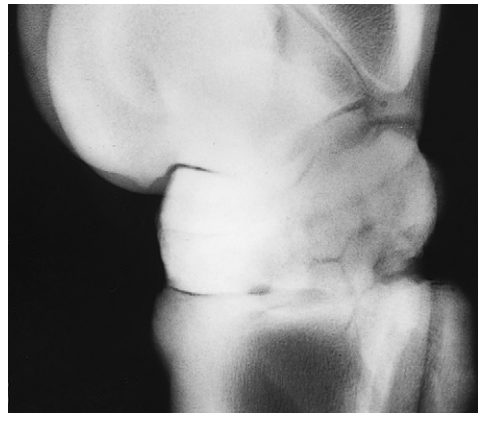


Fig. 115-7 Lateromedial radiographic view of the left hock of a 9-year-old clinically normal Thoroughbred advanced event horse. The centrodistal joint is fused completely. The horse competed successfully for another 8 years before being retired because of a tendon injury.

ligaments may be indicated. Evidence of osteochondrosis of the trochleas of the femur is of concern in a 3-year-old, even if the condition is asymptomatic, whereas mild flattening of the lateral trochlear ridge of the femur in a 10-year-old jumper free from lameness would be of no concern.

NUCLEAR SCINTIGRAPHIC EXAMINATION

Nuclear scintigraphic examination is not a routine part of a prepurchase examination. If the horse is clinically normal, the interpretation of results of whole body screening is difficult. This practice should be discouraged. In selected horses when interpretation of the significance of specific radiological abnormalities may be in dispute, focused nuclear scintigraphic examination may be helpful.

ULTRASONOGRAPHIC EXAMINATION

Ultrasonographic examination of the limbs requires three important pre-requisites: high-class image quality, experience of

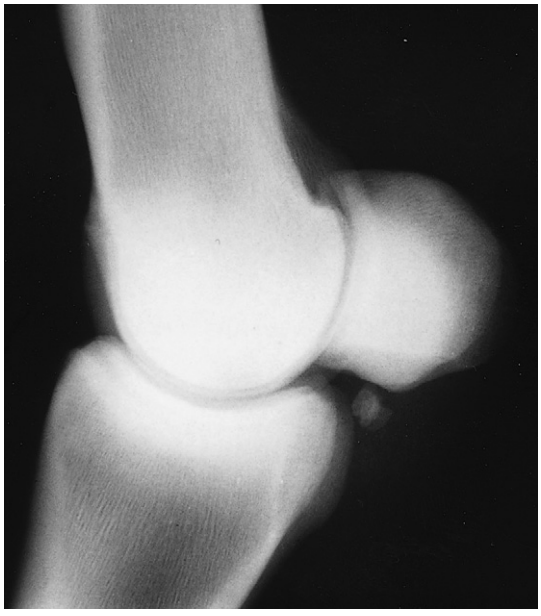


Fig. 115-8 Lateromedial radiographic view of the left forelimb of a clinically normal 7-year-old Thoroughbred. There are two well-rounded osseous opacities distal to the proximal sesamoid bones. Such opacities are usually clinically innocuous.

interpretation (knowledge of normal anatomy and its variations and the ability to detect abnormality and to interpret its potential significance) and client compliance. A number of clinical situations occur in a prepurchase examination when ultrasonographic examination provides invaluable clinical information not otherwise available. These situations include the following:

- An event horse that has competed in a three-day event within the last 6 months but has not competed since or a racehorse that has run recently but has not done fast work since. Strain of the SDFT, unilaterally or bilaterally, is a common injury in event horses and racehorses. Clinical signs of localized heat and swelling may resolve quickly or even pass unnoticed by the vendor (see Chapter 70). Clinical signs of tendonitis may be inapparent at the prepurchase examination but may become evident when fast work is resumed. If the left and right SDFTs are slightly enlarged with rounded margins but are symmetrical, this may pass unnoticed during the clinical evaluation.
- An event or racehorse that has not competed or raced in the last several months during the season. The purchaser may have given a plausible explanation for this, but the clinician should be suspicious that the absence from competition may reflect injury.
- Known previous tendon or ligament injury. Ultrasonographic examination gives more objective information than can be obtained from palpation alone but does not give information about strength of the structure.
- Suspected enlargement of one or more tendonous or ligamentous structures, detection of localized heat, or pain on palpation.
- Moderate enlargement of a digital flexor tendon sheath, especially if unilateral, particularly in a forelimb.
- Enlargement on the palmar (plantar) aspect of the pastern. It can be difficult to determine by palpation alone if thickening reflects localized fibrosis or swelling of a tendon or ligament.

Interpretation of findings in a horse with no identifiable clinical abnormality may not be straightforward, but enlargement of a tendon or ligament usually does reflect previous injury, the future significance of which depends on the intended use of the horse, chronicity of the lesion, severity of the lesion, and degree of healing.

If ultrasonographic examination is recommended but is refused by the vendor or the purchaser, this should be noted on the prepurchase examination certificate.

THERMOGRAPHIC EXAMINATION

Thermography also may provide a useful indicator of low-grade inflammation and help to identify signs of early tendonitis, suspensory desmitis, and splint development. However, a normal thermographic appearance does not preclude the presence of a subclinical tendon lesion.

BLOOD TESTS

The use of blood tests is controversial, and limitations should be discussed with the prospective purchaser. Screening for non-steroidal anti-inflammatory drugs, mood-altering drugs, sedatives, and possibly also corticosteroids is advisable, but the purchaser must be aware that blood tests are less sensitive for these drugs and metabolites than is urine analysis. If the vendor knows in advance that the horse will be tested, this knowledge may provide a deterrent to the unscrupulous. Alternatively, blood may be collected and stored suitably and be analyzed only if a problem arises within the first few weeks of purchase. In Great Britain the Veterinary Defence Society and the Horse Race Forensic Laboratory run a scheme jointly. The vendor signs a form to permit collection of the sample in a specialized container provided by the Horse Race Forensic Laboratory, to which is applied a bar-coded label. The purchaser can elect to have the sample analyzed immediately for a fee or it can be stored at the Horse Race Forensic Laboratory at no charge with the potential for analysis at a later date. This system has proved to be legally robust.

The purchaser must be aware that drugs administered intra-articularly may not be detectable, depending on the nature of the drug and the time of administration relative to the time of examination. In professional dealers' yards in Europe, a high incidence of joint medication to mask lameness occurs.

If the horse is being purchased in one country for export to another, testing for evidence of specific diseases may be necessary. For example, horses entering the United States should be tested for equine infectious anemia, dourine, glanders, and piroplasmosis. A horse from an area where African horse sickness has occurred should be tested. Screening for contagious equine metritis may be indicated. Use of hematological and serum biochemical screening and other assays, such as measurement of cortisol and insulin, as an aid to detect equine Cushing's syndrome in older horses is controversial. Serum testing for equine protozoal myelitis potentially is misleading and should be actively discouraged.

NERVE BLOCKS

In some circumstances, nerve blocks may aid interpretation of clinical findings. A horse may appear completely sound under all circumstances but have an unusually short forelimb stride. Does this reflect bilateral foot pain, or is this gait completely normal and natural for this horse? With the vendor's permission this question could be answered by bilateral perineural analgesia of the palmar nerves at the level of the proximal sesamoid bones.

If a horse is lame, but the vendor claims that the horse has never been lame previously, the situation should be discussed with the purchaser. Re-examining the horse on a subsequent occasion may be best, but the vendor should be advised that

after resolution of the lameness the horse must be worked for at least a week before re-assessment. The veterinarian should ask the vendor to sign a form declaring that the horse has been worked properly and has not received medication, and the veterinarian should collect blood for medication testing when the horse is re-examined.

The examining veterinarian's role is not to perform a lameness investigation; this would be unethical. A lameness investigation is a job for the vendor's own veterinarian.

SUMMARY OF OBSERVATIONS

The veterinarian should summarize the basic assessment of the horse's physical condition for the buyer. Abnormalities of conformation, insignificant swellings, and any clinical abnormality should be discussed and documented. The initial report should be made verbally to the client or the client's agent. When dealing with an agent, informing the agent that the client also will be receiving a complete written report is wise. Comments should be as factual as possible, with minimal personal bias, but findings must be interpreted and the risk assessed and documented.

Although the veterinarian is working for the buyer, the veterinarian does have an obligation to the seller. The findings should not be discussed with anyone other than those involved in the sale. With permission of the buyer, the veterinarian can divulge any and all information to the seller.

A written report reviewing the findings of the examination should be provided to the buyer. The American Association of Equine Practitioners has an excellent set of guidelines for reporting the prepurchase examination. This report can serve as documentation of significant findings for future reference.

The veterinarian should advise the buyer about the risks of purchase, without making the decision for the buyer. The veterinarian is not responsible for assessment of the suitability of the horse for a rider or for determining an appropriate value for the horse. However, if the horse is clearly likely to be unsuitable for the purchaser because of temperament or ease of management or riding, the purchaser should be advised accordingly.

GUIDELINES FOR REPORTING PREPURCHASE EXAMINATIONS

The American Association of Equine Practitioners has approved the following guidelines for reporting equine prepurchase examinations. The spirit of these guidelines is to provide a framework that aids the veterinarian in reporting a purchase examination and to define that the buyer is responsible to determine if the horse is suitable. These guidelines are neither designed for nor intended to cover any examinations other than prepurchase examinations (e.g., limited examinations at auction sales and other special purpose examinations such as lameness, endoscopic, ophthalmic, radiographic, and reproductive examinations). Although compliance with all of the following guidelines helps to ensure a properly reported prepurchase examination, the veterinarian has the sole responsibility to determine the extent and depth of each examination. The American Association of Equine Practitioners recognizes that for practical reasons not all examinations permit or require veterinarians to adhere to each of the following guidelines.

1. All reports should be included in the medical record.
2. The report should contain the following:
   a. A description of the horse with sufficient specificity to identify it fully.
   b. The time, date, and place of the examination.
3. The veterinarian should list abnormal or undesirable findings discovered during the examination and give his or her qualified opinions as to the functional effect of these findings.
4. The veterinarian should make no determination and express no opinion as to the suitability of the animal for the purpose intended. This issue is a business judgment that is solely the responsibility of the buyer that he or she should make on the basis of a variety of factors, only one of which is the report provided by the veterinarian.
5. The veterinarian should record and retain in the medical record a description of all the procedures performed in connection with the prepurchase examination, but the examination procedures need not be listed in detail in the report.
6. The veterinarian should qualify any finding and opinions expressed to the buyer with specific references to tests that were recommended but not performed on the horse (x-rays, endoscopy, blood and drug tests, EKG, rectal examination, nerve blocks, laboratory studies, and so on) at the request of the person for whom the examination was performed.
7. The veterinarian should record and retain the name and address of parties involved with the examination (buyer, seller, agent, witness, and so on).
8. A copy of the report and copies of all documents relevant to the examination should be retained by the veterinarian for a period of years not less than the statute of limitations applicable for the state in which the service was rendered. Local legal counsel can provide advice as to the appropriate period of retention.

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CHAPTER • 116

Lameness in the Show Hunter and Show Jumper

Robert P. Boswell. Richard D. Mitchell, and Sue J. Dyson

HISTORICAL PERSPECTIVE

Show jumping and related competitions have origins in hunting sport and military tradition. The show hunter, most popular in North America, has evolved from the traditions of fox and stag hunting. These horses were expected to provide fast, safe, and athletic passage for the rider, and considerable pride was taken in being well mounted and having the horse admired. Today in the showring these horses are judged for beauty, athletic ability, manners, and way of going. Jumping style is important and must be coupled with consistent performance.

Some competitions encourage the development of a young horse to a higher level of training, others award a mature horse for outstanding performance, and others separate amateur and professional riders. Horses often are selected based on suitability for a particular division of competition.

The modern show jumper has many of its origins with military traditions. Many cavalry officers were by necessity highly skilled and accomplished horsemen. Thus when the era of the modern Olympics began, the equestrian competitors were military men. With the mechanization of the military and the replacement of the cavalry with motorized transport, the private sector became more involved in Olympic show jumping. Many of the early civilian competitors were retired military men. Show jumping has become increasingly more popular, and many talented riders have emerged on the national and international scenes. Since the 1960s many women have entered the sport once dominated by men.

STRUCTURE OF THE SPORT

Show jumping combines athletic effort of the horse and rider. The scoring process is objective, with the winner jumping the course with the fewest rails knocked down and, in the jump off, in the fastest time. Heights of fences range from 1 m at novice level to 1.7 m for advanced competitions. As the jumps get bigger, the potential for injury increases, and many conditions develop from repetitive strain. Many of the fences are set at distances to each other so that the horse must adjust stride length to fit in the appropriate number of strides. A good horse must have explosive power and great athleticism, combined with carefulness—a desire not to hit fences. Contrary to many equine sports, similar numbers of mares and geldings or stallions compete.

Some older horses compete at levels of competition lower than they have reached to be schoolmasters for less experienced riders. These older horses may experience unique problems related to age and use. Today show jumping is a highly diverse and competitive sport enjoyed all over the world from beginners to the level of the Olympic Games. At the top level the sport is entirely professional, with horses changing hands for huge prices and with large amounts of prize money available, putting pressure on the veterinarian to keep horses sound. Competition continues throughout the year with outdoor shows during the summer months and indoor shows during the winter, so horses potentially get little break.

CHARACTERISTICS OF THE JUMPING SPORT HORSE

Many breeds are capable of show jumping and related activities and include Thoroughbred, European Warmbloods, Thoroughbred/Warmblood crosses, and American Quarter Horses and related crosses. The European Warmbloods most often are represented by the Hanoverian, Holsteiner, Trakhener, Dutch Warmblood, Selle Français, Swedish Warmblood, and Irish crossbred breeds. Breeding in continental Europe has become highly specialized and has developed in part through financial support from state governments. Most modern day top-level show jumpers are naturally well-balanced, good-moving athletes. Various pony breeds, such as the Welsh crosses, are used for children (see Chapter 127).

Current preferences are for lighter and taller horses than previously, with moderate muscling. This body type is associated with greater speed and agility, which are assets to a modern show jumper. This body type also benefits hunters because they have graceful movement, with good extension and natural balance. Larger horses, if not excessively heavy, are at an advantage for show jumping because of greater stride length and overall strength and explosive power. The Thoroughbred long has been preferred by American trainers for the hunter ring; however, in recent years Warmblood breeds and American Quarter Horses have gained favor because of a calmer nature and better manners.

Horses often start competing at 4 years of age, reach peak ability at 9 to 12 years of age, and may continue to compete until 18 to 20 years of age, therefore the competition career is exceptionally long.

TRAINING

Training of the hunter/jumper emphasizes using the hindquarters for engagement and collection, which places more weight and stress on the hindlimbs as they are brought forward and under the rider during locomotion. Such a posture is somewhat unnatural for the horse, whose normal inclination is to distribute weight over the forehand. These stresses may contribute to or accelerate the development of problems of the thoracolumbar and pelvic regions and joints of the hindlimbs. Early in the training of a young horse, lameness often reflects musculotendonous problems and relates to lack of accommodation to work.

When training over fences begins, new loads on the hindlimbs occur that place more stress on soft tissues and joints. Stresses on the forelimbs also are increased because they are involved in takeoff and landing. The forelimbs are involved in setting up the jump and aiding in the directional change from horizontal to vertical. On landing the forelimbs

receive considerable impact loads and absorb the entire weight of the horse. Increased load places stress on the foot, joints, and the soft tissues.

Horses must learn to adjust stride length by shortening or lengthening the stride, to jump from a line perpendicular to a fence or at an angle, to turn quickly, to change leading legs in canter, and to jump from variable speeds of approach. Unwillingness to change leads or always favoring one lead when landing after a fence may indicate a problem (see Chapter 100). Much of the basic flat work training is similar to dressage.

Many lameness conditions encountered early in a horse's training begin as subtle performance-limiting problems, progress relatively slowly, and may disappear with further conditioning. However, excessive training leads to the development of important problems such as chronic muscle soreness, fatigue, and behavioral changes. Slow, steady work with a gradual buildup in exercise intensity and duration results in fewer joint and soft tissue problems later.

TRAINING AND COMPETITION SURFACES

Training, warm-up, and competition surfaces may play a substantial role in the development of lameness in a jumper. Soft, deep footing requires much more effort by the horse and is responsible for early fatigue of muscles, tendons, and ligaments. Injuries include gluteal muscle strain and spasm, suspensory desmitis, desmitis of the accessory ligament of the deep digital flexor tendon (ALDDFT), and superficial digital flexor tendonitis. Sandy soils contribute to the development of hoof wall problems. Hard surfaces may result in bone- and joint-related injuries, including foot problems such as subsolar bruising, distal interphalangeal joint synovitis, and osteoarthritis of the proximal interphalangeal and distal interphalangeal joints.

Most outdoor competitions in Europe take place on grass, with a few of the higher-level competitions taking place on all-weather surfaces. The nature of the footing depends highly on the weather and can vary extremely. To enhance traction, screw-in studs often are used in the front and hind shoes. Studs may be used in the medial and lateral branches of the shoe or just the lateral branch to reduce risk of interference injuries. If studs are used in only one branch of the shoe, this immediately creates mediolateral imbalance and the potential for abnormal torque. Studs also alter the dorsopalmar balance, especially if the ground is firm. Horses that flex the carpi excessively during jumping may have to wear a girth guard to protect the sternal region from self-inflicted injuries. If a horse is jumping on firm going, studs concentrate the forces of impact and can predispose the horse to deep-seated bruising of the foot. There is a tendency in horses with poor quality horn for the hoof wall to break in the region where studs are placed.

Excessive work, such as long period of lunging a hunter to calm it down, prolonged competitive efforts, and long show schedules of repeated competitions play a role in the development of many injuries in hunters and jumpers.

Conformation and Lameness

Conformational abnormalities of the foot predispose horses to lameness. Underrun heels, long toe, and a broken hoof-pastern axis frequently contribute to palmar heel pain and distal interphalangeal joint synovitis. Horses with improper medial-to-lateral hoof balance may develop sheared heels, crushed bars, and chronic foot pain. Base-wide or base-narrow forelimb conformation may contribute to injuries to the suspensory ligament (SL), its branches, and the distal sesamoidean ligaments.

Toe-in or toe-out forelimb conformation may predispose horses to proximal interphalangeal and metacarpophalangeal joint problems. Short, more upright pasterns predispose horses to navicular disease and distal interphalangeal joint and proximal interphalangeal joint synovitis or osteoarthritis. These horses are often straight through the shoulder as well and lack the stride and extension necessary for jumping. Long, more sloping pasterns sometimes are associated with sesamoiditis and soft tissue injuries to the distal limb.

Over-at-the-knee conformation may predispose horses to SL strain and should be avoided in selecting a jumper. Offset-knee conformation predisposes a jumper to medial splint problems. However, attention always should be paid to subclinical contralateral hindlimb lameness in a mature horse that develops an acutely painful medial splint bone. Subtle angular limb deformities are rarely a problem, provided proper attention is paid to shoeing and hoof balance.

Extremely sickle-hocked conformation is associated with weak hindlimbs and places more stress on the plantar ligaments and the centrodistal and tarsometatarsal joints. A straighter hock is actually more desirable, but an overly straight hock may predispose to SL strain and distal hock joint pain.

Horses that are extremely straight through the stifle are poor jumping prospects and have a high incidence of instability and upward fixation of the patella. A more angular stifle gives the horse a longer, more powerful stride and is thought to provide more strength for jumping.

A long, sloping hip and croup are desirable characteristics in a jumping horse, providing strength and power. Horses with a flat croup often suffer from thoracolumbar and sacroiliac pain. Asymmetry of the tubera sacrale and tubera coxae can be seen in the absence of lameness and may reflect previous trauma. An experienced rider usually can manage these horses.

Horses with excessively base-wide or base-narrow hindlimb conformation place abnormal stress on the feet and joints. Base-wide horses may have an increased incidence of hock problems, whereas base-narrow animals may suffer more from stifle problems.

SHOEING CONSIDERATIONS

Many lameness problems are a direct result of trimming and shoeing. Neglected feet are frequently a source of lameness, and a poorly shod foot also may contribute to lameness by forcing the horse to transfer abnormal and excessive stresses to other parts of the limb or to other limbs. Many of the Warmblood breeds have relatively tall, narrow upright feet, which are predisposed to the development of thrush and sheared heels. Studs in the shoes may create foot imbalance and may increase the severity of interference injuries. A good cooperative relationship with an experienced farrier is an essential element in lameness prevention and management. This can be a problem for top-level competition horses that are constantly moving from show to show and are being trimmed and re-shod by different people with varying talent.

TEN MOST COMMON LAMENESS PROBLEMS

The following are the 10 most common lameness problems of show hunters and show jumpers:

1. Palmar foot pain
2. Distal hock joint pain
3. Osteoarthritis of the distal interphalangeal joint
4. Proximal suspensory desmitis
5. Thoracolumbar and sacroiliac pain
6. Osteoarthritis or trauma of the metacarpophalangeal joint

7. Stifle pain
8. Osteoarthritis or trauma of the proximal interphalangeal joint
9. Gluteal myositis
10. Superficial digital flexor tendonitis and desmitis of the ALDDFT

Many of these problems are interrelated, and more than one problem may occur simultaneously. We have attempted to list these in relative order of frequency; however, this is not intended to imply that one is more serious than another.

LAMENESS EXAMINATION

Before examining a horse for a suspected lameness problem, several factors should be considered. If the horse is currently competing, will any diagnostic tests or treatment have an effect on the horse's ability to continue competition? Knowledge of the competition rules and drug use guidelines are essential. Has the horse recently competed? Has a change in exercise intensity or duration been made? Does the horse have a new trainer? When was the horse last shod? If the horse has been competing away from home, it is important to determine if another veterinary surgeon already has examined and treated the horse, and if so with what.

The lameness examination should begin as the horse is walked from the stall or paddock, because movements such as a small circle to reverse its direction may offer clues as to which limb or limbs may be affected. The horse should be examined at the trot in hand on a firm, level surface; lunging in small circles in both directions on firm footing may exacerbate subtle lameness. It is sometimes necessary to see the horse ridden to determine which limb is lame.

Proximal and distal limb flexion tests are performed on all limbs. The method and duration of flexion is a personal preference but should be consistent and interpreted with care, bearing in mind that joints and soft tissue structures may be stressed simultaneously. It has become common practice for top-level competition horses to be examined periodically throughout the year to try to detect early warning signs of impending problems. A positive response to flexion is often followed by treating the stressed joint(s). The true value of this practice is difficult to determine objectively.

The response to hoof testers is assessed, and each limb is palpated systematically. An obvious source of pain may be identified, but perineural or intra-articular analgesia often is required to determine the sources of pain. Results are sometimes confusing and always should be related to the clinical examination.

Aseptic preparation is essential for intrasynovial injections to minimize the risk of infection. One author (R.P.B.) also administers gentamicin intravenously before entering any synovial space. The conditions of the work area and the temperament of the horse being examined influence which local analgesic technique to use. Intrasynovial analgesia may be delayed or not performed if no clean, dry place is available for safe injection. Although intra-articular analgesia is considered to be more specific than perineural analgesia, it may influence peri-articular pain. In some instances the response to medication may be helpful in localizing pain to a joint. In situations where subtle lameness makes interpretation of nerve blocks difficult, in horses with multiple-limb lameness, for animals that are difficult or dangerous, or when comprehensive blocking fails to localize the lameness, other techniques such as nuclear scintigraphy should be considered.

When horses are competing regularly, especially away from home, owners, riders, trainers, and peers often put pressure on the veterinarian to treat the horse based on an index of clinical suspicion, rather than a complete lameness evaluation, including local analgesia. Although this can be successful and a positive response to treatment clearly indicates a correct diagnosis, one must bear in mind that some injuries do require rest for the best long-term outcome. A transient response only to treatment or lack of response warrants further investigation of the lameness, and this routinely should include local analgesic techniques.

IMAGING CONSIDERATIONS

Only after successfully localizing the source of pain causing lameness or after an extensive physical examination has provided the veterinarian with a reasonable indication of the problem should the examination progress to diagnostic imaging, including radiography, ultrasonography, and if indicated, nuclear scintigraphy, computed tomography, or magnetic resonance imaging. Routine techniques are used, with no special views. High-quality diagnostic imaging is related directly to the veterinarian's success as a diagnostician.

FAILURE TO MAKE A DIAGNOSIS

Every veterinarian, no matter how astute as a lameness diagnostician, eventually will be confused, unsure, or simply have no idea as to why a horse is lame, and consultation with associates or referral to other experts should be considered. Just as a good relationship with a farrier is paramount to the successful management of many foot-related problems, good relationships with other veterinarians are necessary. The veterinarian must be honest and open about the horse with the owners and trainers. Re-examination at a later date also may be beneficial. Some bone lesions may take a few weeks to become visible on plain radiographs, so the veterinarian should consider re-imaging if the lameness has been localized.

TREATMENT

In recent years the trend has been toward much more aggressive treatment, with many different treatment modalities often being combined to manage a single condition. Although in some circumstances this can be justified, it does mean that the veterinarian is often not sure which treatment really is effective. More targeted treatment actually may be equally effective.

DIAGNOSIS AND MANAGEMENT OF COMMON CAUSES OF LAMENESS

Palmar Foot Pain

Foot Soreness

The most common site of forelimb lameness in a hunter/jumper is the foot. The horse naturally supports 60% to 65% of its body weight over the forelimbs, and impact forces when jumping dramatically increase load and structural stresses in tissues within the hoof capsule. The manner in which a horse is shod has a tremendous significance in the development of hoof- and foot-related problems. Long toe and underrun heels are common hoof conformational defects and frequently contribute to heel pain because of hoof wall separation or bruising in the heel, quarter, and bar areas. The heels themselves may be excessively long and collapsed inward, and the horse actually may be bearing weight on the outer wall. This often results in sensitivity to hoof tester pressure applied to each heel and when the heels are squeezed together. Lameness is often improved by analgesia of the palmar digital nerves.

Lateromedial radiographic views of the foot may reveal that the distal phalanx is abnormally oriented, so that the palmar most aspect of the bone is lower (more distal) than the toe. A flattened and chronically bruised heel and bar area (corns) may be seen after removal of the shoe. In horses with chronic lameness deep digital flexor tendonitis and distal sesamoidean impar desmitis may contribute to pain associated with underrun heels. Clinically relevant lesions recently have been documented using magnetic resonance imaging in show jumpers with poor and good foot conformation (S.J.D.).

Some horses with underrun heels do not respond well to shoeing. Removing the shoes, trimming back the abnormal heel wall, and placing the foot in a support bandage is recommended. One author's (R.P.B.) preference is to fashion a cushion support for the palmar hoof using a two-part putty elastomer material (EDSS, Equine Distal Support System, Inc., Penrose, CO). Using the sulci of the frog for support is believed to suspend the heels and promote the new growth to be more vertical in its orientation. This process, however, requires a long-term commitment by the owner and trainer, because new heel growth may take up to 6 months to be sufficient for the reapplication of shoes.

Subsolar Bruising

Horses with subsolar bruising often respond well initially to Epsom salt poultices and non-steroidal anti-inflammatory drugs (NSAIDs), for example, phenylbutazone, followed by corrective shoeing. Rasping excessive toe from the solar surface proximally up the dorsal wall to create a 45° angle with the ground surface and application of a shoe fitted full in the heels may be of benefit by removing resistance to breakover. Ideal breakover is located between two points; the first is located by extending a line distally along the dorsal surface of the distal phalanx to the bearing surface, and the other by drawing a perpendicular line from the toe of the distal phalanx to the bearing surface. Egg bar shoes may be required to gain adequate heel support. Shoes such as the EDSS natural balance shoe (Equine Digital Support System, Inc.), with the web behind and squared off at the toe, also improve breakover and reduce stress in the palmar portion of the foot. We do not recommend the application of plastic wedge pads because they actually may contribute to further crushing of the heel and promote the heel bulbs to slide forward and grow horizontally. Leather pads may be helpful in some horses if sole pain is present and sole protection is desired. In horses with a chronic problem, long-term use of aspirin (60 g every other day) may be helpful. Hoof growth supplements containing biotin and methionine also may be of benefit, and we recommend feeding of biotin (100 mg daily) to promote hoof wall growth. Careful attention should be paid to the condition of the gastric mucosa with long-term NSAID use in show horses, because gastric ulceration may occur. Concurrent administration of acid pump inhibitors such as omeprazole should be considered.

Extreme sensitivity to hoof testers may be evident along the periphery of the sole at the level of the distal phalanx. Such pain may be associated with bruising, solar margin fractures or inflammation of the distal phalanx caused by chronic concussion from hard ground, or excessive sole pressure from the shoe. These conditions may be more common in North America than in Europe. Radiography is necessary for diagnosis of solar margin fractures of the distal phalanx. Shoeing should be directed at reducing local pressure on the affected areas and improving the overall hoof balance. Egg bar shoes and rim pads are often effective, but soft sole pours that provide extra cushion and shock absorption also help. Two-part putty elastomer is thought to benefit by providing support and lift from the sulci of the frog. Care should be taken with a pour or putty elastomer to avoid overfilling, causing excessive sole pressure.

Subsolar Abscess

Subsolar abscesses occur commonly and result from shoe nails improperly applied, poor environmental conditions, a shoe moving slightly, and poor hoof structure. Onset of clinical signs may be rapid, such as immediately following an event, or within the first several days following shoeing. The additional trauma of jumping exacerbates the condition, leading the rider or trainer to suspect trauma or serious injury. Warmth in the hoof wall, increased digital pulse amplitudes, and focal, extreme sensitivity to hoof testers are usually diagnostic, provided that the hoof horn is not excessively hard. Perineural analgesia is rarely necessary to confirm the suspected diagnosis and also may be confusing, because not all horses respond positively. After blocking and trotting the horse to reassess lameness, re-examination of the solar surface of the foot may reveal purulent drainage from the area of suspicion. Treatment is directed toward liberal opening of the solar surface of the foot at the point of maximum sensitivity to establish adequate drainage. If drainage is not established, bandaging the foot with hyperosmotic agents such as products containing magnesium sulfate (Epsom salts) is recommended. Twice daily soaking of the foot with a hot, supersaturated solution of Epsom salts with the bandage left on is also recommended for 3 to 5 days. Once drainage has been established, the foot is bandaged in a similar fashion, and NSAIDs also may be administered to reduce the inflammation. Antibiotics rarely are indicated but are sometimes used if soft tissue swelling occurs above the coronary band.

Navicular Disease

Initially to think that a horse is suffering from inflammation of the distal interphalangeal joint when the problem is navicular disease is not uncommon, but navicular disease is also overdiagnosed as a cause of heel pain. The diagnosis of navicular disease carries the stigma of a permanent and disabling lameness and is upsetting to the horse owner and trainer. Therefore the veterinarian should make an exhaustive effort to rule out all other possible sources for the pain causing lameness before making the diagnosis.

Navicular disease lameness usually is characterized by a slow, insidious onset. Early signs include shortening of the stride length, tripping, toe stabbing, and an intermittent unilateral lameness, although the lameness is almost always bilateral. Show horses are often more lame the day after a competition. Some horses with no previous lameness history become suddenly lame and are often refractory to standard therapies. Recent experience using magnetic resonance imaging suggests that a proportion of horses that were previously thought to have navicular disease have primary lesions of the deep digital flexor tendon within the hoof capsule (S.J.D.). The response to hoof testers varies, and often the horse shows only resentment when the heels are squeezed together. The wedge test may accentuate lameness. Lameness may be increased after distal limb flexion, but the response is non-specific. Lameness usually is eliminated by palmar digital nerve blocks, and a previously undetected lameness often appears in the contralateral limb. Analgesia of the distal interphalangeal joint or the navicular bursa often improves lameness.

Interpretation of the radiographic appearance of the navicular bone is not easy; many horses with navicular bone pain have no detectable radiological abnormality. We consider that radiolucent cyst-like lesions in the body of the bone, large lollypop-shaped radiolucent areas on the distal border of the bone, and enthesophyte formation on the proximal and distal borders of the bone should be considered significant.

Nuclear scintigraphy is useful in those horses that appear normal radiographically. The solar view is most useful and may reveal increased radiopharmaceutical uptake in the

navicular bone, reflecting abnormal bone metabolism. Lateral pool-phase images are also useful for highlighting horses that may have deep digital flexor tendonitis within the hoof capsule or navicular bursitis. Ultrasonographic examination of the navicular bone via the frog may demonstrate roughening of the flexor surface, but subtle changes easily are missed, and only a limited portion of the flexor surface can be examined.

Therapy for navicular disease includes pain management and corrective shoeing. Clients should be advised that this disease rarely is cured and requires a long-term commitment to its management. Horses with chronic, refractory lameness may require neurectomy.

Trimming and shoeing should be directed toward facilitating breakover, providing support to the palmar aspect of the foot, and in some horses elevating the heels to reduce tension in the deep digital flexor tendon (DDFT). A swelled heel egg bar shoe or the EDSS natural balance shoe is useful. The forward edge of the shoe should be set back from the toe and the branches fitted full at the heel. Leather wedge pads have been used to elevate the heels; however, the foot should be monitored closely to prevent crushed heels. A commercially available aluminum wedge-shaped shoe also may be used to provide heel elevation.

Medical management includes the use of NSAIDs, isoxsuprine, pentoxifylline, and aspirin; intra-articular injection of the distal interphalangeal joint and intrathecal injection of the navicular bursa with corticosteroids and hyaluronan; and rest. Many of these therapies, usually in combination, have proved successful in managing this condition, and we generally would recommend the least invasive therapies first. The degenerative nature of this disease, however, ultimately may result in the failure of any treatment, and palmar digital neurectomy may be considered. Surgical case selection is important, and consideration should be given to the overall condition of the horse and its level of performance. Horses with evidence of deep digital flexor tendonitis and proximal or distal distal interphalangeal osteoarthritis are not considered good candidates because of the possibility of tendon rupture or exacerbation of osteoarthritis. Some horses may experience temporary pain relief from the application of a chemical nerve block using a mixture of corticosteroids, ammonium sulfate, and Sarapin locally injected at the level of the palmar digital nerves. This procedure may work well initially only gradually to lose its effectiveness.

Shock wave therapy recently has been introduced as a non-invasive therapeutic option for pain management in navicular disease. Several investigators have reported good results, but differences in equipment and protocols require further investigation.

Sheared Heels

Sheared heels are a serious problem in hunters/jumpers. Lameness is often insidious in onset, or a critical point of instability may be reached, producing a more acute lameness. Subtle conformational abnormalities and poor hoof balance likely contribute to this condition. The medial heel often is displaced proximally, with the remainder of the foot splayed laterally. When viewed from the solar surface of the foot, the lateral half of the foot is larger and flared compared with the medial half. The medial heel may be painful to hoof testers, and the heel bulbs may be manipulated independently. Analgesia of the medial palmar digital nerve frequently improves lameness, but usually it is necessary to desensitize both heel bulbs before the horse appears sound. Many horses have radiographic lesions of the medial palmar process of the distal phalanx, such as roughening or demineralization of the margin of the bone, presumably because of chronic trauma and inflammation. The therapeutic goal is to stabilize the heel bulbs and reduce pressure on the driven up or proximally displaced bulb. Stabilization most often is accomplished best by improving balance and breakover so that the foot lands flatly and relieving the affected heel from excessive loading during weight bearing. In a jumper, an egg bar shoe is usually satisfactory. Horses with more severely affected feet may benefit from a heart bar, diagonal bar or G-bar shoe. A rim pad with the portion to lie beneath the affected heel cut away may float the heel enough to allow that side to descend into a more normal position. Six to 9 months of persistent treatment are required before a more stable heel structure is established.

Distal Hock Joint Pain

Distal hock joint pain is the second most common reason for lameness in a hunter/jumper and is the most common hindlimb lameness. Conformational defects, developmental abnormalities, and the incredible torsional stresses placed on the distal hock joints during jumping are thought to contribute to lameness. A variety of subtle signs becomes manifest before the onset of clinical lameness. Often the trainer or rider complains of a loss in the horse's stride length, poor impulsion, and a change in the horse's jumping style. Many horses may develop a tendency to switch leads in front of a jump or may have difficulty jumping from a particular lead. The horse consistently may develop the tendency to jump to one corner of the obstacle, and wider jumps appear to require more effort than usual or even necessary. Clinical examination reveals sensitivity to palpation of the muscles of the lumbar area and shortened cranial phase of the stride. The upper limb flexion test may or may not be positive, and some horses are reluctant to move forward immediately into a trot after flexion and may canter away from the veterinarian. The Churchill test is often positive in horses with tarsometatarsal joint pain but may be negative with centrodistal joint pain. Some horses with proximal suspensory desmitis also respond positively to the Churchill test (R.D.M.). Tarsocrural effusion may or may not be present. We (R.P.B. and R.D.M.) rarely perform regional analgesia of the fibular and tibial nerves, because we consider the procedure difficult to interpret and somewhat dangerous to perform. Intra-articular analgesia of the centrodistal and the tarsometatarsal joints is preferred. However, a negative response to intra-articular analgesia does not preclude centrodistal and tarsometatarsal joint pain, and using fibular and tibial nerve blocks to identify some horses with distal hock joint pain may be essential. With practice this is a highly reliable and safe technique (S.J.D.). With subtle hindlimb lameness or complaint of poor performance and no apparent lameness, it may be necessary for the horse to be ridden and for the rider to interpret the results of the block. The veterinarian should allow this with only the most experienced and talented riders.

Radiographic changes vary from none to severe, with what may appear as total radiographic evidence of fusion. Radiographic changes do not necessarily correlate to the degree of lameness. Osteochondral fragments on the distal intermediate ridge of the tibia, or at the distal aspect of the medial trochlea of the talus in a mature athlete in the absence of lameness, are an incidental finding of little if any clinical significance. The severity of the degenerative changes may be similar in the centrodistal and the tarsometatarsal joints; however, the tarsometatarsal joint often has less severe abnormalities. The reason for this is unclear.

Nuclear scintigraphy can be helpful and reveals increased radiopharmaceutical uptake (IRU) in the distal aspect of the tarsus. Occasionally, small, focal, moderate to intense areas of IRU may reflect focal areas of loss of joint space, bone trauma, or tearing of ligamentous attachments.

It has been generally accepted that radiographic evidence of distal joint(s) fusion is desirable, because the suggestion is that once joint fusion has occurred, the inflammation and therefore the pain should disappear. This is an unfortunate myth. Surgical arthrodesis is a currently accepted treatment for horses with osteoarthritis of the centrodistal or tarsometatarsal joints that have become refractory to routine therapies. We challenge this belief and have observed evidence to the contrary. Once arthrodesis has occurred, the normal function of the joint is lost. The distal joints are responsible for the dissipation of the twisting or torsional forces in that area. With this capability gone, these forces are concentrated, and stress fracture of the central or the third tarsal bones may occur or osteoarthritis of the talocalcaneal-centroquatral (proximal intertarsal) joint may develop.

Treatment of horses with distal hock joint pain varies. Intra-articular injections of corticosteroids (20 to 40 mg methylprednisolone acetate or 5 to 7.5 mg betamethasone in each joint) and hyaluronan (2 ml per joint) is usually the first treatment, combined with NSAIDs, and is usually the best and most expeditious means of treatment. With radiographic evidence of severe osteoarthritis we use a long-acting corticosteroids alone. Treating horses with more mildly affected joints with a low corticosteroid dose in combination with hyaluronan may be chondroprotective and extend the useful life span of the joint. Combined therapies have grown popular because of the perception that they work better and the effects last longer. Most horses respond favorably, and the riders comment on how much better the horse performs. Many horses, especially those with demanding show schedules, have the hocks (and perhaps other joints also) routinely injected at intervals predetermined by the history of when the horse has previously become unsound following previous injections. Maintenance treatment is performed about 1 month earlier. This reduces the risks of loss of performance, recurrent lameness, or development of a secondary problem.

Many horses also are treated with the oral nutraceuticals, parenterally administered polysulfated glycosaminoglycans (PSGAGs), and intravenously administered hyaluronan. These products are incorporated into a maintenance program designed to keep the horse comfortable and prolong the interval between intra-articular joint injections. Frequently the large size of these horses means that they are treated with twice the normal dose of PSGAGs. Horses often are treated with intramuscularly administered PSGAGs and intravenously administered hyaluronan the evening before competition. This is thought to enhance the horse's ability by making the joints more comfortable. Occasionally, horses with joints unresponsive to corticosteroids and hyaluronan do respond to intra-articularly administered PSGAGs, with or without corticosteroids. Once the horse is made more comfortable, an exercise program consistent in intensity and duration, emphasizing strengthening the hindquarters and abdominal muscles, improves performance. Recent reports describe shock wave therapy being used to control pain. Shoeing is aimed at encouraging breakover in the center of the hoof by squaring the toe. Some veterinarians prescribe a lateral trailer in an effort to decrease the twisting motion of the limb. Alternative therapies such as muscle massage and acupuncture are used commonly with traditional therapies.

Distal Interphalangeal Joint Synovitis and Early Osteoarthritis

Inflammation of the distal interphalangeal joint is common in jumping horses and usually results in subtle lameness that is frequently bilateral. Palpable joint effusion may or may not be present. Many horses, but not all, respond positively to the distal limb flexion test. Forelimbs are most commonly affected, but the hindlimbs also may be involved. The horse may have a reduction in stride length, and lameness is most obvious when the horse circles on firm footing. The rider or trainer may complain of a reduction in jumping performance, such as a reluctance to leave the ground and landing in a heap. Very hard surfaces for training or competition and surfaces that are too soft, irregular, and unstable may be predisposing factors. Soft footing, although not seemingly likely to contribute to excessive concussion, produces torsion forces on the distal interphalangeal joint that strain the peri-articular soft tissues. An underrun heel, long-toe conformation is probably the single most important contributing factor. The horse often shows a painful response to hoof testers with pressure applied from either heel to the opposite frog and from the center of the frog to the dorsal hoof wall. Lameness usually is improved with analgesia of the palmar digital nerves and eliminated with palmar (abaxial sesamoid) nerve blocks or intra-articular analgesia of the distal interphalangeal joint. Lameness in the contralateral limb may become apparent.

Radiographic changes may be absent or subtle; however, in horses with more chronic or severe disease, reactive periosteal bone may be present on the dorsal aspect of the middle phalanx, and remodeling of the extensor process of the distal phalanx may occur. With chronic, severe osteoarthritis of the distal interphalangeal joint there may be subchondral radiolucent areas in the distal phalanx consistent with collapse of the joint. Prominent radiolucent areas representing synovial invaginations may be present on the distal border of the navicular bone.

Nuclear scintigraphy is useful to rule out bone involvement. An area of IRU in the synovial structures of the joint may be visible in pool-phase studies. Differentiating the palmar pouch of the distal interphalangeal joint from the navicular bursa is difficult, and both structures may be involved in horses with severe lameness.

Therapy for distal interphalangeal joint synovitis depends on the severity of the lameness and the horse's competition schedule. Proper trimming and shoeing as for palmar heel pain is essential. The use of NSAIDs is common. Peripheral vasodilating agents such as isoxsuprine are of questionable value, although higher than standard doses are sometimes effective.

Intra-articular injection of high molecular weight hyaluronan and corticosteroids (triamcinolone or betamethasone) with proper shoeing and an appropriate amount of rest usually yields the best results. Occasionally, repeated injections are necessary after 4 to 6 weeks; however, injections repeated more frequently than once every 3 months should be avoided. Nutraceuticals and parenterally administered chondroprotective agents (PSGAGs) may be beneficial.

Collateral Ligament Injury of the Distal Interphalangeal Joint

Some horses with synovitis of the distal interphalangeal joint that are unresponsive to therapy should be evaluated for possible collateral ligament injury of the distal interphalangeal joint. In a few horses, we (R.D.M. and S.J.D.) have identified thickening and tearing of the collateral ligament ultrasonographically, and unusually, focal increased radiopharmaceutical uptake of the palmar aspect of the distal phalanx, scintigraphically. Differentiating this injury from other more common conditions of the distal phalanx may be difficult. Horses with this injury should be managed with corrective shoeing and rest. Work level is reduced substantially. Extracorporeal shock wave or therapeutic ultrasound therapy combined with a 2- to 3-month period of rest appears to be effective.

Suspensory Desmitis

Injury to the SL is the most common soft tissue injury and certainly one of the most serious injuries of the jumping horse. Early lesions may go unnoticed by even the most skilled horseman, because the rider or trainer is aware of only a vague

problem, from which the horse warms out of fairly quickly. Exercise continues and the injury eventually worsens to the point of causing enough damage to produce an obvious lameness. The lameness usually is more pronounced when the horse is trotted in a circle with the affected limb on the outside and also may be exacerbated in soft footing. Mid-body and branch lesions are diagnosed easily by palpation; however, diagnosis of injury to the proximal aspect of the SL is more challenging. The distal limb flexion test may increase the lameness with mid-body and branch lesions, whereas carpal flexion and upper hindlimb flexion frequently accentuate lameness associated with proximal suspensory desmitis, which can cause confusion with hock pain. A high palmar metacarpal nerve block, direct infiltration of the SL origin, or analgesia of the lateral palmar nerve eliminates lameness in the forelimb. Analgesia of the proximal aspect of the hindlimb SL requires caution, because injury to the veterinarian and patient can occur. Analgesia is best performed with the horse restrained with a twitch and the leg positioned in the veterinarian's lap as if the horse's foot were being examined. Displacing the flexor tendons medially and isolating the SL for injection is thus easier. The SL is infiltrated with local anesthetic solution from the axial aspect of the lateral splint bone, fanning across the SL from lateral to medial. During injection, the local anesthetic solution should go in with considerable resistance if it is being deposited within the SL itself. Alternatively, perineural analgesia of the deep branch of the lateral plantar nerve can easily and safely be performed with the limb bearing weight (S.J.D.). With direct infiltration of the ligament, soundness should be almost immediate. Some risk exists of entering the palmar outpouching of the carpometacarpal joint capsule using direct infiltration in the forelimb, causing potential diagnostic confusion; however, infiltration in the hindlimb inadvertently entering the tarsometatarsal joint is less likely.

Ultrasonographic examination of the SL may reveal obvious lesions; however, finding minimal evidence of damage in horses with acute injuries is not uncommon. Recent nerve blocks may confuse interpretation, so ultrasonographic examination is best delayed for 1 to 2 days. Alternatively, ultrasonographic examination may precede nerve blocks. In our opinion, ultrasonographic imaging of the proximal SL in the hindlimb is difficult. The transducer should be placed on the plantaromedial aspect of the limb to get the best-quality images. A cross-sectional area of greater than 1.5 cm^2 suggests proximal suspensory desmitis, even in the absence of a focal or diffuse loss of echogenicity.

Radiographic examination of the proximal aspect of the third metacarpal or metatarsal bone may demonstrate sclerosis with or without lysis in a dorsopalmar (dorsoplantar) view or subcortical sclerosis in lateromedial views. Incidental radiographic changes can be seen in hindlimbs.

Nuclear scintigraphy has proved useful in characterization and prognosis of horses with proximal suspensory desmitis. The pool phase may reveal increased radiopharmaceutical uptake in the ligament. A unique pattern of uptake is present in the forelimbs and hindlimbs in the bone phase. The lateral view is most important in the forelimb because focal IRU is present on the proximopalmar aspect of the third metacarpal bone (McIII) if bone injury accompanies proximal suspensory desmitis. Proximal suspensory desmitis occurs often without chronic changes in the palmar/plantar cortex of the third metacarpal or metatarsal bone. Dorsal views of the forelimb are less sensitive than lateral views. In the hindlimb the lateral view is equally important. The veterinarian should not confuse as a lesion IRU in the proximolateral aspect of the metatarsal region in plantar views, which is a normal finding. Ultrasonographic abnormalities of the SL and abnormal scintigraphic images confirm injury to the bone at the origin of the SL, indicating a complex injury involving two tissue types and adversely affecting the prognosis.

Therapy for horses with proximal suspensory desmitis varies. NSAIDs, combined with rest and physical therapy, are the most popular and yield the most consistent results. Clients should be advised that treatment may take 6 to 12 months. We recommend stall rest for 10 to 14 days after the injury, with twice daily hand walks. Phenylbutazone is administered (2 g BID for 4 days and then 1 g BID for 10 days). Walking under saddle then is commenced for 20 to 30 minutes once or twice daily for the next 30 days. Follow-up ultrasonographic examinations are performed after 44 days and every 30 to 60 days thereafter. The duration and intensity of exercise gradually are increased based on the ultrasonographic appearance of the SL and the clinical appearance of the horse. Trotting begins once the horse is sound. Many veterinarians in North America recommend an internal blister, using 2% iodine in almond oil infiltrated into the SL. Light exercise is continued immediately following this therapy, with a gradual return to full work. Shoeing should be improved if necessary. In horses with acute proximal suspensory desmitis with no significant ultrasonographic changes, local injection of corticosteroids may decrease inflammation and eliminate pain. We use triamcinolone (12 to 18 mg), betamethasone (30 mg), or isoflupredone acetate. Sarapin (4 ml) also may be added.

Recently a surgical procedure has been reported in which the tight fascia overlying the proximal SL is transected or released so as to reduce any increased pressures or compression created by this apparent compartmental syndrome. Bone marrow aspirate taken from the sternum then is injected in the area of the injury in an effort to stimulate healing. Early results from this technique appear promising; however, further investigation and long-term follow-ups are needed. Neurectomy of the deep branch of the lateral plantar nerve has been used successfully for management of hindlimb PSD (S.J.D.).

Shock wave therapy has been reported to aid healing of proximal suspensory desmitis, especially in horses with bone involvement. This technique provides profound analgesia and therefore a decrease or elimination of lameness. Further investigation is warranted.

Horses with larger lesions within the body of the SL or its branches may benefit from splitting. This appears to allow a more complete healing of core lesions, which otherwise may be slow to resolve. Following surgery, therapeutic ultrasound or infrared laser therapy may aid and reportedly speed healing, although evidence is mainly anecdotal.

Back Pain

Back pain is common in jumpers, and although signs are recognized easily, the etiological diagnosis may be elusive and complex. The trainer or owner may perceive that a horse has back pain from many clinical signs including sensitivity to grooming and saddling, resistance to rider weight, overall body stiffness, poor performance, and pain on palpation of the muscles over the back. Many of these signs are also common to other diseases or injuries and may be secondary to clinical or subclinical hindlimb lameness resulting in an altered gait. Primary back pain may be caused by severe muscle strain, impingement or overriding of the spinous processes, diskospondylosis, sacroiliac desmitis, supraspinous desmitis, osteoarthritis of the facet joints, and sacroiliac joint pain.

The veterinarian should first attempt to rule out any lameness. Horses with distal hock joint or stifle pain may swing the hindlimb outward away from the body or inward toward the midline in an attempt to reduce the degree of joint flexion required to advance the limb. Hindlimb lameness should be suspected in horses with simultaneous gluteal and back pain. Chronic forelimb soreness, particularly involving the foot, also may contribute to back pain because of an inverted way of jumping that somewhat protects the foot on landing. Teeth problems or neck pain also should be considered.

Severe back pain suggests a primary back problem. The horse may move with a shortened, stiff gait and appear to be flat or hollowed out in the back when ridden but appear much more comfortable when moving free in a paddock. Injection of local anesthetic solution into the painful areas of the back frequently changes the horse's movement.

Impinging Spinous Processes

Radiography of the thoracolumbar spinous processes may reveal impingement with sclerosis and bone proliferation and bone lysis. Nuclear scintigraphy can be used to diagnose and support the diagnosis of spinous process impingement and osteitis, although false-negative results are sometimes obtained. Occasionally, areas of IRU are visible in the spinous processes without any radiographic changes, and supraspinous ligament desmitis should be considered.

The treatment for impingement of the spinous processes or supraspinous desmitis is similar. NSAIDs and methocarbamol are prescribed routinely for a prolonged period. Sarapin and corticosteroids frequently are injected between and around the impinging spinous processes. The locations for injection are determined best by placing a radiodense marker on the top of the back during radiography and then identifying the affected vertebrae by clipping the hair.

Muscle Injury

Acute muscle strain may be accompanied by spasm, which is evident as a firm, painful swelling. Immediate application of ice and administration of NSAIDs such as phenylbutazone or naproxen are beneficial One author (R.P.B.) also administers between 20 and 40 mg of dexamethasone once daily for 2 days in horses with acute pain. Later, moist heat and therapeutic ultrasound reduce pain and inflammation. Acupuncture has proved useful in managing back pain no matter what the cause. Chiropractic manipulation may be of some benefit in relieving pain and muscle spasm in an injured back. Pulsating magnetic field therapy is used routinely in show jumpers for the long-term management of back pain. We believe that many owners feel pressure to do everything possible for their horses, and because of anecdotal reports of benefit in people, certainly no harm will come from using such therapy. Saddle fit always should be evaluated critically in a horse with chronic or recurrent back pain, and thermography may be helpful, together with advice available through most good saddle manufacturers.

Fracture of the Withers

Fracture of the withers is not uncommon if a horse flips over backward. The withers area may appear flat and is extremely painful to palpation. The horse plaits in front and holds the neck stiffly. Radiographs are diagnostic. Treatment is directed toward reducing pain and inflammation by applying ice and administering NSAIDs immediately following the injury. Bone sequestrae occasionally develop, and purulent drainage may appear weeks after the injury. However, most fractures are uncomplicated and horses usually may be able to return to work after 6 to 12 months of rest.

Sacroiliac Joint Pain

Sacroiliac strain is common in show jumpers. Many horses suffer from chronic low-grade pain that never seems adversely to affect the ability to perform. With severe pain a horse may stand parked out (the hindlimbs are extended unusually) and rest one hindlimb. Unilateral lameness may develop, and the horse's performance then is affected severely. The horse may experience pain on palpation around the lumbosacral region and directly over the tubera sacrale, but this is not specific. Exerting pressure on one of the tubera sacrale may reveal slight motion and even may be resented by the horse. The horse may offer considerable resistance when one of the hind feet is picked up and the limb is flexed high. Rocking the pelvis may cause the horse to grunt. A hunter's bump is not associated necessarily with this condition, although it may be. Injection of local anesthetic solution deep into the muscles directed toward the sacroiliac articulations should be performed with caution because the horse may lose its ability to stand, which causes severe distress. Nuclear scintigraphy may reveal little if any IRU in this region and is therefore unreliable, but with modern motion-correction software program and improved image quality, useful information is acquired in some horses (S.J.D.).

Rest and time are the most important factors influencing the outcome. These horses require at least 6 months to heal. Severely affected horses should be given stall rest for 30 days, followed by 2 to 3 months of controlled paddock rest. Light exercise then may begin, gradually increasing the intensity and duration of the work. In our experience, acupuncture has been extremely useful for pain management and the treatment of muscle spasm.

Horses with less severe injuries may be managed successfully by local injections of corticosteroids and Sarapin deep into the painful areas. These horses are able to continue to exercise and compete successfully. The procedure involves directing a 9-cm needle from a point just medial to one of the tubera sacrale along the inner surface of the ilium deep toward the sacroiliac articulation. Injection of the acupuncture points on either side of or parallel to the sacrum with the same solution also may be performed.

Chiropractic manipulation often is attempted in horses with sacroiliac injury. Although chiropractic has proved useful as a diagnostic tool and may be of benefit with mild sacroiliac strain, manipulation of the sacrum in more severe injuries never replaces the need for prolonged rest. Pulsating magnetic field therapy, cold laser, and therapeutic ultrasound may be used for the long-term management of chronic sacroiliac problems.

Fetlock Joint

Synovitis and Osteoarthritis

Metacarpophalangeal joint synovitis and osteoarthritis are common in older horses with lengthy careers. Chronic capsulitis results in a thickened, prominent joint capsule with a dramatic decrease in the flexibility of the joint. At least a moderate amount of joint effusion occurs, but many sound horses may have chronic effusion, thickening of the joint capsule, and reduced range of motion. The source of pain is confirmed by a low four-point palmar/plantar nerve block or intra-articular analgesia.

Radiographic examination may reveal osteophyte formation on the proximal aspects of the proximal phalanx and the proximal sesamoid bones, and flattening of the sagittal ridge of McIII. McIII may have a scalloped appearance on the dorsal or palmar aspect in horses with advanced disease. Subchondral lucent areas may develop in McIII or the proximal phalanx and result from severe, focal trauma or end-stage osteoarthritis. The prognosis for horses to return to athletic competition following the development of these lesions is considered poor. Acute fractures and small chips are uncommon in the hunter/jumper. Small, round, smooth fragments on the proximodorsal aspect of the proximal phalanx are seen in forelimbs and hindlimbs and likely are related to osteochondrosis. These fragments rarely are associated with lameness but occasionally may become unstable and require removal.

Nuclear scintigraphy may reveal mild to moderate IRU in the distal aspect of McIII, the proximodorsal aspect of the proximal phalanx, or both. In some horses flexed lateral views are required to separate the bones, especially if radiopharmaceutical uptake in one area is so intense that identifying the adjacent structures is impossible.

In the absence of substantial radiographic abnormalities medical therapy should include NSAIDs, intra-articularly administered corticosteroids and hyaluronan, and orally and parenterally administered PSGAGs. Physical therapies such as

icing, cool water therapy, poultices, sweats, cold laser, therapeutic ultrasound, support wraps, and rest are also beneficial. Arthroscopic exploration of the metacarpophalangeal joint is indicated if the response to medical treatment is transient or poor. Clients should be advised that although the causal problem may be revealed, treatment may not be possible. Debridement of damaged cartilage and subsequent replacement with fibrocartilage may be curative; however, excessive erosion of the articular cartilage warrants a poor prognosis for continued jumping.

Therapeutic shoeing targeted at providing support to the palmar aspect of the foot, such as a wide-web egg bar shoe, may be of benefit (R.D.M. and R.P.B.). Proper medial to lateral hoof balance is also important in reducing torque on the metacarpophalangeal joint.

Sesamoiditis

Sesamoiditis frequently is associated with chronic suspensory branch desmitis. Horses are variably lame and may warm out of the lameness. Distal limb flexion test usually is resented and results in increased lameness. The suspensory branches may be palpably thickened and painful. Intra-articular analgesia of the metacarpophalangeal joint may improve lameness but does not alleviate it. Lameness is abolished by a low four-point palmar nerve block.

Radiographic findings include linear lucent zones or lytic areas within the body of the proximal sesamoid bone (PSB). A generalized loss of bone opacity or proliferative, reactive bone also may be present on the abaxial margins in association with suspensory branch desmitis and insertional lesions of the SL and palmar annular ligament. We find proximodistal oblique views of the PSBs useful.

Nuclear scintigraphy is sensitive to inflammation in the PSBs, and IRU in the PSBs often is intense. A flexed lateral view helps to separate the PSBs from McIII. Dorsal or plantar views are required to distinguish between the medial and lateral PSBs.

Treatment for horses with sesamoiditis includes rest, NSAIDs, and supportive shoeing similar to that prescribed for fetlock joint problems. Shock wave therapy may be helpful.

Stifle Joint Pain

Problems involving the stifle joint are common in show jumpers and frequently accompany problems in the tarsus. Femoropatellar effusion is present variably. Stifle pain may be primary and related to trauma, mechanical problems, developmental diseases, and osteoarthritis, or it may be secondary to other lameness. Primary problems include osteoarthritis, osteochondrosis, meniscal and cruciate ligament trauma, upward fixation or delayed release of the patella, and patellar desmitis. Signs may be subtle at first, with mild shortening of the stride or switching leads at the canter or gallop. Caudal back pain also may be present. The proximal limb flexion test may or may not be positive, and the veterinarian should look closely for subtle gait changes such as a shortening of the cranial phase of the stride. Separation of stifle and tarsal pain may be accomplished by flexing the stifle with the hock slightly extended so that the metatarsal region is held behind the tail and perpendicular to the ground, with the tibia held parallel to the ground. The Churchill test also may help to separate hock from stifle pain. Cranial to caudal motion of the tibia relative to the femur during weight bearing (cruciate test) may produce a change in stride (R.D.M.). However, intra-articular analgesia always is required to confirm the source of pain. Horses with straight stifle conformation are predisposed to upward fixation and instability of the patella. Pain results from subsequent patellar ligament strain and synovitis. Although the limb may be observed to lock in extension, more frequently a slight hesitation in the advancement of the limb is noted. Femoropatellar effusion may be present. Ultrasonographic examination of the patellar ligaments may reveal desmitis characterized by thickening and a focal or diffuse hypoechoic region. Horses with transient upward fixation of the patella benefit from NSAIDs and an increased exercise program designed to increase muscle tone in the quadriceps group, which stabilizes the patella. Failure to respond to these measures may justify a more aggressive procedure, such as local injection of 2% iodine in almond oil at the proximal aspect, middle portion, and insertions of all three patellar ligaments and into the muscle just proximal to the stifle. This procedure has proved effective in treating transient upward fixation of the patella and subtle soreness in the adjacent muscles. Anecdotal reports describe benefit from estrone sulfate, estradiol, and calcium channel blockers.

Cranial Cruciate Ligament and Meniscal Injury

Trauma to the cranial cruciate ligament produces profound lameness. Intra-articular analgesia of the femorotibial joints may or may not abolish the lameness, and radiographs of the stifle may be normal in horses with an acute injury. With chronic injury new bone may be seen cranial to the intercondylar eminences, most obvious in a flexed lateromedial view. Injury to the medial meniscus is most common. Intra-articular analgesia of the femorotibial joint on the affected side usually produces improvement in the lameness. Radiographs may reveal osteophyte formation on the proximomedial aspect of the tibial plateau indicative of osteoarthritis and a subjective decrease in the joint space width on the affected side.

Osteochondrosis and Subchondral Bone Cysts

Osteochondrosis and subchondral bone cysts should be considered in a young Warmblood that has recently started training or increased its training intensity and has developed lameness localized to the stifle. Intra-articular analgesia usually at least partially eliminates the lameness. We recommend blocking all three joint spaces.

Radiographs are usually diagnostic. Arthroscopy may be indicated. Horses with subchondral bone cysts and other lesions on the medial femoral condyle usually have a poorer prognosis than those with osteochondritic lesions of the lateral trochlear ridge of the femur. We recommend radiography of the stifles as part of the routine radiographic study when examining a young Warmblood for purchase.

Nuclear scintigraphy of the stifle with osteochondrosis may reveal little if any IRU. The caudal view is important and should always accompany the lateral views. Visible uptake (to any degree) in the distal medial femoral condyle in the caudal view almost always is associated with a pathological condition (R.P.B.).

Synovitis and Osteoarthritis

Mature horses may develop synovitis and osteoarthritis of any one or a combination of the stifle joints. Often the femoropatellar and medial femorotibial joints are involved; however, lateral femorotibial osteoarthritis may occur alone. Intra-articular analgesia of all three joints should be performed.

The diagnosis may or may not be confirmed radiographically. Osteophyte formation on the proximal medial aspect of the tibia is not uncommon. Nuclear scintigraphy may reveal mild to moderate IRU in the femoral condyle and the opposing surface of the tibia on the affected side in a caudal view.

Intra-articular injection of corticosteroids and hyaluronan usually has favorable results. Each joint should be injected separately with double doses of a high molecular weight hyaluronan combined with methylprednisolone acetate (40 to 80 mg) or betamethasone (5 to 10 mg). NSAIDs, parenterally administered PSGAGs, and intravenously administered hyaluronan are also beneficial and may be used routinely with nutraceuticals as part of a maintenance program. Adequate rest also should be prescribed; however, strict stall confinement is not necessary.

Walking these horses under saddle for 30 minutes once or twice daily is preferred. Alternative therapies such as those described for distal tarsitis are also beneficial. Acupuncture commonly is used for managing horses with stifle pain.

Pastern

The pastern region is subject to considerable stress in the jumping horse. Osteoarthritis of the proximal interphalangeal joint is not uncommon and often is associated with distal interphalangeal osteoarthritis. Horses with base-narrow or toe-in conformational defects are affected most frequently. Distal sesamoidean desmitis is also a common problem. Trainers complain that many horses with osteoarthritis of the proximal interphalangeal joint start out stiff when training and feel much better after warming up, but eventually the lameness becomes persistent. With chronic injury comes obvious thickening in the distal aspect of the pastern. Palmar (abaxial sesamoid) nerve blocks normally eliminate the lameness, although a low palmar (four-point) block sometimes is required. Intra-articular analgesia of the proximal interphalangeal joint also usually produces soundness. The veterinarian must beware that a palmar digital nerve block may alleviate lameness associated with osteoarthritis of the proximal interphalangeal joint because of proximal diffusion of the local anesthetic solution.

Osteoarthritis

IRU usually occurs in the proximal interphalangeal joints of sound jumping horses relative to other joints. With osteoarthritis the intensity of the IRU usually increases. We (R.P.B. and R.D.M.) therefore believe that subclinical osteoarthritis of the proximal interphalangeal joint is common. Radiographic changes of osteoarthritis of the proximal interphalangeal joint include osteophyte formation on the proximodorsal aspect of the middle phalanx. Subchondral osseous cyst-like lesions in the proximal aspect of the middle phalanx may be associated with severe trauma or end-stage osteoarthritis. Acquired osseous cyst-like lesions usually are associated with focal, intense IRU.

Management of osteoarthritis of the proximal interphalangeal joint includes using NSAIDs, intra-articularly administered corticosteroids and hyaluronan, and orally and parenterally administered PSGAGs. The horse should be trimmed and shod to reduce resistance to breakover at the toe and to balance the foot to land as flat as possible. Occasionally, wedge pads provide comfort by opening up the dorsal aspect of the joint space. Recently, shock wave therapy has been suggested to be of benefit to horses with advanced ringbone, but this requires validation. Horses with severe osteoarthritis of the proximal interphalangeal joint may respond poorly to medical management, and arthrodesis may be required. Arthrodesis is often successful in the hindlimbs of jumping horses, but results are often unsatisfactory in forelimbs. Tibial neurectomy also has been used successfully.

Soft Tissue Injury

Injury to the distal sesamoidean ligaments may or may not be accompanied by obvious swelling or pain on palpation. Analgesia of the palmar (abaxial sesamoid) nerves usually eliminates the lameness. Ultrasonographic examination reveals focal or diffuse hypoechoic regions in the affected ligament. Palmar or plantar displacement of the DDFT medially or laterally may be secondary to distal sesamoidean ligament injury. The veterinarian should image all structures carefully in the pastern, because several structures may be injured simultaneously. DDFT injuries are more common in the pastern than in the metacarpal region. Chronic strain at the insertion of the middle distal sesamoidean ligaments may be evident radiographically as enthesophyte formation on the palmar or plantar lateral and medial borders of the proximal phalanx.

Horses with strains and tears of the distal sesamoidean ligaments usually respond to supportive shoeing, NSAIDs, and rest. Horses with acutely torn ligaments may benefit from cast or splint application for about 1 month. Physical therapy such as therapeutic ultrasound may speed recovery. Injury to these ligaments usually requires from 6 to 12 months for convalescence. Returning the horse to work too soon results in thickening and fibrosis of the pastern and prolonged lameness.

Gluteal Myositis

Strain and inflammation of the gluteal muscles is common in jumping horses and usually results from an altered gait secondary to lameness elsewhere in the ipsilateral or contralateral hindlimb. Primary strain may occur in a horse during jumping, when a horse refuses a jump, or after a fall. Palpation of the gluteal muscles at the insertion on the greater trochanter of the femur and over the middle gluteal region reveals milder reactivity in horses with secondary strains and a much more severe response or resentment after a primary muscle injury. Upper limb flexion tests may be positive, especially if the gluteal soreness is secondary to lameness involving the hock or stifle.

Treatment depends on the severity of the muscle injury or inflammation. Identification and appropriate treatment of the primary lower limb lameness, if present, may do much to reduce mild soreness in just a few days. Most horses with mild injury also respond well to NSAIDs, local application of moist heat, dimethylsulfoxide, and therapeutic ultrasound. Methocarbamol (20 mg/kg PO BID) may be of benefit. Acupuncture treatments, and in horses with severe injuries local injection of corticosteroids mixed with Sarapin (mix Sarapin, prednisolone, and betamethasone in equal parts in a 12-ml syringe and inject 3 ml per site), provide pain relief. Injections are performed on either side of the spine into the sorest portions of the muscle at 5- to 8-cm intervals or at specific acupuncture sites. Rest and a reduction in the intensity and duration of exercise also may help. Some horses may require total rest with exercise limited to hand walking for several weeks.

Superficial Digital Flexor Tendonitis, Tenosynovitis of the Digital Flexor Tendon Sheath, and Desmitis of the Accessory Ligament of the Deep Digital Flexor Tendon

Superficial Digital Flexor Tendonitis

Injury to the superficial digital flexor tendon (SDFT) in a hunter/jumper is far less common and severe than in a racehorse, probably because the horse is not trained at high speeds. Injuries are more likely to result from a misstep in deep footing rather than from muscle or tendon fatigue proceeding to failure, but low-grade injuries occur frequently in top-level horses from about 10 years of age. Spontaneous rupture of the SDFT also is seen in old (mid-teens) jumpers. Often no indication of any tendonitis is apparent before rupture. We also have seen several aged horses no longer in competition with spontaneous SDFT rupture. Geldings are overrepresented and there may be an association between this severe trauma and hormonal imbalance.

The diagnosis of superficial digital flexor tendonitis is not difficult, and unlike injury to the DDFT usually is not accompanied by substantial lameness, unless an acute injury occurs in the proximal metacarpal region. Ultrasonographic evaluation is recommended to assess the severity, location, and extent of the injury. Several re-examinations are important to determine the rate of healing and predict the time to return the horse safely to competition.

The therapeutic goals for management of a hunter/jumper with an acutely injured tendon are rapid reduction of the inflammatory response and elimination of edema. This is achieved with the administration of NSAIDs, low doses of corticosteroids, and diuretics. Ice, hydrotherapy, and support bandages also are used and applied as soon as possible, and in

many horses the trainer does these before the veterinary examination. For horses with strains or injury without actual fiber tearing (type 1 to 2 lesions), peritendonous injection of corticosteroids or intralesional injection of hyaluronan may dramatically decrease the size of the tendon and produce favorable cosmetic results. The trainer must be advised not to exercise the horse and that total healing actually may be delayed slightly. Following the injection, the limb should be supported with a firm, modified Robert Jones bandage to prevent the return of any swelling. Tendon splitting is indicated in horses with substantial core lesions (type 3 or 4 with a diameter greater than 0.5 cm). In horses with smaller lesions, injection of hyaluronan intralesionally may reduce inflammation. Therapeutic ultrasound after the initial phase of healing (3 to 4 weeks after injury) encourages resolution of inflammation and promotes healing.

Overall, the time required for tendons to heal sufficiently so the horse may return to jumping depends on the severity of the injury and to some degree the location of the injury. Routine superficial digital flexor tendonitis takes less time to heal than an injury to the SL at the same location. A mild injury takes 3 to 4 months and a severe injury takes about 12 months to heal. Monthly ultrasonographic examinations are used to determine the physical therapy schedule and the optimum time for the horse to return to jumping. Horses often can continue to compete despite active SDFT injuries, although the lesions may progress slowly.

Tenosynovitis of the Digital Flexor Tendon Sheath

SDFT injury within the DFTS frequently is seen in jumpers at any age or level of competition. Tenosynovitis also occurs without tendon injury. Superficial digital flexor tendonitis may result in a chronic tenosynovitis and often plagues older horses. Tenosynovitis is slightly more prevalent in the hindlimb, presumably because of the strain caused by the push off in jumping. The DFTS may be warm and swollen. Digital pressure at the proximal and distal aspects of the DFTS usually is resented. Pressure applied directly over the palmar/plantar aspect of the fetlock also may cause pain. The distal limb flexion test is usually positive. Moderate effusion is usually present in horses with an acute injury, but those with chronic injuries may have a thickened, fibrotic DFTS and much less fluid. The proximal aspect of the sheath may show effusion, yet none may occur distally in the pastern. Lack of effusion likely results from a compartment syndrome with thickening of the DFTS synovium, the SDFT, and possibly the palmar annular ligament, thus preventing the flow of fluid distally past the PSBs. Lameness may vary and may only be evident following flexion of the distal limb. Intrathecal analgesia of the DFTS usually improves lameness. Ultrasonographic examination should include the fetlock and pastern regions and reveals increased fluid, allowing the tendons and a thickened synovium to be evaluated easily. The flexor tendons may appear to be normal.

Therapy for horses with tenosynovitis of the DFTS varies depending on the severity of the problem and the structures involved. Anti-inflammatory therapy consisting of ice, cool water therapy, poultices, NSAIDs, and corticosteroids is effective in the mild injuries. In horses with more severe injuries, intrathecal injection of corticosteroids and hyaluronan is beneficial. Rest is essential. Horses with mild injuries may return to work in 1 to 2 weeks, but those with more severe injuries require more time. Good trimming and shoeing are beneficial. Horses with chronic, unresponsive tenosynovitis may require endoscopic examination or desmotomy of the palmar annular ligament.

Desmitis of the Accessory Ligament of the Deep Digital Flexor Tendons

Desmitis of the ALDDFT is a common problem, especially in older show jumpers, and often results in sudden onset of lameness after landing over a fence, with rapid development of localized swelling. These clinical signs are typical, but ultrasonography is required to determine the location, severity, and extent of the injury and subsequently to monitor healing. Treatment is similar to that for superficial digital flexor tendonitis. A premature return to work may result in recurrent injury and ultimately adhesion formation between the ALDDFT and the SDFT.

CHAPTER • 117

Lameness in the Dressage Horse

Svend E. Kold and Sue J. Dyson

THE SPORT

Dressage is the ultimate athletic challenge in equestrian sports because it combines balance, suppleness, and power in a unique gravity-defying manner. A good horse gives the impression of athletic elegance and expressive animation. The gaits are described using terms such as *balance, suppleness,* and *hindlimb activity.* The first demand is that the horse is completely obedient and has to go wherever the rider wants and carry out movements at his or her request. In doing so the horse has to rely on its rider, to trust and to accept the rider as its superior. The key to the training and development of a dressage horse from the lowest levels to International Grand Prix is gymnastic exercises, with the aim of strengthening the muscles and thereby avoiding injury to joints and tendons associated with an increased workload.

The Federation Equestre Internationale dressage rules state that the object of dressage is the "harmonious development of the physique and ability of the horse." Through the levels of dressage training, the center of gravity of the horse and rider is placed further caudally, obtained by increasing the degree of flexion and loading of the hindlimbs, while at the same time freeing the front end of the horse to create a more airborne, uphill set of movements. This can be obtained only by increasing the power of the hindlimbs, by synchrony in movement between the forelimbs and the hindlimbs, and through the freedom of movement of the back.

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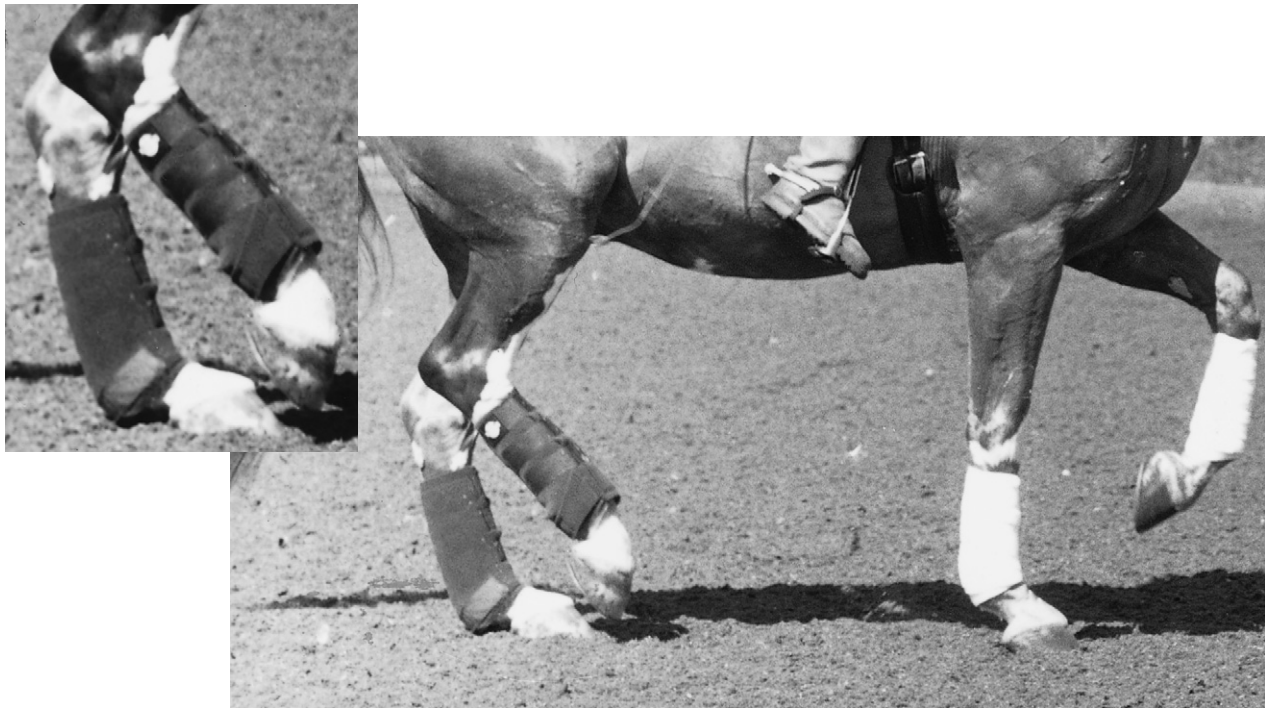

Fig. 117-1 Passage. Note the severe extension of the left metatarsophalangeal joint *(inset)*.

In the German equestrian literature the following terms describe the aims of the correctly trained dressage horse:

- *Takt* (i.e., rhythm)
- *Lossgelassenheit* (looseness and suppleness)
- *Anlehnung* (contact with the bit)
- *Schwung* (energy and swing)
- *Geraderichten* (straightness)
- *Versammlung* (collection)

Dressage is an international sport, although it always has had its main center of excellence in Northern Europe, most particularly Germany, but in later years also in Holland, Denmark, and Sweden. Dressage developed from the military institutes and only in the twentieth century became a truly civilian sport. Even up to the time of the Second World War military officers participated at all the major dressage games.

In Europe the competitive sport has been divided into three levels: L, M, and S. L covers novice level (novice and elementary); M covers medium and advanced medium; and S covers Prix St. Georges, Intermediare I and II, Grand Prix, and Grand Prix Special. The movements required at each of these levels reflects the horse's degree of collection, with the L classes expressing balance and freedom of movement, M classes requiring more collection and lateral movements, and S classes demanding ultimate collection to enable movements of maximum collection and suspension, such as piaffe, passage (Fig. 117-1), and canter pirouettes (Fig. 117-2). However, even the most skilled rider or trainer has difficulty selecting the right horses, because many promising young horses with excellent gaits fail to learn passage and piaffe, probably because of our insufficient knowledge of the biokinematics of collection.[1]

Lateral movements apply specific, unique strains to different structures within the skeleton. In shoulder-in, half pass, renvers, and travers the horse is bent evenly in its neck and body but moves on more than two tracks. In shoulder-in, the horse moves on three tracks (1, outside hindleg; 2, inside hindleg and outside foreleg; and 3, inside foreleg) with the body at an ideal angle of 30° to the direction of movement. In travers (quarters in) and renvers (head to the wall) the horse moves on four tracks. These movements create an unusual strain on the horse's back and an additional twisting movement on the appendicular joints.

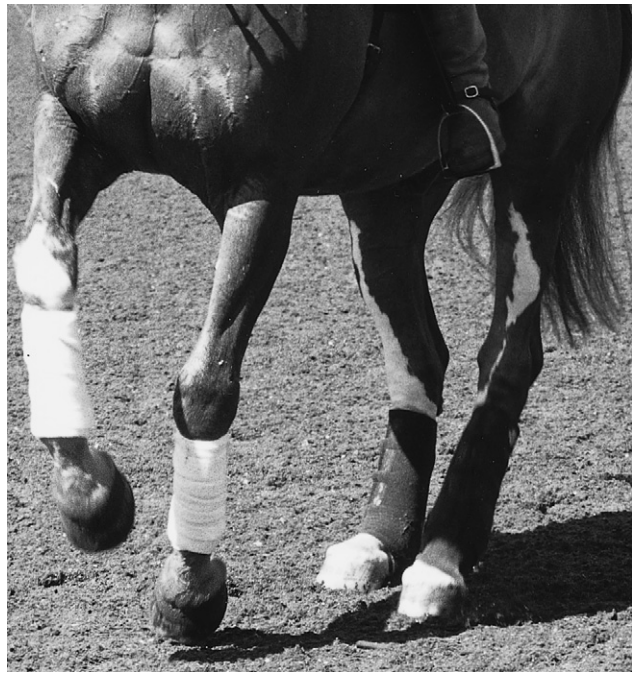

Fig. 117-2 Canter pirouette to the left. The horse intermittently takes all weight on the hindlimbs, resulting in extension of the metatarsophalangeal joints and great strain on the suspensory apparatus.

The increased engagement of the hindlimbs developed through collected work allows for greater storage of elastic

strain energy in the hock joints and pelvis which, via the increased lifting of the forehand, allows for high-energy movements such as medium and extended trot. The term *cadence* is associated closely with working through the back and self-carriage. Self-carriage reflects a level of training in which the horse has learned to balance itself and its rider and additionally has developed its musculature to allow movement with greater range of freedom.

Anlehnung (contact with the bit) is an important concept to understand, requiring the horse to move freely forward with impulsion, to take the bit, and to accept it and react to it without resistance. The Federation Equestre Internationale rules require the horse to work on the bit, that is, with the front of the head positioned in the vertical plane. In recent years the tendency has been toward training dressage horses in an over-bent fashion, with the horse's forehead behind the vertical plane. This is said to be a requirement for developing the trapezius, rhomboidius, and other muscles of the shoulder/withers region and thus enabling a greater lift of the forehand via the shoulder girdle. Although this method of training contradicts the Federation Equestre Internationale requirement for the horse's forehead to be in a vertical plane, top riders are able to place the horse's head in virtually any position according to what is required.

Contact with the bit and *on the bit* frequently are misunderstood terms. The horse must move with energy and impulsion, working through the back for correct contact with the bit. Misinterpretation leads to restriction of the horse by the hands, sometimes resulting in loss of action and gait irregularities.

THE DRESSAGE HORSE

Most dressage horses competing internationally are Warmbloods (WBLs) with a high proportion of Thoroughbred (TB) breeding. Dressage horses today combine the elegance and athleticism of the TBs with the power and trainable mind of the WBLs, which have been selected for many generations for these traits. Few pure TBs reach international standard. The TB has been bred to run fast or show courage jumping obstacles cross-country, which are not of great value when the rider requires complete obedience to perform movements that go as much upward and sideways as forward. Most TBs also lack the strength in all three paces compared with the WBLs, in particular the walk and the trot. Most TBs do not show the same degree of natural engagement of the hindlimbs typical of many WBLs. Many of the greatest TB sires in post-war European dressage breeding (Der Löwe, Velten, and Pik As) have been neither particularly physically impressive nor equipped with more than an average trot. Theoretically, TB stallions in WBL breeding have required a minimum general handicap to ensure that they had been physically and mentally strong enough to stand up to training and race reasonably successfully.

The dressage horse must be naturally well balanced. The head and neck must be set sufficiently high to facilitate working up hill and making easy contact with the bit. The shape of the withers region is important, so that the saddle sits easily in the correct position. The dressage rider spends a lot of time sitting in the saddle in sitting trot; therefore correct weight distribution is critical.

Most dressage horses are broken at 3 or 4 years of age and begin competing in young horse classes at 5 years of age. Medium classes are reached by the age of 7 and many future Grand Prix dressage horses do a small tour at the age of 8 and 9. Once a dressage horse has reached Grand Prix level, the training predominantly involves repetition of movements, maintaining suppleness, and increasing physical power. Thus dressage horses obviously rarely succumb to acute stress-induced traumatic injuries but more likely succumb to repetitive, accumulative subclinical injuries that may surface at irregular intervals. This means that, with the correct training and management, dressage horses can continue to compete at the highest level at an advanced age, often as old as 15 to 20 years. Many of the Lipizzaner stallions at the Spanish Riding School in Vienna are touring and performing adequately well after 20 years of age.

A true link between conformation and soundness is difficult to establish, because what creates an outstanding dressage horse in terms of conformation does not necessarily create a particularly sound dressage horse, and vice versa. However, in a study of 4-year-old Swedish WBL horses, highly significant correlations were found between conformation and movement and between conformation and orthopedic health, whereas no correlation was found between the overall conformation score and competition performance.[2] A series of elite dressage horses had larger hock joint angles and more sloping shoulders than more average horses, whereas good forelimb movements were characterized by a large range of flexion of the elbow and carpal joints during the second half of the swing phase. This is what previously has been referred to incorrectly as shoulder freedom. It is important that a young horse naturally places the hindlimbs well underneath itself, because the approach angle does not seem to be influenced by training.[1]

Wear and tear lesions frequently occur because of a less than ideal joint and limb angulation, but many other factors influence the durability of the horse, including genetic predisposition and less than ideal management conditions before skeletal maturity. The main requirement must be the ability of the horse to balance itself at all paces, because imbalance and asynchrony in movement apply unusual strains on many structures. Holmström[1] found that a large positive diagonal advanced placement (the spatial difference between the contralateral forelimb and hindlimb contacting the ground) correlates with high trot scores and suggests this as an important indicator of the horse's natural balance. The positive diagonal advanced placement does not change with more collection and therefore may become a useful selection criterion. Holmström also found that a group of selected elite horses with high gait scores had significantly larger stride duration, increased hind stance phase duration, and greater diagonal advanced placement than a group of horses with low gait scores.

TRAINING SURFACES

Dressage horses are trained predominantly on artificial surfaces with a high degree of cushion, providing a consistency in the training surface not paralleled in other equestrian sports. All dressage competitions in mainland Europe take place on artificial surfaces, and only in England does dressage at the lower levels (L) still take place on grass. A multitude of artificial surfaces have been developed over the last 20 years. Most are based on silica sand mixed with a variety of rubber and polyvinyl chloride material, together with a binding and dust-limiting agent such as vaseline, which ensures that such surfaces remain frost-free down to –10° C. This standardization of working and competition surfaces unquestionably plays a huge role in the low occurrence of many acute orthopedic problems in the dressage horse. Some trainers, however, consider constant working on ideal surfaces likely to soften the limb structures and recommend that the horses occasionally are jumped or hacked on less ideal surfaces to provide a stimulus for joint, tendon, and ligament adaptation.

Arena maintenance is paramount; drainage is an essential key to a good surface. An adequate drainage system through central and perimeter drains is absolutely essential to maintain

a good arena. Dead corners of deep sand predispose horses to momentary loss of balance and may predispose to the development of lameness. Any sudden change of surface integrity predisposes horses to lameness. Young horses in particular work more easily and confidentially on firmer artificial surfaces, where they can obtain a more confident grip and are less likely to fatigue.

TACK

The horse must be comfortable in its tack if it is going to work optimally. Dressage saddles are designed to position the rider with a deeper seat and with an extended leg position. The surface area over which the weight is distributed must be as large as possible, to avoid focal pressure points. The use of gel pads and layers of numnahs (saddle pads) is not a substitute for good saddle fit. The saddle must fit the horse and the rider and must position the rider appropriately in balance. The fit must be assessed with and without a rider. The shape of the horse's back musculature may change as the horse develops muscular strength and power; therefore a previously well-fitting saddle may become constricting.

Acceptance of the bit is crucial in the dressage horse. Horses vary considerably in the shape of the mouth and the sensitivity of the corners of the lips, bars, and tongue. Great variation also exists in the thickness of the tongue between horses. A slight crack in the corner of the mouth, caused by an inappropriate bit, can cause major problems with proper acceptance of the bit and the horse's willingness to work straight. At S level, horses have to compete wearing a double bridle, that is, the mouth has to accommodate both bradoon (snaffle) and curb bits. These vary greatly in shape and design, and selection of the most appropriate can be critical.

LAMENESS EXAMINATION

Examining the lame dressage horse does not differ in any great detail from examining any other equine athlete. However, examination frequently requires more time being spent observing the horse ridden, because many dressage horses only reproduce the perceived problem, often no more than a resistance, when ridden through certain movements. This, however, does not mean that the horse should not be examined in hand, including walking and trotting on a straight line and lunging on hard, non-slip surfaces (such as gravel) and on softer artificial surfaces. Not only does lunging on tarmac or concrete carry the risk of the horse slipping, with potentially disastrous consequences, but also in most horses such lunging alters the gait so much that it has little value in a lameness examination of an extravagant moving dressage horse. Leading the horse on a circle at a trot also tends to alter the horse's stride. The horse does not have the freedom to move its neck and instead will set its head on the leader's hand.

In many horses the usual rider has to be available to reproduce the described problem, if lameness is not overt. However, one should remember that just as bad riders create lameness, so good riders may hide lameness. The latter may take place completely unintentionally and may involve no more than a corrective change of point of balance of the rider through a corner, but enough that for a long time the problem may not be observable from the ground. Most veterinarians who are not competent riders are not experienced fully to appreciate the subtle differences in high-quality dressage horses and by attempting to ride the horse to better appreciate the problem may create an embarrassing situation. They are better advised to spend more time observing the horse from the ground.

The veterinarian should not just focus on the limbs when watching the horse ridden. It is important to observe changes such as an increased lathering of the mouth, audible change in the rhythm of the stride, or even absence of teeth grinding or grunting after a particular diagnostic test.

For many horses the veterinarian relies heavily on the conceived observations of the rider during the lameness examination; this may involve the appreciation of subtle change of gait, or even just an impression of a stronger rhythm or less heavy contact on the bit after a peripheral nerve block. Many riders feel through their own body that the horse is working crookedly, that is, not straight and is not in complete balance, and will be able to tell the clinician if this feeling has been altered by any of the diagnostic tests.

In many horses, alternating between lunging and ridden work is useful, often going back to lunging with full tack after the horse has been ridden to see a possible difference in the gait from being ridden.

A useful test is to ask the rider to ride deliberately on the wrong diagonal, that is, to sit to the trot in the saddle when the inside forelimb is bearing weight. Horses with forelimb or hindlimb lamenesses and horses suffering from back pain may alter the gait when the weight-bearing diagonal (of the horse) is changed. The difference between the horse's outline and attitude when changing between sitting and rising trot also may add valuable information.

In some horses lameness is *created* by the rider. This most commonly occurs with amateur riders who misunderstand the principles of creating an outline and working the horse forward to the bit. Over-restriction by the hands, with inadequate impulsion, can create gait irregularities. Lower-level trainers are sometimes unable to appreciate these problems and may themselves be unable to work the horse better. Using a good professional rider who is not the trainer to work the horse is therefore preferable. Determining definitively whether the problem is one of riding or of training or a reflection of a genuine lameness may require several days. A rider who sits consistently crookedly can create back pain and loss of hindlimb rhythm and symmetry. Some dressage horses are exuberant and expressive movers and also strong-willed characters that may refuse to go forward properly if ridden by an enthusiastic but less competent amateur rider, especially if the rider is somewhat apprehensive and inclined to be overrestrictive. Nappy (resistant) behavior and unwillingness to work may reflect a pain-related problem, but not necessarily so.

One should remember that not all horses are athletes. Many owners tend to think that all horses can learn to do dressage. Veterinarians must in certain situations be prepared to offer the opinion that the particular horse has too many shortcomings physically or psychologically to be able to perform advanced dressage. A veterinarian may be able to help a horse overcome a specific problem but cannot provide missing athleticism.

The veterinarian should not forget to check the obvious. Dressage horses physically alter during a training lesson. As the muscles over the withers and shoulders expand, a particular saddle that may appear to fit correctly before working the horse may be restricting an hour later, when the horse begins piaffe and passage movements. It is also important to check that the bit is the correct size and is placed correctly in the horse's mouth. Horses' tongues vary greatly in size and when using two bits some adjustments may be required. The corners of the mouth are easily cracked and can become sore. The horse may be apprehensive about taking the bit, may take irregular steps, or may be reluctant to bend properly. Wolf teeth frequently are blamed for reluctance to accept the bit properly and for irregularities in gait. Provided that a wolf tooth is immediately in front of the first upper cheek tooth and is not mobile, the tooth rarely is associated with pain.

Horses with a short poll and a relatively large mandible have difficulty in acquiring the correct degree of neck flexion. In these horses it is also important to check that air flow is not impaired. Restricted airflow is not necessarily accompanied by an audibly abnormal inspiratory and/or expiratory noise.

If a diagnosis cannot be made because clinical signs are too subtle, or if it is difficult to determine whether the presenting clinical problem is pain related, working the horse while treating it with anti-inflammatory medication (2 to 4 g phenylbutazone/day PO) for 2 to 3 weeks may be useful. If lameness returns once the medication is withdrawn, the performance problem can be attributed to pain. The lameness also may be worse, making further investigation easier. If the horse appears to have a low-grade bilateral problem, starting by blocking one limb (front or hind) to see if a contralateral limb lameness subsequently becomes obvious can be useful. This is sometimes misleading, however, and blocking both limbs simultaneously and then re-assessing the overall freedom of movement and balance may be more valuable.

DIAGNOSTIC ANALGESIA

Technically no differences exist between dressage horses and other equine athletes concerning diagnostic analgesia. However, the horse's response should be assessed when ridden and when trotted in hand. Because only minor irregularities in gait are often the point of investigation in lame dressage horses, it is particularly important that the conditions, including the surface, remain consistent throughout the lameness investigation. Starting the investigation on one surface only to find that halfway through the nerve blocks the horse has to be assessed on a different surface is not possible. Indoor arenas obviously are of great assistance in severe winter conditions.

Although the sequence of the nerve blocks in theory should be the same in all equine athletes, known common lameness sites in dressage horses often make focusing on these areas possible to save time and to avoid an unnecessary number of injection sites and undesirable number of clipped sites. If clipping is essential, many riders prefer the entire limb (and contralateral limb) clipped symmetrically rather than many small clipped sites in one limb.

IMAGING CONSIDERATIONS

Imaging of the lame dressage horse is no different from imaging any other equine athlete. However, the frequent lack of overt lameness in a sub-maximally performing dressage horse often makes including every possible diagnostic modality necessary. This is particularly true for evaluation of the neck and the back, both of which are important structures for balance and coordination. These are areas where diagnostic analgesia is less applicable than in the limbs.

Radiographic evaluation of the thoracolumbar region requires fixed or semi-mobile radiographic equipment. Use of a Dodger-T aluminum wedge filter to attenuate the primary x-ray beam facilitates acquisition of high-quality images of the dorsal spinous processes.[3]

Diagnostic ultrasonography of the thoracolumbar and pelvic regions is also useful, used transcutaneously or per rectum to image the supraspinous ligament and epaxial musculature, the synovial articulations (facet joints), and the ventral aspect of the lumbosacral junction.[4]

Nuclear scintigraphic evaluation can be particularly helpful in evaluating the thoracolumbar and pelvic regions. However, no studies have been done on the variation in radiopharmaceutical uptake in a population of normal dressage horses. Such a study may throw light on subclinical orthopedic problems in dressage horses. However, as with all imaging techniques great scientific integrity is demanded to distinguish between normal variations and pathological lesions, and results must be correlated carefully with clinical observations and with other imaging modalities.

Computerized thermographic image analysis may be helpful. Thermography is a non-invasive physiological imaging technique that may detect pathological changes before structural changes are apparent. Thermography is also capable of identifying more than one area of altered tissue metabolism, which may reflect a secondary area of pain. Thermographic data does not lie, but interpretation of data is challenging.

TEN MOST COMMON LAMENESS CONDITIONS

The 10 most common lameness conditions in dressage horses are listed not necessarily in strictly decreasing order of importance or frequency, but they represent a selection of the most commonly encountered lameness problems.

1. Proximal suspensory desmitis (PSD)
   a. Hindlimbs
   b. Forelimbs
2. Suspensory branch lesions
3. Synovitis or osteoarthritis of the forelimb distal interphalangeal joints
4. Desmitis of the forelimb accessory ligament of the deep digital flexor tendon (ALDDFT)
5. Osteoarthritis of the centrodistal or tarsometatarsal joints
6. Synovitis of the middle carpal joint (possibly with palmar intercarpal desmitis)
7. Synovitis or osteoarthritis of the metacarpophalangeal and metatarsophalangeal joints
8. a. Palmar/plantar annular desmitis
   b. Tenosynovitis of the digital flexor tendon sheath: forelimbs and hindlimbs
9. Palmar cortical stress fracture of the third metacarpal bone
10. Thoracolumbar and sacroiliac pain

Proximal Suspensory Desmitis: Hindlimbs

Probably the most important cause of lameness in dressage horses working at medium and advanced levels is PSD. The carrying capacity of the hindlimbs is increased with increased collection required for more advanced work, and movements such as piaffe, passage, and canter pirouettes (see Fig. 117-2) place great strain on the hindlimb suspensory apparatus. Suspensory desmitis is believed to be caused by an accumulation of repetitive strains within the suspensory ligament (SL) and its proximal origin. Detection of PSD is often delayed because of its bilateral nature, which often means that overt hindlimb lameness is not present or noticed by the rider. Accurate diagnostic nerve blocks therefore are required to reveal lameness. When no obvious lameness is present, lameness often is created subsequently in the contralateral hindlimb by diagnostic analgesia of either of the (non-lame) hindlimbs. A negative response can be misleading, and it is sometimes necessary to block both hindlimbs simultaneously, after which there may be a substantial improvement in gait. Direct palpation of the region often fails to indicate a problem because of the deep location of the proximal SL. The clinical diagnosis is confirmed by positive subtarsal analgesia, together with negative intra-articular analgesia of the tarsometatarsal joint.

Extensive ultrasonographic and radiographic changes often reflect a chronic and long-standing problem. Ultrasonographic images reveal enlargement of the proximal SL in the lateromedial and dorsoplantar planes, with areas of reduced echogenicity, often involving one or both of the dorsal quadrants of the

ligament. Radiographic diagnosis requires high-quality radiographs of the proximal metatarsal region; dorsoplantar and lateromedial views are the most useful. Irregularity of the proximal plantar cortex may occur in the region of insertion of the SL origin, with a varying degree of endosteal new bone resulting in trabecular sclerosis over a distance of up to 5 cm. On dorsoplantar radiographs this may be seen as a centrally positioned, triangular-shaped area of sclerosis within the trabecular metaphysis of the third metatarsal bone (MtIII). One should remember, however, that such radiographic changes may be present in an asymptomatic horse because of previous problems, leading to the risk of a false-positive diagnosis. Diagnosis should never be based only on radiographic examination.

Occasionally, nuclear scintigraphy can be useful in horses with early desmitis where no radiographic changes are present and subtle, equivocal abnormalities are detected with ultrasonography. Increased radiopharmaceutical uptake may occur in the proximoplantar aspect of MtIII in bone-phase (delayed) images. Pool-phase (soft tissue) images are rather insensitive, and not all horses have associated increased radiopharmaceutical uptake in MtIII. Thus a negative bone scan does not preclude PSD.

Treatment of this condition often is frustrating because of the chronic nature of the problem at the time of detection. Corrective shoeing using egg bar shoes provides some support in horses with severe hyperextension of the fetlock joint. Prolonged rest (3 to 6 months) often provides a disappointing response, and a controlled exercise program may be more successful. Periligamentous injection of 2 ml of hyaluronan (Hyonate, Bayer AG, Leverkusen, WG) plus a corticosteroid (e.g., 10 to 20 mg of methylprednisolone [Depo-Medron, Upjohn, Crawley, UK] or 10 mg of triamcinolone acetonide [Adcortyl, Squibb & Son, Hounslow, UK]) are now used frequently and are believed to provide an initial reduction in inflammation to enable a reasonably pain-free walking program to be initiated. This frequently is continued for as long as 12 weeks before slow, balanced trotting on a good surface is initiated.

The ultrasonographic appearance of the SL often changes little, even in horses that are returned successfully to full training. Egg bar shoes often are removed when normal training is initiated, because many riders believe that they provide too much breaking action on ground impact of the hindlimbs. The likelihood of recurrence of PSD is high, and special care should be taken not to over-work the horse on deep or holding surfaces. Modification of the training program often is required, particularly in terms of avoiding fatiguing training session in deep or loose surfaces.

Some horses have chronic lameness that fails to respond to therapy. Recently some encouraging results have been achieved by treatment of horses with acute and chronic desmitis with shock wave therapy, using three treatments at 2-week intervals, with substantial improvement in lameness and ultrasonographic appearance of the ligament. Neurectomy has a role in horses that fail to respond to medical management.

Proximal Suspensory Desmitis: Forelimbs

Forelimb PSD is seen more often than hindlimb PSD in younger horses and may result from hyperextension of the carpus in extravagantly moving horses, in particular horses volunteering extended trot. PSD often results from the horse working on less than ideal surfaces. Lameness is often unilateral and acute in onset but is sometimes bilateral. Most sound horses resent firm manual squeezing of the body of the SL, and the proximal region of the SL is difficult to access by palpation; therefore local analgesic techniques are required to verify the source of pain.

In horses with peracute PSD, slight filling in the proximal metacarpal region may occur, but this often resolves within 24 hours. Lameness may be transient unless the horse is worked again. In these horses, the veterinarian relies on a history of acute-onset lameness that is worse with the affected limb on the outside of a circle for diagnosis. Lameness is often easier to feel (by a rider) than to see.

Some improvement is often seen after perineural analgesia of the palmar and palmar metacarpal nerves at mid-cannon level. Lameness is substantially improved or resolved by blocking the lateral palmar nerve or the palmar metacarpal nerves at the subcarpal level. Because of the close proximity of the middle carpal joint capsule, positive subcarpal analgesia should be followed by intra-articular analgesia of the middle carpal joint to exclude articular pain.

Radiography is frequently of little or no value in dressage horses with forelimb PSD. Ultrasonography is required to confirm the diagnosis. Because subtle lesions may be present and may be a reflection of a previous pathological condition (possibly subclinical), high-quality ultrasonographic images of both limbs are required for comparison, together with knowledge of normal variations. Lesions vary from subtle enlargement of the proximal aspect of the SL with normal echogenicity to large areas of reduced echogenicity.

Rest, often for 12 to 16 weeks, together with a controlled, ascending walking program, in hand using a horse walker or ridden, is the treatment of choice. Intralesional injection using 2 ml of a polysulfated glycosaminoglycan (PSGAG [Adequan, Janssen Pharmaceutical, High Wycombe, UK]) may be tried.

The prognosis is good in horses with early PSD, provided that the horse is managed carefully subsequently, avoiding the medium or extended trot in training. In horses with chronic PSD the risk of recurrence is moderately high. Shock wave therapy has been useful in treating some of these horses. In some horses, ultrasonographic appearance may not change substantially, despite a favorable clinical response to treatment, leading to a risk later of false-positive diagnosis in an asymptomatic horse.

Desmitis of the Suspensory Ligament Branches: Forelimbs and Hindlimbs

Desmitis of a branch of the SL is often acute in onset and results in palpable enlargement of the suspensory branch and often moderate lameness. Occasionally, both medial and lateral branches are involved in a hindlimb. The branch is painful to palpation. Diagnosis is confirmed by ultrasonography. Enlargement of the branch is often accompanied by some subcutaneous echogenic material, often with a central (sometimes eccentric) hypoechogenic or anechogenic core lesion. The interface between the ligament branch and the proximal sesamoid bone may be disrupted. This is best seen on longitudinal images and merits a more guarded prognosis. If both branches in a hindlimb are involved, echogenic material may be visible between the branches. The PSBs may or may not show radiographic evidence of enthesopathy, with linear opacities extending from the palmar/abaxial margin. Ectopic mineralization within the SL branch is occasionally seen with ultrasonography and radiography.

Treatment is prolonged rest (4 to 6 months) with a slow return to exercise. Intralesional injections using 1 ml of a PSGAG may be tried but generally have met with disappointing results. Counterirritation (pin firing) has been tried on old, indurated lesions and chronic lameness, but again with disappointing results. Some horses have responded to intralesional treatment with β-aminoproprionitrile fumarate. Encouraging results also have been obtained recently with shock wave therapy (three treatments at 2-week intervals), combined with a controlled walking exercise program for 3 to 4 months.

The risk of recurrence is high. Any predisposing causes such as limb deviations or lateromedial imbalance of the feet should be corrected or at least adjusted.

Synovitis or Osteoarthritis of the Distal Interphalangeal Joint

Pain arising from the distal interphalangeal joint is a frequent diagnosis in all equine sports. The unique anatomical position of the distal interphalangeal joint, with the forces distributed on it through the rigidity of the hoof capsule and the forward thrust of the deep digital flexor tendon and the navicular bone during weight bearing and limb protraction, are likely to be contributory factors. Lateromedial imbalance of the foot contributes to the joint trauma in many horses.

The diagnosis is made from comparing the response to intra-articular analgesia with perineural analgesia of the palmar digital and palmar (abaxial sesamoid) nerves and occasionally intrasynovial analgesia of the navicular bursa. The interpretation of the response to intra-articular analgesia is not black or white. Recent studies have confirmed that intra-articular analgesia of the distal interphalangeal joint is not specific and can influence pain associated with the navicular bone, distal phalanx, and subsolar tissues. A rapid (within 5 minutes), positive response to a small volume (maximum of 6 ml) of local anesthetic solution may be a good indicator of the possible response to subsequent intra-articular medication. A good response to subsequent treatment requires at least 75% clinical improvement after intra-articular analgesia, together with absence of significant radiographic changes involving the distal interphalangeal joint or the navicular bone.

Radiographic changes usually involve the extensor process of the distal phalanx and the dorsoproximal margin of the navicular bone. One should remember that a considerable shape variation exists in the extensor process within sound horses. Minor modeling changes may not be of clinical significance. Small, mineralized fragments proximal to the extensor process may be seen incidentally. Large fragments may require surgical removal to prevent secondary osteoarthritis.

Intra-articular medication using a number of individual drugs or combination of drugs has given encouraging results, although results vary between clinicians and populations of horses. Horses with recent onset of lameness with palpable distention of the distal interphalangeal joint capsule may respond favorably to 10 mg of triamcinolone acetonide or 10 to 20 mg of methylprednisolone acetate, combined with a short period of controlled walking exercise (e.g., 2 weeks of ridden walk or on a horse walker). Injection with hyaluronan probably will provide a similar response in such horses. The most successful long-term results have been seen after a triple series of intra-articular injections of a PSGAG. PSGAGs received some negative press after a research study in North America in 1989 that suggested that PSGAGs have a potentiating effect on a subinfectious dose of *Staphylococcus aureus* in a joint. Subsequently, this preparation has been used sparingly intra-articularly and in many horses only in combination with intra-articular amikacin. We have not seen any negative reaction (inflammatory or infectious) to multiple intra-articular joint injections of PSGAGs and do not use systemic or intra-articular antibiotics. Strict asepsis and a skillful technique are essential. Because of its distal location in the limb, distal interphalangeal joint should always be bandaged for at least 24 hours after injection. Eighty-two percent of dressage horses returned to soundness after PSGAG medication of the distal interphalangeal joint, whereas only 65% of a similar size group of horses competing in cross-country jumping returned to soundness.[5] In most horses the initial triple injection proved adequate, and re-medicating the joint in successfully treated horses was rare. Corrective shoeing by improving the foot-pastern axis and re-establishment of correct hoof balance should be performed with intra-articular medication. Egg bar shoes often are used for an initial 3- to 6-month period.

Desmitis of the Accessory Ligament of the Deep Digital Flexor Tendon: Forelimbs

Desmitis of the ALDDFT frequently occurs as an acute injury associated with sudden onset of lameness and palpable inflammation (heat and swelling) in the proximal metacarpal region. Over-extension of the carpus, because of imbalance in young horses or resulting from imperfect working surfaces, often is believed to be a contributory cause.

The diagnosis is confirmed by ultrasonography in both transverse and longitudinal planes. There is enlargement of the ALDDFT, together with loss of definition of the margins and areas of reduced echogenicity. A definite, hypoechoic core lesion is recognized infrequently.

Horses with desmitis of the ALDDFT respond better to a controlled walking exercise program than to complete box rest. Three to 6 months of controlled walking often is required in horses with severe desmitis. The risk of recurrence is moderately high. Local invasive treatment of desmitis of the ALDDFT seldom has been rewarding, although some horses have responded favorably to treatment with β-aminoproprionitrile fumarate.

Osteoarthritis of the Centrodistal and Tarsometatarsal Joints

Lameness or poor performance (e.g., inability to perform piaffe or passage) associated with pain arising from the centrodistal or tarsometatarsal joints occurs frequently in dressage horses. Likewise, a horse with an outstanding freedom of movement in the trot may have an unexplainably poor canter associated with distal distal joint pain. A poor correlation exists between the clinical signs, including response to intra-articular analgesia of the tarsometatarsal and centrodistal joints, and the radiographic appearance of these joints. Many dressage horses have confirmed pain from the joints but have fairly equivocal radiographic changes. One should remember that the degree of joint collapse and osseous ankylosis always is underestimated from conventional radiographs because of the curvilinear nature of the joints and the low radiodensity of immature bone bridging the joint centrally. Nuclear scintigraphy may be a sensitive indicator of increased bone modeling in the absence of radiological abnormalities or in horses with equivocal changes and also can be helpful in horses with subtle lameness, when the response to local analgesic techniques is difficult to interpret. Alternatively, diagnostic medication with intra-articular corticosteroids may be helpful.

Pain is often bilateral, and often the presenting problem is shortening of the hindlimb stride and an inability to collect. Clinical signs mimicking back pain, rather than overt lameness, may be present.

Most horses respond to intra-articular analgesia of the tarsometatarsal and centrodistal joints, although occasionally a better response is seen after fibular or tibial nerve blocks or after treatment of the joints. Intra-articular analgesia of the tarsometatarsal joint and the response to subtarsal analgesia should be compared. In horses with advanced radiological changes, false-negative responses to intra-articular analgesia may occur.

Horses with few or no radiographic changes may respond satisfactorily to intra-articular medication with a corticosteroid (e.g., 10 to 20 mg of methylprednisolone acetate or 10 mg of triamcinolone acetonide), and/or 1 ml of a PSGAG if lameness is of longer duration. Medical treatment often is combined with a program of controlled walking exercise for 4 to 6 weeks (pending response to treatment), together with alterations of the shoeing. Horses that tend to plait or swing the affected limb axially during protraction may benefit from a lateral extension shoe, which may help to normalize posture and hindlimb gait pattern with an additional effect on secondary back pain.

Repeated medication of the joint(s) may be required at monthly or quarterly intervals. A longer response may be achieved by combined use of corticosteroids and hyaluronan.

Additional PSGAG medication given intramuscularly on a weekly basis (500 mg/ml, 7 × 5 ml) may be beneficial. Glucosamines and chondroitin sulfate given orally on a daily basis often are administered, but clinical efficacy is unproven.

If substantial radiographic changes are present and the joint is changed irreversibly and the horse fails to respond to repeated intra-articular medication, fusion of the joints by surgery or by intra-articular injection of sodium monoiodoacetate can be considered, but the prognosis for high-level dressage is guarded.

Synovitis of the Middle Carpal Joint

Lameness associated with pain in the middle carpal joint has been seen in many dressage horses, in particular young horses that still may be struggling to establish balance and synchronicity in all paces with the additional weight of the rider. Lameness is often mild and most frequently unilateral, being most noticeable when the limb is on the outside of a 10-m diameter circle. Momentary hyperextension of the carpus, often on a tight circle, is believed to be involved.

If the response to analgesia of the middle carpal joint is positive, the proximal SL should be examined by ultrasonography to preclude injury.

Usually no radiographic or ultrasonographic changes involve the middle carpal joint. Arthroscopy of the middle carpal joint sometimes reveals damage to one or both (most frequently the medial) of the palmar intercarpal ligaments, which show edema, petechial hemorrhage, and fraying of superficial fibers.

Most horses respond well to intra-articular medication using a triple series of 1 to 2 ml of a PSGAG given 8 days apart, or treatment with short-acting corticosteroids combined with hyaluronan. Six to 8 weeks of ridden walk or on a horse walker should be followed by a modified training program for at least another 3 months.

Synovitis or Osteoarthritis of the Metatarsophalangeal and Metacarpophalangeal (Fetlock) Joints

Fetlock joint disease is not a common problem in dressage horses and certainly does not seem to be as common in this type of equestrian sport as in others. The absence of galloping across often firm and irregular surfaces associated with cross-country jumping is a likely explanation.

The diagnosis is confirmed by palpation and intra-articular analgesia. In the absence of radiographic changes, intra-articular medication using a corticosteroid (e.g., 10 mg of triamcinolone acetonide), together with 2 ml hyaluronan in horses with synovitis, or 1 ml of PSGAG in horses with more long-standing osteoarthritis, frequently has proved successful. Generally the response to medication is good and the likelihood of recurrence is low in the forelimbs. The response is poorer in hindlimbs. Horses with radiographic abnormalities consistent with osteoarthritis warrant a more guarded prognosis.

Palmar/Plantar Annular Desmitis

Desmitis of the palmar (plantar) annular ligament (PAL) occurs more commonly in forelimbs than in hindlimbs and usually results in acute-onset lameness. The PAL has localized heat and is palpably enlarged, with pain elicited by firm pressure. Mild distention of the digital flexor tendon sheath (DFTS) may occur. Diagnosis is confirmed by ultrasonography. The PAL is thickened, with a diffuse reduction in echogenicity or focal hypoechoic areas. Horses with acute desmitis usually respond well to box rest and controlled walking exercise for 3 months. In the acute phase, non-steroidal anti-inflammatory drugs (e.g., phenylbutazone 2 g bid PO for 5 days) are beneficial.

Tenosynovitis of the Digital Flexor Tendon Sheath

Tenosynovitis of the DFTS often results in sudden-onset lameness associated with distention. Some horses have long-standing distention of the DFTS without lameness (windgalls), especially in the hindlimbs, but subsequently develop clinically important tenosynovitis. Constriction of the DFTS by the PAL may be apparent. Lameness may vary from mild to severe and usually is accentuated by distal limb flexion. If distention of the DFTS is acute in onset, then local analgesia is usually unnecessary. However, if distention of the DFTS has been present for some time, then the source of pain should be confirmed by local analgesia. Intrathecal analgesia of the DFTS usually results in substantial improvement in lameness but often does not alleviate it fully. Perineural analgesia of the palmar (plantar) (mid-cannon) and palmar metacarpal (metatarsal) nerves usually eliminates lameness. Improvement sometimes is seen after perineural analgesia of the palmar nerves at the level of the PSBs. Excluding the metacarpophalangeal joint as a potential source of pain by intra-articular analgesia may be necessary.

Ultrasonographic examination should be performed in the metacarpal and pastern regions. Usually an abnormal amount of fluid within the DFTS allows better visibility of synovial plicae extending from the medial and lateral margins of the deep digital flexor tendon (DDFT) in the distal metacarpal region and the synovial fold on the palmar aspect of the DDFT in the pastern region. These should not be confused as adhesions or tears of the DDFT. In horses with chronic tenosynovitis, the DFTS wall may be thickened, with echogenic bands within the DFTS representing adhesions. Ultrasonography frequently underestimates adhesion formation. The SDFT and DDFT should be inspected carefully, because tenosynovitis may be secondary to a primary pathological tendon condition. Lesions of the DDFT occur more commonly, either as core lesions or marginal tears. The latter can be difficult to detect by ultrasonography. Enlargement of the cross-sectional area of the tendon compared with the contralateral limb suggests a lesion, which may be confirmed only by tensocopic evaluation of the sheath contents. Mineralization within the DDFT warrants a guarded prognosis.

Horses with early tenosynovitis without adhesions respond well to intrasynovial administration of a corticosteroid (e.g., 10 mg of triamcinolone acetonide) or 2 ml of hyaluronan, together with a pressure bandage and box rest with hand walking for 4 to 6 weeks.

If the horse fails to respond adequately to medical therapy, then surgical exploration is warranted. Tenoscopic evaluation should be performed to evaluate the extent of adhesion formation and to detect longitudinal tears in the medial or lateral margins of the DDFT, which may extend proximally under the manica flexoria. Resection of intrasynovial adhesions and lavage, with or without resection of the palmar/plantar annular ligament, may resolve the problem. Horses with lesions of the DDFT that may be debrided and possibly sutured warrant a more guarded prognosis. Surgery is frequently followed by intrasynovial injection of 2 ml of hyaluronan, repeated 4 to 6 weeks later to reduce inflammation and to try to prevent adhesions reforming.

The response to surgery depends on the chronicity of the problem, the number of intrasynovial adhesions, and the presence of lesions of the DDFT. Horses with lesions involving the forelimbs appear to have a better prognosis than those involving the hindlimbs. Surgery is accompanied by 8 to 12 weeks of absence from training, although controlled walking in hand or on a horse walker is essential to try to stretch any adhesions that reform.

Proximal Palmar Cortical Stress Fracture of the Third Metacarpal Bone

Proximal palmar cortical stress fracture of the third metacarpal bone occasionally is seen in dressage horses with an acute onset of moderate to severe lameness. Hyperextension of the carpus and imbalance of limb synchrony in immature horses are believed to be contributing factors.

The diagnosis sometimes can be suspected by inducing pain by digital pressure on the palmaroproximal aspect of the third metacarpal bone. When the horse trots on a firm surface, the lameness tends to increase the farther the horse trots. If the horse turns and then trots again, lameness appears to be improved and then increases again. Lameness usually is improved substantially by perineural analgesia of the palmar metacarpal nerves. If the fracture extends into the carpometacarpal joint, improvement also may be seen after intra-articular analgesia of the middle carpal joint.

On radiographs a fracture may be recognized on a dorsopalmar projection as a linear radiolucent line, usually in the medial aspect of the third metacarpal bone and extending up to 8 cm, possibly with surrounding sclerosis. In some horses a fracture line cannot be seen, although sclerosis is present. In some horses no radiographic abnormality is identified. In these horses diagnosis is confirmed by nuclear scintigraphy.

Treatment is complete box rest for 6 weeks, followed by 6 weeks of controlled walking exercise. The prognosis is good, and the likelihood of recurrence low.

Thoracolumbar and Sacroiliac Pain

Thoracolumbar and sacroiliac region pain are frequent causes of reduced performance in the dressage horse. Although caudal thoracic back muscle soreness may be secondary to primary hindlimb lameness, primary back pain does occur and usually is not associated with overt lameness. The horse may have a history of unwillingness to perform certain movements, stiffness, loss of impulsion and cadence, loss of action or being less easy to work in a correct outline, and sometimes bucking or other nappy (resistant) behavior. The horse usually moves better on the lunge than when being ridden. The most common causes of back pain include the following:

1. An ill-fitting saddle
2. The rider sitting crookedly
3. Primary muscle spasm
4. Impingement of dorsal spinous processes in the mid-thoracic to cranial lumbar regions
5. Osteoarthritic changes involving the synovial articulations (facet joints) in the region of the thoracolumbar junction
6. Sacroiliac disease
7. A combination of lesions in the thoracolumbar and sacroiliac regions

Definitive diagnosis of the cause of back pain can be a diagnostic challenge. Obvious causes such as ill-fitting saddle or the position of the rider should be eliminated first before proceeding with more sophisticated diagnostic tests such as nuclear scintigraphy, radiography, thermography, and ultrasonography. If back muscle tension or spasm is obvious, assessing the response to treatment using physiotherapy (e.g., manipulation and therapeutic ultrasound) or anti-inflammatory medication may be worthwhile before further investigation.

Thermography provides pictorial images of the surface temperature of the body, which gives a physiological identification of changes in tissue perfusion or sympathetic neuromuscular dysfunction. Thermography is a useful tool to demonstrate to an owner the effect of a poorly fitting saddle or the rider sitting crookedly and also may be useful in identifying acute superficial ligamentous or muscle injuries.

Nuclear scintigraphy is much more sensitive than radiography for detecting lesions in the thoracolumbar or sacroiliac regions, but radiography potentially gives more structural information, such as the proximity of the dorsal spinous processes. It is important to verify the clinical significance of any lesions identified by infiltration of local anesthetic solution whenever possible. If dorsal spinous processes are extremely crowded, injecting between them may not be possible, but injecting around them (20 to 50 ml of mepivacaine) usually results in substantial improvement within 15 minutes of injection. Ultrasound guidance is necessary for infiltration around synovial articulations.

Treatment is aimed at removing any predisposing factor and control of pain. Local infiltration with corticosteroids (e.g., methylprednisolone acetate combined with mepivacaine) or Sarapin is successful in some horses. If pain associated with impinging dorsal spinous processes fails to be controlled by medical management, then surgical treatment should be considered. Infiltration of a sclerosing agent, P2G (Martindale Pharmaceuticals, Ramford, Exxes, UK), around the sacroiliac joints produces clinical improvement in some horses with pain associated with these joints.

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CHAPTER • 118

Lameness in the Three Day Event Horse

Andrew P. Bathe

SPORT OF EVENTING

The sport of eventing generally is considered the most all-round test of a horse's athletic ability, and as such the horses tend to be jacks of all trades rather than excelling in any one particular area. The competition consists of three disciplines, namely dressage, show jumping, and cross-country, with the latter being the most influential phase. The Three Day Event or Concours Complet is the pinnacle of the sport (Fig. 118-1) and is run over 4 days: 2 days of dressage, followed by a day for the speed and endurance phases and a day of show jumping. The speed and endurance test consists of roads and tracks at trotting speed, a steeplechase, and the actual cross-country phase over fixed obstacles. This is a severe athletic test, and horses normally only compete in two or maximally three such competitions in a year.

Fig. 118-1 Example of a cross-country fence at CCI**** level. (Courtesy Kit Houghton Photography, Bridgewater, Somerset, UK.)

One Day Events, or Horse Trials, compress the same disciplines into a shorter time frame, but without the roads and tracks and steeplechase phases. The distance and thus time for the cross-country are also much shorter, so that many such competitions may be completed in a season. For most advanced horses One Day Events are used as training for the Three Day Events, although a large number of recreational competitors only aspire to compete in One Day Events. The emphasis of this chapter is the Three Day Event horse, because the extreme demands placed on this horse give characteristic patterns of lameness; whereas the novice One Day Event horse can be considered a standard riding club or recreational horse from a veterinary point of view.

Eventing is an established Olympic sport and a substantial equine industry. The highest grade of competition is the four-star event (CCI****). Traditionally these have been the English events of Badminton and Burghley, although events of this level now have been established in Lexington, Kentucky, in the United States and in Adelaide, Australia. The sport has a high-risk element for rider and horse and a noticeable recent trend has been to try to make courses safer.

The sport includes substantial veterinary involvement. During a Three Day Event, the horse is examined by the official veterinarians on arrival, the day before commencing the dressage, before and after the cross country, and in the morning before show jumping. The horse must be deemed fit to compete—that is, the horse must be sound—throughout the competition, and thus any orthopedic disorders are of great significance. A mild degree of hindlimb gait asymmetry may be acceptable to the Inspection Panel, but any noticeable forelimb lameness normally is not permitted.

HORSE TYPES

Because the Three Day Event places great emphasis on the horse's speed and stamina, there is a preference toward the Thoroughbred (TB) or predominantly TB-cross breeds. A substantial proportion of horses are of uncertain or unknown breeding. Exceptions have been notable, but Warmbloods and classic Irish hunter types normally do not have the endurance required at the top level. Such horses often may do extremely well at the lower levels, however, because they move and jump better than the pure TB. Figure 118-2 demonstrates what could be considered the ideal modern eventing stamp, a highly successful New Zealand TB. A horse's ability and mental aptitude are the most important determinants, and other body types are successful (Fig, 118-3). An average ideal size would be 16.2 hands, but the rules are not hard and fast.

The financial value of event horses is considerably lower than that for racehorses, show jumpers, or dressage horses, so any horse with outstanding ability in one of these phases is likely to be used in that sport first. If the horses do not succeed, then they may be tried as potential event horses. These horses bring with them any injuries they may have accumulated, but because of the different stresses imposed

during eventing, this may not be a problem. For example, event horses tolerate low-grade carpal pathological conditions from previous race training. A small number of horses are bred specifically for eventing. Stallions generally are considered to lack the courage required to compete at the top level, and because the horse takes a number of years to reach its peak, choosing proven sires is a problem. The predominantly TB Irish sport horse and the relatively larger-boned New Zealand and Australian TBs are sought after.

Event horses normally commence dressage and show jumping training at 4 years of age and start competing in prenovice and novice One-Day Events from the age of 5. Depending on the horse's ability and rider's skill and patience, a horse usually competes in its first (graded as a CCI* or CCI**) Three Day Event at 6 to 7 years of age. A significant proportion of horses do not have the ability, courage, or physical durability to proceed beyond the CCI** or CCI*** level to the top grade of CCI****.

Fig. 118-2 An ideal modern eventing stamp. This 16.1-hand high New Zealand Thoroughbred gelding was an individual Olympic and World Championship gold medalist. (Courtesy Badminton Speciality Feeds, Oakham, Rutland, UK.)

Fig. 118-3 Example of a successful but tall and hunter-bred horse. This 17-hand high gelding Thoroughbred-cross Irish Draft horse was a Badminton CCI**** winner and Open European Championship Team gold medalist. (Courtesy Kit Houghton Photography, Bridgewater, Somerset, UK.)

INFLUENCE OF THE SPORT ON LAMENESS

Event horses are generally skeletally mature when they commence training and are not trained at the same speed or intensity as racehorses. They therefore have different patterns of injury, although most of the problems are still related to training. Primary long bone pathological conditions are rare. Soft tissue injuries such as tendonitis are common and often career limiting. The amount of endurance training necessary produces repeated cyclic loading, and problems such as osteoarthritis are common. The other subset of event horse injuries is acute traumatic injuries sustained during competition. Because the horses are jumping large fixed obstacles at speed, they are prone to falls and to direct traumatic fractures.

Fence design has changed in recent years. Square-shaped fences are avoided and rounded top contours are now more common. These fences give horses more leeway to correct mistakes and cause less severe direct impact trauma. The fences may be 1.2 to 1.4 m high, with a 2-m spread and a 2-m drop, so substantial strain can be placed on the supporting structures of the limbs on landing. The quality of the terrain also appears to have an important impact on the incidence of lameness problems. The competitions are run predominantly on turf, the nature of which depends on the soil type, local weather, and management factors. At the lower level, financial constraints generally preclude much improvement on the quality of the ground, but some of the top events try to maintain a permanent track that is tended carefully.

The prolonged period and intensity of training required for horses to reach an elite level means that those horses not metabolically or physically suited to the sport are selected out. For instance, few elite horses have recurrent exertional rhabdomyolysis or navicular syndrome. Three Day Events are regulated by the Federation Equestre Internationale. A strict medication control program is enforced, which permits only emergency medication at the competition after official veterinary approval or the use of a small number of permitted forms of medication, including antibiotics, rehydration fluids, and preparations for treating gastric ulcers.

TRAINING METHODS

Event horses train in all three disciplines, with the actual methods varying greatly between riders. A complete description of training methods is available elsewhere.[1] Most competing horses have natural cross country ability, so little time generally is spent on training for this, because the progression of competitions provides sufficient experience. Event horses normally are selected for natural jumping action, but jumping technique during the cross country phase is different than that needed for show jumping. Therefore event horses may not be as careful when show jumping and can have a tendency to touch poles. Most event horses are show jumped regularly, especially during the off season. The event horse's weakest link is usually the dressage phase. The movements required are no more than a medium level of pure dressage, but the different breeding and level of fitness of these horses means that the major challenge can be to control the horse's temperament. Most of the skills training revolves around this discipline, and a high standard now is required to be competitive at the top level.

Fitness training is a major component of the preparation for a Three Day Event and is where most orthopedic damage is incurred. The horse is likely to undergo a 3- to 4-month training period for the target Three Day Event. Training methods vary greatly between riders and often depend on local factors such as the availability of hills for trotting or all-weather gallops. Restricting the horse to training solely on an artificial surface is not advisable, because this seems to predispose the horse to injury when the horse actually competes on a natural surface.

Event horses normally receive a 6-week initial period of walking and trotting on the roads, and then commence cantering exercise. Different riders vary in their use of conventional or interval training, but usually peak the quantity of work at around three canters of 8 minutes, or shorter if a hill is available. The quality of the work usually is increased closer to the Three Day Event to include faster work. One Day Events are used to monitor the horse's fitness and as additional training. The dressage and jumping training also substantially contributes to the fitness regimen.

CONFORMATION AND LAMENESS

Although the horse's stamp may vary, as discussed previously, the basic conformation must be correct. Eventing is unforgiving to conformational defects compared with show jumping and dressage. The general principles are the same as for any equine athlete. Serious conformational defects include being back at the knee, having upright pasterns and hocks, and a moderate or severe toed-out conformation. Of slightly lesser significance are a long or short back, being over at the knee, or having long, sloping pasterns. Defects such as offset knees and a slight to moderate toed-in conformation seem to be less important. A good foot conformation is always desirable, but many event horses have the TB trait of weak feet with collapsed heels.

The prepurchase examination for a young event prospect, with hopes of a Three Day Eventing career, is likely to be strict. The conformation should be assessed critically at this stage. Although the horse may have sufficient talent, defects in conformation may not allow it to stand up to the 5 or so years of training necessary to reach the top level of the sport. Conversely, it is possible to be more lenient in the interpretation of subtle problems when performing a prepurchase examination on an older, experienced horse. The horse's competitive record should be assessed carefully, because advanced horses are likely to have accumulated wear and tear changes during an extended career. Any conformational or subtle soundness queries can be addressed in the light of the horse's proven ability to perform its task.

TEN MOST COMMON LAMENESS CONDITIONS

Some conditions are more common during training and others are more common at competitions. The overall prevalence is as follows:

1. Thoracolumbar and cervical soreness and restriction
2. Foot soreness (bruising, imbalance, and nail bind)
3. Traumatic osteoarthritis
   a. Distal interphalangeal joint
   b. Metacarpophalangeal and metatarsophalangeal joints
   c. Tarsometatarsal and centrodistal joints
   d. Proximal interphalangeal joint
4. Superficial digital flexor tendonitis
5. Suspensory desmitis
   a. Branch desmitis
   b. Proximal suspensory desmitis
6. External trauma
   a. Lacerations including overreaching injury
   b. Penetrations
   c. Stifle bruising
7. Pain in the sacroiliac region
8. Fractures of the stifle region
9. Other fractures
10. Rhabdomyolysis

CLINICAL HISTORY

A routine lameness history should be obtained, emphasizing and obtaining the following information:

- Horse's competitive level
- Competition targets and timing, ahead or behind in its fitness schedule
- Previous problems
- Recent competitive/training program
- Current medications
- Prodromal signs
- Onset acute or insidious
- Exact nature of problem (limb swelling, lameness, or poor performance)
- Progression of problem
- Response to treatment

LAMENESS EXAMINATION

My standard approach to the lameness examination is found in the following discussion. The regions requiring particular attention and the most rewarding procedures are outlined for horses with subacute or chronic lameness problems. Care should be taken to palpate all limbs and the back thoroughly, because many concurrent or compensatory injuries can be found that way. Because many problems are subtle, the horse should be examined on a variety of surfaces and at different gaits.

Particular attention should be paid to the feet. The size, shape, and conformation should be assessed in relation to the size and breed of horse. The suitability of the shoe type for that horse should be determined, because farrier preference may have been influenced by fashion. The fit of the shoe should be correlated with stage in the shoeing cycle. Fortunately, the trend is to move away from the traditional problem of shoeing the horse short and tight at the heels. The hoof should be palpated for heat and any cracks or defects. The sole should be pared and hoof testers carefully applied over the entire solar surface, assessing the solar compliance and any painful response. Horses with recurrent bruising associated with soft soles may not have demonstrable pain at examination, but the lack of solar rigidity is clearly evident. Percussion may be helpful in a small proportion of horses. An increased digital pulse amplitude can be helpful in determining any inflammation in the foot and is especially important if the amplitude is asymmetrical in the limb or between feet. A subtle increase in digital pulse amplitude is best evaluated after trotting the horse in hand on a hard surface.

The horse should be palpated carefully for distention of the distal interphalangeal and metacarpophalangeal/metatarsophalangeal (fetlock) joint capsules. The range of joint flexion and any resentment to flexion should be assessed carefully. Particular attention should be given to the palmar metacarpal structures for presence of subtle, diffuse filling and any discrete swelling, heat, or pain in the tendonous or ligamentous structures. Owners and riders of event horses tend to be thorough in their own palpation of this region and often know the normal contours of their horse well. However, they

often are misled by distention of the medial palmar vein or diffuse swelling from a more distal inflammatory lesion.

In the hindlimbs, pain on palpation over the cunean bursa and in response to the Churchill test should be determined. The medial femorotibial and femoropatellar joint capsules should be palpated to detect distention. The muscle tension in the back and hindquarters should be assessed carefully. Trigger points and painful foci should be determined, and the range of spinal flexibility in response to running a blunt object along the back should be assessed.

During the static examination the horse should be assessed for symmetry. The horse's condition and degree of muscling should be assessed in relation to its level of fitness. The freeness of stride, dynamic foot placement, and foot flight arcs should be assessed at the walk. At the trot any head nod and the range of gluteal excursion should be assessed to determine any lameness. Lunging in a circle on a hard surface frequently is used to exacerbate subtle lameness. However, it is also critical to see the horse lunged on a soft surface, a portion of the lameness examination that often is omitted. Different lameness conditions may be evident on different surfaces, and this comparison is valuable. For instance, a horse may have a low-grade concussive distal limb lameness evident when lunged on the hard surface. However, the primary problem may be proximal suspensory desmitis, pain from which is evident only when the horse is lunged on a soft surface. Although not decrying the value of evaluating all the lameness problems present, many of these horses have multiple, low-grade sites of pathological conditions to which they have adapted. Evaluating these can be a frustrating business, and it is important to determine the current problem noticed by a knowledgeable owner or rider. Evaluation of the horse while the horse is being ridden can be more difficult, but this form of movement can exacerbate low-grade lameness. Riding may be the only way of observing problems evident during certain movements, such as lack of hindlimb impulsion during a change of gait or when jumping. Seeing the horse ridden by a different, preferably more experienced rider can be helpful in some horses suspected of having back pain or when schooling or behavioral problems are present.

Determining the normal range of soundness is a difficult and contentious issue. Because of age and level of work, most top-level event horses have some degree of orthopedic pathological condition. I prefer to score lameness on a scale of 0 to 10, with 0 being sound and 10 being non-weight bearing. An advanced horse should be sound in the forelimbs when trotted in a straight line, but a large proportion demonstrate a 1 of 10 bilateral forelimb lameness when trotted in a circle on a hard surface. Some horses that are competing satisfactorily may show a greater degree of symmetrical lameness, but asymmetry in lameness may indicate that an important problem exists. Advanced horses can demonstrate up to a 2 of 10 hindlimb lameness when trotted in a straight line, without being penalized for this in a trot-up at a Three Day Event. Many have a 2 to 3 of 10 bilateral hindlimb lameness while circling on a hard surface. Hindlimb lameness evident on soft surfaces is likely to produce lower dressage scores, because the horse will exhibit poor and asymmetrical action.

Flexion tests are useful to exacerbate any subtle lameness problems, but these tests are not specific. Flexion tests can be particularly helpful in evaluating horses with lameness evident only immediately after they have completed an event. These horses can be frustrating, because they are often clinically sound when evaluated subsequently. A persistent positive response to flexion that can be alleviated by diagnostic analgesic techniques is important. Limb protraction, retraction, adduction, and abduction can be helpful in exacerbating upper-limb pain. Turning the horse in a tight circle can be used to assess coordination and flexibility. The range of cervical movement is assessed by observing the horse reach for food (voluntary movement) and by manual manipulation (forced movement). Many horses can cheat and reach the flank by rotation of the upper cervical region, rather than by full lateral flexion of the entire neck.

Different considerations apply when examining the horse with a history of an acute, severe lameness, usually after the cross country phase of a competition. A comprehensive approach of evaluating the acutely lame horse must be performed (see Chapter 13).[2] Unfortunately, localizing signs may be minimal, the horse may be in cardiovascular shock, and initial efforts must be directed at providing support to the horse and the suspected region of injury. Fractures are caused most commonly by external trauma. Particular attention should be paid to the shoulder region for signs of fracture of the supraglenoid tubercle of the scapula and to the stifle for effusion or peri-articular swelling associated with patellar or other fractures. Soft tissue injuries such as severe suspensory desmitis or superficial digital flexor tendonitis may cause acute, severe lameness.

DIAGNOSTIC ANALGESIA

Diagnostic analgesia is an important tool in lameness diagnosis in event horses. Because the horse may have many palpable abnormalities, differential diagnosis is important. However, no localizing clinical signs may be apparent, and diagnostic analgesia is critical to identifying the painful region. Specific treatment can be given if a problem is identified accurately. A positive response to intra-articular analgesia usually means a horse is likely to respond to intra-articular medication, leading to a quicker return to work. In horses with lameness problems too subtle for accurate interpretation of diagnostic blocks, intra-articular administration of corticosteroids may be useful to assess the long-term response to treatment. Even with experienced riders this method of management can have a placebo effect, because the rider may desire for the problem to be veterinary rather than from schooling.

Perineural and intrasynovial analgesic techniques are used commonly. Intra-articular analgesia is particularly helpful because many problems are joint-related, and a quick and definitive diagnosis can be achieved. Owners greatly resist clipping of the hair during the competition season, and clipping is not necessary in fine-coated animals if a thorough scrub is performed. The preferred techniques for the most commonly performed blocks are described subsequently.

The palmar digital nerve block should be performed as far distal as possible, angling the needle axially and distally to the cartilages of the foot. Separate medial and lateral blocks may be helpful to localize the pain to a specific heel. Blocking the palmar nerves at the level of the fetlock is less likely to desensitize the fetlock joint with a block performed just below the base of the proximal sesamoid bones, rather than at the abaxial sesamoid level. The palmar portion of the low four-point block should be performed just proximal to the digital sheath to decrease the risk of proximal migration of local anesthetic solution, taking care to go above or below the communicating branch of the palmar nerves. The lateral palmar nerve block is a satisfactory method of achieving proximal palmar metacarpal analgesia. Because the middle carpal joint is not a likely source of pain in event horses, the risk of confusing pain from this site with that from the origin of the proximal suspensory ligament is minimal.

The distal interphalangeal joint is injected most easily at a site on the dorsal midline, using a vertically directed needle. Six milliliters or less of local anesthetic solution should be used to decrease the risk of diffusion from the joint. For the

fetlock joints I prefer the lateral sesamoidean ligament approach because most horses do not have gross joint distention and the fixed anatomy of this approach is reliable.

In the hindlimbs the distal interphalangeal, proximal interphalangeal, and metatarsophalangeal joints often are overlooked sources of pain. Perineural analgesia normally starts with a basisesamoid block, followed by a low six-point block if necessary. Analgesia of the proximal plantar metatarsal region can be performed most easily by blocking the lateral plantar nerve 1 cm distal to the tarsometatarsal joint. This blocks the nerve before it branches into the plantar metatarsal nerves and carries a minimal risk of inadvertent penetration of the distoplantar outpouchings of the tarsometatarsal joint capsule.

IMAGING CONSIDERATIONS

Radiography

Radiography is the mainstay of imaging, but a few special considerations are necessary in event horses. Lateromedial and dorsopalmar radiographs of the distal extremity can be helpful in assessing foot balance. The palmaroproximal-palmarodistal oblique projection often is under-used in assessing subtle pathological conditions of the navicular bone and the palmar processes of the distal phalanx. The flexed cranioproximal-craniodistal projection of the stifle is especially valuable for assessing the medial aspect of the patella for fractures. It is important to angle the x-ray beam so that the trochlear ridges are not superimposed over the patella, because some fractures or evidence of comminution may otherwise be obscured. A relatively underexposed and undercollimated lateromedial projection is also beneficial to detect small, displaced fragments, which otherwise may be missed.

Ultrasonography

Ultrasonographic evaluation of the palmar metacarpal structures is a vital part of the lameness evaluation of an event horse. Superficial digital flexor tendonitis is a common and career-threatening injury, and if the veterinarian has any doubt about even a subtle problem, an ultrasonographic examination should be performed. Even when primary forelimb lameness is located at a distal site, the most important lesion may be a compensatory tendonitis in the contralateral limb.

It is usually not necessary to clip the coat, particularly if the hair is fine, and diagnostic images can be obtained after thorough scrubbing and the liberal application of alcohol and gel. The coat may be clipped to obtain maximal detail if subtle tendonitis needs to be investigated. Clients are often reluctant to have the coat clipped for a precautionary scan during the competitive season, because they perceive that the horse will be flagged as having a problem when it next competes. In my experience, sufficient detail is visible in fine-coated horses without clipping, although the clients are warned that greater image quality can be obtained with clipping, and if the image quality is non-diagnostic, the coat needs to be clipped. Often the difficulty is not in identifying abnormalities within the tendon but in determining the significance of any changes that are present. Many advanced horses have changes in fiber pattern of the superficial digital flexor tendon. Transverse and longitudinal views should be obtained in a systematic fashion. Different focus, gain, and frequency settings optimize evaluation of different structures. The cross-sectional area of the superficial digital flexor tendon should be obtained routinely, because sequential monitoring of this may allow the early detection of tendonitis. Determining the cross-sectional area also assists in assessing the current significance of chronic lesions, which is an important and helpful part of the ultrasonographic examination and should not be omitted.

Ultrasonography also can be helpful in assessing articular and peri-articular pathological conditions in structures such as the patellar ligaments. Examination of the ventral sacroiliac ligaments per rectum also can be valuable when pain in this region is suspected.

Scintigraphy

Nuclear scintigraphy is a useful technique for evaluating some lame event horses. Scintigraphy commonly is used in horses with hindlimb lameness, back problems, multiple limb lameness, and forelimb lameness with an equivocal or negative response to diagnostic analgesia. Image quality can be a concern, because event horses are skeletally mature and the degree of pathological bone conditions is low. Normal bone uptake can be limited, except in horses with acute trauma. Good technique is therefore essential to obtain diagnostic images, and postprocessing techniques, such as using motion-correction software can be helpful. Case selection is also important, because the more chronic and low-grade the problem, the lower the likelihood of finding an obvious focal region of increased radiopharmaceutical uptake (IRU). Examples of conditions for which scintigraphy can be rewarding include non-localized foot pain (pedal osteitis and insertion injury of the deep digital flexor tendon attachment), stress fractures (although these are rare overall), and osteoarthritis of the thoracolumbar facet joints.

Normal scintigraphic patterns are described poorly in the literature, especially for non-racehorses. Regions that commonly have IRU in event horses, but without associated pathological conditions, include the distal phalanx, the proximal interphalangeal, fifth and sixth cervical and sixth and seventh cervical articulations, and the distal tarsal bones. The distal tarsus appears to have active bone remodeling when evaluated scintigraphically, but many event horses do not show lameness or a positive response to either flexion tests or local analgesia. In a study evaluating the accuracy of scintigraphy in horses with confirmed distal hock joint pain, we found a positive predictive value of 0.70 and a negative predictive value of 0.91 for focal IRU.[3] Because of this high false-positive rate, scintigraphy should be used with thorough clinical examination. Although relying on scintigraphy in fractious horses may be tempting, authenticating the diagnosis using diagnostic analgesia is important.

Thermography

Thermography has been used for more than 25 years but is still a developing diagnostic modality. The technology has improved recently, and thermography units are now affordable. The handheld infrared imaging cameras are particularly attractive. Thermographic imaging provides a sensitive representation of skin surface temperature, but many confounding variables make interpretation of these images difficult. To develop expertise involves a steep learning curve, and a considerable amount of time and experience are necessary to make consistently useful interpretations. Major advantages include that the technique is non-invasive and is quick to perform. Thermography is similar to scintigraphy because it is a physiological rather than an anatomical imaging technique. Given the high prevalence and importance of soft tissue injuries in event horses, thermography has applications in detection and in monitoring response to therapy. Because of low specificity, however, thermography should be used with other imaging techniques.

Thermographic imaging is useful in evaluating foot balance and in differentiating various types of inflammation of the foot (Fig. 118-4; Color Plate 10). Examinations before and after exercise are particularly useful for this purpose. Similarly, pre-exercise and post-exercise thermograms are helpful in identifying specific muscle injuries. Accurate

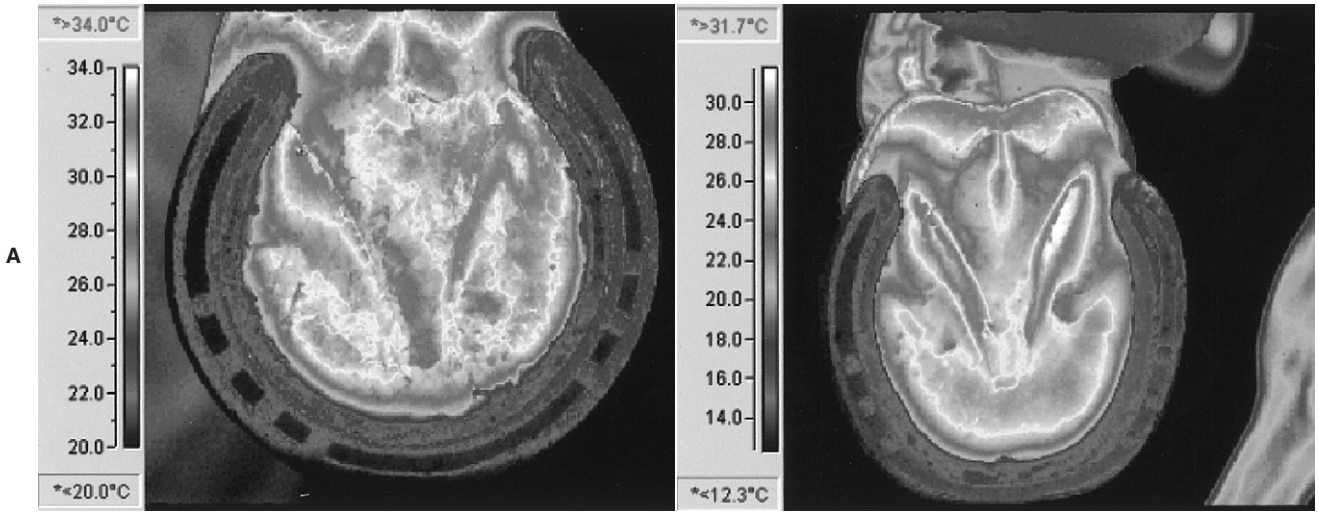

Fig. 118-4 Examples of different thermographic foot patterns (solar views). **A,** This hoof has a medial corn, manifested as a focal hot spot (*white*) within an area of increased temperature. **B,** This hoof has subacute laminitis, with a pattern of increased heat in the region of the tip of the distal phalanx.

identification of local muscle strain permits treatment to be focused on the affected area, with a consequently shortened convalescent period. Thermography is also a useful tool in evaluating neck and back problems, although interpretation is more complex in these regions.

Thermography can be particularly useful in monitoring superficial digital flexor tendonitis and can be used as part of a routine screening procedure, with regular examinations in the run up to a Three Day Event. Thermography can detect small lesions before clinical signs are evident, or it can be helpful in determining if a chronic lesion is active (Fig. 118-5; Color Plate 11). If thermography suggests a lesion is present, then ultrasonographic examination is indicated. During the convalescent phase after injury, regular thermographic screening allows detection of any signs of inflammation in the affected region, as the plane of exercise is increased. Care must be taken to avoid artifacts from bandaging, previous clipping, and topical medication.

SADDLE PRESSURE ANALYSIS

Computerized saddle pressure analysis using a force-sensing array system allows an objective assessment of pressure distribution beneath the saddle (Fig. 118-6; Color Plate 12). Poor saddle fit is an important problem in event horses and is discussed in greater detail (see pages 991 and 992). Computerized saddle pressure analysis is straightforward to perform and is complementary to conventional saddle fitting. By allowing an objective assessment, computerized saddle pressure analysis can be useful to confirm a problem to a rider, owner, or saddler. The better systems allow dynamic assessment of saddle fit at exercise, which is not otherwise possible.

PROCEEDING WITHOUT A DIAGNOSIS

Although in general a diagnosis can be made in most lame event horses, factors such as the experience level of the veterinarian, the thoroughness of the lameness workup, the number of imaging modalities available, and the nature, severity, and stage of the disease process affect diagnostic ability. If a horse is seen repeatedly on a first-opinion basis, stepping back and re-assessing the horse as if from the start is sometimes necessary. It may be necessary to refer the horse for a second opinion or for advanced imaging techniques, such as scintigraphy, if these have not been performed. In some horses the precise diagnosis continues to remain elusive. In horses with obscure forelimb lameness, I routinely perform an ultrasonographic examination of the palmar metacarpal soft tissue structures, because tendonitis and desmitis are important and highly prevalent. Serial cross-sectional area measurements of the superficial digital flexor tendons should be obtained, because the most significant problem with an undiagnosed low-grade lameness may be a compensatory tendonitis in another limb. Any evidence of tendonitis indicates that exercise level should be decreased. Similarly, any persistent clinical signs of swelling in the palmar metacarpal structures should prompt a cautious approach. Even in the absence of ultrasonographic changes, mild swelling should alert the veterinarian to the possibility of a subclinical problem with tendonitis or desmitis. It is important not to rely too much on ultrasonography for diagnosis of soft tissue injuries, because early lesions may not be apparent.

Generally in horses with low-grade, undiagnosed lameness the response to a period (few days to weeks) of rest should be assessed. If the response is poor, then most horses with an undiagnosed, low-grade hindlimb lameness can be continued in work, with or without the use of a systemic non-steroidal anti-inflammatory drug (NSAID) such as phenylbutazone. Greater caution normally is advised in horses with undiagnosed forelimb lameness, because the risk of developing a career-limiting injury with continued exercise is greater compared with similar injuries in the hindlimbs. If a subtle problem persists, then it may be necessary to increase exercise intensity to exacerbate the problem to a point where diagnostic local analgesia can be performed. If a competition is imminent, the owner or rider may apply considerable pressure to treat the most likely problem in the hope of rendering the horse sufficiently sound to compete.

If lameness is severe without initial diagnosis, then the horse should be given box rest, and the workup should be repeated until a diagnosis is achieved. If an upper limb soft tissue problem or a back problem is suspected, then a physiotherapeutic or chiropractic/osteopathic opinion can be helpful. Although veterinary opinion is divided on the validity and value of these techniques, an owner frustrated by the lack of a veterinary diagnosis is likely to turn to these. Horses with

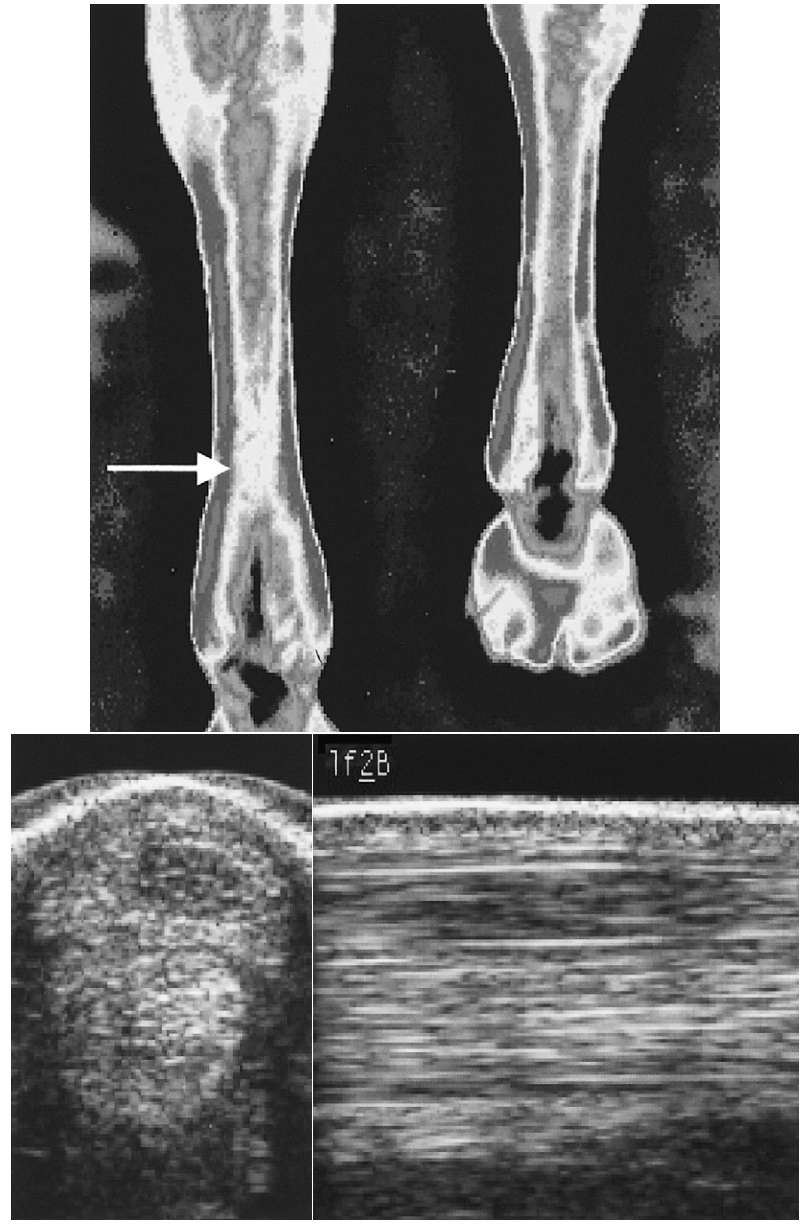

Fig. 118-5 Palmar thermographic image and subsequent transverse (on the left) and longitudinal ultrasonographic images of an advanced event horse 10 days after successfully completing a Three Day Event. The horse was having a routine examination, and no clinical localizing signs were evident in the tendon. The thermogram *(top)* demonstrates a focal hot spot over the left distal superficial digital flexor tendon (*arrow*), and the ultrasonographic images reveal a hypoechoic core lesion in the same region.

poor performance may have schooling or behavioral problems. Assessment by an experienced and different rider can be helpful.

SHOEING CONSIDERATIONS

Foot problems are vitally significant in event horses, and the importance of high-quality farriery in minimizing the incidence of lameness cannot be over-emphasized. Regular and good farriery is important in maintaining good medial to lateral and dorsal to palmar (plantar) hoof balance. Types of shoes vary between the traditional fullered hunter-type shoes, continental flat shoes, four-point style shoes, and various types of bar shoes. For routine shoeing the conventional fullered shoe offers the advantage of superior grip. Many farriers prefer the extra width of the flat section shoe, because this shoe is easier to fit, has sufficient width at the heels, and gives extra sole cover. If not applied correctly, flat-section shoes may cause excessive sole pressure in horses with flat feet. Traction devices are necessary if flat section shoes are put on horses working on tarmac. Natural balance shoeing has become popular recently in event horses, but although the technique suits some horses, others may actually develop lameness because of improper shoeing technique. However, the natural balance concept of a more palmar location for the breakover point has been well accepted. Seeing horses shod with the shoe set well back is now common, and quarter clips rather

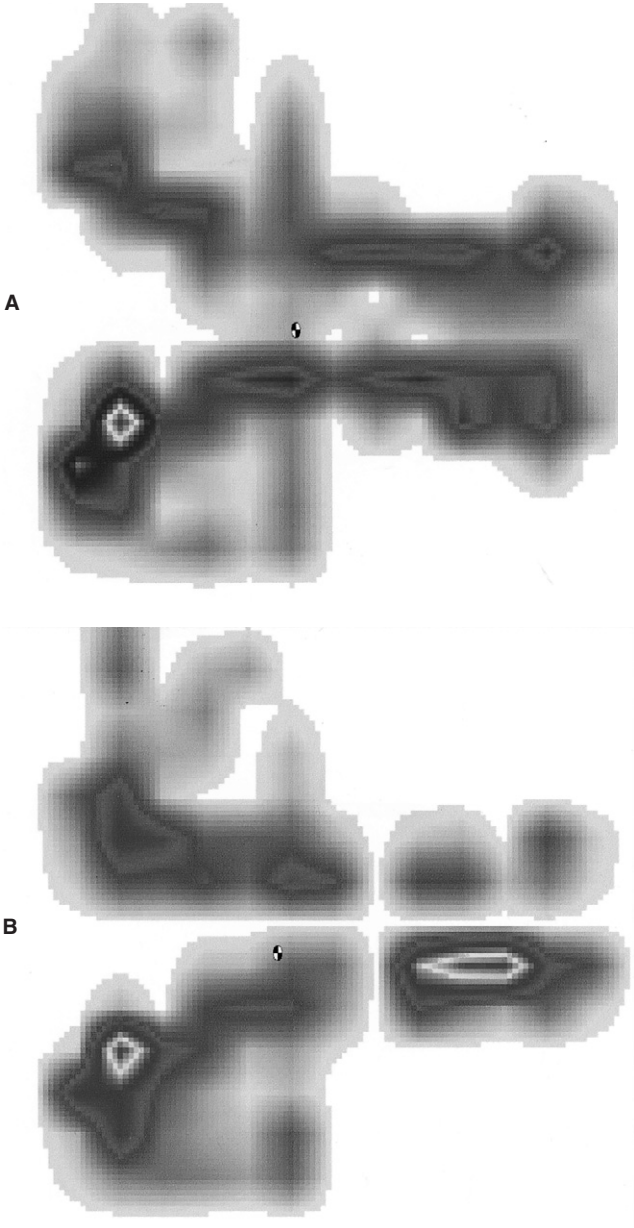

Fig. 118-6 Computerized saddle pressure analysis images. Cranial is to the left, and left is to the bottom. **A,** This image shows a poorly fitting saddle, with a focal pressure point in the left wither region. **B,** This image demonstrates failure of a gel pad to alleviate the pressure point and the development of an additional pressure point caudally.

than toe clips are most common. This is helpful in horses with long-toe conformation, osteoarthritis of the distal interphalangeal joint, or navicular syndrome. A rolled-toe shoe may give a similar effect, although bringing the breakover point back to the same degree is difficult, and care must be taken to set the shoe back sufficiently.

The most common therapeutic shoe used in event horses is the bar shoe. The egg bar shoe is used commonly to manage horses with navicular syndrome and other causes of palmar heel pain and to provide support in horses with collapsed heels. Egg bar shoes are heavy if they protrude beyond the heel bulbs and may be pulled off. Shoe loss is an important issue in event horses, more so than in other sport horses. As a compromise the straight bar shoe may provide additional foot stability while reducing the risk of shoe loss. In those horses requiring egg bar shoes, overreach boots can be used to reduce the risk of shoe loss, and by using a more palmar point of breakover, the front feet may leave the ground more quickly compared with horses with conventional shoes. In horses with weak heels or quarters, laminar separation, or focal osteitis of the distal phalanx, an egg bar shoe may provide inadequate support and a heart bar shoe can be beneficial. This shoe is heavy, does not project as far beyond the heels as does the egg bar shoe, and transfers some portion of weight-bearing load to the frog. In horses with pain localized to one heel or quarter, a half bar shoe can provide sufficient support and is lighter than a full heart bar shoe.

Pads and cushions are used infrequently in event horses. Although pads may provide short-term benefit in horses with solar pain, they promote excess movement of the clenches and premature shoe loosening. Full pads cause a poor solar microenvironment. Modern synthetic hoof repair materials can be beneficial in horses that have lost portions of the hoof wall, because the defect can be filled and shoe nails can be placed subsequently. However, routine use of hoof repair materials to augment hoof wall in horses with poor-quality, cracking, and flaking feet should be discouraged and may even cause the problem to persist. If used, repair material should be removed at the end of the season, and the horse should be turned out without shoes. The foot may break up initially but will grow back stronger without the repair materials.

Steel shoes are used routinely, but some riders switch to aluminium shoes at a Three Day Event. The foot must be of sufficient quality to cope with the lower degree of support offered by the more flexible aluminium shoe, and switching is not recommended if the horse has any history of foot-related lameness. Any speed and recovery benefits of the lighter shoes do not appear to be obvious in event horses. Synthetic shoes rarely are used.

Road nails or studs often are used, especially in broad section shoes, to decrease the risk of slipping on the roads during walking and trotting exercise. Improper use of road nails or studs may cause hoof imbalance. If the grip point is positioned near the middle of the foot (rather than near the heel), the foot may rock over this point during weight bearing. Event horses commonly wear studs for competition. Some advocate that a single stud be worn simply on the basis that if the stud is on the outside, the risk of the horse treading on itself is less. With a single stud, the foot can still twist, causing less jarring to the limb overall. Conversely, a single stud severely imbalances the foot, and because the roads and tracks phase of a Three Day Event often includes sections of metaled road or tracks, this could induce excessive strain on the joints (Fig. 118-7). Studs should be avoided for the roads and tracks phase if logistically possible. However, some horses will only jump with confidence if two or more studs per foot are applied. The studs always should be chosen based on ground conditions, and a blunt inside stud decreases problems from tread injury. Care should be taken to avoid positioning a stud hole over an area of defective hoof wall. Horses are normally re-shod one week before a Three Day Event, because if a horse is pricked or any foot soreness occurs after shoeing, the horse has time for recovery.

TACK CONSIDERATIONS

Saddle fit is of great importance in event horses. Injuries to the withers and back may manifest as poor performance in the dressage or show jumping phases of competition. Different saddles are required for the different disciplines of eventing, predisposing the horse to fitting problems. The fit of the saddle to the horse is sometimes a secondary consideration

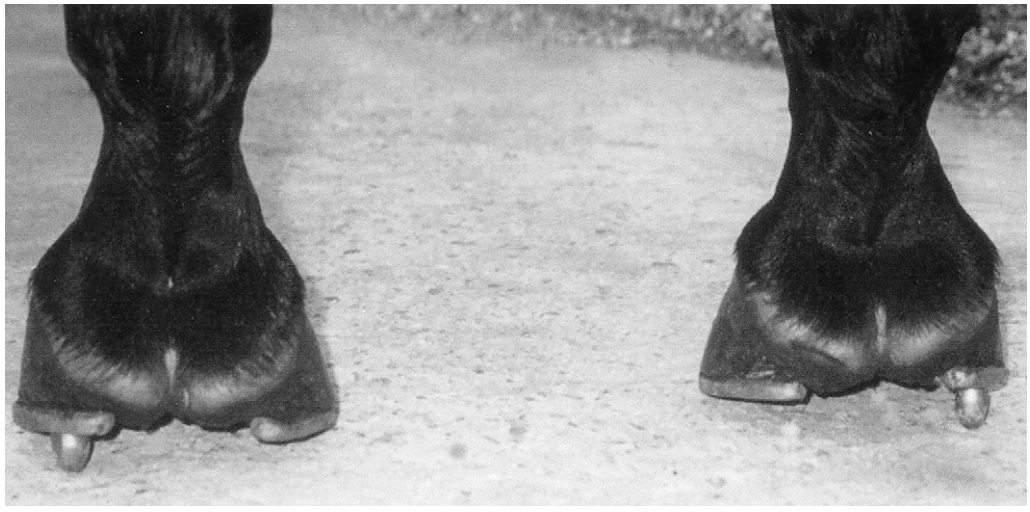

Fig. 118-7 Palmar view of horse with single studs, showing the degree of mediolateral imbalance induced when the horse is on a hard surface.

to riders who may have a particular saddle in which they feel secure. Event horses often have high withers and fitting a saddle is often difficult. Saddle fit can be affected adversely by the tendency of event horses to lose weight dramatically in the run up to a Three Day Event. Therefore a saddle that is fitted 2 weeks before an event can sit too low by competition time. Unfortunately, the use of pads and numnahs to correct poor saddle fit is not effective. Computerized saddle pressure analysis has shown that even gel pads are ineffective in alleviating focal pressure points, and that numnahs and pads placed beneath well-fitting saddles can be detrimental (see Fig. 118-4).[4] Thick or multiple pads elevate the saddle and put pressure on the midline and over the dorsal spinous processes, a situation that impedes spinal flexibility.

Opinions vary on the benefits of protective leg wear. Tendon boots are used almost universally for the cross country phase, because the risk of direct traumatic injury to the distal limb is high. However, the additional insulation increases the temperature of the distal limb. There are theoretical concerns that elevation in temperature could damage tenocytes and predispose the horse to tendonitis, although no clinical evidence indicates that this occurs. Some boots have reinforced sections to give additional protection against speedy cut injuries to the palmar aspect of the tendons, but the large rigid section may rub against the tendons at exercise and cause abrasions. Conversely some of the flimsy boots do not give sufficient protection against this kind of laceration. Bandages give a greater degree of conformity, but they are more difficult to apply correctly and require inclusion of a reinforcing layer if they are to provide substantial protection. Neither bandages nor boots reduce risk of tendon strain but simply protect against direct trauma.

DIAGNOSIS AND MANAGEMENT OF LAMENESS

Thoracolumbar and Cervical Soreness and Restriction

Neck and back soreness are common clinical findings in event horses, although they do not always limit performance. Soreness may be secondary to lameness as occurs, for example, when pain on palpation of the brachiocephalicus muscle is found in horses with distally located forelimb lameness. Primary problems may be subtle, and determining the importance of clinical findings can be difficult. To know an individual horse's normal degree of sensitivity and flexibility can be extremely helpful, because any changes can be correlated with the onset of a performance problem. Questioning a knowledgeable owner, groom, or physiotherapist can be helpful. Sometimes the significance of any soreness can be assessed only by the horse's response to treatment.

Event horses are particularly prone to muscle soreness when exercise intensity is varied or increased. Intense dressage training initially causes a transient period of lumbar muscle soreness, especially during the sitting trot. Areas of focal pain and muscle spasm may be easily palpable, but assessing flexibility and range of movement is also important. Radiographs are not helpful in diagnosing the significance of dorsal spinous process impingement, and clinical signs, scintigraphic examination, and positive response to local analgesia are necessary to confirm if a lesion is active. Ultrasonographic examination can be helpful in diagnosing supraspinous ligament injury. Thermography is helpful in detecting acute soft tissue injuries in the thoracolumbar region (see Chapter 25).

Many horses with neck and back pain respond well to physiotherapy. If range of movement is decreased, then mobilization of the affected region is beneficial (see Chapters 94, 96, and 98), which then can be followed by continued stretching exercises, performed by the owner or rider. Massage is beneficial but yields only temporary improvement. Various modalities such as laser, therapeutic ultrasound, and neuromuscular stimulation can be helpful. Initial results with extracorporeal shockwave therapy have been encouraging. In some horses, chiropractic manipulation is beneficial. Acupuncture also can be effective in the right hands. Given the range of physiotherapeutic treatment options available, the degree of experience necessary for optimal treatment results, and the nature of these therapeutic modalities, I suggest that veterinarians should work with an experienced and qualified therapist. It behooves the veterinarian to be familiar with these techniques, because case selection and palpation skills can improve.

Horses with lower cervical restriction caused by osteoarthritis of the dorsal cervical articular facets may show a positive response to intra-articular or peri-articular medication with methylprednisolone acetate (80 mg) injected under ultrasonographic guidance. Each affected facet joint is injected, and the horse is rested for 2 weeks, after which time improvement is normally dramatic. Horses with mild to moderate impingement of the thoracic dorsal spinous processes

can be treated successfully using local injection of methylprednisolone acetate (80 mg) and Sarapin (4 ml). The horse is lunged or long reined for 2 weeks and then returned to exercise. The duration of effect can be as short as 6 weeks, but in a large proportion of horses, the problem resolves without repeat medication. Marks provides a review of the various veterinary options in treating back pain.[5] The importance of management and riding factors in treating these conditions cannot be overemphasized. Rehabilitation and reschooling of horses with moderate to severe pain are necessary to build up the local musculature and to develop flexibility.

Foot Soreness (Bruising, Imbalance, and Nail Bind)

Foot soreness is a common clinical problem in event horses. Temporary solar bruising is frequent, especially in flat- and thin-soled horses. Foot imbalances predispose horses to soreness, and medial to lateral hoof imbalance is commonly present. In horses with collapsed heels, pain to hoof testers and corns are commonly found. Horses with persistent foot lameness may have laminar separation or focal osteitis of the distal phalanx, and scintigraphy is helpful in differential diagnosis. Poor foot conformation in many horses can predispose them to nail bind or pricking when shod. Shoes often are pulled off, which can lead to breakup of the wall and further problems with shoe security. Regular, high-quality farriery is more import in maintaining good hoof quality than is the feeding of supplements or applying topical applications. Good stable hygiene is also important. Shoeing aspects have been discussed (see page 990).

Osteoarthritis

Distal limb joints are especially prone to traumatic joint disease, especially in the forelimb. Osteoarthritis of the distal interphalangeal joint is most common, followed by that of the metacarpophalangeal joint. Osteoarthritis of the proximal interphalangeal joint is rare. Diagnosis is confirmed by observing a positive response to intra-articular analgesia, effusion or fibrosis, a positive response to flexion, and low-viscosity synovial fluid. Radiographs are often unremarkable in horses with early or low-grade osteoarthritis. Response to intra-articular medication is normally excellent. Combining hyaluronan with a low dose of corticosteroid, such as triamcinolone acetonide (5 mg) produces the greatest therapeutic effect. Hyaluronan alone produces an inconsistent response, and low doses of triamcinolone have been shown to be chondroprotective. Medium viscosity hyaluronan usually is used initially based on economic factors, but high molecular weight products may give a greater effect in some horses. Systemic administration of hyaluronan (intravenously) or polysulfated glycosaminoglycan (intramuscularly) is not nearly as effective as is specific intra-articular medication. Systemic medication does not carry the same risk of iatrogenic infection and is easier to administer and so is used frequently as an adjunct therapy. Prophylactic use of systemic therapy frequently is used in horses in the run up to a Three Day Event or to manage those with pathological conditions of multiple joints or generalized stiffness. Feeding of nutraceuticals such as chondroitin sulfate and glucosamine is also common practice. Although in vitro evidence of efficacy is substantial, no convincing clinical studies demonstrate efficacy in vivo. In fact, studies have demonstrated a lack of oral absorption of chondroitin sulfate in horses.[6,7]

In horses with osteoarthritis of the distal interphalangeal and metacarpophalangeal joints that has been non-responsive to medication, and especially with radiographic evidence of osteochondral fragmentation, arthroscopic evaluation can be beneficial. Focal areas of pathological conditions of cartilage and subchondral bone may be identified, and the horse often responds positively to debridement. Access is limited in the distal interphalangeal joint, but lesions often are found on the extensor process.

Osteoarthritis of the tarsometatarsal and centrodistal joints is not infrequent, but the distal tarsal joints show scintigraphic evidence of remodeling even in normal event horses. Horses with authentic osteoarthritis of these joints usually respond well to medication with low doses of long-acting corticosteroids (40 mg of methylprednisolone acetate). The condition rarely seems to progress sufficiently to necessitate chemical or surgical arthrodesis.

Superficial Digital Flexor Tendonitis

Although not the commonest lameness problem in event horses, tendonitis is the most substantial cause of wastage because of prolonged convalescence and high recurrence rates. Although tendonitis can occur as a single-event injury, especially if the horse falls, stumbles, or trips badly, the condition most commonly results from repetitive cyclic loading. Unlike the suspensory ligament, tendons in adult horses do not strengthen in response to exercise and therefore are prone to develop accumulated microdamage during intense training. This damage can give the prodromal signs of slight filling or heat in the palmar metacarpal structures. The clinical signs of a tendon strain then may develop acutely after a training canter or competition. For horses to develop subclinical tendonitis after a Three Day Event is not uncommon. The condition may not be apparent during the rest period after the competition but develops into clinical tendonitis when the horse resumes training or competitive work, even after 3 to 6 months. Close monitoring is thus essential, and I recommend routine ultrasonography after each Three Day Event. Ultrasonographic evaluation is important to assess the severity of injury and to determine a prognosis and an appropriate treatment plan. Lesion length and percentage of cross-sectional area involvement should be determined. Tendons may increase in cross-sectional area up to 10% normally with intense training, but this tends to return to baseline at the end of the season. If ultrasonographic examination is performed soon after injury, the extent of the lesion may not be apparent and severity may be underestimated. Severity of tendon damage is assessed most accurately between 1 and 4 weeks after injury.

Initial treatment of event horses with tendonitis is aimed at limiting inflammation and preventing any further tendon damage. The horse should be given box rest for 2 to 4 weeks, and anti-inflammatory therapy should be commenced. Systemic NSAIDs appear to decrease swelling and pain, and no evidence indicates that they impede healing. Conversely, prolonged corticosteroid administration may interfere with fibroplasia, although a single systemic dose helps decrease inflammation without any apparent detrimental effects.

Support bandages or firm stable bandages should be applied for the first few weeks to reduce swelling. Topical dimethylsulfoxide can be useful to decrease inflammation early after injury but should not be used for more than 5 days, because it can weaken collagen fibers and blister the skin. Local treatment with cold water hosing and ice application is beneficial while the signs of acute inflammation persist. For horses with mild to moderate injuries, the benefits of being able to perform local therapy seem to outweigh the greater external support that can be provided by application of a Robert Jones bandage.

In horses with severe tendonitis in which loss of support (sinking) of the metacarpophalangeal joint has occurred, external support using a Robert Jones bandage is recommended. A heel wedge should be applied if the animal is severely lame. Heel elevation theoretically does not decrease load on the superficial digital flexor tendon but appears to provide analgesia to horses that stand with the heel off the ground. In horses with severe breakdown injury, support with a proprietary splint, such as a Kimzey LegSaver, (Kimzey, Woodland, CA) is recommended.

Controlled exercise, to encourage development of a longitudinal fiber pattern but without placing excessive load on the tendon to damage the healing fibers, is the mainstay of recuperation. Serial ultrasonographic examinations are helpful to determine the appropriate rate of progress and the response to increasing exercise. After the period of box rest, walking in hand normally is commenced. Mechanical horse walkers appear detrimental in the early stages of healing, because constant turning places excessive load on the weak tendon. However, such walkers are extremely helpful after the initial 4 to 6 weeks of walking in hand, when duration of walking is increased. A major problem during this stage is degree of tolerance shown by the horse to this restricted level of exercise. Many horses are too excitable to be walked safely in hand, but they behave appropriately on a horse walker or if ridden under saddle. Exercise programs must be tailored to account for rider competence and the horse's behavior. Once trotting has been commenced, the horse normally settles down into the exercise regimen.

A large number of treatment options exist for managing event horses with tendonitis. Tendon splitting, usually performed percutaneously in the standing horse, appears beneficial in horses with acute core lesions. Tendon splitting may decompress the core lesion and allow neovascularization but is most effective when performed in the first 10 days after injury. Intralesional injection of hyaluronan or a polysulfated glycosaminoglycan has not proved beneficial. Initial clinical studies investigating intralesionally administered β-aminoproprionitrile fumarate unfortunately lacked representative control groups. The drug has received a mixed reception since becoming licensed and is not the panacea that some initially promoted it to be. The drug has been used in a limited number of event horses that returned to compete at the top level.

Use of intralesional growth factors is an area of current research interest. Insulin-like growth factor 1 and equine growth hormone, which induces the production of insulin-like growth factor 1, have been studied. I have had success in a small number of horses using transforming growth factor β (TGF-β) in horses with tendonitis. The drug appears to promote fibroplasia. Although this gives a quicker healing rate and horses have returned to work successfully, whether the expected increase in strength and decrease in elasticity are desirable is theoretically debatable. I have had rewarding results using TGF-β in horses with chronic tendonitis. When ultrasonographic evidence of poor infilling exists, intralesional injection of TGF-β improves fiber pattern, and horses are able to withstand increased exercise intensity. After treatment, some horses have returned to CCI**** level and have completed successfully in up to three events.

Desmotomy of the accessory ligament of the superficial digital flexor tendon (superior check desmotomy) also can be beneficial in selected horses. Because of the risk of general anesthesia, I reserve its use for horses with recurrent tendonitis or those in which the ultrasonographic appearance of the tendon deteriorates inappropriately during the controlled exercise regimen. In a small number of horses in trotting exercise after tendonitis, increased cross-sectional area and decreased echogenicity are observed. Even after the plane of exercise is reduced to walking for another 6 to 8 weeks or even longer, the ultrasonographic appearance of the tendon does not change. After desmotomy, however, these horses tolerate an increased plane of exercise without ultrasonographic deterioration or subsequent recurrence of tendonitis. Because the procedure may increase the risk of suspensory desmitis after surgery, desmotomy should not be used in horses with a concurrent pathological condition of the suspensory apparatus.

Systemic medication with a polysulfated glycosaminoglycan is used as an adjunct treatment in horses with tendonitis. Ease of administration and lack of potential complications make such treatment popular, but no evidence indicates that any systemic medication, or feed supplements for that matter, has beneficial effects in tendon healing.

The total convalescent period depends on severity of initial injury, how well the injury appears to heal as viewed by ultrasonography, and the degree of compliance with the controlled exercise regimen. In horses with mild tendonitis (subtle loss of fiber pattern and ≤10% increase in cross-sectional area) as few as 6 weeks of walking exercise is sufficient. Horses with loss of fibers and frank tendonitis require a convalescent period of 9 to 15 months. Recurrence rate is less in horses that are given at least a year to recuperate before commencing full work. After tendonitis, many horses can be managed successfully to complete one Three Day Event, but sustaining the horse through a number of Three Day Events without recurrence is more difficult. A small number of horses seem to suffer recurrent clinical signs regardless of management protocol and these are best restricted to One Day Events in which they will often then compete successfully for many years.

Suspensory Desmitis

The two main levels of suspensory injury in Three Day Event horse are branch desmitis and proximal suspensory desmitis. Mid-body desmitis is rare. Suspensory branch desmitis can be considered an occupational disease of event horses. Advanced horses commonly develop inflammation and enlargement of the branches after a Three Day Event, and the condition is often transient with no associated lameness. Viewed by ultrasonography, the branches have periligamentous fibrosis with no areas of obvious hypoechogenicity, although some loss of longitudinal fiber pattern may occur. In many horses this pathological condition is missed or ignored, and the horse is just turned out. Even a short period of controlled exercise often decreases the risk of recurrence, however. Some degree of synovitis of the metacarpophalangeal joint commonly occurs and usually responds favorably to intra-articular medication.

Proximal suspensory desmitis is a common cause of forelimb lameness. Lameness may be acute in onset and inflammation obvious after exercise, or the disease can be insidious without any localizing signs. Diagnostic analgesia is mandatory, and ultrasonographic examination is valuable. Radiographic and scintigraphic examinations are useful adjunct tools. Most horses have true desmitis, although a small number have enthesopathy and associated bone remodeling, with significant IRU and minimal ultrasonographic abnormalities. Most horses respond well to 4 to 9 months of controlled exercise, and no convincing evidence exists that any intralesional medications are beneficial. Extracorporeal shock wave therapy has been used to treat horses with suspensory desmitis and may provide quick analgesic effect. However, this may encourage a premature return to full exercise, thus increasing the risk of recurrence. Shock wave therapy may well have a role in horses with chronic, active desmitis, especially those with bone involvement that does not respond appropriately to controlled exercise.

I feel that proximal suspensory desmitis in the hindlimb is a most important but underdiagnosed condition. Similar to that seen in the forelimb, hindlimb proximal suspensory desmitis may cause acute or chronic lameness. The prognosis is generally much poorer in the hindlimb, however, because of the development of a local compartment syndrome and associated pressure on the plantar metatarsal nerves. Thus most horses remain chronically lame, even after the ligament appears healed on ultrasonographs. I have had success in treating chronic lameness resulting from hindlimb proximal suspensory desmitis using local infiltration of triamcinolone (10 mg) in horses with static or healed ligaments but with persistent lameness. Although lameness abates, treatment needs to be repeated at 6-month intervals and may be detrimental to the ligament in the long term. Thus a surgical technique has been developed for use in

horses with chronic desmitis that has yielded extremely good results.[8] The surgical technique involves the resection of a 3-cm segment of the common branch of the plantar metatarsal nerve, distal to its origin from the lateral plantar nerve. This is combined with incision of the fascia (fasciotomy) overlying the origin of the suspensory ligament. This technique combines decompression with analgesia, and has allowed horses to return to a normal level of work within 3 months. The horses are restricted to walking only for the first month, after which time turnout and ascending exercise are permitted. I have long-term follow-up data on 20 horses, and all returned to the previous level of work (including CCI****). Recurrence of proximal suspensory desmitis was seen in only two horses.

External Trauma

Event horses are prone to traumatic lacerations during competition, the most common being overreach injuries to the heel bulbs. Standard principles of treatment apply, although owners often apply pressure to minimize healing time during the competition season. Aggressive early treatment is advised, and horses with deep foot or pastern wounds should be managed with a fiberglass cast for 10 to 14 days, a treatment that may shorten the convalescent period. Wounds should be assessed carefully for synovial penetrations. Thorn penetrations from brush or hedge fences can lead to infectious arthritis of the carpal or stifle joints, with only minimal clinical signs evident initially.

Direct trauma from jumping solid fences is common. Trauma directly over bone can lead to a severe, transient lameness. Acute lameness from this type of bone bruising can be difficult to differentiate from a fracture on an initial clinical examination. Trauma over the soft tissues may lead to the development of a considerable hematoma or edema, so local cold and pressure are indicated in the early stages.

Pain in the Sacroiliac Region

A low-grade form of sacroiliac disease is common in event horses and is manifest as a loss of impulsion and scope when jumping, with a reduced hindfoot flight arc. Most advanced event horses have bony pelvic asymmetry when critically assessed. Clinical signs include pain on palpation around the tubera sacrale and resentment of gentle lateral rocking action when the horse is in a weight-bearing position, with the opposite hindlimb held up. Often associated muscle soreness and spasm are present in the surrounding musculature, notably the middle gluteal muscle. Most of these horses appear to have sacroiliac joint instability, but minimal evidence of IRU is seen on scintigraphic examination. Clinical signs can be localized by assessing the response to infiltration of local anesthetic solution around the dorsal aspect of the sacroiliac joint, using a spinal needle angled ventrocraniolaterad from just caudal to the opposite tuber sacrale. Ultrasonographic evaluation of the ventral sacroiliac ligaments per rectum also can be helpful to localize any pathological condition. The condition usually occurs when the horse is unfit and being brought into work or when the plane of exercise is increased. Sacroiliac pain often is self-limiting because the musculature increases as the plane of exercise increases and stabilizes the sacroiliac joint. Administering a systemic NSAID such as phenylbutazone helps the horse work through the period of lameness. Physiotherapy can assist in treating the local muscle spasm, and some horses improve with chiropractic or osteopathic manipulation. Horses with refractory pain sometimes improve after the local infiltration of methylprednisolone (120 mg) or a sclerosing agent (50 ml; e.g., P2G Solution, Martindale Pharmaceuticals, Romford, UK) using the same approach as for local analgesia. Horses are continued in the same plane of work, and improvement normally is reported within a week. The duration of response varies, and some horses do not require repeat treatments.

Stifle Fracture

Fractures of the patella are common in event horses,[9] and the imaging aspects have already been discussed. For medial fractures, surgical removal offers the best prognosis. The original surgical description is for an incision 15 to 25 cm in length, but I prefer to use arthroscopic guidance, enabling me to create a minimal surgical incision (5 to 7 cm) centered directly over the fragment. This is satisfactory for small fragments and also allows for thorough evaluation of the femoropatellar joint and easy removal of any loose fragments.

Other Fractures

Fractures caused by direct trauma of the distal phalanx and second and fourth metacarpal bones are not uncommon. Occasionally, condylar fractures of the third metacarpal or metatarsal bones or sagittal fractures of the proximal phalanx occur during training or competition, although they are rare. The standard considerations apply for evaluation and treatment. Vertebral or upper limb fractures can occur after a fall, and the initial evaluation can be complicated by adrenaline dominance.

Rhabdomyolysis

Recurrent rhabdomyolysis is rare in advanced-level horses, because horses prone to this disease are selected out at the lower levels. Sporadic episodes are common at Three Day Events, however. Many potential trigger factors exists, including the stress of travel, stabling away from home, the competition itself, dehydration, climate changes, electrolyte imbalance, and dietary changes. I have noted a particularly high incidence in horses that were switched to haylage products immediately before a competition. This practice is done frequently because of the greater convenience of the small, packaged bales. A period of at least 4 to 6 weeks is recommended to allow adaptation to the new diet. The clinical manifestations at a Three Day Event can vary between collapse and recumbency on the roads and tracks phase to slight stiffness developing many hours after the completion of the cross-country phase.

To reduce the risk of rhabdomyolysis, attention should be paid to avoiding the trigger factors. Vitamin E levels are frequently low in event horse diets. Many commercial electrolytes do not contain sufficient quantities of salt, and horses may be at risk if owners follow manufacturers' recommendations. Routine blood sampling to monitor the muscle-derived enzymes aspartate aminotransferase and creatine kinase is performed frequently, but considerable fluctuation in asymptomatic event horses can occur,[10] making interpretation difficult. This variation tends to decrease as horses become fitter, as the muscle cells appear less leaky. With the recognition of polysaccharide storage myopathy as a cause of rhabdomyolysis, some event horses have been successfully managed on a diet high in fat and low in soluble carbohydrates, although actual diagnosis is difficult to confirm.

PREVENTION OF LAMENESS

When considering the time involved to train a horse to the top level and the potential for a long athletic career, preventing lameness is especially desirable in event horses. However, many important factors are beyond the veterinarian's influence. The number and frequency of competitions, having the opportunity to choose events with good going and an appropriate riding surface, and a high standard of riding ability are factors difficult to control. The veterinarian may be able to assist with other factors such as a high standard of farriery and optimizing training programs. Regular veterinary monitoring can be valuable by allowing the early detection and treatment of any

lameness problems. Monitoring for incipient tendonitis can be useful by performing periodic clinical examinations, using thermographic and ultrasonographic examinations, and by monitoring serum markers of tendonitis. Serial determination of markers such as cartilage oligomeric matrix protein holds promise. Initial studies have shown that cartilage oligomeric matrix protein increases as training intensity increases, and higher cartilage oligomeric matrix protein levels are found in horses that subsequently developed tendonitis.[11] The concurrent use of all of these maximizes the veterinarian's ability to detect subclinical tendonitis.

Many medications and supplements are sold with the aim of decreasing the risk of orthopedic disease. In racehorses, intramuscularly administered polysulfated glycosaminoglycan and intravenously administered hyaluronan have been shown to decrease the number of races missed, although the incidence of injuries was not decreased. Thus these drugs seem to be acting as anti-inflammatory rather than as disease-modifying agents. Glucosamine has disease-modifying properties in vitro, has been shown to be efficacious in human studies, and is a sufficiently small molecule that absorption would be expected. A role for oral glucosamine supplementation in horses may be found, but further studies are required. Many commercial products are available with unproven efficacy and variable and uncertain composition.

The future of lameness prevention may be the modulation of skeletal development by early training. Waiting until the horse is skeletally mature before breaking it in means that the different loads of carrying a rider and performing athletic exercises are imposed on a skeleton with little adaptive capacity. Conversely, overworking an immature skeleton can lead to a high incidence of orthopedic injury, such as is seen in young racehorses. A compromise position may be found that theoretically could lead to the early development of a skeleton adapted for the loads that will be placed on it and with the development of a higher-quality extracellular matrix that will better withstand the work that it has to perform.

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CHAPTER • 119

Lameness in Endurance Horses

Martha M. Misheff

DESCRIPTION OF THE SPORT

The ancient Bedouin raced their horses long distances across the desert, British and American cavalry units used endurance tests as part of their military training, and nineteenth-century Austrians had a Vienna to Budapest ride, but organized endurance riding is a young sport. The first modern endurance ride, the 100-mile (160-km) Tevis Cup from Nevada to California has been run every year since 1955. The Tom Quilty Gold Cup, a prestigious 160-km endurance race named for its founder, was established in 1966 and is held every year in a different part of Australia. The oldest endurance organization, the American Endurance Ride Conference, has been in existence only since 1972. The American Endurance Ride Conference International was established in 1991.

The first Federation Equestre Internationale (FEI) European Championship was held in Florac, France, in 1984, and the first Endurance World Championship in Rome, Italy, in 1986. Since then many recognized international competitions have been held under the auspices of the FEI, including the Endurance World Championship, held every 2 years in a different country. Many countries have endurance or long-

lameness problems. Monitoring for incipient tendonitis can be useful by performing periodic clinical examinations, using thermographic and ultrasonographic examinations, and by monitoring serum markers of tendonitis. Serial determination of markers such as cartilage oligomeric matrix protein holds promise. Initial studies have shown that cartilage oligomeric matrix protein increases as training intensity increases, and higher cartilage oligomeric matrix protein levels are found in horses that subsequently developed tendonitis.[11] The concurrent use of all of these maximizes the veterinarian's ability to detect subclinical tendonitis.

Many medications and supplements are sold with the aim of decreasing the risk of orthopedic disease. In racehorses, intramuscularly administered polysulfated glycosaminoglycan and intravenously administered hyaluronan have been shown to decrease the number of races missed, although the incidence of injuries was not decreased. Thus these drugs seem to be acting as anti-inflammatory rather than as disease-modifying agents. Glucosamine has disease-modifying properties in vitro, has been shown to be efficacious in human studies, and is a sufficiently small molecule that absorption would be expected. A role for oral glucosamine supplementation in horses may be found, but further studies are required. Many commercial products are available with unproven efficacy and variable and uncertain composition.

The future of lameness prevention may be the modulation of skeletal development by early training. Waiting until the horse is skeletally mature before breaking it in means that the different loads of carrying a rider and performing athletic exercises are imposed on a skeleton with little adaptive capacity. Conversely, overworking an immature skeleton can lead to a high incidence of orthopedic injury, such as is seen in young racehorses. A compromise position may be found that theoretically could lead to the early development of a skeleton adapted for the loads that will be placed on it and with the development of a higher-quality extracellular matrix that will better withstand the work that it has to perform.

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CHAPTER • 119

Lameness in Endurance Horses

Martha M. Misheff

DESCRIPTION OF THE SPORT

The ancient Bedouin raced their horses long distances across the desert, British and American cavalry units used endurance tests as part of their military training, and nineteenth-century Austrians had a Vienna to Budapest ride, but organized endurance riding is a young sport. The first modern endurance ride, the 100-mile (160-km) Tevis Cup from Nevada to California has been run every year since 1955. The Tom Quilty Gold Cup, a prestigious 160-km endurance race named for its founder, was established in 1966 and is held every year in a different part of Australia. The oldest endurance organization, the American Endurance Ride Conference, has been in existence only since 1972. The American Endurance Ride Conference International was established in 1991.

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distance riding associations, all of which have been established only in the last 30 years.[1] All of the organizations require that horses be at least 5 years of age to compete at distances of 80 km (50 miles) or more, and all of the organizations have stringent veterinary controls.

Competitors undergo an initial veterinary inspection. The total distance of the ride is divided into segments. After riding each section the competitor must pass a vet gate or veterinary checkpoint where a panel of veterinarians evaluates the horse for lameness and metabolic criteria. The heart rate must be at or under a maximum criterion (usually 64 beats per minute) set before the ride by the veterinary commission. A cardiac recovery index often is used, in which a second heart rate is measured 1 minute after the commencement of an approximate 80-m trot, during which time the horse is evaluated for lameness. Although not a criterion for elimination in and of itself, a positive cardiac recovery index, in which the second heart rate is elevated more than a few beats above the first one, is an indication that the horse merits further evaluation. Other metabolic parameters, such as mucous membrane color, capillary refill time, skin turgor, and auscultable intestinal motility are checked and recorded. Any saddle sores, girth rubs, or bit-related lesions also are noted. A heart rate above the maximum allowed, lameness, injury, and synchronous diaphragmatic flutter are grounds for elimination. Endurance horses are not permitted to compete on medication, and regulatory bodies conduct random testing for those substances that are not permitted. Some jurisdictions routinely test the first several finishers. Although smaller competitions often use the same veterinarians for control and treatment, many of the larger competitions now have separate veterinarians dedicated to treatment. Typically a horse that is eliminated by a control veterinarian for a lameness or metabolic problem is referred to the treatment veterinarian for further evaluation. If the treatment veterinarian deems that treatment is prudent, treatment may be provided at a field clinic at the ride site, or the horse may be transported to a local veterinary clinic with which prior arrangements have been made. Ride site treatment clinics at the larger venues are able to offer relatively sophisticated care, and only those horses that require continued care or further investigation are referred.

TYPE OF HORSE

Most endurance rides are open to horses of any breed, but horses of Arabian extraction are most popular for endurance riding because of their light build and stamina over long distances. Larger, heavier framed horses are less able to sustain the speed required over the distances traveled. A few Middle Eastern endurance rides are restricted to purebred Arabians or Arabian crossbreeds.

TRAINING METHODS

Methods differ considerably among trainers, but most would agree that horses take 2 to 3 years of training to make good endurance racers. Training distance and frequency depend largely on individual circumstances (terrain, weather, and availability of pasture or turnout paddocks), but in general endurance horses are ridden at least several times a week. A typical program might include rides of 10 to 20 km with 40- to 50-km rides at weekly or 10-day intervals. Horses are ridden at a walk, trot, and canter with the proportion of the time spent at each gait varying considerably among trainers. Horses aiming for a 100- to 160-km ride usually complete 60 km or more in a training ride on at least two occasions before the competition. Some endurance organizations require a horse to complete a qualifying ride at a lesser distance before being permitted to compete at the next distance level. Overtraining of endurance horses tends to be more of a problem than under-training, particularly by novice trainers. In some countries endurance competition is rapidly evolving from an amateur sport in which the same person owns, trains, and rides the same horse for many years, competing for pleasure with little financial reward, to large multiple-horse stables run by professional trainers with riders that may never have ridden their horses before, competing for high-value prizes. Even if the prize money is a paltry sum, compared with other sports, the value of a horse winning or running successfully in a prestigious international competition may increase exponentially. This amateur to professional shift has meant that the speed required to win international competitions over flat terrain has increased dramatically. Speeds at high profile rides over mountainous terrain have not changed substantially. Essentially the sport has changed at some venues from endurance riding to endurance racing. The winner of the 1998 World Championship in the United Arab Emirates averaged 17.77 km/hr for 160 km. The winner of the 2000 World Championship in France averaged 16.96 km/hr over 161.7 km, and at some competitions the winning speed has increased to greater than 25 km/hr. This is the equivalent of a continuous fast canter or gallop. Years ago this would have been unheard of, and opinions differ as to whether this is desirable progress. From a veterinary standpoint, at rides where speeds are increasing we are beginning to see injuries more like those seen in flat racing horses, as well as serious metabolic abnormalities, because horses tend to be pushed harder when high prize money is at stake.

COURSE TERRAIN

Endurance horses compete and train over some of the most highly variable terrain of any sport horses. They go up rocky mountains, through creeks, across sandy deserts, along tarmac and gravel or dirt roads, across grassy fields, and on other surfaces depending on where the ride is held. Course terrain has a bearing on the types of injury in a predictable way. On rocky ground more horses have stone bruises and painful joint injuries, whereas on soft or sandy ground more horses have ligament and tendon injuries.

CONFORMATION AND LAMENESS

Lameness in endurance horses essentially can be divided into ligament and tendon injuries, muscle problems, injuries to joints and feet, and miscellaneous less common causes. Lameness also can be separated into two categories: first, transient problems that may be cause for elimination on the day of competition but then resolve, and second, more persistent problems that are likely to be recurrent. Endurance horses, compared with other athletic horses, tend to be scrawny, scraggy-looking beasts. Like human marathon runners, the good ones are ectomorphs. Because endurance horses often compete over an 8- to 10-year period, most horses with glaring conformation defects tend to weed themselves out by attrition. Plenty of moderately toed-in, toed-out, sickle-hocked, post-legged (straight-legged), calf-kneed, bench-kneed, or club footed endurance horses are used. Provided the conformational abnormality is not extreme, the overall function of the horse may not be compromised. A toed-in horse may develop splint exostoses that impinge on the suspensory ligament (SL), causing desmitis that may be recurrent; such horses ultimately may be unsuitable for endurance use.

LAMENESS EXAMINATION AND PROCEEDING WITHOUT A DIAGNOSIS

A lameness examination in any horse is performed most easily and efficiently when the lameness is visible, and the endurance horse is no exception. Making a specific diagnosis concerning the cause of a horse's lameness is not possible if the horse is not lame at the time of examination. Watching the horse trot while lame, applying hoof testers, and noting any palpable abnormalities and response to flexion or other manipulative tests usually help to narrow the possible causes. Adhering to the principle of starting at the bottom and working upward during diagnostic analgesia is a particularly useful means of localizing the source of pain.

Sometimes, despite a careful and complete lameness examination, diagnostic analgesia, and ancillary diagnostic aids such as radiography, ultrasonography, and scintigraphy, a specific diagnosis cannot be reached. In some horses lameness can never be eliminated by local analgesia. In others pain can be localized to a specific region, but no lesion can be found. A horse with lameness localized to a specific region by local analgesia—with no identifiable radiographic, scintigraphic, or ultrasonographic abnormalities—should be re-examined by the most appropriate imaging modality after 2 to 4 weeks. Stress fractures are particularly notorious for not being visible on the first examination and being readily visible later. If the lameness has resolved and no abnormality is identified when the horse is re-examined, returning the horse to light training is reasonable. If lameness recurs, the lameness examination should be repeated.

Horses with a lameness that cannot be localized to a specific area should be evaluated for neurological disease, because this can be a cause of or mimic lameness. Generally, horses that have a lameness that cannot be localized are subjected to full body scintigraphy. If lameness resolves, scintigraphy can be repeated before training is resumed. This is probably less important in endurance horses than in Thoroughbreds, because of less risk of stress fractures turning into catastrophic fractures. Not all owners want to pursue a diagnosis to this degree, and sometimes despite every attempt at imaging and re-imaging the veterinarian is still left with a lame horse and no diagnosis. When this point is reached, benign neglect and a prolonged rest period (8 to 12 months) sometimes work miracles.

TEN MOST COMMON CAUSES OF LAMENESS

The following sections deal with the 10 most common causes of lameness in endurance horses (Box 119-1).

Suspensory Desmitis

The leading cause of chronic or recurrent lameness in endurance horses, regardless of terrain, is suspensory desmitis, most commonly proximal suspensory desmitis.[1-3] Suspensory desmitis may occur in a forelimb or hindlimb but is more common in front. Lameness typically develops in the later stages of a race, when the horse becomes fatigued, but is more likely to occur in the earlier stages in an unfit horse. Lameness from proximal suspensory desmitis may be sudden in onset and severe or more insidious. Often little swelling occurs, but careful palpation may elicit pain. Lameness associated with forelimb proximal suspensory desmitis often is accentuated by distal limb flexion. The diagnosis is confirmed by local analgesia. The origin of the SL may be blocked by the following methods:

1. Blocking the deep branch of the lateral palmar nerve at the level of the accessory carpal bone (proximal to its bifurcation into the medial and lateral palmar metacarpal nerves) and the lateral palmar nerve

Box • 119-1

Ten Most Common Causes of Lameness in Endurance Horses

Lameness	*Comments*
1. Suspensory desmitis	Suspensory desmitis is a common cause of chronic or recurrent lameness. More frequently localized to the origin than to the body or branches.
2. Foot problems	Bruising of the foot is a common cause of elimination during competitions. Laminitis is seen with increasing frequency as a sequela to serious metabolic abnormalities. Navicular problems are uncommon.
3. Myositis	Myositis may be a nuisance disease causing generalized shortening of the stride, bilateral forelimb or hindlimb lameness, and elimination from competition. In its more serious forms, coupled with metabolic abnormalities, myositis may be life-threatening.
4. Superficial digital flexor tendonitis	Tendonitis may be acute or chronic, low grade or severe. In general, endurance horses have a better prognosis for return to competition than do Thoroughbreds or horses that race faster over shorter distances.
5. Osteoarthritis of the fetlocks	Osteoarthritis can be present without causing lameness. Diagnostic analgesia helps in determining whether treatment is necessary.
6. Distal hock joint pain	Radiographic changes do not always correlate with clinical signs. Pain often is treated empirically and frequently occurs with sore feet and sore back (the terrible triad).
7. Paravertebral myalgia	Paravertebral myalgia is related to rider fatigue, occurring as the horse experiences muscle fatigue, and also is caused by sore feet and hocks causing gait alteration.
8. Splints	Splints are a common, often nuisance problem necessitating a short break from training.
9. Gluteal myalgia	Gluteal myalgia is related to strenuous work over long distances and may occur with primary forelimb lameness. Severe inflammation may occur with rhabdomyolysis.
10. Lumbosacral or sacroiliac pain	Pain also is related to rider fatigue and repetitive stress over long distances.

2. Direct infiltration
3. A high palmar and palmar metacarpal nerve block (high four-point)

Inadvertent desensitization of the carpometacarpal and middle carpal joints is rarely a problem with the latter blocks, because lameness in endurance horses seldom is localized to the carpus. If a high index of suspicion of proximal suspensory desmitis exists, but a horse does not improve with one of the three blocks, the block should be repeated or one of the other two should be tried, because some variability in response occurs. Ultrasonography is used to confirm diagnosis of proximal suspensory desmitis. Abnormalities include loss of echogenicity of the most dorsal fibers close to the third metacarpal bone immediately distal to the carpometacarpal joint, seen best in longitudinal images (Fig. 119-1). The farther distally the fiber loss extends (from zone 1 into zone 2), the worse the injury. The key to determining whether acute injury has occurred is symmetry. Both suspensory origins should be compared. Bilateral change often is present, but the clinically affected side appears less echogenic. If the SL appears normal with ultrasonography, inflammation of the ligament may exist but without accompanying detectable structural change. Horses in such condition have a better prognosis than those in which lesions are identified by ultrasonography. It is important to recognize that a horse that has undergone any strenuous work may have some degree of structural change in the proximal SL,[4] such as reduced echogenicity or fiber malalignment. Therefore diagnosis of proximal suspensory desmitis depends on response to local analgesia and comparison of the ultrasonographic appearance of the SL in each forelimb. Proximal suspensory desmitis is particularly treacherous to a horse's career because the initial lameness often responds to a short period of rest. The horse's caretakers are lulled into a false sense of security, and the horse is returned to work. Most of the time lameness recurs, which is why ultrasonographic evaluation is critical to management success. If substantial structural change is present, strenuous work should be avoided for at least 8 months. Radiography and scintigraphy are useful ancillary diagnostic aids, particularly in horses with avulsion of the origin of the SL (Fig. 119-2).

Initial management of horses with proximal suspensory desmitis should be aimed at reducing inflammation. Systemic corticosteroids (a single injection of triamcinolone acetonide, 0.03 mg/kg) along with 2 to 3 weeks of non-steroidal anti-inflammatory drugs (NSAIDs) and local therapy (ice, poulticing, and bandaging) are beneficial. The horse should be restricted to hand walking until no lameness is apparent trotting in hand on a hard surface without the influence of NSAIDs. If no structural change has been identified, the horse may walk under tack for an additional 2 weeks. The horse should be re-examined ultrasonographically to confirm that no substantial structural change is present, because lesions visible by ultrasonography can lag behind clinical signs. Assuming the ligament is structurally normal, training then may progress to gradually increasing periods of trotting for 1 month, and then normal training may be resumed. A horse with structural abnormalities of the SL should have the same initial local and anti-inflammatory therapy and 6 weeks of hand walking and then be re-evaluated with ultrasonography. If healing is satisfactory and the horse is quiet, turnout into a small paddock is recommended. Ultrasonographic examinations are repeated at 6-week intervals until 30 weeks, at which time trotting may be resumed if healing is satisfactory. Time for rehabilitation depends on the horse's temperament, the amount of help available, and the facilities available, but the best success is achieved with a controlled and gradual increase in exercise over 8 to 12 months. The bottom line has never changed: horses that sustain substantial damage to the SL or superficial digital flexor tendon (SDFT) need a long time off (8 months to 1 year) and will always be at higher risk of re-injury. The reason that conventional wisdom becomes conventional is that it has withstood the test of time.

Foot Problems

Problems with the feet are a common cause of elimination from endurance competition and are a common cause of chronic or recurrent pain. Foot problems may occur in the forelimb or hindlimb but are more common in front. Many

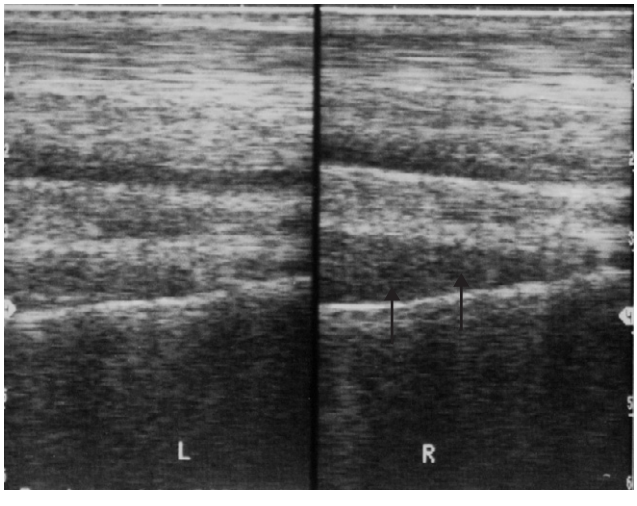

Fig. 119-1 Longitudinal ultrasonographic images of the palmar metacarpal soft tissues. The left forelimb is on the left. Proximal is to the right. There is marked loss of echogenicity and fiber pattern in the proximal aspect of the right forelimb suspensory ligament *(arrows)*. Some loss of echogenicity is also seen in the proximal aspect of the suspensory ligament of the non-lame left forelimb.

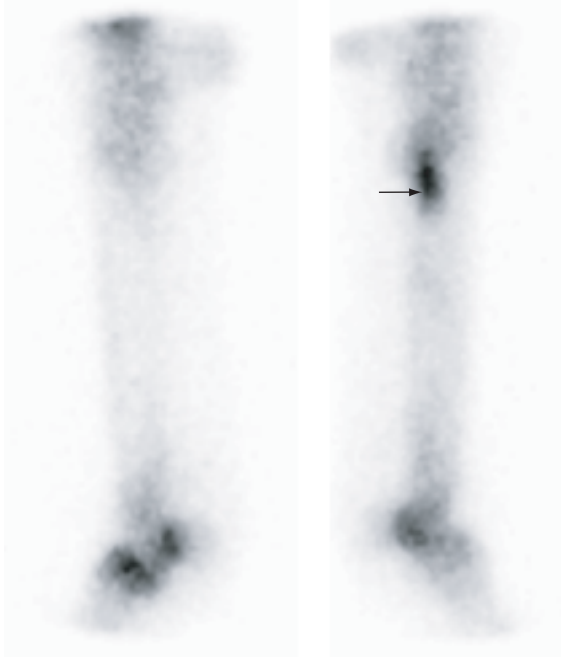

Fig. 119-2 Lateral bone-phase scintigraphic images of the left and right forelimbs. There is intense focal increased radiopharmaceutical uptake in the proximopalmar aspect of the right third metacarpal bone *(arrow)* at the site of attachment of the suspensory ligament.

problems are transient in nature, from bruising or a dislodged shoe. The importance of a properly trimmed, well-balanced, well-shod foot cannot be overemphasized. Because endurance horses go farther in a 24-hour period than any other horses, all of these things have a great impact, particularly on the support structures of the limb. Incredibly, the long-toe, low-heel syndrome still exists, even in endurance horses. This type of trimming and shoeing can lead to direct heel trauma and can increased strain on the SL and flexor tendons. Farriers who are accustomed to working with horses that wear keg shoes may be impatient with the requirements of endurance horses, which can sometimes require a more innovative approach aimed at providing the foot with increased protection and support. Inflammation of the solar structures occurs frequently in horses moving on hard ground. Lameness is manifest as a shortened, choppy gait, and hoof tester examination reveals a painful response around the solar margins. Lameness often is more pronounced in one foot. Diagnostic analgesia (abaxial sesamoid block) in the limb in which the lameness is most noticeable may result in a switch in lameness to the other side. Inflammation of the solar structures may or may not be accompanied by remodeling of the distal phalanx (pedal osteitis). Radiographic changes associated with remodeling of the distal phalanx include decreased radiopacity at the solar margin and increased size and number of vascular channels, resulting in a ragged appearance. Management is aimed at protecting the solar margins and reducing concussion. Wide webbed shoes, orthotic shoes, full pads, or rim pads can be used to accomplish these goals. Inflammation of the distal interphalangeal joints also may occur and can be treated with intra-articular medication. Sore feet often occur in horses with hock and back pain (the terrible triad), and each area must be addressed separately, but the horse must be considered as a whole. Navicular syndrome is uncommon in Arabian and Arabian crossbreeds but should be ruled out using diagnostic analgesia and imaging.

Endurance horses are subject to traumatic laminitis, which often becomes apparent in the first 24 to 72 hours after a competition, and may range from mild but frightening to disastrous. Separation of the hoof wall from the coronary band may occur, serum often oozes from the coronary band, and a bounding digital pulse is present. These clinical signs are alarming but do not always correlate with the final outcome. In some horses the outer layer of hoof wall peels away from the coronary band, but the horse is not lame and no rotation or sinking of the distal phalanx occurs. The episode is marked by a distinct defect in the hoof wall that must grow out. Horses without rotation or sinking usually are able to return to endurance competition. Sometimes horses with the worst outcome (death) do not show much in the way of early warning signs. Any horse that begins to shift its weight or act uncomfortably after competition should be scrutinized carefully. Predicting which horses will have a satisfactory outcome and those which will not is not possible, therefore aggressive treatment aimed at halting the progression of laminitis should always be instituted (flunixin meglumine [0.25 mg/kg intravenously tid], acepromazine [0.025 mg/kg intramuscularly qid], pentoxyfylline [8.5 mg/kg PO tid], and dimethylsulfoxide [1 g/kg intravenously diluted to 10% to 20% solution sid to bid]). However, the horse should receive fluid therapy before administration of NSAIDs or acepromazine.

Exertional Myopathy

Exertional myopathy, rhabdomyolysis, or myositis in endurance horses is not so much a lameness problem per se as a part of a larger picture of fatigue and metabolic abnormalities. Exertional myopathy is manifest as stiffening and shortening of the stride in all limbs and may be accompanied by trembling and profuse sweating. The condition usually occurs in the earliest or latest stages of a competitive ride. Horses with severe exertional myopathy often have pronounced swelling and hardening of the gluteal muscles. Asymmetry of the gluteal muscles may be present. Fluid therapy (0.9% sodium chloride) is the mainstay of treatment. Horses with severe exertional myopathy require large volumes (50 to 80 L) of fluids. Horses with myoglobinuria should urinate normal colored urine before flunixin meglumine (0.55 to 1.1 mg/kg or 250 to 500 mg/450 kg) is administered. When in doubt as to the status of renal function, it is better to give a lower dose and repeat as necessary. Blood urea nitrogen and creatinine should be monitored if possible. Highly portable, relatively inexpensive, user-friendly blood analyzers now make it possible to monitor kidney and muscle enzymes, and electrolytes and packed cell volume conveniently in the field. Dimethylsulfoxide (l g/kg intravenously in a 10% to 20% solution) also may be beneficial. Dantrolene may be useful but is not available in an intravenous form and is expensive to use orally at recommended dosages (15 to 25 mg/kg). Horses that have suffered an episode of exertional myopathy or myositis should not be returned to training until muscle enzymes return to normal.

Superficial Digital Flexor Tendonitis

Tendonitis of the SDFT may be acute or chronic, low-grade or severe. Acute ruptures or partial ruptures do occur occasionally during competition. Initial management is directed at trying to reduce swelling and inflammation and to relieve pain. Ice, bandaging, and NSAIDs are used. Dehydrated horses should not be treated with NSAIDs until they are rehydrated. A horse that sustains a rupture or partial rupture of the SDFT has a poor prognosis for return to endurance competition. Horses with less severe damage of the SDFT can be more challenging to diagnose and treat. Training injuries occur frequently. The SDFT may be warm and tender on palpation, but lameness is usually not apparent. Ultrasonography should be performed to determine if fiber damage exists. The ultrasonographic appearance does not change initial management, but it determines whether the horse may continue training after a short rest period or whether a more prolonged rest period is required. Those horses with transient heat and tenderness, but no swelling or fiber damage, usually can be safely returned to training within 2 to 4 weeks after the resolution of clinical signs. If the tendon is enlarged or if fiber separation has occurred, a substantially longer absence from training and competition is required. Horses that sustain tendon fiber injuries in training or competition are managed similarly to flat racing horses. Management with one injection of triamcinolone acetonide (0.03 mg/kg), local application of ice for the first few days, bandaging, poulticing, and NSAIDs for 2 to 3 weeks is beneficial in reducing inflammation. Surgical splitting of those tendons with central core lesions, autogenous bone marrow grafting of split SDFT[5] and desmotomy of the accessory ligament of the SDFT may be beneficial, but whether these therapies are more beneficial than rest alone is unproven. Because the career of endurance horses tends to span many years, time is on the veterinarian's side, and the tendency is to treat endurance horses with superficial digital flexor tendonitis conservatively rather than surgically. Endurance horses with tendonitis of the SDFT have a better prognosis for return to competition than horses that race over short distances at higher speeds.

Osteoarthritis of the Metacarpophalangeal Joint

Osteoarthritis of the metacarpophalangeal joints and the distal joints of the hock occurs with similar frequency. This is hardly surprising, given the ongoing nature of the degenerative process and the fact that endurance horses may compete until they are 18 to 20 years old. Osteoarthritis of

the metacarpophalangeal or metatarsophalangeal joints may occur with or without joint effusion and may be unilateral or bilateral. Osteoarthritis is far more common in the forelimbs but also occurs in the hindlimbs. Flexion of the joint produces pain, and the joint often has a decreased range of motion and thickening of the joint capsule. Radiographic changes may be subtle, such as narrowing of the joint space, or may consist of more severe peri-articular osteophyte or enthesophyte formation and joint remodeling. Radiographic changes do not always correlate with clinical significance, so diagnostic intra-articular or perineural (low palmar and palmar metacarpal blocks) analgesia is used to confirm the site of pain. Acute, traumatic synovitis or capsulitis of the metacarpophalangeal joint may occur occasionally, but in general, long-term management of chronic osteoarthritis is the more common scenario, and management is no different from that in any other athletic horse. Local therapy (ice, poultice, and sweats), intramuscularly administered glycosaminoglycans, intravenously administered hyaluronan, and judicious use of an intra-articularly administered hyaluronan and corticosteroid combinations are beneficial. Intra-articular corticosteroid injections should be low dose (20 to 40 mg methylprednisolone acetate with 20 mg sodium hyaluronan per joint), used with a rest period of 3 to 10 days, and should be spaced as far apart as possible, no more than 2 to 3 times a year, because the idea is to prolong the horse's career. Competitions should be selected carefully and spaced appropriately.

Distal Hock Joint Pain

Distal hock joint pain is common in the endurance horse. Osteoarthritis of the tarsus usually affects the tarsometatarsal and centrodistal joints and is often bilateral, with one limb being more affected than the other. Joint effusion is not palpable, but occasionally during intra-articular injection of the tarsometatarsal joint one gets the impression that the pressure and volume of synovial fluid is increased. Affected horses respond positively to hindlimb flexion and are sensitive to the Churchill test (see page 56). Radiographic abnormalities include joint space narrowing and peri-articular osteophyte formation. Radiographic changes do not always correlate with clinical signs, but intra-articular analgesia is used less frequently in the hindlimb compared with the forelimb. If the clinical picture suggests distal hock joint pain, intra-articular medication is administered. A positive response is empirical evidence of a correct diagnosis. Distal hock joint pain frequently occurs concurrently with paravertebral muscle pain and sore front feet, the terrible triad mentioned previously. This probably results from a horse with sore front feet altering its gait in such a way as to cause strain in the paravertebral muscles and hocks. The best results are obtained when all three problems are treated simultaneously; otherwise, the pain in one area and gait alteration and soreness in another become a never-ending cycle.

Paravertebral Myalgia

Paravertebral myalgia is caused by a fatigued, unbalanced rider. Unfortunately, diminished rider capability usually is occurring at the same time that the horse is experiencing its own muscle fatigue. Horses ridden by fit, experienced riders are less subject to battering of the paravertebral muscles than those ridden by novices. Similarly, those horses carrying live weight are less likely to be battered by flopping lead pads than those carrying dead weight, because a fit, competent rider is able to adjust his or her weight distribution to help a tiring horse. Horses competing in FEI rides often have to carry a minimum weight of 70 to 75 kg.

A horse that has strained or injured the paravertebral muscles appears stiff or rigid, and instead of flexing and extending the spine normally when palpated, tends to squat or crouch. Paravertebral myalgia also occurs in horses that alter the gait from sore feet or sore hocks. Treatment consists of removing the inciting cause and reducing inflammation. Most horses respond to a 2-week rest period with NSAIDs and injection of the paravertebral muscles with an anti-inflammatory agent. The longissimus dorsi muscles are injected bilaterally about 3 cm lateral to the spine at 5 or 6 sites about 5 cm apart, from the mid-thoracic area moving caudally, with methylprednisolone acetate (200 mg) or estrone sulfate (50 mg), mixed with an aqueous solution of soluble salts of the volatile bases from Sarraceniaceae (Sarapin, 10 ml). Complementary therapies (acupuncture, chiropractic manipulation, and physical therapy) may be beneficial in certain horses but require well-trained and experienced therapists under veterinary referral.

Splints

Periosteitis and exostosis of the second and fourth metacarpal and metatarsal bones (splints) are usually nuisance problems that may necessitate a 4- to 6-week break from training. Radiographs of horses with splint enlargements should be obtained to rule out fractures. Ice, NSAIDs, and bandaging can be used to reduce inflammation. Cryotherapy also can be used to reduce inflammation and sometimes enables horses to train after several days. Most splints resolve with time and treatment, although a non-painful enlargement remains. If the exostosis continues to enlarge, training should be discontinued to avoid impingement on and damage to the SL. Sometimes suspensory involvement occurs before the problem is recognized. These horses require a longer rest period. Exostoses that enlarge to the point that they impinge on the SL and are far enough distal to allow removal should be removed surgically before the rest period.

Gluteal Myalgia

Inflammation of the gluteal muscles occurs with relative frequency in horses subjected to strenuous work over long distances. The superficial and middle gluteal muscles are painful on palpation and the horse may crouch down or move away during palpation. Swelling or asymmetry may be apparent in the acute stages, with elevation of serum muscle enzyme concentrations. Severe inflammation of the gluteal muscles and accompanying rhabdomyolysis is a serious problem that is discussed with metabolic abnormalities (see page 1002). NSAIDs must never be administered in the acute stages of gluteal muscle inflammation unless it can be ascertained that hydration and renal function are not compromised. Horses with less severe gluteal muscle inflammation usually respond to NSAID therapy and a short (2- to 3-week) rest period. Soreness in the gluteal muscles often occurs with forelimb lameness, because horses alter hindlimb gait to protect themselves. It is important to identify and treat the primary source of pain. Once that is accomplished, the gluteal muscle inflammation will resolve.

Lumbosacral and Sacroiliac Pain

Lumbosacral and sacroiliac pain occur in endurance horses from repetitive stress over long distances. Rider fatigue and diminished rider capability are again likely to be important components in the development of pain in the surrounding musculature. Muscle spasm is likely to exacerbate any existing instability in the sacroiliac region. Horses exhibit stiffening and shortening of stride, and pressure over the tubera sacrale produces a painful response. Scintigraphic examination shows increased radiopharmaceutical uptake. Manipulation and deep muscle massage to alleviate muscle spasm, performed soon after injury, may be helpful. NSAIDs in conjunction with rest help resolve inflammation.

METABOLIC PROBLEMS

Avoidance, diagnosis, and management of metabolic problems are the most controversial topics currently facing the sport of endurance. The pressures alluded to earlier are manifest in an increasing number of horses that require treatment for metabolic abnormalities. On one hand, veterinarians are becoming more adept at recognizing subtle signs of exhaustion and are becoming more willing to treat horses aggressively before they get into serious trouble. On the other hand, more horses are getting into serious trouble. The exhausted horse syndrome was described many years ago[6] and unfortunately is still evident today. Exhausted horses undergo massive but poorly understood fluid and electrolyte shifts that lead to multiple organ system compromise. Most commonly the first signs are a persistently elevated heart rate and a profound ileus that do not respond to fluid therapy, electrolyte supplementation, or analgesics. Synchronous diaphragmatic flutter may be present. Horses with ileus should be checked for the presence of gastric reflux. It cannot be stressed enough or overemphasized that exhausted horses, or those with severe myositis, must not be treated with NSAIDs until they are adequately rehydrated. To do so risks renal failure, and these horses are already in a high-risk category. If lameness or myositis is present, the temptation always is to treat the horse immediately with phenylbutazone or flunixin meglumine. *This temptation must be resisted*. Lame horses, even those without evidence of metabolic compromise, should be rehydrated before administration of NSAIDs. Horses with acute tendon or ligament injuries should have the affected limb placed in ice while they are undergoing rehydration before NSAID administration.

Exhausted horses or those with severe myositis should receive at least 10 L, and preferably 15 to 20 L of intravenous 0.9% sodium chloride (or other available isotonic fluid), before NSAID administration. Collapsed horses or those on the verge of collapse may be given prednisolone sodium succinate (0.22 to 1.1 mg/kg or 100 to 500 mg/450 kg). Two intravenous catheters, one of which is 10 or 12 gauge, should be placed. Catheters should be 140 mm (5.5 inches) long and should be sutured in place to prevent dislodgment and extravasation of fluid. Time is always available to do a sterile skin preparation before catheter placement; most of these horses recover, but infectious phlebitis is an unwelcome complication. Sterile skin preparation materials should be handy in a catheter kit. Exhausted, dehydrated horses are also at risk of pleuropneumonia and laminitis. Some horses already have been stressed before competition by being shipped long distances. Profound leukopenia may precede the onset of pleuropneumonia. Laminitis may strike several days after the initial episode of post-race exhaustion. Treatment for laminitis, discussed elsewhere (see page 1000), should be instituted in horses that are depressed, inappetant, leukopenic, or febrile.

Prevention of Metabolic Problems

Most metabolic problems could be avoided by common sense, but in the heat of competition, common sense is often lost. Both horse and rider inevitably get tired, but a shrewd rider who knows his horse should know when the horse has had enough. If a horse is not eating and drinking at rest stops or is reluctant to move forward, the horse should be stopped. Horses must be allowed to drink at rest stops. Hay fed to a well-hydrated horse acts like a sponge or water reservoir in the large intestine. Endurance horses should be encouraged to eat hay and should have free access to water before competition so that they will have a fluid reserve available for absorption from the large intestine. Providing oral electrolyte replacement is helpful if the horse is drinking, and using electrolyte preparations may encourage a horse to drink. Concentrated electrolyte preparations that are force-fed, however, may actually be detrimental if the horse does not drink, because by raising the tonicity of the gastrointestinal lumen, more fluid is drawn into the lumen from the circulation, contributing to dehydration.

Synchronous diaphragmatic flutter, or thumps, a condition in which the phrenic nerve is stimulated by atrial depolarization, causing contraction of the diaphragm and consequent thumping of the flank in time with the heartbeat, is a sign of serious electrolyte imbalance, most commonly hypochloremic metabolic alkalosis. Sometimes, a low total or ionized calcium level can be demonstrated. Rest, food, and water may be all that is required for the condition to resolve. If treatment is required, however, horses with synchronous diaphragmatic flutter invariably respond to intravenous calcium supplementation (100 to 300 ml 20% to 23% calcium borogluconate diluted in 2 to 3 L of saline solution or 5% dextrose solution given over 15 to 20 minutes to effect). It has been proposed that horses subject to synchronous diaphragmatic flutter be fed a diet that is low in calcium before competition, which may enable more efficient mobilization of calcium from bone reserves during periods of stress.[7]

Recently an additional examination has been added at selected veterinary checkpoints on some rides. The purpose of the additional examination is to try to identify those horses that, because of aggressive cooling and perhaps high circulating catecholamines, pass the initial veterinary examination but then deteriorate during the mandatory hold period. Without this second look these deteriorating horses return to the trail and may deteriorate further until treatment is required. Although identifying all those horses that are going to have metabolic problems is probably not possible, an additional examination appears promising in at least identifying some of them. Therefore implementing the second look should be supported and encouraged by riders, trainers, ride organizers, and veterinarians.

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CHAPTER • 120

Lameness in the Polo Pony

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HISTORY OF THE SPORT

Polo was the first equestrian sport in recorded history. With strong ties to military traditions, the game originated in China in AD 272 and was often substituted for war games in preparation for military battle. Polo evolved into an organized sport and spread into Greece, India, and China, where the British colonies adopted the game. During the nineteenth century, the game became more refined in Great Britain and eventually found its way into the northeastern United States in 1876. Today polo continues to be one of the fastest and most dangerous equine sports in the world. Polo is no longer a sport for only the wealthy, but an increasing number of small clubs start up each year that attract people of moderate incomes to take lessons, buy horses, and begin to play. The sport has become more complex, with international professional players competing year-round on different teams around the globe. Professional polo coaches, umpires, trainers, and breeders have thus emerged, solidifying polo as a genuine equine sport industry. Playing seasons in the northern and southern hemispheres are followed by nomadic players, grooms, horses, and spectators. During the winter months in the United States, thousands of horses enter Florida and California, where the tropical climate is inducive to world-class polo tournaments. During the spring, summer, and fall seasons these horses travel across the Midwest, up the northeast coast and into Canada. The high-goal season begins in the summer in England and Spain, but the season in Argentina commences in the fall.

POLO AS AN INDUSTRY

Today three types of polo are played: outdoor, indoor (arena), and snow polo. Outdoor polo is by far the most popular and is played on a large, finely manicured grass field measuring 274 m (300 yards) by 137 m (150 yards) (Fig. 120-1). Injuries are related to fatigue (because of the distances covered), stopping, turning, and speed. Arena polo is played in much smaller arenas and is more common in collegiate settings. Injuries tend to be less frequent and are usually impact related. Snow polo is regarded as a novel exhibition sport played on the surface of a frozen lake and produces surprisingly few injuries. Obviously, footing and surface conditions often can be responsible for the type of lameness seen. Heavy, soft, grass polo fields and deep, sandy, uneven exercise tracks are frequently responsible for proximal suspensory desmitis, suspensory branch desmitis, and metacarpophalangeal joint sprains. Hard fields, exercise tracks, and polo field sideboards may cause hoof and pastern region injuries and hard, fast ground predisposes horses to superficial digital flexor tendonitis.

In outdoor polo each team is composed of four players, and each member brings an average of seven to eight horses to the field. A game normally runs for six chukkers (a chukker is 7 minutes), and a different horse is used for each chukker. Therefore a single match may have 50 to 55 horses playing over 1½ hours. The number of horses that are required to mount a polo team make each owner's total investment much larger than that for other equine disciplines.

Polo requires the speed and stamina of a Thoroughbred or a Thoroughbred-cross horse, the ability to stop and turn quickly, and the boldness to meet and collide with other horses at high speed. Although called ponies, polo horses stand 15 to 16 hands tall, and mares are preferred to geldings at a ratio of 10:1. Most trainers look for a fine neck and throatlatch, a good strong shoulder, powerful hip, quiet disposition, and a responsive, light mouth. Many horses have not raced, so few racetrack-related injuries are found in polo ponies. Argentina, New Zealand, and Australia are the only countries that specifically breed large numbers of horses for polo competition. Horses indigenous to these countries tend to have more bone than those in the United States and Europe, rendering them slightly more durable. Argentina has historically produced the most horses used solely for polo. Since 1970

Fig. 120-1 Outdoor polo is the most popular format for the game today. Close proximity of horses and riders explains why polo ponies often develop injuries related to direct trauma. Polo is only played right-handed.

thousands of Argentine horses have been imported into the United States and Europe, primarily because such a large selection was available at low cost. During the 1970s, inexpensive American ex-racehorses were sold as polo prospects, but many had numerous orthopedic problems. Today the price of high-goal Argentinian polo ponies continues to rise, and as the cost of importing horses into the United States increases, economic demands necessitate a greater influx of American Thoroughbreds into polo.

Most horses are 3 to 4 years old when introduced to the game, and 2 years of training and playing generally are required before a pony becomes seasoned. Exceptionally talented horses are playing high-goal polo at 6 years of age. By the age of 12 to 14 years, speed usually has begun to diminish, and horses are sold to less demanding players. By the age of 15 to 16 years, depending on temperament, polo ponies may be sold to beginners before being retired.

Neck reining is paramount in training a polo prospect, because the rider uses only one hand for control. Wide range of movement, the ability to stop and turn quickly, and the ability to exhibit rapid bursts of speed are required. How well and smoothly the horse performs these maneuvers often determines the number of years the horse stays sound and competes successfully. In addition to schooling, fitness training consists of daily galloping (legging up). Ponies often are tied together in sets of four to five. This time-saving practice teaches the horses to travel more calmly together in close contact but can result in traumatic injuries to the lower limbs (Fig. 120-2).

Polo ponies are shod with special rim shoes in front that allow for traction and pivoting without applying excessive torque to the lower limb. Medial and lateral heel calks on the hind shoes are helpful for stopping abruptly but often result in coronary band and pastern region lacerations to other horses during competition. For safety reasons, the Great Britain Polo Association only permits a lateral calk on each hind shoe, whereas the United States Polo Association allows medial and lateral heel calks. The size limit for calks is regulated but seldom enforced. Therapeutic corrective shoeing is problematic in playing horses (those being used in polo competition), because they may lose traction and maneuverability. Some common shoeing modifications include squared, rolled, and rocker toes; elevated and full-shod heels; and padded soles. The standard support and protection afforded the horse during exercise are leg wraps and coronet boots. All legs are wrapped with double-layer rolled cotton bandages. Impact-resistant European racing boots may be added to cover the metacarpal regions to protect against mallet and hoof trauma. More recently, especially on previously injured limbs, cotton leg bandages have been replaced with neoprene fabric wraps that extend below the fetlock joint and provide additional support. Despite these additional protective barriers, horses may still injure tendons and receive skin laceration during a game or practice.

Drug testing of polo ponies is not yet compulsory in the United States, and no mention of prohibitive medication is addressed in the United States Polo Association rulebook; however, limited testing is done in Great Britain and France. Attending veterinarians often work for many competing teams within the same tournament and prompt assessment of injuries is important. Low doses of non-steroidal anti-inflammatory drugs (NSAIDs) commonly are used, especially in horses with wounds and solar bruising. The general aim is to have as many sound horses as possible sharing the workload during a match to avoid the practice of double-chukkering (same horse used for two chukkers).

Minor conformation abnormalities in polo ponies often can be overlooked, but some faults predispose ponies to specific injuries. Long toes and underrun heels may result in tendonitis of the deep digital flexor tendon (DDFT) and palmar heel pain. Toed-in horses are prone to develop lateral suspensory branch desmitis, whereas toed-out horses are more likely to injure the medial branch. Horses with long pasterns and long third metacarpal bones (McIII) are at increased risk of tendonitis of the superficial digital flexor tendon (SDFT).

The most common sources of lameness in polo ponies are similar to those seen in most other equine sports. Polo ponies are at higher risk for traumatic injury because of the high-impact play and the practice of tethering of horses in close proximity to other horses during shipping, exercise, and polo tournaments. Causes of lameness often seen concurrently include palmar heel pain and proximal suspensory desmitis

Fig. 120-2 Daily legging up is an important part of training polo ponies. Ponies often are tied together in sets of four to five, and although this practice saves time, horses in close contact are at risk of traumatic injury.

and osteoarthritis of the fetlock joint with chronic suspensory branch desmitis.

TEN MOST COMMON LAMENESS PROBLEMS

The following are the 10 most common lameness problems in polo ponies:

1. Tendonitis of the superficial digital flexor tendon
2. Osteoarthritis of the metacarpophalangeal joint
3. Proximal palmar metacarpal pain and suspensory desmitis
   a. Proximal suspensory desmitis and third metacarpal bone disease
   b. Body suspensory desmitis and splint bone disease
   c. Suspensory branch desmitis and sesamoiditis
4. Injury to the hoof and distal phalanx
5. Palmar heel pain including navicular disease
6. Osteoarthritis of the distal interphalangeal joint
7. Desmitis of the accessory ligament of the deep digital flexor tendon
8. Splint bone disease
9. Distal hock joint pain
10. Gluteal myositis and back pain

LAMENESS EXAMINATION

Horses should be stabled overnight so that they cannot warm out of subtle lameness. The horse is first examined in the stall and then as it walks from the stall. The horse is observed at a trot in a straight line on a hard surface and is circled in both directions. Most polo ponies are reluctant to lunge. If necessary, the horse may be observed under saddle, but Argentine ponies generally resist trotting when ridden.

A systematic examination at rest is begun with the hoof and hoof tester evaluation and then continued proximally in the limb, noting evidence of pain, swelling, or obvious injury. Findings always should be compared with the contralateral limb, especially when palpating the body of the suspensory ligament (SL). Joints are assessed for range of motion and a painful response to flexion. Lower limb flexion is followed by carpal or tarsal flexion. Walking the horse briefly between flexion tests to allow an aggravated response to wear off is wise.

One of us (P.J.M.) attends to many older polo ponies that have effusion of the metacarpophalangeal joints, manifest a positive response to flexion, and even may have visible and radiographic evidence of osteoarthritis, but lameness often is abolished using low palmar digital analgesia. Palpation may reveal one or more fractured splint bones with callus, but the rest of the limb should be examined, because the cause of lameness may be elsewhere.

If a definitive diagnosis cannot be made, diagnostic analgesia is performed. Because drug tests are not performed, local anesthetic solutions can be used for diagnostic purposes in actively competing horses. When performing a nerve block, it is important to remember that the block may affect a larger area than intended, primarily related to diffusion of local anesthetic solution to surrounding tissue. High palmar analgesia can mask middle carpal joint pain, and an abaxial sesamoid nerve block can eliminate pain associated with the fetlock joint. For this reason the horse should be observed at the trot shortly after injection of local anesthetic solution and then again after an appropriate wait.

To reduce time and money spent on lameness diagnosis one author (P.W.) prefers to block large areas during the initial examination. Specific blocks then are performed, if necessary, the following day. For example, a horse that shows neither sensitivity to hoof tester examination nor an increased digital pulse may go sound after an abaxial sesamoid nerve block. The following day the same horse may show slight improvement after palmar digital analgesia and a 100% improvement with intra-articular analgesia of the distal interphalangeal joint. However, two authors (P.J.M. and M.W.R.) prefer to start distally and work proximally in systematic fashion.

Intra-articular analgesia is used extensively in polo ponies because it is more specific than perineural analgesia. Although intra-articular analgesia requires aseptic preparation and carries a small risk of infection, clients are generally receptive. If lameness is localized to a specific joint on clinical examination, therapeutic agents such as corticosteroids and hyaluronan can be added to local anesthetic solution to confirm diagnosis and initiate treatment simultaneously. One author (P.W.) uses combination diagnostic and therapeutic arthrocentesis typically in the distal interphalangeal and proximal interphalangeal joints. The horse's immediate response to local analgesia is noted, and response to therapy is usually evident 2 to 3 days later. Another author (P.J.M.) frequently uses combination diagnostic and therapeutic injections in the distal interphalangeal and distal hock joints. If treatment is not combined with intra-articular analgesia, injection of the joint with therapeutic medication should be delayed for 2 to 3 days. Combination injections can also be used simultaneously to diagnose and treat back pain. The dorsal aspect of the dorsal spinous process and the interspinous space can be infiltrated with a combination of local anesthetic solution, Sarapin, and a corticosteroid. Response to infiltration is evaluated immediately by riding the horse after injection, and response to medication is evaluated over the next several days.

Because metacarpophalangeal joint disease and splint bone injury are common sources of pain in the polo pony, one author (P.J.M.) prefers specifically to differentiate these sources of pain by first performing intra-articular analgesia of the metacarpophalangeal joint and then later performing a low palmar block. If low palmar analgesia is performed first, both potential sources of pain are eliminated. If pain is detected on palpation of bony exostoses of the splint bones, these areas can be blocked first, before a systematic blocking strategy is followed. One author (P.J.M.) refers to this as the splint block. This block is performed by first blocking the palmar metacarpal nerve distal to the exostoses. If improvement is not seen, the palmar metacarpal nerve just proximal to the exostoses is then blocked (2 ml of local anesthetic solution). A biaxial splint block can be performed if exostoses are found medially and laterally. This block should be done well below the origin of the SL to clearly differentiate proximal suspensory desmitis from splint bone disease. Splint disease, mainly from direct trauma from mallets and calk trauma, is common in the hindlimb. Diagnostic analgesia is performed as described in the forelimb.

Hindlimb proximal suspensory desmitis has become a common diagnosis because we are now more aware of it. In the United Kingdom a variation of the high plantar nerve block is commonly used to diagnose proximal suspensory desmitis. Three ml of local anesthetic solution is injected deep to the proximal aspect of the lateral splint bone and 2 ml each is placed over the medial and lateral plantar nerves. If this block is unsuccessful in abolishing pain, each hock joint compartment is blocked subsequently. This procedure then is followed by fibular and tibial nerve blocks. In Argentina chemical neurolysis (long-term nerve block) of the fibular and tibial nerves frequently is performed for horses with distal hock joint pain or proximal suspensory desmitis (P.J.M.).

UNDIAGNOSED LAMENESS

In some horses the lameness is inconsistent and/or subtle, and diagnostic analgesia cannot be performed. Nerve trauma on

the medial aspect of a proximal sesamoid bone (PSB) may cause episodic, transient severe lameness. An option in a horse with mild inconsistent lameness is to treat with phenylbutazone (2 g PO bid) for 5 days and then reassess the horse. If lameness resolves and does not return after treatment is discontinued, the horse gradually is put back into work. In horses with inconsistent lameness that fail to respond to rest or therapy, we recommend nuclear scintigraphic examination. Exercise intensity can be increased in horses with subtle lameness but is done so with caution. Lameness may become more apparent to the point which diagnostic analgesia can then be performed. In a horse with recurrent episodes of hindlimb lameness, the veterinarian should be aware of the possibility of an ilial stress fracture (S.K.).

Occasionally a polo pony becomes acutely non-weight bearing, with lameness lasting only a few minutes and resolving before examination is possible. If this sort of episode becomes recurrent in the same limb and physical examination reveals no significant findings, we refer the pony for nuclear scintigraphic examination.

Consultation with colleagues and seconds opinion are always options. It is also important to consider the option of extended turnout. Because the career of a polo pony can last 12 to 15 years, owners are often willing to give the horse 6 to 12 months of turnout to avoid any further injury. If subtle lameness resolves with phenylbutazone therapy, the polo pony can compete because there is no drug testing in polo competition. This option must be elected with caution, however.

Several lameness problems may exist simultaneously in a polo pony, a fact that makes observing the primary or baseline lameness difficult. Subtle signs such as the failure of a horse to stop appropriately, a horse that jumps on after stopping, or a horse that turns one way or the other when stopping (which is probably from outside hindlimb pain; horses turn away from lameness) may reflect low-grade lameness. If these observations have been made, having the horse ridden to witness the problems firsthand is useful. In one author's experience (P.J.M.) the most common source of pain in this type of situation is from the distal hock joints.

IMAGING CONSIDERATIONS

Conventional and computed radiography are the mainstays of imaging, with the front feet and front fetlock joints and hock joints being examined most frequently. The introduction of computed radiography has provided great advantages, because with the exception of faulty positioning, obtaining non-diagnostic radiographs is almost impossible. Exposure can be adjusted at the time of processing, and subtle details that would be difficult to see on conventional radiographs can be detected and scrutinized easily with computed radiography. Images can be enlarged, and the contrast and brightness can be improved, which are important factors in the diagnosis of incomplete fractures.

Scintigraphic examination is particularly useful in polo ponies with undiagnosed lameness and in those with palmar heel pain, but it is not always helpful in horses with chronic lameness (S.K.). Motion-correction software has been an important innovation.

Ultrasonography is extremely important in evaluating the damage and healing processes in tendons and the SL in the forelimb and hindlimb. Transverse views are used more frequently in identifying lesions, whereas longitudinal images aid in assessing healing. Ultrasonographic evaluation of the supraspinous ligament is often useful in horses with obscure hindlimb lameness. We have not found thermography particularly useful in our practices.

Diagnostic arthroscopy in horses with osteoarthritis of the metacarpophalangeal or carpal joints can be valuable in evaluating the condition of joint surfaces. Palmar intercarpal ligament injury has been diagnosed in ponies with lameness localized to the middle carpal joint but lacking radiographic and scintigraphic signs. Tenoscopy and bursoscopy also can be useful diagnostically and therapeutically.

SUPERFICIAL DIGITAL FLEXOR TENDONITIS

Tendonitis of the SDFT is the most common soft tissue injury seen in polo ponies and is by far the most common reason for early retirement. Tendonitis can be divided into three categories by location on the limb—high (proximal), mid-metacarpal, and low (distal)—or by cause: trauma, speed, and fatigue.

We believe that most peripheral injuries of the SDFT result from tendon trauma while the limb is bearing weight. However, peripheral injuries commonly are seen in other sport horses, such as Standardbred racehorses, in which direct trauma is usually not a factor (M.W.R.). These injuries occur much more frequently at the mid-metacarpal region on the lateral aspect and to a lesser extent on the palmar surface of the tendon (Fig. 120-3). Proximal tendonitis also can be caused by trauma (P.J.M.). These areas have a high degree of exposure to swinging mallets and flying hooves. Despite new protective boots the SDFT is still traumatized with surprising frequency. Traumatic tendon injuries are generally noticed 1 to 2 days after the incident and are characterized by a slight widening of the tendon (not a banana-shaped profile). Lameness is usually not present, but the area is warm and tender to palpation. Some horses have recurrent heat and swelling that resolves quickly with topical and systemic anti-inflammatory therapy. Peripheral lesions may involve 20% or less of the cross-sectional area (CSA) of the tendon. However, careful ultrasonographic examination of the medial and lateral borders of the SDFT and critical evaluation of longitudinal images are necessary. Recurrent tendonitis leads to typical

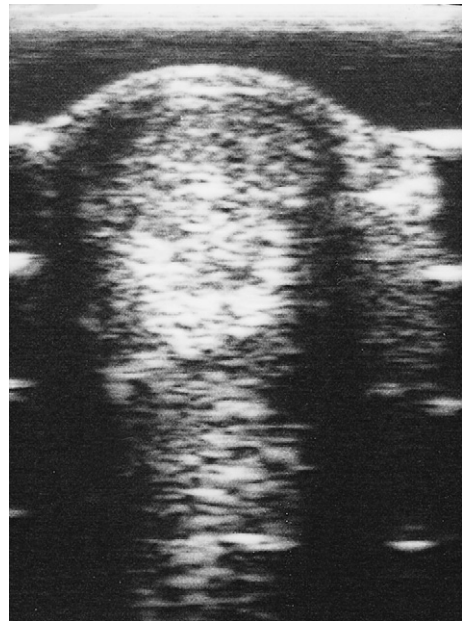

Fig. 120-3 Transverse ultrasonographic image of the palmar metacarpal region of a polo pony with traumatic tendonitis of the lateral aspect of the superficial digital flexor tendon. Lateral is to the left. After initial diagnosis the horse continued to play, and the lesion progressed.

swelling and later lameness commonly found with moderate or severe tendonitis of the SDFT.

Core lesions and lesions of the SDFT adjacent to the DDFT are thought to be injuries related to speed and fatigue. Hard, fast ground may a predisposing factor. Tendonitis of the SDFT may result in a banana-shaped profile of the metacarpal region (Fig. 120-4). Core lesions compromising between 20% and 25% of the CSA of the SDFT are serious, and the risk of recurrence is high. Horses with small CSA tears that extend more than 2.5 cm in length, or those with distally located tendonitis involving SDFT impingement by the palmar annular ligament, are at high risk of recurrence. Despite appropriate therapy these horses often have chronic and recurrent lameness, and ultrasonographic evaluation reveals a lesion that often fails to heal.

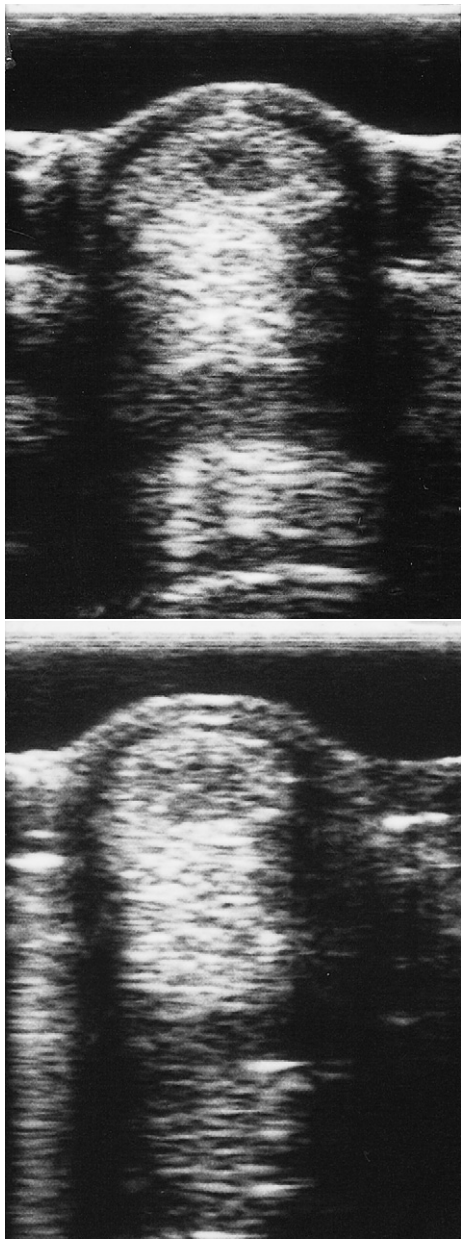

Fig. 120-4 **A,** Initial and **B,** 40-day follow-up transverse ultrasonographic images of the palmar metacarpal region of a polo pony with a typical core lesion of the superficial digital flexor tendon. Tendon splitting was done immediately after the initial image was obtained. The core lesion is more echogenic in the follow-up image but can still be seen.

Initial treatment for any polo pony with tendonitis regardless of location or cause includes cold therapy (ice boots and cold-water hosing), application of sweats and compression wraps, and administration of NSAIDs. Peritendonous injections of corticosteroids and hyaluronan may help to reduce inflammation, but it is important that corticosteroids are not injected directly into a tendon. Peritendonous corticosteroid injection is frowned on in the United Kingdom (P.J.M.). A combination of injection and rest or surgical management has been successful (M.W.R.). Tendon splitting within a week after injury appears to be beneficial in decompressing the lesion. Tendon splitting must be done early, because granulation tissue forms quickly and cannot be decompressed. After appropriate sedation, perineural analgesia, and aseptic preparation, 30 to 40 incisions with a No. 11 scalpel blade in a stabbing manner (a fan-shaped pattern is avoided) are used to decompress only the affected tendon segment. One author prefers using a double-edged tenotomy in a fan-shaped pattern (M.W.R.). The procedure can be performed with the limb in a weight-bearing or flexed position, but a weight-bearing position is preferred. With experience the texture of diseased tendon can be differentiated from surrounding normal tendon structure. Bandages are applied, and horses are confined to a box stall and given controlled, increasing hand walking over the next 8 to 10 weeks. Application of a cast to support the fetlock and to provide pressure is preferred by one author (S.K.) to tendon splitting. Topical blisters may be beneficial in increasing local circulation or inducing fibrosis and may eventually improve cosmesis and can be used alone or with tendon splitting (P.W. and P.J.M.). Some veterinarians favor bar firing or an internal blister using ethanolamine (S.K.). Controlled exercise is recommended for a minimum of 4 to 6 months before any form of turnout exercise is given, but client compliance with this timetable is poor unless a horse walker is available. Unfortunately, most horses are turned out to pasture after blistering within 1 month of injury, a conventional practice we feel is harmful to tendon healing. Total time of turnout exercise (after controlled exercise) is 8 to 12 months. Follow-up ultrasonographic evaluation is important to monitor healing and to determine when exercise level can be stepped up.

Other forms of therapy can be considered. Intra-lesional β-aminoproprionitrile fumarate injections have not been used extensively in polo ponies, because of the difficulty in garnering owner compliance with the required extensive rehabilitation program. Intra-muscular administration of polysulfated glycosaminoglycans (PSGAGs) may be beneficial (S.K.). Desmotomy of the accessory ligament of the SDFT (superior check desmotomy) has been useful in horses with large CSA core lesions (≥25%) and those with small CSA tears that are >2.5 cm in length. Combining the surgical procedures of desmotomy and tendon splitting early after injury has been successful in returning horses with tendonitis of the SDFT to polo. After surgery, horses are given stall rest and hand walking for 4 to 6 weeks, with a total of 4 to 6 months of controlled exercise, before being turned out or conditioned for polo. Desmotomy of the palmar annular ligament occasionally is performed when the SDFT is injured and enlarged just above the ligament. This surgery interrupts a self-perpetuating cycle of injury that develops between the enlarged tendon and the thickened palmar annular ligament. One author (P.W.) believes that horses with distal tendonitis of the SDFT involving the palmar annular ligament should initially be treated with peritendonous injection of short-acting corticosteroids followed by compression wraps, not only to reduce the size and cosmetic appearance of the SDFT but also to prevent the need for desmotomy of the palmar annular ligament.

The most common reasons for therapeutic failure in horses with tendonitis of the SDFT are lack of ultrasonographic follow-up after tendon splitting to determine if additional

therapy is needed, poor owner compliance concerning walking the horse and stretching the injured tendon in the early stages of healing, not allowing for adequate rest before the horse is returned to work, and failure to perform ultrasonographic examination at the time of initial injury, resulting in an inability to evaluate maximal medical improvement before returning the horse to unsupervised work.

OSTEOARTHRITIS OF THE METACARPOPHALANGEAL (FETLOCK) JOINT

Osteoarthritis of the fetlock joint is the most common articular problem in polo ponies and the most common articular problem necessitating early retirement. Ex-racehorses with mild osteoarthritis, osteochondrosis, or chip fractures may be sold for use as polo ponies and have a high risk of developing lameness. The fetlock region of any polo pony with forelimb lameness should be examined carefully. Lameness apparently originating from the fetlock joint based on the clinical observations of pain on palpation and a positive response to distal limb flexion must be differentiated from suspensory branch desmitis and splint bone disease. Intra-articular analgesia is important for differentiation. A full set of radiographs should be obtained. One author (P.J.M.) recommends using large cassettes to assess the fetlock joint and distal aspect of the splint bones. Common radiographic findings include fragmentation of the dorsoproximal aspect of the proximal phalanx, radiolucent areas in the distal aspect of McIII (Fig. 120-5), proliferative new bone formation on the palmar aspect of the PSBs, and mineralization of the proximal and distal fetlock joint capsule attachments (Fig. 120-6).

Intra-articular injections are the mainstay of treatment. One author (P.W.) injects both long- and short-acting corticosteroids and hyaluronan. Another author (P.J.M.) prefers PSGAG (Adequan) therapy or short-acting corticosteroids and hyaluronan or a combination of atropine, short-acting corticosteroid, and hyaluronan. Intra-muscular injections of PSGAGs at weekly intervals for 1 month, or possibly the entire season, are recommended. Horses that do not respond well to intra-articular injections are candidates for arthroscopic evaluation, because many of these horses have considerable cartilage damage. Daily icing, poulticing, and NSAID administration can help reduce inflammation in horses with chronic osteoarthritis. In horses with chronic osteoarthritis, radiographic changes may be extensive, but many horses are serviceably sound (Fig. 120-7).

Chronic proliferative synovitis (villonodular synovitis) is common in polo ponies with chronic osteoarthritis of the fetlock joint and may be associated with capsular tearing (S.K.). The dorsal aspect of the fetlock joint develops an apple-shaped appearance, with only mild or moderate effusion. Chronic proliferative synovitis is most common in horses previously used as racehorses and becomes evident after several years of polo. Plain

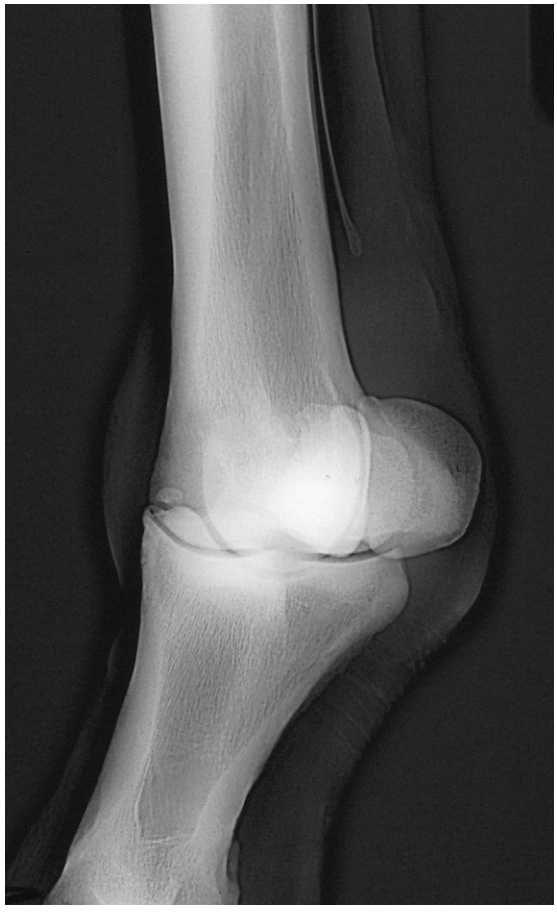

Fig. 120-5 Dorsolateral-palmaromedial oblique radiographic view of a metacarpophalangeal joint of a polo pony with osteoarthritis. Soft tissue swelling is apparent, a rounded osteochondral fragment is on the proximal dorsomedial aspect of the proximal phalanx, and proliferative changes involve the proximal aspect of the proximal phalanx and proximal sesamoid bones.

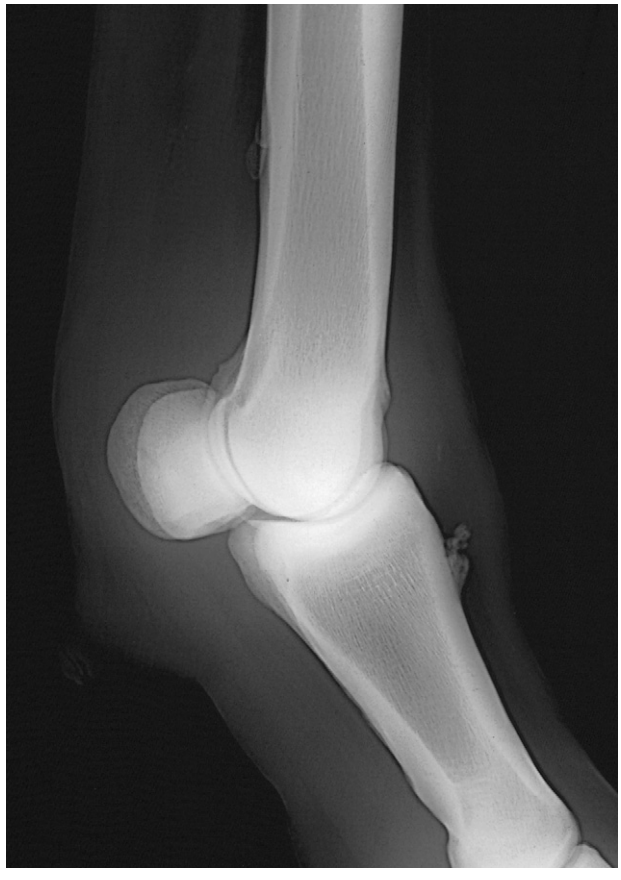

Fig. 120-6 Lateromedial radiographic view. Mineralization at the insertion of the common digital extensor tendon and metacarpophalangeal joint capsule on the proximal aspect of the proximal phalanx is a common radiographic finding in polo ponies with osteoarthritis of the fetlock joint. Although the finding is important, it does not preclude successful playing.

radiographs may reveal an abnormal contour of the distal dorsal aspect of McIII, and positive contrast radiographs may be diagnostic. We prefer ultrasonographic examination, because ultrasonography helps differentiate between horses that are surgical and non-surgical candidates. Chronic proliferative synovitis masses of 1 cm or larger should be removed arthroscopically to maximize long-term prognosis, whereas horses with smaller masses respond well to rest and intra-articular corticosteroid injections. Intra-articular atropine sulfate also has been used successfully (S.K.) to reduce effusion.

SUSPENSORY LIGAMENT

Suspensory desmitis is seen at three levels: proximal suspensory desmitis, suspensory body desmitis, and suspensory branch desmitis.

Proximal Suspensory Desmitis

Proximal suspensory desmitis occurs frequently and diagnosis is confirmed using high palmar or lateral palmar analgesia. Proximal suspensory desmitis results from polo ponies exercising on soft, uneven footing and is not related directly to playing polo. Lameness is usually only visible at a trot and varies in degree. The cranial phase of the stride is shortened, and lameness is usually most prominent with the affected limb on the outside of a circle. Palpation of the proximal palmar metacarpal region often reveals neither pain nor clinically appreciable enlargement of the SL. Polo ponies with proximal suspensory desmitis often fail to improve with rest and NSAID administration. Longitudinal ultrasonographic views are most useful in diagnosis of proximal suspensory desmitis. CSA measurements on transverse views are occasionally helpful if the same area is measured in the affected and contralateral limbs. One author (P.W.) has found that variation in CSA measurements leads to misdiagnosis, because even in normal polo ponies obtaining reliable repeat measurements is difficult. Hypoechogenic muscle tissue should not be confused as a lesion (S.K.).

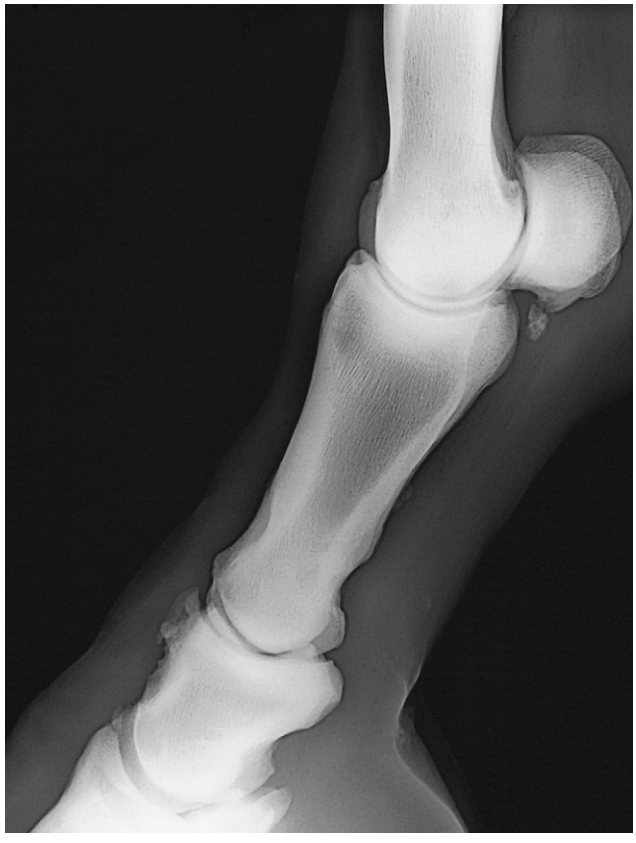

Fig. 120-7 Lateromedial radiographic view of the distal forelimb of a serviceably sound polo pony. There is modeling of the proximal sesamoid bone and the dorsoproximal aspect of the proximal phalanx, and osteoarthritis and osteochondral fragmentation of the proximal and distal interphalangeal joints.

Proximal suspensory desmitis causes more subtle lameness and clinical signs in polo ponies than in other sport horses. Subtle injury and enlargement may cause the ligament to be pinched or compressed by overlying dense fascia, especially in hindlimbs. Horses with long-standing proximal suspensory desmitis often have sclerosis of McIII visible in lateromedial or dorsopalmar views. Absence of sclerosis does not rule out proximal suspensory desmitis, because horses with soft tissue injuries often lack bony involvement. Avulsion fractures of McIII associated at the origin of the SL, incomplete longitudinal fractures of McIII, and stress reaction can occur independently or concomitantly to proximal suspensory desmitis. Radiographic and ultrasonographic examination may reveal small or large fragments, or proliferative changes and radiolucency, associated with the palmar cortex of McIII. Computed radiography, xeroradiography, and nuclear scintigraphy are beneficial in diagnosing bony injury and differentiating it from proximal suspensory desmitis. Follow-up radiographs may be necessary, because avulsion or longitudinal fractures may not show up on initial radiographs.

Even without treatment, almost all horses with proximal suspensory desmitis and bony causes of proximal palmar metacarpal pain recover within a 3-month rest period. In polo ponies with proximal suspensory desmitis in which a quick return to work is mandated, local injection into and around the origin of the SL of a combination of short-acting corticosteroids and PSGAGs hastens resolution of clinical signs. Ponies are walked for 1 week and then put in light work the second week. By the third week they may be galloped and are able to play shortly thereafter. Owners tend to keep polo ponies with proximal suspensory desmitis in work if the end of the season is near, because ponies are turned out routinely for 3 to 6 months after the season. Owners may gamble successfully by continuing to play the horse through the end of the season without permanently damaging the ligament. Proximal suspensory desmitis in a hindlimb, although rare, has a much more guarded prognosis, and one author elects neurectomy (S.K.).

In the United Kingdom and Europe, injection of corticosteroids into the proximal SL is frowned on because this medication may slow healing and may mask the presence of fractures (S.K. and P.J.M.). This is especially true in horses with acute injuries. Shock wave therapy is popular and may prove beneficial once additional clinical studies are available. In horses with chronic proximal suspensory desmitis, once bony involvement has been ruled out, local injections of corticosteroid and Sarapin or internal blister may be warranted. Horses with known bony injury should be given rest. Recently fasciotomy and bone marrow injection has shown promise in polo ponies with chronic, recurrent desmitis.

Body Suspensory Desmitis

Body suspensory desmitis is a serious and often career-ending injury. Diagnosis is straight forward if the SL is thickened and painful. Ultrasonographic examination is crucial in assessing SL damage, but radiography is important to evaluate the medial and lateral splint bones, because splint bone disease often is associated with suspensory desmitis in the polo pony. Radiographs should be obtained even if obvious areas of pain or bony and soft tissue swelling associated with the splint bones are absent. Treatment of polo ponies with acute desmitis

includes immediate application of cold or ice therapy, alternating with topical sweats, and administration of NSAIDs. Periligamentous infiltration of short-acting corticosteroids early after injury improves cosmetic appearance and may minimize adhesion formation between the SL and splint bones. Injections are performed in polo ponies only if the owner agrees the horse is in need of long-term rest. The horse gradually is returned to hand walking in 5 to 7 days and can be turned out after 3 weeks. Sclerosing agents (e.g., ethanolamine) injected into the SL may be helpful (S.K.). Two authors (P.W. and P.J.M.) feel that long-term box stall rest may increase the chance of adhesion formation. One author (M.W.R.) prefers controlled exercise rather than turnout exercise. Ultrasonographic evaluation of ligament healing is important. One author (P.J.M.) has observed many polo ponies with distal body suspensory desmitis that involves the bifurcation and invariably at least one branch. If desmitis at the suspensory bifurcation is severe, the polo pony many never fully recover. The best results are seen with a combination of periligamentous injections of dimethylsulfoxide (DMSO) and corticosteroids and long-term rest. Performance level may need to be dropped to junior polo, and even at this level lameness may be persistent or recurrent.

Suspensory Branch Desmitis

Suspensory branch desmitis is common in polo ponies. The lateral branch is injured more frequently than the medial branch, and occasionally both branches are injured simultaneously. Pivoting of the distal limb at high speeds is likely the cause of suspensory branch desmitis. Faulty conformation is another important predisposing factor. Horses that are toed in tend to develop lateral suspensory branch desmitis, whereas those that are toed out tend to develop medial suspensory branch desmitis. Palpation of the branches while the fetlock joint is in partial flexion is a preferred technique of one author (P.W.). Firm palpation and lower limb flexion followed by trotting exacerbates the degree of lameness. Ultrasonographic examination of the branches is accomplished easily and allows assessment of the degree of suspensory branch desmitis. Radiography should be performed to evaluate the distal aspects of the splint bones, because fracture and fracture displacement are common in ponies with suspensory branch desmitis. Mineralization in a branch close to attachment on a PSB may occur in horses with chronic injury (Fig. 120-8). Horses with acute injuries are treated identically to those with suspensory body desmitis. The appearance of the branch can be restored cosmetically to near normal over time with periligamentous injection of short-acting corticosteroids. The branch can be split 2 to 3 weeks after injury, although results in the United Kingdom have been disappointing (S.K.). Horses with suspensory branch desmitis need about 6 months of layup time before returning to polo training. One author (P.J.M.) feels that adhesions between the inflamed branch and surrounding tissue or the ipsilateral splint bone negatively influence prognosis, and corticosteroid injections may limit adhesion formation. Surgical adhesiolysis and distal ostectomy of the fractured splint bone may be useful in horses with chronic suspensory branch desmitis and splint bone fracture, but the cosmetic appearance is usually less than desirable. Counterirritants still are used in Europe with variable results. We feel that pin firing (hot firing) is not successful for horses with suspensory body desmitis, but it can be useful for horses with suspensory branch desmitis as a last resort. Shock wave therapy may be useful, but clinical studies are currently lacking.

INJURY TO THE HOOF AND DISTAL PHALANX

Polo more than other types of equine sporting activity predisposes horses to direct hoof trauma. Direct trauma results from swinging mallets; horses stepping directly on the hard polo balls used during playing; interference or direct impact from hooves of nearby horses; and from horses stepping on wooden sideboards at great speed. Calk or stud injuries to the hoof wall and pastern region are most common. Careful evaluation of wounds for involvement of deeper structures such as the proximal interphalangeal and distal interphalangeal joints is necessary. A common lameness that occurs during polo competition is called *getting stung*, referring to a sudden crippling lameness, lasting only a few minutes, generally resulting from a blow to the hoof or pastern by a mallet or hoof of another horse. The horse is usually sound within minutes of the incident with no clinical evidence of injury. Occasionally a fracture occurs that may not be evident radiographically for up to 14 days.

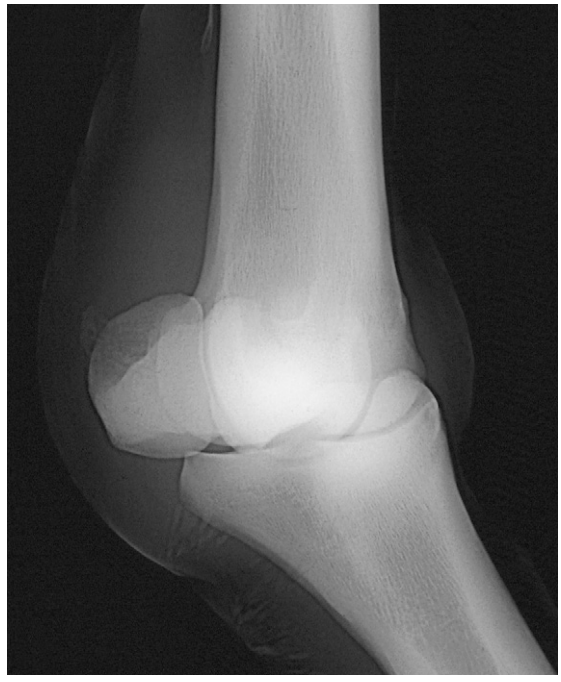

Fig. 120-8 Dorsolateral-palmaromedial oblique radiographic view of metacarpophalangeal joint, with mineralization in the lateral branch of the suspensory ligament at the attachment to the proximal sesamoid bone. Note also the soft tissue swelling.

Fractures of the Distal Phalanx

Acute fractures of the palmar processes of the distal phalanx often occur from mallet blows, and because polo is played right-handed, fractures usually are seen in the medial aspect of the left forelimb and lateral aspect of the right forelimb. Oblique fractures are sometimes difficult to see in conventional radiographic views and several proximodistal oblique views may be necessary. Rarely, fractures of the margin or of the extensor process of the distal phalanx are seen. Management of polo ponies with distal phalanx fractures is similar to that in other sport horses.

PALMAR HEEL PAIN INCLUDING NAVICULAR DISEASE

Palmar heel pain in polo ponies with Thoroughbred and Thoroughbred-cross ancestry appears to be decreasing, primarily because of the successful efforts of farriers and owners. The problem may be worse in American Thoroughbreds than in Thoroughbreds originating from the United Kingdom,

Australia, and New Zealand (P.J.M.). The long-toe and underrun heel complex and the tendency to shoe front feet with shoes with short branches that sit tight at the heel (to prevent front plates from being pulled off by hind feet) gradually have been corrected. Squared or rolled toe shoes, with or without elevated heels (world plates and natural balance plates), have reduced the number of polo ponies with broken pastern foot axes and have reduced DDFT tension due to prolonged breakover. Owners have allowed blacksmiths to reset polo plates more frequently during the playing season and to perform more frequent four-point trims during the off season than in previous years. Palmar heel pain is often difficult to differentiate from navicular disease. In both conditions the horse may show a painful response to hoof testers and lameness is abolished using palmar digital analgesia. Differentiation may be possible using distal interphalangeal analgesia, radiography, and scintigraphy. Many polo ponies with palmar heel pain have secondary pain at the origin of the SL, which is thought to be caused by alteration of gait.

A common source of palmar heel pain is laminar tearing at the heels. Sudden stops force the horse to use the heels of the front feet as brakes. Corns or heel bruising can cause poor performance and lameness. Corns are diagnosed easily using hoof tests and by carefully inspecting the seat of the corn for hemorrhage or discoloration. One author (P.J.M.) has seen many polo ponies toward the end of the polo season with bilateral, biaxial horn staining resembling chronic corns that apparently did not result in overt lameness. Management of polo ponies with palmar heel pain resulting from corns is similar to that used for undiagnosed palmar heel pain, including the application of wedge pads to relieve heel pain for a few games, or by using the four-point trimming method with natural balance or straight bar shoes and acrylic rubber frog and heel support. One author (S.K.) has found that four-point shoeing provides poor grip and traction and considers it unsuitable for polo ponies. These shoeing techniques appear to decrease stress on the heels and the DDFT and navicular region. Hoof growth stimulants and NSAID administration may help horses with palmar heel pain.

Osteitis of the Distal Phalanx (Pedal Osteitis)

Although definition and accurate diagnosis of osteitis of the distal phalanx remain obscure, many polo ponies with palmar heel pain have scintigraphic and radiographic evidence of disease in the margins of the distal phalanx. A lateromedial radiograph may show new bone projecting distally from the palmar aspect of the distal phalanx. This finding should be interpreted carefully on radiographs taken during purchase examinations.

Navicular Disease

Navicular disease in a polo pony is characterized by chronic forelimb lameness, which is abolished using palmar digital analgesia, intra-articular analgesia of the distal interphalangeal joint, or analgesia of the navicular bursa, often with little radiographic abnormality. Nuclear scintigraphy may be helpful to differentiate navicular disease from other causes of palmar heel pain. Most horses with early navicular disease respond positively to intra-articular administration of hyaluronan and corticosteroids in the distal interphalangeal joint. Horses with advanced navicular disease or those unresponsive to therapy may be dropped from medium- or high-goal polo. The administration of NSAIDs and isoxsuprine and the application of corrective shoeing techniques (see "Palmar Heel Pain") are valuable. Those ponies unresponsive to distal interphalangeal intra-articular injections may improve after injection of the navicular bursa, best performed under radiographic guidance. Palmar digital neurectomy is considered undesirable and rarely is performed today. Chemical neurolysis (long-term foot block) is of limited value. Cryoneurectomy can give limited relief but the palmar digital nerves regrow and lameness recurs. Concomitant or solitary injury of the DDFT does not appear to be as common in polo ponies as in other sport horses (P.J.M.). Shock wave therapy may offer a viable solution for these horses.

OSTEOARTHRITIS OF THE DISTAL INTERPHALANGEAL JOINT

Early (synovitis) and chronic osteoarthritis of the distal interphalangeal joint can cause lameness in the polo pony, but diagnosis can be challenging due to the lack of specificity of analgesic techniques in the foot. For example if more than 6 ml of local anesthetic solution is injected into the distal interphalangeal joint, and lameness is evaluated after 3 to 5 minutes (S.K.) or 10 minutes (P.W.), pain from other areas of the foot including the sole can be blocked inadvertently. This effect can be avoided by using smaller amounts of local anesthetic solution. Horses with synovitis have distal interphalangeal effusion and manifest a painful response to lower limb flexion. However, this test may be positive in horses with many sources of pain, including either the navicular bone or the fetlock joint. Management of horses with osteoarthritis of the distal interphalangeal joint usually includes intra-articular medication. Short- or long-acting corticosteroids are preferred to injection with hyaluronan. One author (P.J.M.) combines corticosteroids and DMSO, because DMSO may improve distribution of corticosteroids to all parts of the joint and the navicular bursa. The distal interphalangeal joint is the only joint in which one author (P.W.) uses PSGAGs, combined with a single intravenous dose of gentamicin sulfate (6.6 mg/kg). Another author (P.J.M.) favors using PSGAGs in horses unresponsive to injections with corticosteroids. Most of these horses have clinical and radiographic evidence of advanced osteoarthritis. In some polo ponies lameness does not improve directly after intra-articular analgesia but inexplicably resolves 24 to 36 hours later, when presumably the effects of the corticosteroid begin. After intra-articular injection, horses are given 3 weeks of limited exercise and a tapered dose of NSAIDs. Weekly intramuscular administration of PSGAG for a minimum of 30 days and the application of corrective shoes to ease breakover are recommended.

DESMITIS OF THE ACCESSORY LIGAMENT OF THE DEEP DIGITAL FLEXOR TENDON

Trainers and owners commonly confuse chronic desmitis or acute tears of the accessory ligament of the deep digital flexor tendon (ALDDFT) with bowed tendons. Desmitis of the ALDDFT occurs in polo ponies, especially older ponies (S.K.), but is less frequent than tendonitis of the SDFT. Mild desmitis is characterized by a meaty, non-tender swelling of the proximal palmar metacarpal region between the SL and DDFT. Horses with severe or complete tears can have prominent swelling at or near the junction of the ALDDFT with the DDFT, but neither lameness nor the response to palpation is commensurate with the degree of damage. Diagnosis must be confirmed and healing monitored using ultrasonographic examination. Initially, horses are managed with rest, cold therapy including ice, application of sweats, and administration of NSAIDs. Local infiltration of short-acting corticosteroids around (but not in) the ALDDFT can help cosmetic appearance. Stall rest for 2 weeks is recommended, and thereafter horses can be turned out for 3 to 4 months. Follow-up ultrasonographic examination reveals an enlarged, hyperechoic ALDDFT. Prognosis for future soundness and return to polo is usually excellent but swelling persists. Chance of recurrence is slim.

SPLINT BONE DISEASE

Traumatic exostoses (Fig. 120-9) caused by swinging mallets and fractures of the splint bones are common in polo ponies and are sometimes referred to as *bamboo fever.* Although trauma is usually the inciting cause of exostoses, injury of the SL may play a role (P.J.M.). Horses with splint exostoses resent direct palpation, but many do not manifest overt signs of lameness. If lameness is present but the exostosis is only mildly painful, a splint block (see page 1005) should be performed to confirm the diagnosis. Splint disease can make a veterinarian look foolish, because an acute injury may be confused with proximal suspensory desmitis, but subsequent development of a large golf ball-size swelling reveals the true diagnosis. Oblique radiographic views are most helpful for evaluating the splint bones.

Local infiltration of corticosteroids and Sarapin is used to treat polo ponies with acute splint exostoses. Other injections include a combination of corticosteroids, Sarapin, calcitonin, and medroxyprogesterone acetate. Calcitonin (400 IU) and medroxyprogesterone acetate (200 mg) are used commonly in Europe (P.J.M.). Rest, compression wraps or sweats, and the administration of NSAIDs are useful. Polo ponies with chronic exostoses seem to benefit from pin freezing (cryotherapy), and this is the treatment of choice, with a quick return to work in 7 days. Cryotherapy appears to stimulate remodeling of chronic proliferative splint exostoses, and often a cosmetically acceptable limb profile is seen within 6 months. Despite the resulting white spots, clients seem to accept this form of therapy because of a high success rate. Thermocautery (hot firing) has merit, and early results of shock wave therapy appear promising (P.J.M.).

Proximal splint bone fractures may require surgical fixation, and certainly horses need prolonged stall rest and a slow return to work. If fractures are non-articular and fragments are not displaced or are displaced minimally, prognosis is favorable.

Polo ponies with non-displaced diaphyseal splint bone fractures respond well to cryotherapy. Cryotherapy not only produces local analgesia, but also induces deep fibrous tissue formation that stabilizes fracture fragments. Probes are applied in firm contact with the skin covering the abnormal splint for 1 minute at each site at sites 1.5 cm apart. Ponies with non-displaced splint bone fractures are given rest for 4 to 6 weeks. Distal splint bone fractures can be diagnosed easily using longitudinal ultrasonographic evaluation or radiography. These fractures usually are associated with chronic suspensory branch desmitis, which is thought to cause bowing of the distal splint bone and subsequent displacement and proliferative changes (Fig. 120-10). Distal fracture fragments generally are removed surgically in the standing position, and concomitant splitting of the involved SL branch(s) often is performed, although results in United Kingdom have been disappointing (S.K.).

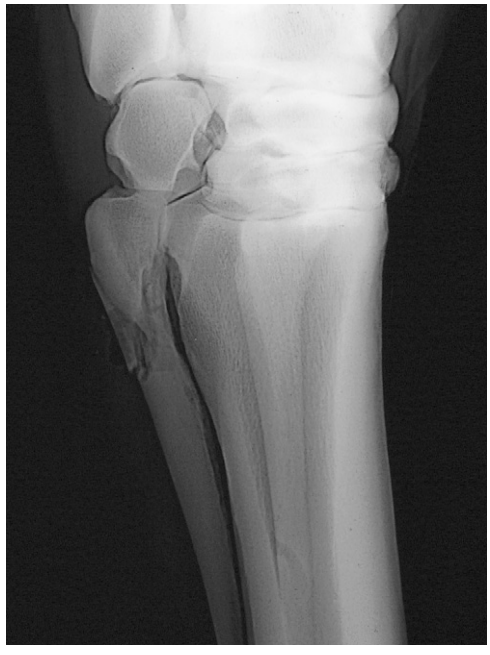

Fig. 120-9 Dorsolateral-plantaromedial oblique radiographic view of the metatarsal region. There is a comminuted fracture of the proximal aspect of the fourth metatarsal bone caused by mallet injury. This fracture usually does not require internal fixation, and prognosis for future soundness is good.

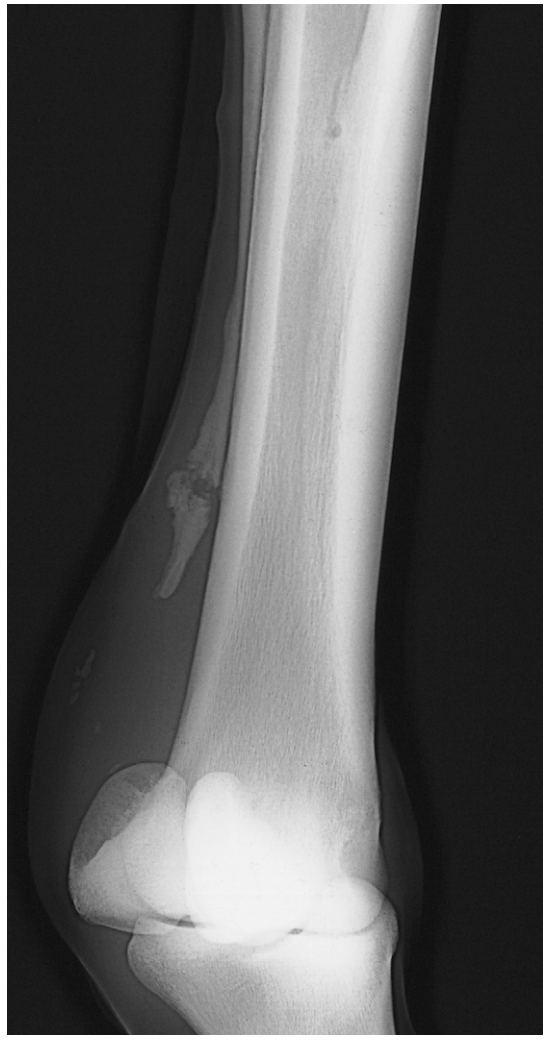

Fig. 120-10 Dorsolateral-palmaromedial oblique radiographic view of the metacarpal region. A chronic displaced fracture of the fourth metacarpal bone is associated with suspensory branch desmitis. The second metacarpal bone is bowed away from third metacarpal bone; chronic proliferation has resulted from instability; sesamoiditis is apparent, and the suspensory branch shows mineralization.

DISTAL HOCK JOINT PAIN

Polo ponies can play successfully with moderate to severe radiographic changes in the distal hock joints (Fig. 120-11). Most of the distal hock joint pain appears to be subclinical, meaning lameness is not the most noticeable clinical sign. Most polo ponies with distal hock joint pain are noticed by owners or riders to lack quick jump-out speed and the ability to stop abruptly and are noticed to be running through the bridle. These ponies generally improve if they are given ample warm-up time before playing. Distal hock joint pain appears to be one source of lameness with which a polo pony can live, but such pain is a major cause of poor performance and may induce compensatory lameness such as proximal suspensory desmitis in the forelimbs. Proximal limb flexion tests may elicit little response, and detecting pain or effusion using careful palpation is difficult in some horses. Because lameness is not necessarily proportional to radiographic changes, scintigraphic examination is helpful, especially in polo ponies with subtle hindlimb lameness. Diagnostic analgesia is an important tool if lameness is perceptible, and in horses with bilaterally symmetrical lameness, analgesia of one side may induce obvious contralateral lameness. Combining diagnostic and therapeutic injections is commonplace, because the practice saves time and money. The tarsometatarsal and centrodistal (distal intertarsal) joints are injected most commonly with methylprednisolone acetate, but in some ponies the tarsocrural joint also is injected. Intramuscular administration of PSGAGs and intravenous injection of hyaluronan throughout the playing season appear to be helpful in allowing horses to play up to potential. Horses are kept in work and placed on low doses of NSAIDs throughout the season, and training is limited.

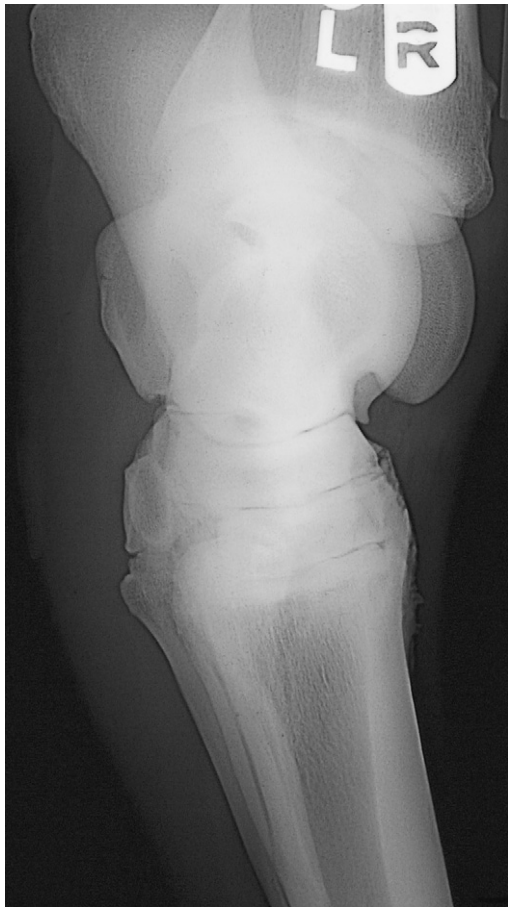

Fig. 120-11 Dorsomedial-plantarolateral radiographic view of a left hock with radiographic evidence of advanced osteoarthritis. This polo pony was not lame.

Although rare, avulsion injury and dislocation of the SDFT from the tuber calcanei is seen nearly once each year in a busy polo pony practice, resulting in extreme panic by the pony, necessitating sedation. The SDFT usually dislocates laterally, but diagnosis may be difficult before swelling develops. In our experience, surgical techniques including primary repair, mesh augmentation, and laterally located screws combined with full-limb cast application are neither successful nor necessary. Most horses respond well to confinement in a small pen and subsequent turnout for 6 to 12 months. Turnout exercise is recommended as soon as possible, and manually forcing the SDFT completely laterally is helpful if luxation is incomplete (P.J.M. and S.K.). Despite the fact that the SDFT remains displaced, causing a slight mechanical lameness, horses tend to perform well at the canter and gallop, with a fair to good prognosis for medium- and low-goal polo. Some polo ponies with lateral dislocation of the SDFT in one limb develop the same condition in the opposite limb 1 or 2 years later (P.J.M.).

GLUTEAL MYOSITIS AND BACK PAIN

Gluteal myositis often accompanies subclinical low hindlimb lameness and is often a compensatory problem. One or both hindlimbs shows a shortened cranial phase of the stride. Deep palpation of the gluteal muscles elicits pain, although the muscles never feel as hard as they do in ponies with rhabdomyolysis. If possible, it is important to determine if gluteal myositis is a primary or secondary problem. Guteal myositis can be differentiated easily from rhabdomyolysis, because serum creatine kinase and aspartate aminotransferase levels are invariably normal. Polo ponies with gluteal myositis can be treated with local injections directly into the gluteal muscles and between the biceps femoris and semitendinosus muscles. The horse is allowed light work and is placed on NSAIDs. One author (P.W.) has had modest success using a 5-day series of rubeola virus immunomodulator. If gluteal myositis is secondary, the primary source of pain must be identified and managed successfully.

Back pain in polo ponies is often secondary to lameness or results from mismanagement, including use of ill-fitting saddles, overweight amateur riders, and mouth problems. Poor dentition from lack of, or inappropriate, tooth floating procedures causes sharp molars to come in contact with gag bits. Horses carry the head and neck high to avoid pain and tend to hollow the back. Back pain can become a permanent or chronic problem if horses are mismanaged continually. Back pain generally is characterized by a painful response to palpation along the lateral edges of the longissimus dorsi muscles. The horse exhibits a crouching gait when mounted and during initial walking under saddle.

Intramuscular injections of corticosteroids combined with Sarapin (2:1 ratio) are performed along the length of the longissimus dorsi muscle, from the caudal border of the trapezius muscle to the level of the tuber coxae, 15 cm lateral to the dorsal midline. Injection sites are placed every 15 cm, and 5 ml of the mixture is administered at each site. Because back pain may be compensatory to primary distal hock joint pain or other lameness, it is important to evaluate the hindlimb and pelvis carefully. Primary management of the distal hock joint pain in horses with lameness in this region and local treatment of acupuncture points in the back is commonplace.

Dorsal spinous process (dorsal spinous process) impingement can be a source of back pain, and one author (P.J.M.) feels this is a major cause of back pain in the polo pony.

Diagnosis should be confirmed by assessing the effect of local analgesia and/or performing scintigraphy (S.K.). Treatment involves injection of the spaces between the dorsal spinous processes with corticosteroids and Sarapin. Shock wave therapy may be beneficial (P.J.M. and P.W.). Horses with back pain are given NSAIDs and are kept fit during a 4- to 6-week period using ponying (being led from another horse) exercise. In Europe a common management regimen includes paddock exercise as much as possible, lunging exercise with the horse's head down and little warm-up before polo games. Internal blisters have been used with some success, but care must be taken when injecting these compounds, because deep muscle abscesses can develop. Faradism is useful for longissimus dorsi and gluteal muscle strain (S.K.).

OTHER CONDITIONS

Fractures of the Cranial Thoracic Dorsal Spinous Processes (Fracture of the Withers)

Fracture of the withers is fairly common. Polo ponies are often tied next to each other for long periods, and a frightened horse occasionally rears up and flips over (Fig. 120-12), resulting in fractures of the longest dorsal spinous processes at the withers. Often up to four dorsal spinous processes are fractured, with ventral displacement of the fragments resulting in a flattened appearance of the withers. Ponies are usually only mildly painful to palpation but are generally reluctant to lower the head when grazing. The affected horse may travel with a painful, stiff, extended head and neck carriage, and some horses grunt with every stride. The prognosis for return to polo is excellent after rest for 4 to 6 months, as long as secondary infection does not develop. Special consideration should be given to saddle fit; a croup strap may be necessary to keep the saddle from sliding forward.

Osteoarthritis of the Proximal Interphalangeal Joint and Other Pastern Region Injuries

Osteoarthritis of the proximal interphalangeal joint (high ringbone) occurs occasionally in polo ponies and is seen most frequently in green horses playing on rough terrain in the western United States and generally results from irregular footing. Pain originating from the proximal interphalangeal joint is difficult to diagnose in horses with acute disease without radiographic changes, but it can be identified scintigraphically. Osteoarthritis of the proximal interphalangeal joint is often difficult to manage, and although motion of this joint is limited, lameness can be inappropriately severe. Rest (60 to 90 days), NSAID therapy, and intra-articular long-acting corticosteroid therapy are recommended.

A common finding in oblique radiographic views of the pastern region is periosteal reaction on the medial aspect of left forelimb and the lateral aspect of the right forelimb proximal phalanges (Fig. 120-13). Because polo always is played right-handed, powerful neck shots always hit the right side of each forelimb. Lameness is usually insignificant and short-lived. These radiographic changes should not be confused with those resulting from enthesitis at the attachment sites of the distal sesamoidian ligaments. To differentiate osteoarthritis of the proximal interphalangeal joint from these proliferative changes, intra-articular analgesia or pinpoint perineural or local analgesic techniques should be performed.

Sesamoiditis

Inflammation (sesamoiditis) of the PSBs can be caused by direct mallet or hoof trauma and by stress-related injury at the

Fig. 120-12 Polo ponies commonly are tied to rigid bars and in close proximity to each other. Occasionally a polo pony flips over and fractures the thoracic dorsal spinous processes at the withers.

suspensory branch insertions. Traumatic sesamoiditis occurs in the medial PSB in the forelimbs from interference injury and in the lateral PSB in the forelimb and hindlimb from mallet trauma. Diagnosis of sesamoiditis is straightforward using radiography and scintigraphy, but ultrasonographic evaluation provides information about the suspensory attachment as well. One author (P.J.M.) feels that shock wave therapy is a promising treatment modality for sesamoiditis.

Fractures of the PSBs are rare, and response to surgery is similar to that of other sport horses. Ponies with fractures of the base of the PSBs rarely return to athletic soundness with or without surgery. Polo ponies with basilar fractures and desmitis of the oblique distal sesamoidean ligament often can be helped by using shock wave therapy (P.J.M.). An apical fracture of the PSB may be mistaken for mineralization of the suspensory branch. Polo ponies with apical fractures of the PSBs have a good prognosis, provided that SL injury is not concurrent. Horses with apical fractures associated with sesamoiditis and insertional suspensory branch desmitis often have recurrent lameness. It is important to differentiate true fractures from sesamoiditis or a commonly seen radiographic abnormality in older polo ponies, one to three stress lines in the PSBs, which most often are considered incidental changes.

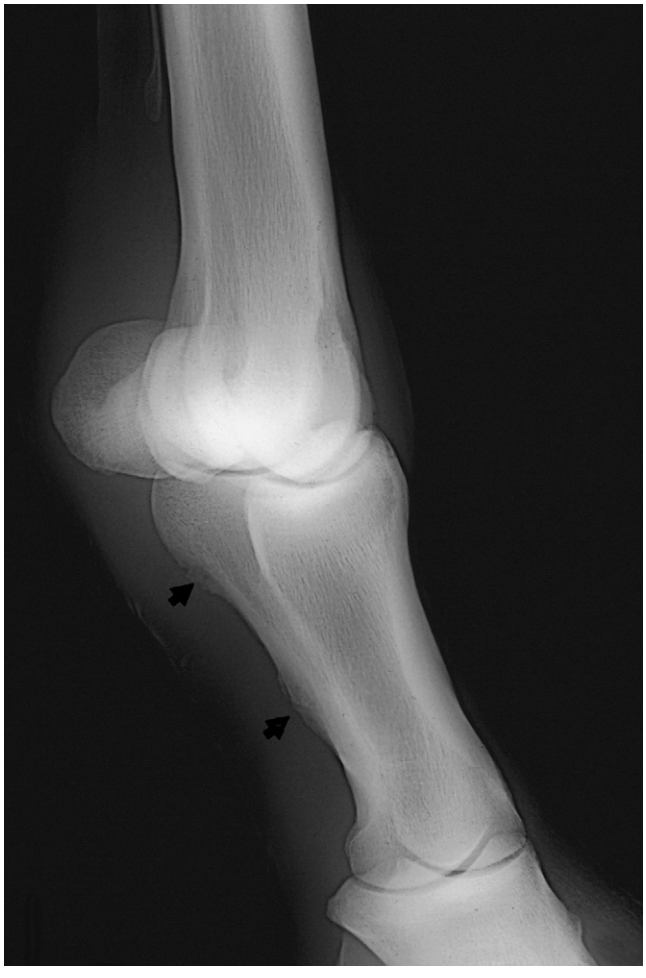

Fig. 120-13 Dorsomedial palmarolateral oblique radiographic view. Proliferative changes (*arrows*) along the medial aspect of the left forelimb proximal phalanx are typical of those seen from mallet injuries. Polo is played right-handed, and trauma usually involves the medial aspect of left forelimb and lateral aspect of right forelimb proximal phalanges.

Digital Flexor Tenosynovitis and Desmitis of the Palmar Annular Ligament

Tenosynovitis of the digital flexor tendon sheath (DFTS) is common in polo ponies. Debate exists as to whether this syndrome is caused by desmitis of the palmar annular ligament or whether the palmar annular ligament is a passive structure causing only constriction of the inflamed tendon sheath. The terms *tenosynovitis* and *desmitis of the palmar annular ligament* are sometimes used synonymously, but tenosynovitis with mild thickening of the palmar annular ligament is more common than primary desmitis. A normal or slightly thickened palmar annular ligament can restrict SDFT movement in horses with tendonitis, but this is a separate entity (see page 1006). In polo ponies desmitis of the palmar annular ligament can be solitary, diagnosed in horses with healthy flexor tendons. It can lead to tenosynovitis or can accompany tenosynovitis. Primary desmitis of the palmar annular ligament occurs most frequently from interference injury, when the hind feet strike the forelimb palmar annular ligament, or from direct mallet trauma. Initial trauma may be minor, but continued trauma can lead to substantial injury (Fig. 120-14). Whether primary or secondary, tenosynovitis with thickening and compartmentalization of the DFTS can occur proximal and distal to the palmar annular ligament. Diagnosis can be made by combining the results of clinical examination and intrathecal analgesia. Ultrasonographic evaluation is crucial and should include dynamic studies in which the flexor tendons and DFTS are evaluated for possible adhesions between tendons, DFTS, and the palmar annular ligament. Concomitant conditions such as osteoarthritis of the fetlock joint, sesamoiditis, and demineralization of the PSBs at the medial and lateral attachments of the palmar annular ligament should be assessed radiographically. Initial management is to apply cold therapy and sweats and to

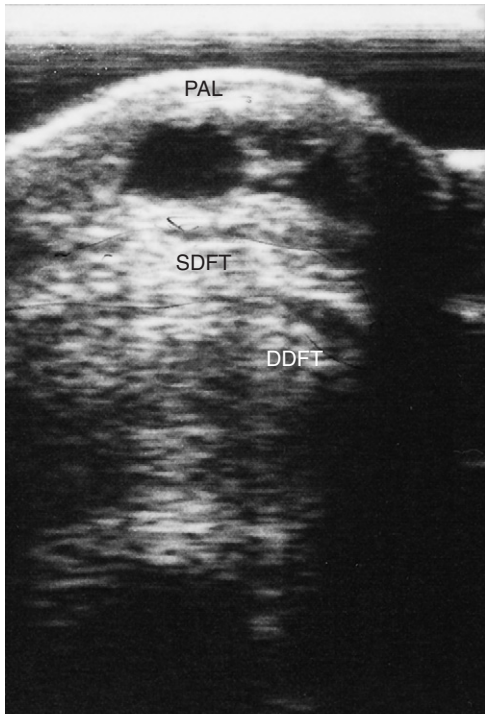

Fig. 120-14 Transverse ultrasonographic view of the distal palmar metacarpal region. An acute injury caused by direct trauma resulted in a chronically thickened palmar annular ligament. *PAL*, Palmar annular ligament; *SDFT*, superficial digital flexor tendon; *DDFT*, deep digital flexor tendon.

administer NSAIDs. Decompression of the DFTS and injection of hyaluronan and corticosteriods then is followed by 3 weeks of stall rest. Tenoscopy is valuable in identifying horses with tendon tears and to perform adhesiolysis. Desmotomy of the palmar annular ligament in horses with chronic desmitis or in those with chronic tenosynovitis may be helpful. Definitive therapy should be instituted early and aggressively to minimize adhesion formation.

Desmitis of the Distal Sesamoidean Ligaments

Desmitis of the distal sesamoidean ligaments (bowed pastern) is seen occasionally in polo ponies and usually involves the straight distal sesamoidean ligament. Diagnosis is made by detecting pain and swelling, by performing perineural analgesia (abaxial sesamoid block), and by using ultrasonographic examination.

Polo ponies with recurrent injury may not resent palpation, and ultrasonographic examination is necessary to differentiate this from other injuries. Proliferative changes along the base of the PSBs and abaxial surface of the proximal phalanx are often seen. These injuries may be career ending and at best are performance limiting. Long-term rest is necessary. Some owners request pinfiring.

Carpal Region Lameness

The carpus is an uncommon source of lameness in the polo pony. Pre-existing chronic radiographic changes are often incidental findings, even in horses with reduced range of carpal flexion and mild effusion. Carpal chip fractures can occur, but they are unusual. Tearing of the medial palmar intercarpal ligaments can cause lameness. Transient traumatic carpitis occasionally is seen after direct trauma from a mallet or ball. Rupture of the carpal sheath is a rare cause of lameness in the polo pony.

One author (P.J.M.) has seen two horses with massive swelling in the distal antebrachium, and ultrasonographic evaluation revealed rupture of the carpal sheath. Prognosis is not known. Fractures of the proximal aspect of the splint bones can lead to osteoarthritis of the carpometacarpal joint. Fracture of the medial splint bone is most serious, but polo ponies usually respond well to conservative management. Horizontal fracture of the accessory carpal bone may occur after collapse or a fall. Ponies are occasionally hit on the forehead by a hard struck polo ball, rendering them unconscious and causing the hind hoof to hit the back of the carpal region. Fracture can also occur if a horse trips inadvertently on a loose polo wrap. Prognosis appears to be indirectly proportional to fracture displacement.

Vertical (longitudinal) fracture of the accessory carpal bone occurs rarely in polo ponies that fall onto a flexed distal limb. Treatment is conservative. Physiotherapy, forcing full carpal flexion, is helpful to restore normal mobility (S.K.). Tenosynovitis of the carpal sheath can occur primarily or after fracture of the accessory carpal bone and in ponies with tendonitis of the SDFT and DDFT, and in horses with desmitis of the accessory ligament of the SDFT (ALSDFT), but this condition is rare.

Desmitis of the Accessory Ligament of the Superficial Digital Flexor Tendon

In the United Kingdom, between 10 and 15 polo ponies each year are diagnosed with desmitis of the accessory ligament of the SDFT, which may occur because of the fast and aggressive play of modern polo (P.J.M.). Awareness of the condition and use of ultrasonography may have resulted in increased recognition.

One high-goal player had five horses with this problem in a single year. Diagnosis is made by eliminating the distal limb and by palpating pain and swelling in the distal antebrachium and in the carpal sheath. Conservative management is usually successful, including the periligamentous injection of hyaluronan and corticosteroids. Surgical resection of the accessory ligament of the SDFT may be indicated, particularly in polo ponies with considerable fibrosis and bony proliferation on the caudal distal radius, or those that stand over at the knee.

Upper Forelimb Lameness

Lameness associated with the antebrachium, elbow, and shoulder regions is uncommon in polo ponies. Occasionally, fractures of the radius or olecranon and wounds with bony sequestration occur from kick trauma. Repair of fractures of the olecranon has been successful. Shoulder region soft tissue trauma, fracture of the supraglenoid tubercle of the scapula, or suprascapular nerve injury occurs occasionally from falls, dangerous play, or wrecks.

Other Hindlimb Lameness

The distal hindlimb is subject to the same type of bony injuries as seen in the forelimb, with the exception of navicular disease. Fractures of the distal and proximal phalanges and PSBs occur with the same frequency as in the forelimb, but prognosis is better in the hindlimb. In the hindlimb, osteoarthritis of the fetlock joint, flexor tendonitis, suspensory desmitis, and desmitis of the ALDDFT are not nearly as common as in the forelimb.

Two soft tissue problems occur specifically in the hindlimbs. A form of severe suspensory desmitis occurs in older polo ponies with exceedingly straight hock conformation. The hind fetlock joint drops and the pastern region is parallel to the ground. Lameness, however, is often minimal, and despite the abnormal angle of the fetlock joint these horses can continue to play for years with the help of support wraps. The second specific soft tissue injury of the hindlimb is spontaneous rupture of the common digital extensor tendon above the fetlock joint. This injury results in lameness and hyperflexion of the fetlock joint at the trot. These horses respond well to conservative management, with support wraps and rest, and prognosis is excellent.

Lameness from stifle injuries is uncommon. Osteochondrosis occasionally causes effusion and lameness or poor performance if present bilaterally. Collateral ligament injury may occur from severe bumps. Kick wounds to the tibial crest can cause pronounced lameness and may result in a fracture. Polo ponies with a history of poor stopping or turning ability respond favorably to injection of counterirritants (blisters) around the patellar ligaments and the insertion of the vastus lateralis and rectus femoris muscles. Horses are kept in a working schedule following these injections and typically may be played successfully several days later.

Pelvic injury in polo ponies is infrequent but may result from dangerous play, such as a high-angle ride off behind the saddle (S.K.), and is best diagnosed using scintigraphy. Stress fractures of the ilium and traumatic fracture of the acetabulum occur but are rare. Disparity in height of the tubera sacrale often is seen, but with no effect on performance.

Rhabdomyolysis

Rhabdomyolysis, or tying up, is fairly common, especially in mares. Two types of horses are predisposed to this syndrome. Unfit horses placed into work too rapidly often develop a stiff gait at the trot and tenderness to palpation over the dorsal musculature. Muscle enzyme levels are only moderately elevated and horses respond well to rest and NSAIDs. The most common form of rhabdomyolysis is seen in fit polo mares after an extended layup period for illness, unrelated lameness, or foul weather that prevents horses from being played regularly. Rhabdomyolysis can also occur on cold, clear mornings

or blustery days during which a drop in barometric pressure occurs. Horses generally become affected after exercise, when they return to the barn or trailer; are reluctant to move; and have firm, tight gluteal regions. Epaxial muscles rarely are involved. Clinical signs may be confused with colic because horses with rhabdomyolysis often paw, stretch out, and sweat profusely. Muscle enzyme levels are elevated greatly. Management is similar to that described for other sport horses (see Chapter 84). Nutritional management appears to be important in Europe where bran, which is high in phosphorus (reduces calcium), is fed to reduce recurrence.

Gracilis Muscle Tear

Gracilis muscle tears are seen in several polo ponies each year and are characterized by lameness at the trot and dramatic swelling in the medial thigh region. Abduction of the flexed hindlimb elicits a painful response and a large hematoma generally develops soon after the injury. The administration of NSAIDs, administration of DMSO intravenously, and the topical application of cold water are beneficial. If necessary seroma fluid can be drained surgically 5 to 7 days after injury, and exercise is limited for a minimum of 2 weeks.

Equine Protozoal Myelitis

Mild neurological signs typical of equine protozoal myelitis or lameness associated with the disease can be confused with other causes of musculoskeletal pain, in particular hindlimb lameness. Equine protozoal myelitis is prevalent in the United States but is rare in England (P.J.M.). Occasionally a polo pony in the United Kingdom that was imported from the United States develops clinical signs consistent with equine protozoal myelitis under the stress of the latter half of the season.

CHAPTER • 121

The Western Performance Horse

THE CUTTING HORSE

• Jerry B. Black

DESCRIPTION AND HISTORY OF THE SPORT

The cutting horse was born of necessity long ago on the open grass plains of West Texas. This was the era of Western history that included big cattle drives from the open ranges of ranches such as Burnett and the 6666 Ranch, Waggoner Ranch, the Pitchfork Ranch, and the Matador Ranch to Dodge City, Kansas. Cutting horses enabled big country ranches, where no barbed wire fences existed, the only means of working vast herds of cattle. In those days the task of the horse was simple, at least by definition. Guided by the rider, the cutting horse entered a herd of cattle quietly and deliberately. A single cow was cut, or separated, from the herd. The natural instinct of the cow is to return to the safety of the rest of the herd. The cutting horse, through breeding and training, controlled the calf with a series of moves and countermoves. The speed, agility, balance, and quickness of the cutting horse kept the cow from the herd, where other cowboys would hold the cut. The horse and rider would re-enter the herd again and again, cutting cattle out until the work was done. Only the top hands earned the right to ride the best horses of the remuda, the cutting horses.

The unique skills of the cutting horse were a great source of pride to the frontier cowboy. This often led to impromptu or jackpot cuttings on the open range or, from about 1900, in outdoor pens of the large ranches. From this love of the cutting horse and the subsequent competition to determine who had the best horse came the roots of cutting as we know it today. The first cutting horse contest for money was held at the 1898 Cowboy Reunion in Haskell, Texas. Twelve cutting horses competed for a purse of $150. From this start, regular events occurred on ranches of the Southwest and at the Fort Worth Stockyards. Rules and prizes varied greatly, but the ability of the cutting horse to separate a single calf from the herd always was and continues to be the goal of the competition. From these roots the National Cutting Horse Association was formed in 1946 during the Fort Worth Exposition and Fat Stock Show. The stated purpose of the organization was to standardize the rules and judging of competition and to preserve the tradition and history of the cutting horse with the ranching and livestock industry.

Today, competitions approved by the National Cutting Horse Association occur throughout the United States and Canada. In addition, many association members from other countries such as Australia are conducting competitions outside North America. The format of these competitions and other Western performance horse disciplines, such as reining, present a unique challenge to the equine veterinarian.

TRAINING

Training of the cutting horse begins at 2 years of age. Usually 60 to 90 days are spent in basic training before the horse is introduced to cattle. This generally is accomplished by turning one cow into a round pen that is 38 to 54 m in diameter. The horse is taught to mirror the movements of the cow as the cow moves around the perimeter of the arena. This process of training a cutting horse is repetitive and is done several days a week for months. The object of training is for the horse to develop an ability to perform identical movements with the cow. Simply put, when the cow stops or stops and turns, the horse does the same maneuver. This type of training is accomplished by asking the horse to stop with the aid of the bridle and turning the horse to move with the cow. The key to training is a complete and balanced stop. With time, the stop ultimately is followed by the instinctive ability of the horse to read the movement of the cow and to turn in the direction the cow is going. Because this ability to watch the cow and respond to its movement is instinctive to the working stock horse, breeding is of the utmost importance. Without this

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genetic instinct the horse simply does not respond to the movement of the cow and does not initiate the stop or turn necessary to continue to track the animal. A good cutting horse trainer knows in a relatively short period if a young horse has the instinct and athletic ability to be a successful cutting horse. The finished cutting horse must perform the necessary moves to keep the cow in proper position away from the herd, without any hand cues from the rider, relying on instinct to read the movement of the cow alone. Reining the horse is permitted only to make the cut of a single cow out of the herd. After the cut is successfully made, the reins are placed in a relaxed position on the horse's neck, and only leg cues are permitted from the rider during the actual working time. The ability of the working cow horses to contain the individual cow provides the excitement of competition in cutting.

Training of a cutting horse prospect that has shown good potential continues when it is a 3-year-old, preparing it for the first major competitions, the futurities. The futurity is the first of the horse's aged event competitions that continue for 4 years. No horse can compete in aged events beyond 6 years of age. Aged events consist of two elimination go-rounds, followed by semi-finals and the final competition. Substantial musculoskeletal stress is placed on these athletic performance horses, with multi-day competitions over a short period. In addition, the horses usually are practiced on cattle daily, including the day of competition, to sharpen performance skills. Competition in these aged events is heavy, with the major shows having more than 500 entries in a single age division. Purses in this type of event can exceed a total of $1 million. The nature of this aged event competition, with large purses in numerous events over a 4-year period, has caused the cutting horse economy to grow rapidly over the last several years. Select yearling and training sales are conducted annually that are beginning to parallel the racing industry in financial return on sales. This has contributed to the current popularity and resurgence of breeding of the cutting and Western stock horse, which in turn will ensure the preservation of the tradition and heritage that this horse played in the history of the great Old West.

LAMENESS EXAMINATION

The increasing popularity of the cutting and reining horse for show and performance is occurring nationally and internationally. Sales of this type of horse have increased to Europe, South America, and other countries. In North America the revival of interest stems primarily from excellent programs instituted by associations such as the National Cutting Horse Association and the National Reining Horse Association. These associations encourage owner participation at the amateur level in cutting and reining events. This type of performance horse creates a new diagnostic and treatment challenge for attending veterinarians, partially because of the rigid training schedules necessary for the 3- to 6-year-old horse to compete in futurities and aged events.

Hindlimb lameness presents one of the more interesting diagnostic challenges to the equine clinician. The lameness is often difficult to diagnose and even more difficult to manage. A systematic approach must be developed to achieve an accurate diagnosis consistently. The veterinarian must use a routine that is repeated with each horse and must allow sufficient time to complete a thorough examination. Western stock horses may be difficult to evaluate while being led. These horses usually are not taught to lunge and are often difficult to trot in hand. A 10- to 15-m round pen with firm footing has proved to be beneficial for evaluating lameness of this and other types of horses.

DIAGNOSIS AND MANAGEMENT OF SPECIFIC LAMENESS

Hindlimb lameness is more common than forelimb lameness in cutting horses. Mixed lameness with swinging and supporting components is common in the hindlimb, especially in upper limb lamenesses such as those involving the hock, stifle, and hip or sacroiliac region. Hindlimb lameness may be associated with two sources of pain; for example, chronic hock lameness and secondary lumbar and gluteal myositis. This section focuses on selected hindlimb lamenesses of the hock, stifle, and thoracolumbar regions.

Selected Lameness of the Tarsus

Osteoarthritis of the Distal Tarsus (Distal Hock Joint Pain)

Osteoarthritis of the distal tarsal joints is seen most commonly in horses that have repeated, excessive compression and rotation of the hocks at high speed, and a high incidence occurs in the young working cow horse, and in cutting and reining futurity prospects, reflecting the demanding training schedules of 5 to 6 days a week at 2 and 3 years of age. Sickle hocks, cow hocks, and narrow hocks also may predispose horses to lameness. Osteoarthritis also may develop secondary to partial collapse of the central and third tarsal bones, and affected horses often develop lameness within the first year after birth. In our practice, routine survey radiography of the tarsus in 20, 2-year-old cutting horse prospects before training began revealed evidence of osteoarthritis in 11 (55%).

Clinical signs include reduced height of the foot flight arc, resulting in abnormal toe wear, and a shortened cranial phase of stride. Hard work increases the degree of lameness, although most horses are lame and stiff when first taken out of the box stall and improve to some degree during the initial warm-up. Trotting in a circle increases the degree of lameness. Gait alteration or lameness may be observed with the affected limb on the inside or outside of the circle. Cutting, reining, and stock horses are reluctant to stop properly. Flexion of the hock often increases the degree of lameness. Palpation of the distal medial aspect of the hock may reveal exostosis and soft tissue thickening. Deep palpation of the area can cause a painful withdrawal response. Thoracolumbar pain is present in about 50% of horses.

Diagnosis is based on a positive response to intra-articular analgesia and radiography. Radiographic abnormalities are often only seen in a dorsolateral-plantaromedial view in 2- to 4-year-old cutting horses, findings that differ from those seen in other young sport horses.

Therapy varies depending on the degree of lameness. Rest is generally not helpful in horses with advanced osteoarthritis, and obtaining trainer compliance if the lameness is subtle is difficult. Training usually is continued with the help of non-steroidal anti-inflammatory drugs (NSAIDs), such as phenylbutazone (2 g sid or 1.5 g bid) and intra-articularly administered anti-inflammatory drugs. Shoeing changes include removing excessive toe, squaring the toe of the shoe and extending both branches of the shoe for more heel support. Half-round shoes help aid breakover in some horses with cow-hocked or sickle-hocked conformation. Changes in training schedules include more paddock or free-choice exercise and longer warm-up periods before training. Training in deep surfaces, overtraining, or conditioning in circles should be avoided. Varying the gait frequently during training and conditioning helps the horse to stay more comfortable.

Intra-articular medication is used to keep chronically lame horses in competition. A combination of methylprednisolone acetate (Depo-Medrol; 50 mg) and hyaluronan (10 to 20 mg) is injected separately into the centrodistal (distal intertarsal) and tarsometatarsal joints in horses with advanced osteoarthritis.

The veterinarian should not rely on communication between the two joints. If good results are achieved, these injections are repeated as necessary every 8 to 12 weeks. Horses with early osteoarthritis respond favorably to intra-articular treatment with hyaluronan (Hylartin-V; 20 mg) and triamcinolone (Vetalog; 3 to 6 mg). Intravenous injections of hyaluronan (Legend; 40 mg in 7-day intervals, series of three) or intramuscular injections of polysulfated glycosaminoglycan (Adequan IM; 500 mg in 5-day intervals, series of four to eight) are used frequently as concurrent therapy. Combinations of intermediate-acting corticosteroids and hyaluronan administered intra-articularly have been used in horses that are lame immediately before leaving for circuit shows or important multi-day competitions such as cutting horse, snaffle bit, and reining horse futurities.

Therapeutic levels of NSAIDs may be necessary during competition if allowed by the breed, performance, or state drug regulations governing the event. Phenylbutazone (2 g sid or 1.5 g bid) is usually effective. However, many stock horse trainers feel that this drug tends to dull the mouth and sides of the horse, thus limiting bit and spur response. Other NSAIDs that are effective include flunixin meglumine (Banamine; 1 mg/kg sid) or ketoprofen (Ketofen; 2 mg/kg sid). Horses vary in response to the therapeutic effects of each NSAID. If one drug is not effective, a different one should be assessed.

Surgery has been an important adjunct to osteoarthritis therapy in horses requiring repeated intra-articular injections or continual therapy with NSAIDs. Horses with mild to moderate radiographic changes but normal joint spaces respond favorably to cunean tenectomy. Horses with substantial intra-articular changes and joint space collapse are treated best surgically with a combination of cunean tenectomy and fenestration of the affected joint or joints using a 3.2-mm drill bit and creating three to four tracts. The horse is returned to work as soon as possible after surgery to encourage ankylosis. Hand walking is begun the day after surgery, and light riding at a walk may begin 2 to 3 weeks later. Light riding exercise continues for another 3 weeks, and full training begins 45 to 60 days post-operatively if the horse is reasonably comfortable. Phenylbutazone, 2 g once daily as needed, is used initially if obvious lameness persists. Most horses show almost immediate improvement after surgery. This improvement may be caused partially by the release of intraosseous pressure after the fenestration procedure, plus cessation of the rotational effect of the cunean tendon on the distal tarsus. Radiographic evidence of ankylosis occurs over a prolonged period. Soundness does not seem to be related to radiographic evidence of ankylosis.

Prognosis varies depending on the degree of osteoarthritis, the number of joints involved, and the type of competition in which the horse is engaged. Surgery offers the best prognosis for horses with chronic lameness.

Arthrosis of the Tarsocrural Joint

Distention of the tarsocrural joint capsule is usually from osteochondrosis or trauma. Osteochondrosis lesions occur on the intermediate ridge of the distal tibia, the trochlear ridges of the talus, and the lateral or medial malleolus of the distal tibia. Trauma is related to quick turns, hard stops, loss of balance, and poor footing. Faulty conformation, such as overly straight angulation of hock and stifle joints, may be a predisposing factor.

Distention of the tarsocrural joint capsule is observed most easily on the dorsomedial aspect of the hock, but swelling also occurs in the plantar pouches, laterally or medially. The horse may have pain on palpation, and lameness may follow trauma. The hock flexion test may or may not be positive, depending on the degree of joint capsule distention and synovitis. Radiographic examination is essential to determine the cause and should be repeated in 10 to 14 days if initial radiographs are normal.

Osteochondrosis is treated surgically. Traumatic distention of the tarsocrural joint is treated by intra-articular injection of intermediate-acting corticosteroids and hyaluronan, 2 or 3 times, 14 to 21 days apart. Intra-articular injections often are followed by hyaluronan (40 mg) administered intravenously weekly for 3 weeks. All injected hocks are bandaged concurrently to help reduce joint effusion. Pressage elastic contour bandages (Jupiter Veterinary Products, Harrisburg, PA) provide adequate pressure and are easy to maintain. The horse is given rest for 3 to 6 weeks.

Exploratory or diagnostic arthroscopy is justified in any horse that does not respond to conservative therapy, permitting identification of subtle osteochondrosis lesions not detectable radiographically and soft tissue injuries, and providing joint lavage.

The prognosis is good if treatment is initiated early, and in horses with osteochondrosis lesions or severe trauma, all fragments and debris are removed soon after the synovitis is recognized. If conformation is the predisposing cause, the prognosis is poor.

Selected Lameness of the Stifle

The stifle is a large, complex joint composed of two articulations: the femorotibial and femoropatellar joints. My experience has been that during arthroscopy of the femorotibial joint, despite high intra-articular fluid pressure, obvious distention of the femoropatellar joint capsule does not occur. Thus little or no distention of the femoropatellar joint capsule occurs in association with disease of the femorotibial joint. When performing intra-articular analgesia of the stifle, all three compartments should be injected separately.

Osteochondrosis

Osteochondrosis of the trochlear ridges of the femur is seen commonly in young horses. Clinical signs include distention of the femoropatellar joint capsule and varying degrees of lameness, depending on the amount of joint surface involved. Diagnosis is confirmed radiographically. Arthroscopic surgery is the treatment of choice to debride all diseased cartilage and bone and to remove all free-floating bone and cartilage. Aftercare consists of 45 to 60 days of stall rest, followed by an equal amount of stall and paddock confinement. Training generally resumes 3 to 6 months post-operatively. Intra-articularly administered hyaluronan (20 mg) followed by intramuscularly administered polysulfated glycosaminoglycans (PSGAGs; 500 mg in 5-day intervals, series of four to eight) 2 to 3 weeks after surgery has helped to reduce post-operative synovitis.

Subchondral Bone Cysts

Subchondral bone cysts of the medial condyle of the femur are the most frequently recognized bony lesions of the stifle in my practice. All horses with clinically relevant signs are lame at the walk or trot in one or both limbs. The degree of lameness varies greatly among horses. Some horses are subtly lame, requiring riding or repeated flexion to produce a recognizable lameness. Others have acute, severe lameness and are unwilling to trot. Moderately lame horses tend to swing the toe medially during protraction. This phenomenon is unlike the horse that carries the stifle out or abducts the limb with femoropatellar joint or patellar ligament lamenesses. Lameness may be more obvious with the affected leg on the inside of a circle.

Subtle distention of the femorotibial joint capsule may be palpated between the medial patellar and medial collateral ligaments. Some horses resent deep digital pressure over the medial femoral condylar region.

Diagnosis of subchondral bone cysts is based on clinical signs, response to intra-articular analgesia using 30 ml of mepivacaine and radiography. Conservative treatment for the most part yields only temporary improvement in the lameness and is used in our practice only when a performance horse needs to compete for the remainder of the season or when

finances prohibit surgical intervention. Conservative treatment consists of intra-articular injections of hyaluronan, with or without corticosteroids such as betamethasone or triamcinolone. An intramuscularly administered PSGAG given in a series of four to eight injections at 5-day intervals also is used. Many trainers report a pronounced effect about 24 hours after administration of PSGAGs. Therapeutic levels of systemic NSAIDs may be also necessary during multi-day competitions. Owners should be informed that continued training and competition over an extended period might lead to secondary osteoarthritis.

The treatment of choice in my experience is curettage and fenestration of the subchondral bone cyst. Before 1988, this procedure was done through an arthrotomy incision. Although the surgery was successful in most horses, wound dehiscence and prolonged hospitalization were of great concern. Currently the surgery is performed by arthroscopy, with the horse placed in dorsal recumbency and the limb in flexion. This position provides adequate visibility and good access to the cystic lesion via the instrument portal. Postoperative hospitalization is minimal, and to date no post-operative complications have been seen. The patient is confined for 60 days after surgery. Hand walking for 10 minutes daily is allowed during confinement. Free-choice exercise for an additional 2 to 4 months is allowed. Training usually resumes 6 months post-operatively or earlier, if the horse is sound.

The prognosis for horses with surgically debrided subchondral bone cysts has been good (60% to 70%) in my practice. Most of these horses return to a competitive level of performance, if given adequate rest.

Upward Fixation of the Patella

Partial or complete upward fixation of the patella is a common cause of stifle pain, which can eventually produce articular changes of the patella. In my experience this condition can occur in any type of body conformation and hindlimb angulation and may be related to the anatomical formation and depth of the notch on the proximal medial trochlear ridge. Lack of condition and loss of condition are contributory factors. Poor coordination between extensor and flexor groups of the stifle and lack of quadriceps development may explain why upward fixation is seen in young animals at the beginning of training. I have examined two horses in which upward fixation of the patella was secondary to a subchondral bone cyst in the medial femoral condyle. Upward fixation may have been caused by alteration of gait and foot placement because of pain in the medial femorotibial joint. Upward fixation resolved after arthroscopic treatment of the subchondral bone cyst. The duration of locking varies from an almost instantaneous release, with only slight backward jerk evident, to a complete locking that can last for hours and may require surgical release.

Diagnosis is based on clinical signs. Often, although no obvious upward fixation occurs in extension, the leg snaps with an audible click while in an extended position. Occasionally, pushing the patella over the top of the trochlear ridge when the leg is in extension can produce the locking. Clinical signs often are exacerbated if the horse can be walked down a steep slope. The diagnosis is sometimes based almost entirely on the owner's or trainer's description of the condition.

Treatment should remain conservative when at all possible. If complete upward fixation has occurred for any period, the femoropatellar joint usually shows effusion. Treatment should be aimed initially toward reducing inflammation and resting the tissues involved. Systemic corticosteroids (20 mg dexamethasone [Azium] IM sid) for 1 to 3 days, followed by 3 to 5 days of NSAIDs (2 g phenylbutazone bid) is a usual treatment schedule. Hand walking for 5 to 10 minutes is allowed if no further upward fixation occurs, but no free-choice exercise is allowed. Excessive toe is removed, and wedged shoes or wedge pad and flat shoes are used if the heel is low. Half-round shoes allow the horse to break over in its most comfortable and natural position.

Once the initial inflammation has subsided, a conditioning program is started. Long warm-up periods are essential. Thirty minutes of walking and trotting, followed by an increasing amount of extended trotting on the straightaway is recommended. Once the horse is becoming conditioned, trotting in the hills is prescribed, where possible. The concept of conditioning is to improve quadriceps development and tone and to improve overall coordination. Horses that are underweight should be fed to gain weight and to improve the overall body condition and the condition of the muscles involved in movement of the stifle.

Horses that do not respond to conservative treatment may require an internal blister or medial patellar desmotomy. Internal blister is accomplished by local infiltration of 2% iodine in peanut or almond oil injected directly into the body of the medial patellar ligament. Care must be taken to avoid the accidental penetration and injection of the femoropatellar joint with the counterirritant solution. Desmotomy should be reserved as a last form of therapy because the postoperative complications include fragmentation of the apex of the patella, soft tissue fibrosis, and mechanical alteration of gait.

Femorotibial Pain

Subtle soft tissue injuries may occur in the femorotibial joint, resulting in low-grade lameness that is most evident when the horse trots in circles. Such injuries often occur as training is increased. A typical example is a young cutting horse that is being worked hard on cattle before a futurity. The horse has a shortened cranial phase of stride and lowered foot flight, causing toe drag. Results of hindlimb flexion tests are generally negative. Mild distention of the medial femorotibial joint capsule may be palpable. Diagnosis is based on clinical signs, response to intra-articular analgesia of the femorotibial joint, and the absence of radiographic abnormalities.

Treatment comprises intra-articular medication with hyaluronan and corticosteroids such as triamcinolone (6 mg), plus intramuscularly administered PSGAGs. Systemic NSAIDs are given in decreasing doses over 10 to 14 days. All trailers and calks are removed from shoes. A wedge pad may be added with an egg bar shoe or extended branch shoe, depending on the amount of heel support needed.

Training is resumed after 14 to 21 days of rest. Long warm-up periods and extended straightaway trotting is recommended to condition the muscles of the upper hindlimb.

Prognosis is good if a consistent training schedule is maintained. Horses with irregular training schedules and frequent periods of several days off between exercise sessions tend to have recurrent problems.

Thoracolumbar Injuries

Thoracolumbar Myositis

Soft tissue injuries of the thoracolumbar region produce back soreness and are the most common injuries in a working stock horse. Thoracolumbar myositis may coexist with hindlimb lameness, such as distal hock osteoarthritis, or may be a primary traumatic lesion, frequently caused by the extraordinary forces of rotation and propulsion placed on the hindlimbs. Other factors include rigid training and competition schedules such as the fall futurities for 3-year-olds that result in an overworked young horse.

Local myositis involving the muscles of the thoracolumbar and pelvic region can have a profound effect on the performance of a stock horse. A cutting horse has three basic components to work: the stop, turn, and ability to track the cow in mirror image across the arena at high speed. Localized back pain results in decreased performance in all of these,

without obvious lameness. The trainer perceives the horse as simply not trying. Consequently the horse with back pain is forced to try even harder and soon falls into the overworked category.

Clinical signs of thoracolumbar myositis include pain to palpation of the affected muscle groups and associated spinous processes, obvious discomfort during saddling or mounting, subtle bilateral or unilateral hindlimb lameness, unwillingness to stop in form, and overall lack of performance. Flexion tests are seldom positive, unless the back problem coexists with distal tarsal disease. One may reasonably believe that arthrosis of vertebral articulations in the lumbar and lumbosacral region exists in some of horses. However, because of the depth and mass of the muscles involved, distinguishing the exact pathological condition or even the exact site of the injury is impossible.

Therapy is aimed at reducing inflammation and controlling the associated muscle pain and spasms. Prolonged rest periods from training always are indicated but in reality are difficult to achieve because of the rigid schedule of preparation for competition. For example, an average futurity horse being prepared for the National Cutting Horse Association futurity in December of its 3-year-old year accumulates a $20,000 to $24,000 debt in training and entry fees alone before competition. Convincing an owner and trainer that the horse should be allowed to rest immediately before the futurity is difficult, if appropriate therapy has even a remote chance of being effective.

The systemic use of skeletal muscle relaxants such as methocarbamol (Robaxin; 10 mg/kg PO bid for 5 to 10 days) has been effective in treating generalized back pain. Dexamethasone (10 mg PO bid for 3 to 4 days) is indicated in horses with acute pain. Chronic back pain may be treated successfully during competition with a single dose of triamcinolone acetonide (12 to 16 mg IM), while concurrently administering methocarbamol orally. NSAIDs generally have not been effective, unless the back pain is secondary or coexists with distal tarsal osteoarthritis. Care must be taken to comply with any medication rules.

Specific localized pain may be treated successfully by local injection of methylprednisolone acetate (200 to 400 mg) and Sarapin (50 ml). Treatment is repeated every 10 to 14 days until pain subsides.

Other management considerations are important for recovery. Horses with low, underslung heels of the hind feet should be shod using raised heels. Evaluation of the fit of the saddle, type of pad and specific pressure points when ridden should be considered. Other modalities of therapy, such as pulsed electromagnetic field and ultrasound, have been useful in keeping a horse in competition. Long warm-up periods without the rider for 30 to 45 minutes by ponying (leading from another horse) at a walk and trot always are indicated. The trainer must be cautioned that overwork and severe fatigue must be avoided at all times.

Sacroiliac Desmitis

Strain and subluxation of the sacroiliac joint are not uncommon in the working stock horse because of twisting and rotation of the back and pelvis during work. This rotation is complicated by the weight of tack and rider, who is attempting to maintain balance and remain stationary on top of the horse during sudden hard stops, turns, and bursts of speed.

Many of the clinical signs observed in horses with thoracolumbar myositis are also common in those with sacroiliac desmitis, because the epaxial muscles go into spasm to provide stability to the traumatized sacroiliac joint. However, bilateral or unilateral lameness with stiffness and alteration of gait usually is associated with sacroiliac desmitis. Protrusion of the tubera sacrale may be evident when the horse is walking away from the observer. Flexion of the contralateral limb for 2 minutes may result in elevation of the tuber sacrale, hip hike, and stiffness of the affected limb. Digital palpation adjacent to the tuber sacrale and over the gluteal regions usually elicits pain. Local infiltration of local anesthetic solution may result in improvement, but rarely are clinical signs fully alleviated.

Deep intramuscular injections of methylprednisolone (400 mg) and Sarapin (50 ml) into the region of the sacroiliac joint have been effective in treatment. Disposable needles at least 10 cm long are necessary to reach the affected area. Strict aseptic technique must be followed. Injections usually are repeated after 2 to 3 weeks. Concurrent systemic therapy with NSAIDs is beneficial. Horses must have rest, and 2 to 6 months out of training is often necessary, with strict stall confinement for the first 30 to 45 days.

Proper therapy and management of injuries to the thoracolumbar and sacroiliac regions generally are rewarding if initiated early in the course of the disease. Horses with chronic recurrent problems usually can be managed to allow some level of competition.

THE ROPING HORSE

• Robin M. Dabareiner, G. Kent Carter, and Richard Galley

TEAM ROPING HORSE

Description of the Sport

A unique handicapping system implemented in the early 1990s has contributed to team roping becoming a rapidly growing equestrian sport. Team roping began as a rodeo event many years ago, evolving from the everyday work of cowboys on ranches. If a cow needed to be treated on the open range, the only method of restraint was to secure the head and heel of the animal, or to team rope it. The cowboys soon began wagering among themselves to see which team of a header and heeler could accomplish this feat in the shortest time. Currently nearly 1 million people compete in team roping competitions in North America.

Because of the large number of participants, team roping has become of great economic importance. Many team roping organizations exist nationally, but the most prestigious is the United States Team Roping Championships. The numbering or handicapping system of the team ropers was begun by the United States Team Roping Championships and has become standard. A number, from 1 to 9, with 9 being the highest level of ability, is assigned to each of the team ropers. This number is based on various factors, including ability, previous prize earnings, experience, age, and physical handicaps.

The roping categories also are assigned a numerical value that cannot be exceeded by the total handicap numbers of the two participating ropers. The highest level of roping (11) is the open roping, which allows the world champions to compete together, and the lowest beginner level is 4. This method allows the lower-level ropers to compete as a team with the world champions and to level the playing field of competition at all levels. The entry fees of the participants usually generate the purse money in a jackpot fashion. A portion of the entry fee is held out by the producer of the roping to pay for the arena, the cattle, and advertising, and the rest of the fee is placed in the purse money to be divided among the winners.

Dally team roping is a timed event involving five basic elements: the header, the heading horse, the heeler, the heeling horse, and the steer. The steers that are used for the team roping event are usually horned cattle called Corriente cattle,

often from Mexico. Other types of cattle used are longhorns or other native homed breeds ranging in weight from 170 to 320 kg.

A typical run in dally team roping begins with a steer contained in a chute at the end of an arena. The heading box is to the left of the chute and the heeling box to the right of the chute. When the header calls for the steer, or asks that the steer be released from the chute, the chute gate is opened and the steer is allowed a head start called the score. If the header leaves the heading box before the steer crosses the score line, or reaches the predetermined head start, then the team is issued a penalty of 10 seconds. The timing of the run is begun when the steer crosses the score line.

When cued by the riders, the horses leave the roping box much as a racehorse leaves the starting gate to attain maximum speed as quickly as possible. As the header approaches the steer, now with the heading horse running at full speed, the horse is trained to rate off, or to slow up slightly, once the horse reaches the hip of the steer to position the steer properly so that the header may rope the steer. Team roping has three legal head catches: both horns (a clean horn catch), a half head (1 horn and the nose of the steer), or a neck. All other catches are considered illegal, and the team is given a no time.

After the header successfully catches the head of the steer and dallies (wraps in a full circle) the rope around the saddle horn, the heading horse drops its hindquarters and slows somewhat as it sets the steer and brings the steer's head around to the left. As the steer's body progresses to the left, the heading horse also is turned to the left and is moved out in front of the steer to allow the header to pull it across the arena at about a 90º angle to the original direction of travel, maintaining a constant slower speed, thus allowing the heeler to get into position to rope the hindlegs of the steer.

As the header sets and turns the steer, the heeler turns left following the steer and positions just behind and slightly to the left of the steer as it is taken across the arena. As the heeling horse follows the steer in this position, maintaining a constant speed equal to that of the header and steer, the heeler properly times his swing and then releases the heel rope, placing the loop under the steer and ropes the hindlegs of the steer. If only one hindleg is caught, then the team is issued a 5-second penalty. As the slack is taken out of the heel loop and as the dally is made on the saddle horn, the heeling horse is signaled to drop its hindquarters and come to an abrupt stop.

As the heading horse progresses away from the heeler with the steer still in tow, the ropes come tight, and when tight, the heading horse is cued to spin around to the right while maintaining a tight rope to face the steer. When the facing is complete, the rope is tight and in a straight line from the saddle horn of the header to the head of the steer and is tight from the hindfeet of the steer to the saddle horn of the heeler, and then the flagman signals the end of the run and the time is taken.

The roping can be accomplished by experts in 6 to 7 seconds but requires thousands of hours of practice to achieve this and to minimize the danger to all of the participants. Runs recorded in the range of 3.6 to 3.8 seconds have been made by the World Champion caliber team ropers.

Conformation

The horses, predominately geldings, used in team roping are usually American Quarter Horses (QHs), preferred because of exceptional athletic ability, quick acceleration over short distances, and a good mind. Other breeds are rarely used. The head horse must be larger and faster than the heel horse. Head horses typically weigh 545 to 590 kg and are heavily muscled to tow the steer across the arena. The heel horse is smaller and quicker with more cow sense than the head horse. Horses that are trained for cutting but no longer are competing often make good heel horses because of size and cow sense.

Team ropers prefer a mature, experienced horse, and so horses are usually over the age of 10 years, and many top ropers have horses in the mid to late teens. Repetitive injuries are therefore common, but because horses often have had several owners, a complete medical history is rarely available.

Training

The training of heading and heeling horses involves thousands of team roping runs and many thousands of miles of hauling. The horse must anticipate every variable of a team roping run, so that rider intervention is unnecessary. This allows the roper to focus on nothing but the speed of the run, which is an important factor contributing to injury. The current high demand for a finished team roping horse has greatly increased the value of these horses, and because replacement is difficult, veterinary advice is now sought more often.

Lameness Examination

Team roping horses experience many of the same problems as any of the Western performance horses. The hocks and the right front suspensory ligament (SL) are placed under tremendous pressure as the heading horse sets the steer. The right front is placed cranially and laterally to decelerate and brace against its forward motion and the weight of the steer, thus placing tremendous strain on this limb (Fig. 121-1). The distal tarsal joints are loaded with the rider's weight, the horse's weight, the deceleration of the full-speed forward motion, and the weight of the steer as primarily rotational forces. This occurs as the horse is asked to set and get under the steer and pull the steer's weight forward across the arena. After a successful catch by the heeler, the heading horse must face or spin around to the right while the hocks and distal hindlimbs are loaded as described, and do so with the addition of a backward motion to maintain tightness of the rope. When attempting to diagnose lameness, it is extremely important to remember that often in the early stages little or no lameness may be present. The complaint from the owner or trainer is often that the horse is coming untrained or is exhibiting increasing behavioral problems. It is imperative that the veterinarian is familiar with how the team roping horse works to arrive at a diagnosis. The history of a heading horse may include a reluctance to go willingly into the heading box, or reluctance to turn and face the front of the heading box and stand relaxed as the roper prepares to ask that the steer be released. As the problem worsens, many horses may rear up and spin around in the heading box, or lunge out of the box and refuse to re-enter. The horse may overrun the steer or even run past the steer, rather than rating off as it is supposed to do. The horse may duck or drop the left shoulder excessively as the header attempts to dally the rope and as they set the steer. The way the horse handles the steer may change, and the horse may tend to lunge across the arena with the steer in tow rather than taking the steer at an even, controlled gait. The horse may be reluctant to face at the end of a run. As the steer is followed to the end of the arena after the run, the horse may prance and jump around, rather than loping in a relaxed fashion as it follows the steer. These problems usually reflect pain, especially of the distal tarsal joints, but horses are seldom clinically lame at this stage.

The heeling horse often has a similar history, with problems in the heeling box, not making the corner properly as the steer is set and turned, or of nickering and bouncing out of the stop at the completion of the run while maximal tension is on the ropes. A common complaint by the rider is that the horse is not stopping anymore after the heeler's rope is thrown (Fig. 121-2). Horses are often unwilling to drop the head and relax after a run and tend to prance out of the arena.

Fig. 121-1 A header roping the steer and making a 90° turn to set the steer for the heeler. The weight of horse and rider result in excessive loading of the right forelimb of the head horse (grey horse).

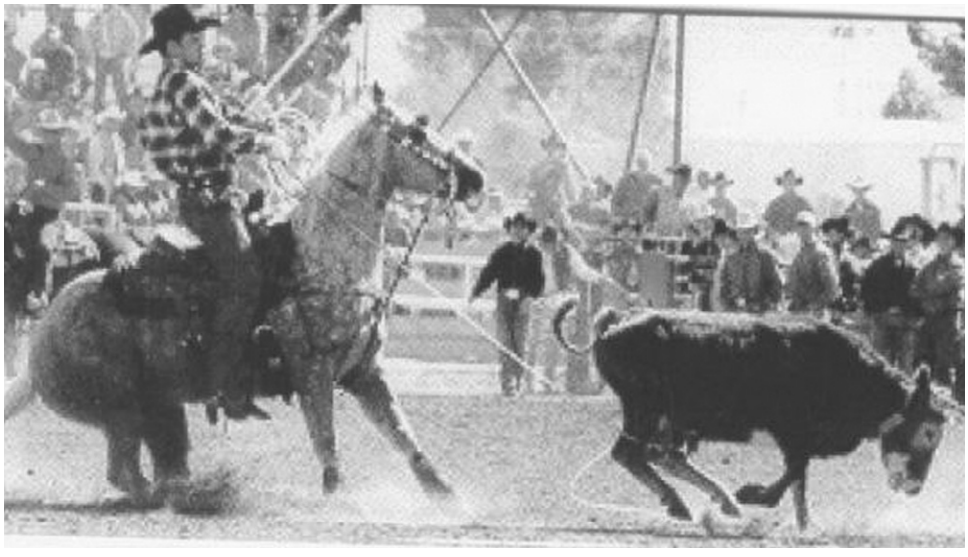

Fig. 121-2 A heel horse stopping after the heeler throws his rope. Note the flexion of the hock joints during the stop and the extreme forward placement of both hindlimbs.

Clinical examination often reveals pain on palpation of the caudal lumbar area, the gluteal muscles, and over the point of both hips. This pain often is accompanied by the complaint that the horse is sore in the kidneys. If only the distal tarsal joints are involved, effusion usually is not detectable, but the horse may be reluctant to allow palpation of the medial aspect of the tarsus, as if anticipating pain. The craniomedial aspect of the distal tarsus may be enlarged. High-quality radiographic examination of the hocks is essential; lesions may be missed unless the radiographs are examined under magnification.

Intra-articular analgesia is often ineffective because the horse has developed performance problems in response to the pain experienced during every run, and the pain is anticipated, even though the area has been blocked. Thus horses become intractable in the roping box because they dread the pain that they will experience during the run. However, horses often respond well to intra-articular therapy when they realize, after several runs, that the pain has been lessened or stopped.

Diagnosis and Management of Specific Lameness

Suspensory apparatus injury in the right forelimb is common, especially in a heading horse, and involves the SL, the accessory ligament of the deep digital flexor tendon, the distal

sesamoidean ligaments, or a combination of these. These injuries usually cause lameness of varying degrees, and diagnosis by clinical examination is straightforward. Ultrasonography should be used to confirm the diagnosis. Distal sesamoidean ligament injury may be more difficult to diagnose but often results in acute, moderate to severe lameness (grade 2 to 3 of 5) after a run, associated with pain on palpation of the palmar aspect of the pastern. Perineural analgesia of the palmar digital nerves may improve the lameness, but analgesia of the palmar (abaxial sesamoid) nerves is needed for complete soundness. Fractures of the middle phalanx are also common, so the horse should be examined by radiography and ultrasonography. Ultrasonographic evidence of enlargement or reduction in echogenicity in the straight or oblique distal sesamoidean ligaments is diagnostic (Fig. 121-3). Radiography is usually negative if lameness is acute; however, chronic injury is often associated with periosteal proliferation on the palmar aspect of the proximal phalanx associated with oblique distal sesamoidean ligament injury or entheseous new bone on the proximopalmar aspect of the middle phalanx associated with straight sesamoidean ligament injury. In hindlimbs deep digital flexor tendonitis, tenosynovitis of the digital flexor tendon sheath, and proximal suspensory desmitis are common, especially in the left hindlimb of head horses.

When turning to face the steer, the head horse turns 180° at fast speeds, putting a rotational torque on the distal aspect of the left hindlimb (Fig. 121-4).

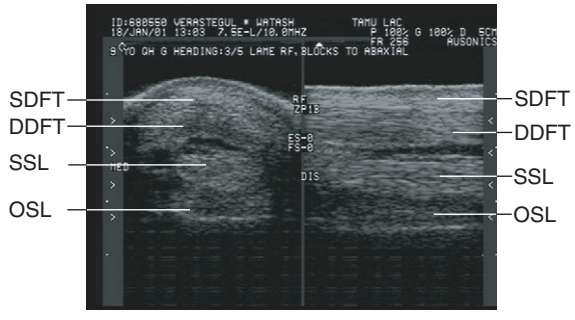

Fig. 121-3 Transverse *(left)* and longitudinal *(right)* ultrasonographic images of the mid-pastern region of the right forelimb of a head horse. There is a hypoechoic lesion in the straight sesamoidean ligament (SSL). *SDFT,* Superficial digital flexor tendon; *DDFT,* deep digital flexor tendon; *SDSL*, straight distal sesamoidean ligament; *OSL*, oblique sesamoidean ligament.

Management of these soft tissue injuries includes rest consisting of confinement and controlled exercise (Box 121-1), ranging from 4 to 6 months for horses with desmitis of either forelimb SL or the accessory ligament of the deep digital flexor tendon, to 8 to 12 months for horses with distal sesamoidean ligament injuries, deep digital flexor tendonitis, and hindlimb injuries (Box 121-2).

Given the usual QH conformation of large body size and small feet or short, upright pastern conformation, navicular disease and injuries to structures within the palmar aspect of the foot (navicular syndrome) are common. Diagnosis is based on the response to perineural and intra-articular analgesia and high-quality radiographs. Navicular bone pain results in a slowly progressive lameness, often bilateral, that responds to intra-articular medication of the distal interphalangeal joint, NSAIDs, and corrective shoeing. Soft tissue injuries of the palmar aspect of the foot (e.g., impar or collateral ligament desmitis and injury to the deep digital flexor tendon in the region opposing the flexor surface of the navicular bone) are also common in head horses and usually are acute, occurring

Box • 121-1

Exercise Regimen after Mild Injuries to Forelimb Suspensory Ligament or Accessory Ligament of the Deep Digital Flexor Tendon

Weeks 1-4	The horse is given stall rest or a small run (6 × 10 m) with support bandage on limb.
Weeks 5-8	Confinement is continued with 15 minutes of hand walking twice daily.
Weeks 9-12	Confinement is continued with 30 minutes of hand walking daily.
Weeks 13-16	If horse is sound at the trot, then confinement is continued, but the horse is walked under saddle or is ponied 15 minutes a day plus 5 minutes of trotting. Five minutes of walking and trotting are added every third day. The horse is re-examined by ultrasonography before returning to roping activity.

Fig. 121-4 A head horse (on the right) turns to face the heeler at the end of the run. Note the strain placed on the left hindlimb.

during a roping event. Lameness is improved by perineural analgesia of the palmar digital nerves or intra-articular analgesia of the distal interphalangeal joint. Often no radiographic abnormality is apparent. Extended rest is required for full recovery.

Osteoarthritis of the distal hock joints is common, and head horses seem especially at risk. Most respond well to intra-articular medication of the centrodistal and tarsometatarsal joints.

Injury to the back and pelvic area is seen, especially in the heading horse. Strain or tearing of the sacroiliac ligaments can occur. Diagnosis can be augmented by the availability of a level surface near a level reference point. When examining a horse with only the horizon or surroundings as reference, noticing a subtle change in one side of the pelvic area may be difficult. A level surface near a door, especially one with horizontal lines or ridges, offers a grid effect that makes asymmetry more apparent if the horse faces the door and is viewed from behind. Radiographic evaluation is difficult and often unrewarding, but ultrasonographic examination may reveal longissimus lumborum and gluteal muscle tearing and can be used to evaluate the sacroiliac area. Lameness predisposing to back pain must be eliminated. Rest combined with topical heat (hot towels) and dimethylsulfoxide helps many horses, but often months of rest are needed before the pain is eliminated. Treating the painful areas with local injections hastens the recovery time. We prefer a combination of 5 ml methylprednisolone acetate, 5 ml Sarapin, and 5 ml prednisolone diluted with 30 ml mepivacaine and depositing 3 to 5 ml in several sites around the painful muscle or ligament. The horse is given 2 to 3 weeks of light riding, with no roping activity, and then is re-evaluated by ultrasonography. Most horses return to roping activity within 4 weeks from the time of injury.

Obviously all team roping horses are also susceptible to any of the same injury problems that other equine athletes experience. Most of these injuries produce overt lameness, and diagnosis is usually straightforward.

Box • 121-2

Eight- to 12-Month Tendon Rehabilitation Program

Weeks 1-12	The horse is given stall rest or is confined to a small run (6 × 10 m) with 15 minutes of daily hand walking. Support bandages are used for first 30 days.
Weeks 13-16	The horse is re-examined by ultrasonography. If the lesion is healing, confinement continues, but hand walking is increased to 15 minutes twice daily.
Weeks 17-24	Confinement is continued. The horse is hand walked 30 minutes a day or walked under saddle for 20 to 30 minutes a day.
Weeks 25-32	If the horse is sound at the trot and greater than 70% healing has taken place as visible by ultrasonography, the horse is walked under saddle or ponied for 20 minutes and trotted for 5 minutes. Walking and trotting are increased by 5 minutes every third ride until the horse is walking 45 minutes and trotting 30 minutes. If the horse remains sound, treatment proceeds to next level. If lameness develops, treatment reverts back 30 days in the schedule and the horse is re-examined by ultrasonography.
Weeks 32-36	Riding is increased to include loping for 30 days. If horse remains sound, the horse can return to roping activity. The horse can be turned out into a larger pasture.

Shoeing Considerations

Because rope horses are often middle-aged, are used frequently, and commonly have navicular problems, maintenance of proper hoof balance is critical in keeping these horses sound. Long toes and collapsed heels are common. From a solar view the heels grow too far forward and are left unsupported and thus are at risk of abnormal concussion. We recommend trimming the heels back to the widest part of the frog and setting the shoe further back on the foot, so the heels of the shoe end at the widest aspect of the frog. Care must be taken to fit the front shoes properly so that little of the medial side of the shoe is exposed, because head horses have a tendency to grab this portion of the shoe and pull a shoe off, especially the left front shoe. Easing the breakover of the limb can be accomplished by rockering the shoe. A rim shoe or steel natural balance shoe provides good traction and allows an easier breakover by the rounded and rockered toe region of the shoe construction. The natural balance shoe has a wider web than a normal shoe and is beneficial for horses with sore feet acquired from performing on harder ground. If trimming alone cannot establish a correct hoof-pastern angle, a 1° to 2° wedge pad is recommended. We prefer a cutout pad to a full pad that often traps moisture and can lead to thrush problems. Care must be taken to avoid pad pressure over the central region of the frog in horses with navicular pain.

In horses with deep digital flexor tendonitis, we recommend applying a 2° to 3° wedge pad for the initial 4 to 5 months of rest to decrease tension on the tendon. The wedge is reduced gradually over three shoe resets, once the horse is sound. In horses with distal hock join pain, we try to ease breakover of the hindfeet by squaring the toes or by setting the shoe back under the toe 0.3 cm. Avoiding any type of trailer or extension on the rear shoes is also preferable, especially on the heading horse, because this can aggravate pain associated with distal hock osteoarthritis.

CALF ROPING HORSES

Description of the Sport

Calf roping originated on ranches of the Old West when sick calves were roped and tied down for medical treatment. Success in calf roping depends on the teamwork between a cowboy and horse. After the calf is given a head start (like a scoreline in team roping), horse and rider chase the calf, and as the rider ropes the calf, the rider dismounts and runs to the calf. As the rider dismounts, the horse must sit back on its hind end and come to a sudden stop, which takes the slack out of the rope and stops the running calf. This allows the cowboy to catch the calf and throw the calf to the ground, termed *flanking the calf.* Once on the ground the contestant ties three of the calf's legs together with a pigging string. The horse is trained to work the rope as the cowboy ties the calf, meaning to back up if needed to keep the rope tight, thus keeping the calf still for the rider to tie. When the cowboy completes his tie, he throws his hands up in the air as a signal to the judge

that his run is complete. The calf must stay tied for 6 seconds. A 10-second penalty is added if the calf roper breaks the barrier at the beginning of the run. An 8- to 9-second run is considered good, and Jeff Chapman of Athens, Texas, set the arena record in 1997 when he roped and tied a calf in 6.8 seconds.

Conformation

American QHs are used for calf roping and must be athletic and well trained. Because the rider must dismount during the event, calf horses are usually not tall, often being 14.2 or 14.3 hands high. Calf horses also have wide-base and muscular frontquarters and hindquarters. Many ropers believe that the performance demands on a calf horse are greater than that of a team roping horse.

Diagnosis and Management of Specific Lameness

Calf horses have lameness similar to that of team roping horses with a few exceptions. Hindlimb lameness is more common in horses used for calf roping. Osteoarthritis of the distal hock joints is the most common problem, but stifle lameness also occurs frequently. Collateral ligament damage or meniscus tearing occurs, but diagnosis is usually not apparent until after bony changes are seen radiographically, months after the initial injury. Bony reaction at the proximal medial aspect of the tibia is diagnostic. With rest, NSAIDs, and intra-articular medications, these horses often can return to roping, but at a lower level, perhaps as a calf roping horse for a child or beginner. Hindlimb proximal suspensory desmitis, traumatic fetlock osteoarthritis, and fractures of the proximal sesamoid bones and middle phalanx are common. The most common forelimb lameness is palmar heel pain, often resulting from improper shoeing.

Shoeing Considerations

The same shoeing considerations are used for balance and protection of the navicular region in the forelimbs as described for team roping horses. In the hindlimbs if the toes of the feet are too short, so that the horse has a broken forward hoof-pastern axis, then the hind toe digs deep into the ground and the limb stops abruptly, causing sudden torque on the distal limb. Having more toe length and a shoe that fits full with the heel branches of the shoe extending to the heel bulbs is preferable, so that the horse slides as it stops. This not only protects the heel bulbs from the ground surface, but it also puts less strain on the distal limb. Horses used for calf roping often wear skid boots, protective leg gear aimed at minimizing friction between the ground surface and the plantar aspect of the hindlimb fetlock joints, because the horse skids to a stop.

THE REINED COW HORSE

• Van E. Snow

The National Reined Cow Horse Association was initiated in 1949 as the California Reined Cow Horse Association and changed its name in 1970. The purpose of the association is to preserve the training traditions of the vaqueros, the horsemen of early California, who trained their horses for ranch work. As a result of selective breeding and refined training techniques, reined cow horses today are able to achieve more with livestock than was ever thought possible.

The National Reined Cow Horse Association held its first Snaffle Bit Futurity in 1970 in Sacramento, California. Bobby Ingersoll, a renowned cow horse trainer, had the idea to showcase the best all-around cow horses in the world. The competition involves showing the horse in three different disciplines: herd work (cutting), rein work, and cow work (working a cow down a fence). In the Snaffle Bit Futurity the top 20 horses from the first round of competition come back and do another round over 2 days, starting again with even scores. The sport of reined cow horse has evolved from 1970, when 27 horses competed for $3900, to 1999, when hundreds of horses competed at their annual futurity for nearly $1 million.

The professionals compete on multiple horses in four divisions: snaffle bit, hackamore, two rein, and bridle. Competition consists of multiple levels, including professional, non-professional, amateur, and limited open. An auction is held each year at the reined cow horse futurity at which horses that range from 1 to 12 years of age and breeding stock horses are available for sale.

Affiliate groups of the National Reined Cow Horse Association are organized in Canada, Germany, Belgium, and Australia, and a strong interest in the group exists in most of Europe and in South and Central America. No restrictions exist as to the breeds allowed to compete in the futurities. Numerically the American QH dominates, but Paint horses often compete successfully, and an Appaloosa has won.

The competition requires that the horse be accomplished in its ability to run, stop, turn, and read and control a cow. These horses also must have a high level of endurance. These qualifications do not necessarily dictate a particular body type, but the reined cow horse tends to be a taller, leaner horse than the cutting horse. Stallions, mares, and geldings compete equally, as do all genders and age groups of riders.

Reined cow horses are typically begun in training when they are less than 2 years old and start to compete at 3 years of age. They have about 20 months to learn all of the events and to become well enough conditioned to withstand the rigors of competition. This requires that the training surface, shoeing, and judgment of the trainer be optimal throughout the 20-month period.

Most trainers have become knowledgeable in the prevention and the early detection of lameness. Musculoskeletal evaluations are done quarterly on the futurity prospects, beginning in the fall of the 2-year-old year. These evaluations include a complete lameness evaluation, including flexion tests. At this time good baseline information on each horse is established, and future insidious problems can be detected before they become unmanageable. In the past, it was commonplace for a trainer to blame himself or herself or the horse's attitude for not being able to accomplish a certain task, when in fact low-grade lameness was developing. The trainer would continue to train the horse, and often a serious lameness would develop. For the trainer to suspect a subtle lameness and to have the horse evaluated is much more common now. Lameness detected at this point is usually manageable.

The reined cow horse continues to compete beyond 3 years of age. As 4-year-olds they can continue in the snaffle bit or begin in the hackamore or bridle class. The 5-year-olds are shown in the hackamore or bridle class, and the 6-year-old in the bridle class. Hackamore and bridle horses compete in the reining and cow work divisions, but they do not do herd work. The intention is to finish the horse's education by the time the horse is 6 years old.

TRAINING SURFACES

The training surface has a strong influence on lameness in terms of incidence, degree of severity, and type of injury. In general the arena or round pen is best constructed as follows: The original ground is graded to a 1.5% grade from one corner to the other and is compacted to 95%. A 15-cm thickness of base material such as limestone rock dust is then added, watered, and compacted to 95%. The type of base used is dictated by what is available in the area, but it should be smaller-

sized particles (not small rocks), which have interlocking edges. A 1.5% grade should be maintained. Sand is then added to the top of the base to a thickness of at least 5 cm and not more than 12 cm in the arena, and from 10 to 15 cm in a round pen. Adding the sand at the minimum levels first and adding more later if necessary is recommended. In general, less injury occurs with thinner layers of sand, but performance may be enhanced with thicker layers of sand. Fine beach sand is the best, because it has low abrasive characteristics and is light. These qualities are important because the sand gets between the skin and protective boots and also comes in direct contact with the back of the pasterns and fetlocks. If the sand is coarse and abrasive, it damages the skin and causes lameness. The density of the sand is important because the horse has to push the sand during sliding stops, and if the sand is too heavy, it causes injury and destroys the horse's confidence.

Water content of the training surface is also an important consideration. In general, applying the water a few hours before use is a good idea, so that the water is distributed evenly. If the training surface is too deep with sand or if the base is not consistent, I see more soft tissue injuries such as tendonitis, suspensory injuries, curbs, and sprained backs. If the training surface is too hard, I tend to see bone injuries such as osteitis of the distal phalanx, navicular syndrome, sesamoiditis, or fractured proximal sesamoid bones.

TRAINING AND ITS RELATIONSHIP TO LAMENESS

The types of injuries commonly seen tend to vary as the training progresses. The young 2-year-olds have hoof and sole problems during the initial breaking process, before they are shod, associated with an increased digital pulse and sensitivity to hoof testers. Subchondral bone cysts in the medial femoral condyle and osteochondrosis of the fetlock may manifest at this time. Chip fractures, present but unnoticed when the horse was a foal or yearling, may become clinically apparent when ridden work starts.

The work during the latter half of training for a 2-year-old involves more speed and collection, and low-grade lamenesses may develop that later become persistent problems. These lamenesses include suspensory desmitis, navicular syndrome, and hock and stifle pain. The 3-year-old year involves even more collection, cow work at speed, and harder stops. We generally see a continuation of the 2-year-old problems, if they are not managed well, and also more acute injuries to soft tissue and chip fractures of the fetlock and carpus.

CONFORMATION AND LAMENESS

Conformation does have an effect on the development of lameness, but I feel that conformation often is overemphasized. The component of conformation that has the biggest effect on soundness is body conformation and the degree of balance in motion. I have seen many horses whose conformation was not impressive when viewed at a standstill but were impressive when evaluated in motion. Balance in motion is that somewhat immeasurable quality that gives one the impression that the horse could jump, stop, turn, or do almost anything on any given stride without effort. In my experience, these horses remain sound while performing to an exceptional level, whereas horses that have less balance suffer more frequent injury. Although static conformation related to lameness is important and should be evaluated, in my opinion, conformation in motion is equally important.

The most common conformation problems that I see are toed in and toed out. These problems predispose the horse to lameness but are present to some degree in nearly all horses. Most often these conformational abnormalities are associated with injury to the SL and also can be associated with injury to the collateral ligaments of the digit. I try to manage these conformation faults before they cause injury to the horse by trimming and shoeing. A horse that toes out initially contacts the ground with the outside toe quarter with improper trimming. The hoof then slides along the ground and begins to rotate the toe out. The inside heel then makes contact with the ground, and the hoof rotation stops. However, this rotational force continues up the limb and places unnatural strain on the soft tissues below the carpus and eventually causes damage. I recommend removing more wall from the hoof at the point of contact and beveling the toe so that breakover is accomplished easily over a wide area. My goal is to get the entire hoof to contact the ground evenly and eliminate the rotational force. Horses that toe in generally contact the ground at the inside toe quarter. Horses with this problem need more hoof removed from the inside toe, the goal being to cause the foot to land flat.

TEN MOST COMMON LAMENESS CONDITONS

The following are the 10 most common lameness conditions in the reined cow horse:

1. Suspensory desmitis
2. Osteoarthritis of the centrodistal and tarsometatarsal joints
3. Navicular syndrome
4. Hoof-related problems such as bruises, abscesses, and cracks
5. Superficial digital flexor tendonitis
6. Subchondral bone cysts, osteochondrosis, and traumatic injuries of the stifle
7. Fetlock arthrosis
8. Sesamoiditis
9. Distal interphalangeal joint synovitis or osteoarthritis
10. Osteitis of the distal phalanx

LAMENESS EVALUATION

I begin the lameness examination by obtaining as complete a history as possible. I establish the duration of lameness; any change in lameness, if the horse has been rested or kept in work; any response to treatment; alteration in lameness with work; when the horse was last trimmed and shod; any changes in footing, type of work or shoeing; if lameness was associated with turnout or work; if the horse has received any intramuscular injections; if the horse kicks the paddock or stall; if the horse is turned out with others; if the horse stumbles; any known trauma; whether the lameness occurred insidiously or suddenly; and if the horse stands normally. Generally, the best history is obtained from the grooms and trainer, although some owners can provide information.

I watch the horse at the walk, trot, and canter, loose in a 9-m round pen, and on firm footing on a 2% slope. The degree of lameness often is amplified when the horse is loose, on hard ground, and allowed to work on a slight slope. Some lameness characteristics are best seen at a slow trot and some at an extended trot. The round pen enables one to evaluate the horse's ability to hold leads. Detecting low-grade ataxia is also easier when the horse works free, especially during downward transitions.

Lameness may be manifest in how a horse does a sliding stop; lameness may be accentuated after several sliding stops from a canter. A horse may slide stronger on one hindlimb

than the other. The complaint is often made that the horse is not stopping as well as previously, although lameness is not noticed. A horse may begin a sliding stop normally but then pick up the painful limb halfway through the stop and replace it. The stop is not held evenly. The stop becomes normal if pain is removed by local analgesia.

Another common situation occurs when the horse is cutting a cow, the horse runs off at one end or does not come back with the cow as strongly at one end. The hindlimb farthest from the cow before the weak turn is generally the lame limb. If this horse is asked to do rollbacks in a round pen, the horse is much weaker when it reverses direction one way. Generally the hindlimb on the outside of the circle after the weak rollback is the lame limb.

Competition is now so great that the difference between winning and losing is sometimes a matter of which horse feels the best. Performance diminishes before outright lameness is noted. Hindlimb lameness is particularly common in reined cow horses because of hock or stifle pain or proximal suspensory desmitis.

Flexion tests include flexion of the digit in all limbs and carpal, hock, and stifle flexions. Notes are made on the results and whether the horse resented flexion. The digit is flexed in an oblique plane (applying medial and lateral torque), and any resentment or diminished elasticity is noted.

All of the soft tissue structures are palpated, and any enlargements and sensitivity are noted. The hoof testers always are applied to the front feet, and to the hind feet of horses with hindlimb lameness. Any lameness or weakness is classified in degrees (grade 1 to 5); by the limb involved; by weight bearing or non-weight bearing or mixed status; by the effect of walking in a circle or on a straight line, on an incline or decline; by flexion tests; and by the effects of prolonged digital pressure over sensitive areas.

If lameness is serious enough possibly to be caused by fracture, radiography is performed first; otherwise, diagnostic analgesia is used to establish the source of pain. Intra-articular analgesia is more specific than regional analgesia, which can be done at a later time if necessary, so I often start with intra-articular blocks, especially in forelimbs. I rely on the flexion tests to direct the start point. If flexion of the distal limb is normal but carpal flexion is positive, I usually use perineural analgesia of the proximal palmar metacarpal and palmar nerves (high four-point). If lameness persists, I perform intra-articular analgesia of the carpus. If flexion of the distal limb is positive, I perform intra-articular analgesia of the distal interphalangeal joint first and then proceed to the pastern and fetlock joints if necessary to eliminate the lameness. If intra-articular analgesia is not helpful and the flexion test is still positive, I perform perineural analgesia of the palmar digital nerves, followed by the digital nerves (abaxial sesamoid) and later a low four-point block. Knowing whether the problem is articular is useful, because horses with articular problems are often more amenable to treatment.

For intra-articular analgesia I do a three-step povidone-iodine (Betadine) and alcohol skin preparation and use sterile gloves. I place a subcutaneous bleb of local anesthetic solution before injection and combine local anesthetic solution with gentamicin sulfate (40 mg).

In a hindlimb I usually use perineural analgesia proximal to the fetlock, local infiltration around the region of the SL, intra-articular analgesia of the centrodistal and tarsometatarsal joints separately, and intra-articular analgesia of the femoropatellar and medial femorotibial joints. The lateral femorotibial compartment rarely is involved. If the response to a low four-point block is positive but the response to plantar (abaxial sesamoid) nerve blocks is negative, intra-articular analgesia of the metatarsophalangeal joint is performed the following day.

IMAGING CONSIDERATIONS

Once the region of pain is localized, the area is examined using radiography and ultrasonography to identify the exact structure that has been damaged and to assess the degree of damage. The more specific I can be at this point, the more reliable the prognosis and treatment are. If the diagnosis is not specific, scintigraphy often is used. Generally a differential diagnostic list is generated based on the physical examination. Developing a therapeutic plan based on a tentative or vague diagnosis is a mistake. Nearly all diagnoses are tentative until the pain has been localized as specifically as possible through the use of diagnostic analgesia and appropriate imaging.

PROCEEDING WITHOUT A DIAGNOSIS

Sometimes the cause of lameness cannot be discovered, even after all of the joints and nerves have been blocked and a complete scintigraphic examination has been performed. I then treat the horse for myofascial pain with drugs or chiropractic therapy. If the horse responds to these therapies, then this is indirect evidence of the cause. Another possible cause is equine protozoal myelitis. Horses with equine protozoal myelitis may show chronic lameness without other neurological signs.

LAMENESS AND SHOEING CONSIDERATIONS

Proper foot balance is critical to resolving all lameness problems successfully. The foot should be trimmed to land evenly. Any medial-to-lateral imbalance should be removed by trimming. If the point of contact is the lateral toe quarter, then enough of the lateral wall should be removed so the foot lands flat. The foot should be trimmed or a wedge used to achieve a hoof angle that is the same as the pastern angle (about 55° in forelimbs and 57° in hindlimbs). A square-toed shoe that has a wide bevel, which extends from the medial to lateral toe nail holes, commonly is recommended and is useful. The shoe should be set back so the dorsopalmar axis is shortened. The heels are trimmed so the shoe comes in contact with the wall at the widest section of the frog. These specifications are designed to remove rotational forces up the limb that occur in horses with medial to lateral imbalances, to provide mechanical advantage to the palmar/plantar aspect of the limb that occurs in horses with with a long toe and low heels, and to reduce concussion to the digit that occurs in horses with the two-phase foot impact associated with foot imbalance.

An egg bar shoe is used for horses with moderate to severe tendonitis, suspensory desmitis, or an apical sesamoid fracture. A reverse shoe is used sometimes for horses with navicular syndrome, ringbone, sesamoiditis, suspensory desmitis, tendonitis, constricted palmar/plantar annular ligament, carpitis, and tarsitis. Horses with bruising in the sole are shod with soft dental acrylic pads. Horses with heel and quarter cracks are managed with egg bar shoes.

TREATMENT OF LAMENESS

Suspensory Desmitis

For horses with suspensory desmitis, it is important to optimize foot balance as previously described. Corrective shoeing is designed to minimize any further physical damage and therefore prevent further pain and inflammation. Other treatments provide short-term results and are aimed at reducing the existing inflammation.

Horses with minor to moderate fiber disruption are treated with perilesional injections of Sarapin and corticosteroids. If

the lesion is proximal, a sterile skin preparation is recommended, with the addition of gentamicin sulfate (40 mg) in case the carpometacarpal joint is entered inadvertently. The drugs are injected under the loose tissue between the SL and the accessory ligament of the deep digital flexor tendon. Middle carpal joint pain may occur concurrently and is treated using intra-articularly administered corticosteroids, provided that no radiographic abnormalities are apparent. Physical therapy in the form of electrical stimulus modalities or low-level light laser therapy is started 3 days after injection and continued as needed through the intended competition. Magnets of 600 to 1200 G strength placed over the site have been used successfully and are kept in place for 60 days and then as needed for soundness.

The lesion is monitored monthly using ultrasonography. If conservative treatment fails, extracorporeal shock wave treatment is used and administered with the horse under general anesthesia. Up to four treatments have been used, combined with rest for 60 to 90 days.

Centrodistal and Tarsometatarsal Joint Pain

Intra-articular injection of corticosteroids is effective at relieving pain and inflammation in the centrodistal and tarsometatarsal joints. Horses are allowed to rest for at least 10 days after injection and then work gradually is increased as soundness dictates. The systemic use of hyaluronan and acetylglucosamine is helpful for maintenance. Orally administered chondroitin sulfate and glucosamine products also have been useful. If medication is ineffective, joint drilling or forage to induce joint ankylosis, or peri-articular extracorporeal shock wave treatment can be used effectively.

Navicular Syndrome

Navicular syndrome is diagnosed based on a positive response to palmar digital analgesia and analgesia of the distal interphalangeal joint, radiography, and scintigraphy. Radiography can be misleading, and in my opinion scintigraphy is the most reliable method of diagnosis. Corrective shoeing is essential. Isoxsuprine hydrochloride (600 mg) is given orally once daily for 60 days. Aspirin (15 g) also is given orally once daily for 60 days. If this combination provides relief, it is continued for another 60 days or until the horse finishes the futurity season. Injection of the distal interphalangeal joint with corticosteroids, or a combination of corticosteroid and hyaluronan, often provides temporary relief (about 30 days).

If the horse fails to respond to the corrective shoeing and medications, shock wave treatment is indicated and has been effective, except in horses with entheseous new bone at the attachment of the collateral ligaments of the navicular bone. Palmar digital neurectomy is used as a last resort.

Traumatic Hoof Injuries

Horses with bruises and cracks in the hooves respond well to egg bar shoes with soft dental acrylic pads or metal pads. A horse with a quarter crack is treated by trimming the wall palmar/plantar to the defect, so that no contact is made with the shoe, that is, floating the heel. The air gap is up to 6 mm deep and is reopened daily using a hacksaw blade to relieve pressure between the hoof wall and the shoe, until the defect has grown out. If the crack is moving and bleeding, a copper patch is attached to the wall with screws to span the defect and therefore stabilize it.

Superficial Digital Flexor Tendonitis

Ultrasonography is used to characterize size and position of the lesion in horses with superficial digital flexor tendonitis. If a lesion is small, the owner or trainer usually decides to continue preparing for the futurity. It is important to try to determine the circumstances that caused the lesion to develop to eliminate further damage. Possible causes for tendonitis include poor hoof balance, deep footing, fatigue, speed, overexertion from working a difficult cow, uncontrolled exercise (turnout), interference from hindlimbs, tight bandages, poorly fitting support boots, and hanging a leg on a hot walker, tie rope, or fence.

Therapy for tendonitis involves depositing corticosteroids in the loose subcutaneous tissue in the proximity of the lesion. The leg is wrapped in a gelocast for 3 days and then is treated with ice and laser or electrical stimulus therapy. The horse is walked in hand or under saddle for 15 minutes twice daily for 7 days. Training then is resumed. The leg is maintained in a support wrap continuously. Shoeing, good footing, and conservative training are critical to the healing process.

Stifle Pain

If no abnormalities are detected in the stifle radiographically, the affected joints are treated with intra-articularly administered corticosteroids and hyaluronan. The horse is prescribed weekly injections of systemic acetylglucosamine, which continue through the futurity. Subsequent intra-articular injections are not performed sooner than 6 months and preferably not at all.

If radiographic evidence of osteochondrosis of the trochlear ridges of the femur exists, the same therapy is effective. The prognosis for horses with subchondral bone cysts of the medial femoral condyle is dismal, and I do not recommend that these horses continue training to be futurity horses.

Metatarsophalangeal Joint Pain

Metatarsophalangeal joint pain may be caused by traumatic arthritis, fracture of a proximal sesamoid bone, sesamoiditis, fragments detached from the proximoplantar aspect of the proximal phalanx, or peri-articular soft tissue injury. If no radiographic lesions are present, the horse is treated with intra-articularly administered betamethasone and hyaluronan, followed by 10 days of rest. Systemic glucosamine is given every 2 weeks as maintenance therapy. Orally administered glucosamine and chondroitin sulfate also seem to be useful in managing metatarsophalangeal joint pain. Shoeing imbalances must be corrected concurrently.

Sesamoiditis

The foot must be properly balanced to treat sesamoiditis. Egg bar shoes with a square toe and a wide roll or bevel are used. The shoe is set back to minimize the leverage on the palmar/plantar aspects of the fetlock. The hoof angle is optimal when it matches the pastern angle. Isoxsuprine hydrochloride (600 mg) and aspirin (15 g) are given orally once daily for 60 to 90 days. Electromagnetic energy is applied to the affected region every other day for 3 months and thereafter as needed for soundness. The amount of training is dictated by the degree of soundness.

Distal Interphalangeal Joint Pain

The foot must be properly balanced to treat distal interphalangeal joint pain. Soft dental acrylic pour-in type pads and shock-absorbing rim pads can be helpful. Intra-articular injection of a corticosteroid and hyaluronan is followed by the systemic use of acetylglucosamine and hyaluronan. It is imperative that training is continued on a soft surface.

Osteitis of the Distal Phalanx

Soft dental acrylic pour-in type pads and shock-absorbing rim pads are used to treat horses with osteitis of the distal phalanx. The horse is treated with isoxsuprine hydrochloride (600 mg), aspirin (15 g), and phenylbutazone (2 g) given orally once daily for 60 days and then as needed for soundness. Training must be done in soft footing to reduce concussion to the foot. Extracorporeal shock wave therapy has recently been introduced to manage horses with osteitis of the distal phalanx.

BARREL-RACING HORSES

• Robin M. Dabareiner and G. Kent Carter

Barrel racing began in 1948 when a group of Texas ranch women started riding horses around a cloverleaf pattern of barrels and the fastest horse around the course was the winner. Barrel racing has evolved into a multimillion-dollar industry called the Women's Professional Rodeo Association. From this original organization many other barrel racing associations, which host numerous futurities for horses 4 or 5 years of age or younger and derbies for older horses, have arisen. In 2000, five futurity events were held nationwide, with each futurity paying out more than $800,000 in prize money from which the winner is guaranteed more than $100,000. The major competitions are divided into rodeo, futurities, derbies, open jackpots, and professional levels. Futurities and derbies are classified by age of the horse. Open competitions are open to any horse or rider and are highly competitive. Within the open jackpots, a new classification of barrel racing has developed, termed a *3D* or *4D competition*, which includes a handicapping system that allows less experienced horses or riders to compete with seasoned horses and riders.

Barrel racing is a timed event with the clock beginning when a predetermined line is crossed. Three barrels (55-gallon steel drums) are positioned in a triangle or cloverleaf (Fig. 121-5). The contestant must turn the left or right barrel first (most choose to turn the right-hand barrel first) in a manner that the path always crosses. The distance covered for the pattern varies with the size of the arena, but generally the distance from the starting line to the first barrel is 14 to 18 m. The distance between the first and second barrel is 21 to 27 m and between the second and third barrel is 27 to 32 m. The horses must start at the alleyway or entrance to the arena, run at full speed to the first barrel, slow down and complete a 360° turn around the barrel, speed up and run to the second barrel, turn 360°, head to the third barrel, turn 360°, and then sprint to the finish line. The fastest time to complete the pattern varies with distance of the pattern and arena size, but a good run for a large arena pattern is 15 to 16 seconds. An electric eye timing system usually is used, and times are recorded in hundredths of a second. If a rider hits a barrel and tips it over, a 5-second penalty is imposed. If the horse and rider fail to negotiate the pattern correctly, they are disqualified. The arena is often leveled after each horse.

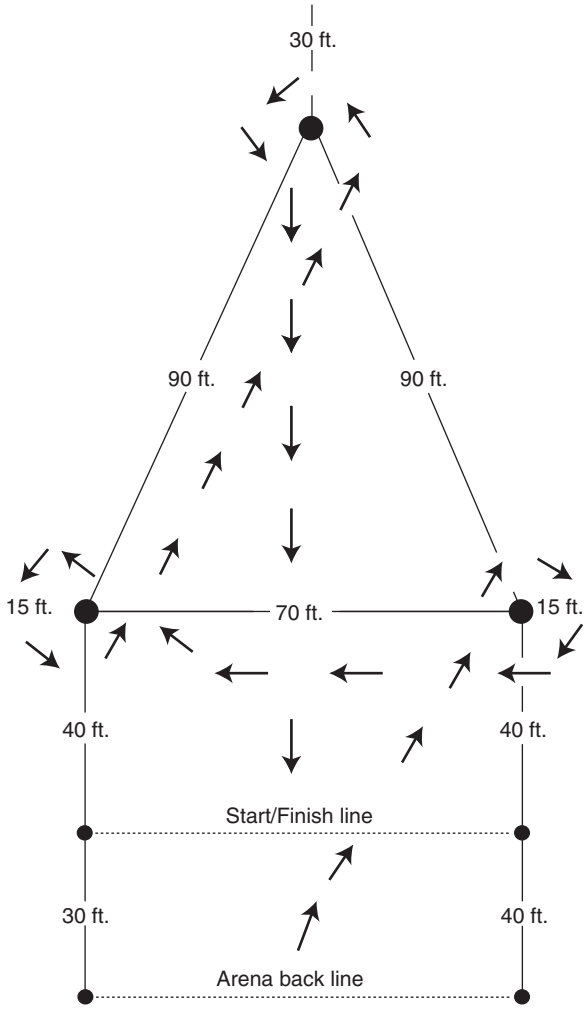

Fig. 121-5 Diagram of barrel racing cloverleaf.

CONFORMATION

Horses are predominately American QHs and Appendix horses (QH and Thoroughbred crosses), but occasionally Thoroughbred, Paint, or Arabian horses are seen. Speed, quick acceleration, and agility to slow down quickly to turn the barrel are essential. Many barrel horses have QH racehorse pedigrees. Some prefer stockbred horses, which usually have a calmer personality. Geldings are preferred. Horses usually range from 14.3 to 15.1 hands. Barrel horses are not usually as muscular and wide based as calf roping horses. They usually have long, leaner muscle mass that provides the speed and flexibility needed to perform the 360° turns.[4]

TRAINING

Futurity competitions are popular because of the amount of prize money, and training usually starts at 2 years of age. Futurity horses often have short careers because of the pressure to perform at a high level of competition at only 4 to 5 years of age, resulting in advanced osteoarthritis, often in the distal tarsal joints. Successful futurity horses seldom have long-term careers because of behavioral problems or performance-limiting injuries.[1]

Horses that are 5 years of age or older, started slowly, and are allowed to mature before performing in serious competition may compete professionally for 5 years.[2] The average age of a professional barrel horse is 10 to 15 years. Horses may compete in 50 to 100 rodeos per year and spend much time traveling. An experienced barrel horse is trained by practicing turns but rarely uses the barrels because the horse learns to anticipate the pattern and tires of it. Many professional horses are ponied (led from another horse), or are ridden just enough to keep them fit for competition, but they are never really worked unless competing.[4,6]

Older, semi-retired horses that are well trained to the barrel pattern, but not fast enough for upper-level competition, may be used for a child or beginner. These horses do make repetitive barrel pattern runs in a practice pen for the rider to learn and often have use-related injuries, requiring much maintenance to withstand the number of practice runs needed to teach a beginner.

LAMENESS EXAMINATION

A barrel horse does not run at top speed over long distances like flat racehorses; therefore fatigue-type injuries like

superficial digital flexor tendonitis or fetlock and carpal chip fractures are seen rarely.

Some of the common injuries seen in barrel horses are attributed to the twisting and turning motion of the horse at high speeds (Fig. 121-6). Other musculoskeletal problems vary by the horse's age and level of competition. A young futurity barrel horse may run up the fence, that is, the horse does not turn around the barrel but runs past it up the arena, or it may refuse to enter the arena. No lameness may be detectable, and the veterinarian has to determine whether the young horse is mentally stressed and showing behavioral problems or is responding to pain. Complaints about an older, experienced horse usually result from pain, but behavioral problems occur more often in young horses. Most barrel racers train their own horses, and the quality of training is inconsistent, and performance changes may be induced by the rider.

The teeth are evaluated to eliminate bitting problems as a cause of performance changes. Radiographic examination of the hocks is performed because of the high incidence of distal hock joint pain. The stifles also are examined to eliminate osteochondrosis as a potential source of pain. If no radiographic abnormalities are detected, several options are presented to the owner to help determine if the horse is responding to pain or is overtraining. One option is to administer phenylbutazone (2.2 mg/kg daily for 2 weeks and then 1.1 mg/kg daily for 2 weeks) for 30 days and to keep the horse in barrel-race training. If the horse returns to normal performance, then we conclude that the horse is most likely experiencing pain. If the problem does not resolve, then we try to address any behavioral problems. If problems persist, we often suggest giving the horse a 3- to 6-month break from barrel racing and have the horse work cattle or perform other less intense activities. A second option is to increase the workload and try to establish lameness that can be identified and then located with diagnostic analgesia, but few owners select this option. Because of high incidence of distal hock joint pain and the poor correlation between clinical signs and radiographic abnormalities, the centrodistal and tarsometatarsal joints can be medicated with hyaluronan, with or without corticosteroids, and the response assessed. Nuclear scintigraphic examination can be performed but often is unrewarding; however, owners may be encouraged then to increase the horse's work to see if overt lameness develops.

Fig. 121-6 A barrel horse turning a barrel. Note the pressure on the inside hindlimb. This horse is on the backside of the barrel coming out of the turn and is in correct position. At this point in the barrel turn a horse with hindlimb pain becomes uncollected and swings wide around the barrel.

An experienced horse may begin to have a change in behavior or performance rather than show an actual lameness. Behavioral problems occur more often than in other Western performance events, because the horse may experience pain at the same spot in relation to the barrel, regardless of arena size, and thus the horse begins to dread making the barrel turn.[3] A common complaint is that the horse is coming up out of the turn on the first barrel, meaning that the horse heads into the turn normally, but as the horse comes into the backside of the barrel turn, it does not want to stay in the ground or push off aggressively to the next barrel. Instead the horse comes uncollected, bounces out of the barrel, and makes a wide turn around the barrel that adds time to the run. Such behavior often is associated with distal hock joint pain, especially involving the hindlimb on the inside of the turn. The horse may not run between the barrels as it should or is slower, 3 or 4 tenths of a second off its time. Trotting the horse in hand in a small circle (1.5- to 3-m radius), mimicking the barrel turn, is helpful. This may be the only time the horse shows lameness. Determining which barrel is the problem barrel is helpful. An owner may complain that the horse will not stay in the ground for the first barrel but works fine for the second and third barrels. The horse may perform fine for the first barrel but does not make a good turn around the second barrel. The first barrel (usually going to the right) is the hardest barrel and most often a problem, because the horse is running at full speed when it reaches the barrel and must slow quickly to make the 360° turn.

When the horse is turning the barrel, the inside hindlimb appears to be under the greatest pressure (see Fig. 121-6). If the owner complains that the horse is swinging wide or pulling away from the first barrel, the horse does not want to turn to the right and is probably trying to avoid inside hindlimb pain. The outside forelimb is also under much stretching pressure as the horse goes into a barrel turn, but we also have seen horses that do not want to take the first barrel to the right and have right (inside) forelimb pain. Hindlimb lameness is more common than forelimb lameness, and less correlation exists between horses resisting a particular direction of the turn relative to a specific forelimb lameness.

DIAGNOSIS AND MANAGEMENT OF SPECIFIC LAMENESS

Distal hock osteoarthritis is the most commonly diagnosed problem, causing reduced performance or lameness. Diagnosis is based on intra-articular analgesia, radiography, response to treatment, and sometimes nuclear scintigraphy. The centrodistal and tarsometatarsal joints are treated using methylprednisolone acetate (40 to 60 mg per joint) or triamcinolone acetonide (6 mg per joint) alone, or in combination with hyaluronan (20 mg per joint). Ideally the horse should have 7 to 10 days of turnout or light exercise before returning to barrel racing, but often the competition schedule does not allow this. Response to treatment may take up to 3 weeks. If the horse does not respond to treatment, we re-evaluate the horse for lameness.

Hindlimb proximal suspensory desmitis is common in older, seasoned horses and can be a career-ending injury. Deep sand in arenas may contribute to the high incidence. Moderate lameness (grade 2 to 3 of 5) often is exacerbated by upper limb

flexion. The horse usually has no swelling or minimal swelling and no heat and pain. Local analgesia is essential to verify the source of pain. Radiography of the proximal metatarsal area is often negative, although if the condition is chronic, sclerosis or entheseous new bone may be present. Ultrasonographic examination may reveal an enlarged and hypoechoic SL, but some horses have no detectable structural abnormalities. The latter are treated by local infiltration of triamcinolone acetonide (9 to 12 mg) and reduced work. With acute, mild proximal suspensory desmitis horses often return to work in 2 to 4 weeks. We also recommend confining the horse to a small area to avoid further damage from excessive uncontrolled exercise. If ultrasonographic abnormalities are detected, the horse is confined to a small area with no riding for 60 to 90 days before reexamination. Complete healing often takes 6 to 12 months, and some horses never return to full athletic function.

Suspensory branch injuries are common in hindlimbs, especially the right, resulting in mild hindlimb lameness (grade 1 to 2 of 5), which is improved by a low plantar nerve block. Normally detectable ultrasonographic abnormalities are present, but radiographic abnormalities of the proximal sesamoid bones are unusual. Horses respond well to 60 to 90 days of small area confinement, followed by a gradual return to conditioning and resumption of competition after 4 months. We recommend the use of boots to protect the hindlimb soft tissues. If lameness is improved by a low plantar block but ultrasonographic and radiographic examinations are negative, intra-articular analgesia of the metatarsophalangeal joint and intrathecal analgesia of the digital flexor tendon sheath are performed. If the response is positive, the joint or digital flexor tendon sheath is treated with hyaluronan (20 mg) and triamcinolone acetonide (9 mg). These horses usually can continue competing but may have recurrent lameness.

QH and Appendix QHs make up most of the barrel horses. Sore feet, navicular disease, and palmar foot pain are the most frequent causes of forelimb lameness, with poor hoof conformation and farriery and hard arena surfaces being predisposing factors. Diagnosis and management are similar to those described for the team roping horse.

Proximal suspensory desmitis is also common in forelimbs. Deep arenas and long toes and low heels are predisposing factors. The prognosis for barrel racing horses with forelimb proximal suspensory desmitis is good, with most horses responding well to rest and controlled exercise for 3 to 4 months. Tendon protection boots are recommended.

Metacarpophalangeal joint synovitis results in mild lameness (grade 1 to 2 of 5) that is exacerbated by fetlock flexion. The horse often strongly resists fetlock flexion. Lameness and response to fetlock flexion are eliminated by intra-articular analgesia. Radiographic examination is usually negative. The horse is treated with intra-articularly administered triamcinolone acetonide (6 mg) and hyaluronan (20 mg). The problem may or may not recur. If recurrence is frequent, diagnostic arthroscopy is recommended.

Many times horses have undergone several types of alternative therapies (e.g., equipment changes, acupuncture, chiropractic, and herbal therapy) in an attempt to correct the perceived problems before being checked by a veterinarian. Often, for some inexplicable reason, an accurate diagnosis and conventional medical therapy is sought only after failure of these other treatments. Many owners rely on diagnosis from a fellow competitor, and request treatment that was successful in another horse. This makes client communication difficult.

SHOEING CONSIDERATIONS

Traction, especially in the deep and often muddy arenas in which horses compete, is critical. A rim shoe is used most commonly on all feet, with one rim higher than the other to provide added traction. The rounded toe provides easier breakover than a normal flat steel shoe. Hind shoes may have square toes.

Recently the natural balance shoe has gained popularity as owners are becoming more aware of good farriery principles and are trying to correct long-toe, low-heel problems. The natural balance shoe is available in steel or aluminum. The steel construction is preferred because of the added weight, which provides better traction than the aluminum shoe. The natural balance shoe has a rockered toe that eases breakover of the forelimb and is built with a wider web than a normal shoe, which benefits a sore-footed horse competing on hard surfaces.

OTHER CONSIDERATIONS

Exercise-induced pulmonary hemorrhage is common in barrel horses. The owner may complain that the horse has lost a step or has become slower. If a musculoskeletal reason for the performance change cannot be determined, a respiratory evaluation is indicated. Obvious bleeding from the nostrils rarely is seen, but endoscopic examination within 30 minutes of the barrel race often reveals tracheal hemorrhage. Presently no drug testing or rules prevent drug administration for competition. Most horses receive phenylbutazone or flunixin meglumine 4 to 6 hours before competition and furosemide (4 to 8 ml per horse).

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THE EUROPEAN WESTERN PERFORMANCE HORSE

• Franco Ferrero

The Western performance sport horse has developed in Europe since the mid-1980s. Most horses are American QHs and are used for reining, a sport most popular in Germany and Italy. The Italian reining and cutting futurities for 3-year-olds offer the highest prize money. The rules governing competition usually are based on the American rules, with a few notable exceptions in Germany. Horses entered in the Reining Breeder's futurity must be 4 years of age. Strict rules govern which bits and harness are acceptable. Excessive use of force, spurs, or the whip may result in suspension of the rider.

Reining QHs usually are broken at 18 months of age and may undergo intense training in preparation for the snaffle-bit futurity at 3 years of age, but these horses rarely compete at high levels when older. Only a limited number of horses compete after 6 years of age.

TEN MOST COMMON LAMENESS CONDITIONS

The 10 most common causes of lameness in reining QHs 2 to 4 years of age are as follows:

1. Foot pain
2. Hock pain
3. Carpal pain

4. Fetlock pain
5. Stifle pain
6. Splints
7. Superficial digital flexor tendonitis
8. Desmitis of the accessory ligament of the deep digital flexor tendon
9. Suspensory ligament desmitis
10. Shoulder injury

Causes of foot pain include subsolar abscessation, solar bruising, osteoarthritis of the distal interphalangeal joint, fracture of the palmar process of the distal phalanx, osteitis of the palmar processes of the distal phalanx, and navicular syndrome.

Hoof tester examination is an essential diagnostic tool. Oblique radiographic views of the distal phalanx are invaluable for assessing the palmar processes. Poor foot conformation is often an important predisposing factor for the development of foot pain. The foot is often upright with overgrown angles of the walls and heels, flattening or convexity of the sole at the toe, and an underdeveloped frog. The shoes are often too small. Bar shoes also may prevent the heels from digging into the ground.

Osteoarthritis of the distal hock joints is common. Horses of 2 years of age have a high incidence of partial collapse of the central and third tarsal bones with secondary osteoarthritis. Carpal problems occur more commonly than in North America and include synovitis of the antebrachiocarpal and middle carpal joints, osteoarthritis, sagittal fracture of the third carpal bone, and chip fractures of the distal aspect of the radial carpal bone. Conformational abnormalities such as carpus valgus, calf knees (palmar deviation of the carpal joints), tied-in knees, and offset knees may be important predisposing factors. The limited number of blood lines available for breeding may influence the incidence of these conformational abnormalities. Bench or offset knees also may predispose horses to the high incidence of splints causing lameness, especially in 2-year-old horses.

CHAPTER • 122

The Tennessee Walking Horse

James T. Blackford and James C. Sternberg

DESCRIPTION OF THE SPORT

The Tennessee Walking Horse, commonly called the Walking Horse, is a light horse breed developed in middle Tennessee for use on southern plantations during the eighteenth century. The average Walking Horse is 15 to 16 hands tall and weighs 500 to 600 kg. The Tennessee Walking Horse is a composite breed, created by crossbreeding Canadian and Narragansett Pacers, Morgan, Standardbred, and Thoroughbred horses. The Tennessee Walking Horse Breeders' and Exhibitors' Association was formed in 1935. Because of the versatility of the Walking Horse, along with a characteristic racking gait within the breed, a second breed association was recognized in 1971, the Racking Horse Breeders' Association. This group of horses is commonly referred to as the Racking Horse.

The Walking Horse performs three gaits: the flat-foot walk, running walk, and canter. Both walks are four-beat gaits, with one foot up and three feet in various phases of striking the ground. The footfall sequence is left hind, left front, right hind, and right front. High-stepping forelimbs with an extended reach characterize the flat-foot walk. The hind foot overreaches the imprint of the front foot by 15 to 55 cm in a straight, smooth, gliding motion. This over-stride is unique to the breed and is referred to as the big lick. The horse's head and neck nod, and the ears flick forward and backward in rhythmical fashion with the rise and fall of the front feet. The appearance is that of pulling with the forelimbs and driving or pushing with the hindlimbs. The speed of the flat-foot walk is from 4 to 8 miles per hour. At the running walk, the gait is basically the same but faster (10 to 20 mph). The canter is a three-beat gait, with a left or right lead. In the canter the horse lifts the forehand, giving an easy rise and fall, in a rolling motion. The gait is referred to commonly as the rocking-chair canter because of the high, rolling movement of the horse's body.

The Racking Horse gait is a bilateral four-beat gait, with one foot striking the ground with the other three limbs in various phases of elevation. The gait often is referred to as the single-foot gait. When shown, the Racking Horse performs the show walk, slow rack, and fast rack. The show walk is a smooth, collected, slow and easy four-beat gait. At the slow rack the horse's head is held with the neck arched and ears erect. The fast rack is similar in form to the slow rack, but the horse displays speed and leg action. The natural head nod must not be exaggerated at the slow or fast rack.

In both breeds the collected gaits of the show horse shift the center of gravity caudally, compared with most other light breeds, with increased loads on the hindlimbs resulting in a high incidence of hindlimb lameness.

Both breeds are used for show, trail, and pleasure riding. Horses are shown at halter, harness, and under tack, with English or Western saddles. Horses that are shown wear a light shoe similar to a standard keg shoe, a plantation shoe, or a performance shoe, depending on the class. The plantation shoe cannot exceed 3.8 cm in width and 1.3 cm in thickness, and the heel calk cannot exceed 2.5 cm in height. The performance shoe, or stack, is a shoeing technique used to accentuate the show horse gaits. The shoe is constructed of several layers of flat or wedge pads, stacked one on top of another, placed between a nail pad at the solar margin and a metal shoe on the contact surface. Pads are made of leather, plastic, or hard rubber. The amount of height or extension the shoe provides must not exceed 50% of the natural hoof wall length, measured from the coronary band to the tip of the toe. Pleasure horses are commonly flat shod.

Although the highest population of Walking and Racking horses is presently in the southeast United States, they are becoming more popular across North America, especially for trail and pleasure riding, because of the physical endurance and

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5. Stifle pain
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Although the highest population of Walking and Racking horses is presently in the southeast United States, they are becoming more popular across North America, especially for trail and pleasure riding, because of the physical endurance and

gentle disposition of the breeds. The show season generally is considered to be year-round, picking up in the spring and peaking in the fall with the national championships. The Tennessee Walking Horse National Celebration is held in Shelbyville, Tennessee, each year and is an 11-day event ending with the crowning of the national champions on the Saturday before Labor Day. The Racking Horse Celebration is held each year in Decatur, Alabama, during the last 2 weeks in September.

LAMENESS EXAMINATION

Lameness evaluation of the gaited horse is best performed with the horse under complete tack with a rider. It is impossible to duplicate the head set, balance, and shifting of the horse's center of gravity caudally without a rider. The horses generally exercise in a large, oval arena, and to perform a good evaluation, one needs to provide a place where the horse has room to work in all gaits. The ground surface in this area should be firm. A veterinarian's impression when evaluating a show horse in performance shoes for the first time will be that the horse's problem must be in the forelimb and more specifically the foot. In light of the negative publicity the stacked shoe receives, focusing one's attention on this area is easy. Even as the horse is walked at halter, this impression may not change, because of the awkward nature of the horse handling the bulky shoe. In our experience, few problems are associated directly with the shoe, except hoof wall avulsions, when the shoe is accidentally pulled off in the show ring. Measures to control bleeding must be taken immediately, followed by good wound care. Hoof wall composites then can be used to restore hoof wall function. If an elevated shoe is removed completely and suddenly from an immature, developing horse early in training, removal causes a mechanical flexor contracture. The horse knuckles over at the fetlock joint because of the pain associated with pulling of the deep and superficial digital flexor tendons.

Unfamiliarity with the standard gaits of Walking and Racking horses, especially when shod with a stacked shoe, may result in confusion with ataxia, particularly at faster speeds. Anecdotally, our impression is that a high incidence of cervical vertebral malformation occurs in Tennessee Walking Horses, so this should be kept in mind.

Tennessee Walking Horses are stoical and seem to have a high pain tolerance. Problems encountered in the flat-shod Walkers or Rackers mirror the lameness problems seen in other light breeds; therefore the following discussion focuses on show horses in stacked shoes. The hoof tester evaluation is of limited value on the front feet of performance-shod horses. Careful palpation and manipulation of the limbs are performed routinely. Careful evaluation of the foot and shoe should not be overlooked. Pressure-shoeing techniques by unscrupulous owners and trainers, which causes soreness and increases the big lick, occasionally arise in a performance-shod horse and should not be overlooked. The technique is performed by over-trimming the hoof wall and thus increasing sole pressure. A carefully placed nail in the nail pad encroaching on the sole or the sensitive laminae may be noted. A foreign object, such as a stone inserted between the pad and the sole inflict discomfort. Digital pulses, tapping on the hoof wall with hoof testers or a shoeing hammer, and response to nerve blocks may determine the problem.

Problems seen in the forelimbs commonly are associated with imbalances in the shoeing, leading to an uneven height of the forelimb carriage. To evaluate the forelimb carriage, the observer should draw an imaginary line at the chest level, watching the carpal action to determine which leg is carried lower to the imaginary line as the limb advances, indicating the problem limb. When this problem is detected, careful evaluation of the shoeing technique and comparison between limbs are indicated. Correction of foot imbalance may resolve the problem. Osteoarthritis of the distal interphalangeal joint (low ringbone) frequently is seen in older horses but is not always correlated with lameness. Proximal suspensory desmitis usually is caused by slipping in an elevated shoe and may be associated with localized pain on palpation. Bicipital bursitis is seen occasionally. The horse may have asymmetry in shoulder motion and pain on palpation of the area, especially when the limb is raised. Abduction or adduction of the shoulder is performed. Like most breeds, sole abscesses are common in flat or performance-shod horses, as is laminitis.

The highest percentage of lameness seen in our practice with Walking and Racking horses is associated with the hindlimbs, because of loading of the hindlimbs during exercise. As each hindlimb strikes the ground and the horse's weight is carried through the stride, a rotational, twisting motion also is seen throughout the entire limb before the leg is advanced. This is especially noticeable in the Walking Horse at the faster gaits. The shifting weight and the twisting motion increase stress on the joints and surrounding support structures. Osteoarthritis of the distal hock joints and stifle is common. A positive response to the Churchill test is helpful in isolating pain to the distal hock joints. Careful palpation of the medial patellar ligament is particularly important in this type of sport horse, because at one time almost all of the show horses were subjected to medial patellar desmotomy with hopes of improving the over-reaching stride of the rear legs. Although the medial patellar desmotomy site only occasionally is found to be the origin of the lameness in these horses, the possibility should not be overlooked. The practice is not as common today as in the past; however, many trainers from the old school feel that having the desmotomy performed is important. Upper- and lower-limb flexion tests are performed. Local analgesia is used to define the source of pain. Trochanteric bursitis occurs in Walking and Racking horses and is related to the rotational, twisting motion of the hindlimbs. The horse has pain on deep palpation over the greater trochanter of the femur, during upper limb flexion tests, and when the hip is stretched forward or backward, abducted or adducted, and rotated.

Other conditions are encountered in these horses. Some have muscular weakness in the hindlimbs, especially in young unconditioned horses. When weakness is combined with straight hindlimbs, instability of the patella is common. Chronic hip and stifle soreness caused by a trailer on the outside of the shoe may be an underlying cause of lameness. Osteochondrosis occurs in the forelimbs and hindlimbs. We see a substantial number of horses that are thought to be lame but have equine protozoal myelitis.

CHAPTER • 123

Lameness in the American Saddlebred and Other Trotting Breeds with Collection

Scott D. Bennett

DESCRIPTION OF THE SPORT

The American Saddlebred, Morgan, Hackney Pony, National Show Horse, and Arabian (see Chapter 124) show horses are described as trotting breeds with collection. Lameness in disciplines such as dressage, road horses, and road ponies is similar. The American Saddlebred and National Show Horse have five gaits: walk, trot, canter, slow gait, and rack. The slow gait and rack are manmade gaits. These horses also perform in three gaited classes (walk, trot, and canter), fine harness classes, pleasure driving, pleasure-gaited classes, and equitation. Morgan and Arabian horses are shown similarly, but without the slow gait or rack. Hackney ponies are shown in harness, pleasure driving, and road pony classes. Show classes are further divided for professional, amateur, and juvenile riders. Equitation, hunt seat, Western, and numerous young horse and in-hand halter classes are available.

In road horse classes, Standardbreds, Morgans, American Saddlebreds, or Standardbred-cross horses are shown at the walk, trot, and road gait pulling a bike similar to a sulky used for Standardbred racehorses. These horses usually are more animated in gait than Standardbred racehorses and go both ways around the ring when performing. The road gait is a high-speed performance gait.

To understand lameness in the gaited show horse, the veterinarian must first understand the difference in locomotion between running and gaited horse disciplines. Concussion (impact) is a part of every gait. How the horse distributes concussion is related directly to athletic ability and the longevity of a horse's career. Better equine athletes are more efficient in the distribution of concussion through the limbs and body. The superior equine athlete appears capable of using energy of concussion efficiently and distributing it for dispersion and recovery. Normally kinetic or stored energy from proper distribution of concussion causes recoil of the tissues receiving the energy of concussion. Tissue injury results in an inability to disperse concussive energy properly. Maintaining healthy hoof wall, bone, cartilage, tendons, ligaments, and muscles in a good conditioning and gait management program is essential. The veterinarian must be familiar with the gaits, because gait analysis is an important part of evaluating poor performance and subtle lameness.

Gaits of the show horse are complex and must be synchronous to maintain distribution of concussion. Synchrony must be achieved in up to five gaits and is altered by the different gait specifications. The normal load distribution between forelimbs and hindlimbs in the show horse is about 35% and 65%, respectively (see Chapter 2). The show horse does not have to perform at speed. Compared with many sport horses, synchrony of concussion and weight distribution are totally different, and because much concussion is dispersed through the hindlimbs, hindlimb lameness is more important. In some other sports horses the head and neck are raised and lowered with the stride, a movement that assists in balance, energy distribution, and propulsion. The show horse, like the dressage horse, maintains a fixed and flexed head and neck carriage. This further shifts the balance and energy of concussion to the hindlimbs.

Show horses carry more body weight for a fleshier look than the greyhound-like racing counterparts. Riders of show horses, as a rule, also are heavier than racing jockeys.

Longevity of show horses compared with racehorses is related directly to speed of performance, which is dramatically less. Racehorses must change energy distribution at high speed quickly, potentially leading to catastrophic breakdown, but such actions and injuries in show horses are rare. A show horse often can remain competitive into the late teens and early twenties, but chronic wear and tear may result in lameness.

Although show horses do not perform at speed, high head carriage and high leg action and motion are strenuous. Show horses perform numerous gait changes and transitions going both directions of the ring, and for a high-level (stake class) five-gaited class to last from 30 to 40 minutes is not unusual.

Because five-gaited movements and transitions are complex and arduous, compensatory lameness is common. A methodical approach to lameness diagnosis must be used to differentiate primary and compensatory lameness. A superior show horse distinctly separates its different gait movements, raising each carpus above the horizontal, with a high hock action. The horse drives off its hindlimbs with a flexed high head and neck carriage. Responsiveness to the bit, with an alert expression and attitude, and forward placement of the ears are desirable. Just as racehorses are bred for speed, show horses are bred for animated motion.

AMERICAN SADDLEBRED

The American Saddlebred has a long history and aptitude for different gaits and is derived from many different lineages. The breed was developed by free men in a young free country where the best horses could be bred to the best. The American Saddlebred was developed as a horse of usefulness and beauty that could work in the field, pull a buggy or carriage, and have gaits that were smooth for travel under saddle.

The ability of the American Saddlebred to perform lateral gaits (slow gait and rack) came from the Narragansett pacers, which were among the earliest known easy-riding pacers. The Narragansett pacers were derived from French Canadian pacers of Arabian and Andalusian descent that were bred 100 to 200 years before the American Revolutionary War and had a comfortable saddle gait. Early settlers brought these horses, known as saddlers, to Kentucky. During the late 1830s and 1840s, many of these easy-riding saddlers were bred to the Thoroughbred foundation sires, Denmark and Montrose. These crossbreeds were then bred to horses of trotting blood, from which the Standardbred breed was developed. Offspring of these crosses became favorite mounts of cavalry during the

Civil War, because they had an easy gait, versatility, and an ability to withstand the pressures of war. On April 7, 1891, the American Saddlebred Breeders Association was founded in Louisville, Kentucky, and became the first all-American breed registry. In recent years the American Saddlebred has gained popularity in South Africa and has been crossed with European Warmblood and carriage bloodlines (e.g., Dutch Carriage Horse). During the 1980s, the National Show Horse was derived from American Saddlebred and Arabian lineage.

The American Saddlebred ranges in height from 152 to 178 cm (15 to 17.2 hands; average 160 cm [15.3 hands]) and varies in weight from 455 to 545 kg. Colors include chestnut, bay, black, gray, golden (palomino), and spotted (chestnut, black, or bay mixed with white). As described in *Modern Breeds of Livestock*,[1]

> *the American Saddlebred has a strikingly long neck and considerable arch to the neck. The American Saddlebred is refined in appearance; has long, sloping pasterns that give spring to the stride; has a long, level croup; is strong and short-coupled; and has a back with high, well-defined withers above the level of the hips. The American Saddlebred is famous for refinement, smoothness, proportion, and a beautiful and handsome presentation and projects an alert, curious, expressive personality.*

There are shows for American Saddlebreds throughout the United States and South Africa. The World Championship Horse Show is held each year in Louisville, Kentucky. Shows are run under the guidelines of USA Equestrian, which establishes rules, regulations, and drug-testing procedures. A veterinarian must be aware of current drug and medication rules, and failure to do so may result in fines and penalties to the horse, owner, and trainer.

SADDLEBRED GAITS

The five gaits of the American Saddlebred and other gaited horses are as follows[2]:

1. *Walk and flat walk*—The flat walk is a relaxed, elastic, ground-covering, and collected four-beat gait, maintaining proper form and consistency in stride. The gait is required in pleasure classes. The animated walk is a highly collected gait, exhibiting much primp at a slow, regulated speed, with good action and animation. The gait can be a two- or four-beat gait and is performed with great style, elegance, and airiness of motion.
2. *Trot*—The trot is a natural, two-beat diagonal gait. A balanced trot features coordinated motion with straight, true shoulder motion of forelimbs, with flexing hocks carried close together. The gait is executed in a highly collected manner and should display the horse's athletic ability.
3. *Slow gait*—The slow gait was developed from the pace to be a four-beat gait with each of the feet striking the ground separately. In the takeoff the ipsilateral front foot and hind foot start almost together, but the hind foot contacts the ground first. The slow gait is a highly collected gait with most of the propulsion coming from the hindquarters, whereas the forequarters assist in the pull of the final beats. The slow gait is not a medium rack. The slow gait is a restrained gait, executed slowly, but with true and distinct precision, and speed is penalized. The gait is high, lofty, brilliant, and restrained, denoting the style, grace, and polish of the horse.
4. *Rack*—The rack is a four-beat gait in which each foot meets the ground at equal, separate intervals. The gait is smooth and highly animated, performed with great action and speed in a slightly unrestrained manner. Desired speed and collection are determined by the maximum rate at which a horse can rack in form. Racking in form should include the horse remaining with a good set head and should be performed by the horse in an effortless manner from the slow gait, at which point all strides become equally rapid and regular. Any tendency to become trotty, pacey, or hitchy-gaited is penalized.
5. *Canter*—The canter is slow, lofty, and fluid, with a definite three-beat cadence. High action, a good way of going, and proper collection are paramount. The propulsion is in the hindquarters, with the leading forelimb sustaining the concussion of the final third beat. A brief interval occurs when all feet are off the ground. The gait is an ambidextrous gait, executed on the lead that is toward the center of the ring to relieve stress and aid in balance.

TEN MOST COMMON LAMENESS CONDITIONS

The 10 most common lameness conditions in show horses are the following:

1. Distal hock joint pain (tarsitis)
2. Gluteal myositis and back pain
3. Palmar heel pain, including contracted heels, sheared heels, quarter cracks, and navicular syndrome
4. Osteitis of the distal phalanx (pedal osteitis)
5. Osteoarthritis and osteochondrosis of the tarsocrural joint
6. Osteoarthritis and osteochondrosis of the fetlock joint
7. Osteoarthritis and osteochondrosis of the stifle joint
8. Suspensory desmitis
9. Tendonitis and desmitis, including deep digital flexor tendonitis, superficial digital flexor tendonitis, and desmitis of the accessory ligament of the deep digital flexor tendon
10. Splint exostosis

LAMENESS EXAMINATION

The history of lameness and poor performance must be discussed with the trainer and rider to seek their perception. This should include noting any problems with the bridle and the way a horse pulls on the bit and bridle. Show horses with hindlimb lameness often fight the bit and try to lower the head, an observation known as diving in the bit. Horses that become one-sided in the bit may have contralateral hindlimb or ipsilateral forelimb lameness. Faulty bit and bridle placement may cause gait abnormalities particularly of the hindlimbs. Keeping the head up and fixed in position in a horse that dives in the bit is difficult. A horse with forelimb lameness is more likely to raise up out of the bridle. A horse cannot produce a gait properly or do proper gait transitions when being pushed into an uncomfortable bridle or if the rider is using the bit improperly. It is important to determine if the rider is using the bit to balance the horse or themselves. A rider using the bit poorly can induce a gait abnormality. Bit and bridle responsiveness is often a wild card that must be played during examination of the show horse for gait abnormalities and lameness.

Problems with a particular gait may indicate the source of lameness. Back pain is seen in horses that have difficulty in the canter, a condition known as *being broke in the middle*. This occurs with asynchronous movement of the forelimbs and hindlimbs. Broke in the middle also can be caused by stifle pain that causes a reduction in the cranial phase of the stride. Most commonly, however, broke in the middle is caused by stringhalt. Stringhalt prevents the limb from moving forward

at a time when the forelimbs are required to go faster, causing a mismatch in synchronization between the forelimbs and hindlimbs. Distal hock joint pain and back pain can cause a hitching motion of the hindlimbs, jerking the lame limb caudally and leaving the hocks behind the motion.

Examination first begins in the stall before the horse is worked. Careful palpation with emphasis on the tendons, ligaments, joint capsules, and bulbs of the heels should be performed. Palpation of the back and gluteal muscles before working is important, because the horse can warm out of soreness in these areas. Digital pulse amplitudes should be assessed.

The horse should be evaluated during movement under tack. Harness horses, road horses, and ponies should be examined while working with and without the over-check (checked up and without the check). Five- and three-gaited horses should be examined performing each gait going in both directions. Often horses are only lame while going in one direction or only in the turns. Horses with lameness from the hock distally are often worse with the lame limb on the inside, whereas those with pain located more proximally are lame with the lame limb on the outside of the turn. Forelimb lameness is usually worse with the affected limb on the inside.

In most show horses flexion tests can be performed with a rider or in harness. The horse's temperament may make this difficult, but I find that the horse being ridden or jogged in the cart after flexion is helpful.

DIAGNOSTIC ANALGESIA

Diagnostic analgesia in show horses is similar to that described for other sport horses. I start distally and work proximally.

IMAGING CONSIDERATIONS

Conventional and computed radiography are used extensively. Scintigraphy is most useful in horses with complex lameness, because primary and compensatory issues are difficult to differentiate. The solar scintigraphic view is mandatory to evaluate horses with palmar heel pain in which radiographs are negative, because modeling of the navicular bone and distal phalanx should be assessed carefully. Areas of increased radiopharmaceutical uptake in the distal phalanx may indicate excessive pressure that can be relieved by corrective shoeing. Thermography is of value in diagnosing tendonitis, sole pressure, muscle inflammation, and suspensory desmitis. Ultrasonographic examination is useful to confirm and assess damaged soft tissues and healing. Diagnostic arthroscopy is used occasionally.

SHOEING GAITED HORSES

Shoeing gaited horses to assist with motion and gait transitions is an art form in itself. In general a long toe and high heel in front help to delay breakover and cause high knee action. In the past, weighted shoes and lead weights screwed into the bottom of shoe pads were used to induce animation. In recent years a transition to lighter is better has occurred, and now the focus is on fitness and training techniques to teach the horse to elevate its limbs to achieve animation. Shoeing depends to a great extent on the horse's ability, conformation, and desired gait performance (e.g., five-gaited, three-gaited, or harness). For example, a three-gaited horse may have a long foot with long toe and heel length to delay breakover in the forefeet and hindfeet. This gives the extreme highly animated knee and hock action expected from a top three-gaited horse. In contrast, in a five-gaited horse a lower hind heel angle is used to assist with a longer hindlimb stride needed for the slow gait and rack. Compared with three-gaited horses, lighter and shorter front feet are maintained in five-gaited horses to promote speed at the trot and rack.

Shoeing with high heels and long toes, with or without pads, predisposes the American Saddlebred to contracted heels, sheared heels, and quarter cracks. The recent use of cushion polymer compounds to maintain frog pressure is helpful, because frog pressure is lost with high heels and pads. Cushioned polymers placed in the collateral sulci (grooves) of the heel and over an atrophied frog have dramatically reduced hoof problems. Medial to lateral hoof balance is paramount.

SPECIFIC LAMENESS CONDITIONS

Distal Hock Joint Pain and Distal Tarsitis

There are two types of show horses, those that have distal hock joint pain and those that are getting it. Show horses with distal hock joint pain typically jerk the hindlimb caudally (hitching) and leave the hocks behind in motion. Many horses stab the toe, rather than landing normally with the heel first.

Distal hock joint pain without radiographic abnormality is common in 2- and early 3-year-olds just starting to rack. As training and showing proceed, radiographic evidence of osteoarthritis, such as loss of joint space, can become apparent as early as 5 years of age and may be severe by 12 years of age.

The tarsometatarsal joint is by far the most valuable point of intra-articular injection in show horses. Occasionally, injection of the centrodistal joint also is required. I prefer to use hyaluronan and a low-dose corticosteroid combination and recommend oral supplementation with glucosamine and chondroitin sulfate–containing products. Magnetic therapy appears beneficial. Horses with lameness that does not improve are considered candidates for shock wave therapy. Arthrodesis of the distal hock joints, using a drilling technique combined with laser ablation, is used in horses with severe pain. Although cunean tenectomy was once widely used, effects of the procedure are short-lived, and I do not recommend it. If the horse is hitching through the turns of a show ring, a 45° flat outside trailer is used on the hind shoe for support. The shoe is set back, and the toe is squared or rolled to improve breakover.

Gluteal Myositis and Back Pain

Show horses are prone to gluteal myositis and back pain, which are often secondary to primary distal hock joint pain. A willing horse hyperflexes its back to compensate for lower hindlimb pain. The middle gluteal muscle passes over the greater trochanter of the femur and the trochanteric bursa. Gluteal myositis and tendonitis are common sequelae to distal limb pain, but they can cause primary lameness. Trochanteric bursitis (whorl bone disease) can accompany gluteal myositis.

In horses with subacute gluteal myositis, upper limb flexion may stretch the gluteal muscles and produce a transient improvement in gait. Transient improvement may be seen by gently massaging the greater trochanter and gluteal muscles. However, in horses with chronic myositis with involvement of the trochanteric bursa, deep massage and pressure may make lameness worse. Horses with subacute primary gluteal myositis commonly have a tight-rope trot (plaiting). Plaiting is associated with distraction and rotation of the hindlimb, with subsequent lateral movement of the hip, motion that may cause gluteal muscle strain. Horses that plait usually have base-narrow conformation, and corrective trimming in the form of spreading the stance (lowering the outside hoof walls) may help.

Back pain is identified easily using digital pressure along the thoracolumbar region, abaxial to the spinous processes. Many horses are so painful that one can almost put them on the ground with digital pressure. Diagnostic analgesia usually

is not necessary. However, small amounts of local anesthetic solution injected at numerous sites along the affected muscles may give enough relief to allow evaluation for lameness that has been hidden by back or gluteal pain.

Management of horses with gluteal myositis and back pain requires a multifaceted approach, including local injection of Sarapin and corticosteroids. In most horses non-steroidal anti-inflammatory drugs (NSAIDs), methocarbamol, electrical stimulation, magnetic therapy, therapeutic ultrasound, and anti-concussion saddle pads are used in various combinations. Exercise regimens using stretching and flexing during the warm-up period are also beneficial. Acupuncture and chiropractic modalities often are used and can be of benefit if performed by skilled practitioners. Shock wave therapy may prove to be beneficial. Diagnosis and treatment of primary lameness problems, if present, are of utmost importance.

Palmar Heel Pain

Because show horses are shod intentionally with a high heel and long toe to produce high motion, they are prone to contracted and sheared heels. Full or wedge pads are often applied to achieve the desired motion, and the lack of frog pressure can cause atrophy of the soft tissues of the heel. Without proper frog support, the bulbs of the heel contract and sheared heels often develop. As the heels contract or sheared heels develop, more stress is applied to the quarters, predisposing the hooves to quarter cracks. Differentiation of causes of palmar heel pain is critical but difficult, because palmar digital analgesia affects these conditions and navicular syndrome similarly.

Recently more concern has arisen about maintaining frog pressure. Soft, acrylic polymers are now used for frog support in horses with frog atrophy. Turnout for several months, during which the horse is barefoot, may help. Expansion springs can be used to assist in re-establishing proper heel conformation. Severe quarter cracks are repaired using the techniques of lacing, applying screw compression plates, or nailing. Floating the heel and quarter located under the crack is important to reduce weight bearing, allowing showing to continue.

Navicular disease is not uncommon, but in horses without abnormal radiological findings the diagnosis should be confirmed using scintigraphy. The aforementioned conditions of the hoof capsule and supporting soft tissues are much more common.

Treatment of soft tissue causes of palmar heel pain involves maintaining comfort while proper anatomy is re-established. Corrective shoeing, NSAIDs, and long-term foot blocks often are used. If navicular disease is confirmed, intra-articular treatment of the distal interphalangeal joint using hyaluronan and corticosteroids is recommended. Drugs aimed at improving peripheral perfusion or decreasing intra-osseous pressure such as isoxsuprine may be useful with NSAIDs, long-term foot blocks, and corrective shoeing. Shock wave therapy appears promising in managing palmar heel pain.

Osteitis of the Distal Phalanx

In horses with high stepping gaits the distal phalanx is prone to injury. Trauma to the solar margin such as bruising and fracture occurs. A careful evaluation for improper sole pressure or medial to lateral hoof imbalance should be performed. Well-exposed and well-positioned radiographs are essential. Digital venography may reveal compression of blood vessels within the hoof capsule and is useful to pinpoint a location of trauma.

Fractures of the distal phalanx are rare. Palmar scintigraphic views are useful to diagnose fractures and other areas of distal phalanx trauma and can help to formulate a corrective shoeing plan. Thermography may be useful for diagnosis of distal phalanx trauma.

For horses without distal phalanx fracture, management includes corrective balancing and shoeing to relieve improper sole pressure and to provide support, NSAIDs, and isoxsuprine. If effusion of the distal interphalangeal joint is present, intra-articular treatment with hyaluronan and corticosteroids may help.

Osteoarthritis and Osteochondrosis of the Tarsocrural Joint

Lameness of the tarsocrural joint is not as common as distal hock joint pain. Osteochondrosis of the tarsocrural joint in show horses is similar to that described for other sport horses. The most common location is the distal intermediate ridge of the tibia. Although show horses with osteochondrosis lesions may compete successfully without surgical intervention, effusion and capsulitis are indications that surgery should be performed. Prognosis after arthroscopic surgery is favorable. However, prognosis for for show horses with trochlear ridge lesions is guarded. Osteophytes and small fragments of the distal, medial trochlear ridge are not a major source of lameness.

The diagnosis of osteoarthritis of the tarsocrural joint is derived from the results of physical examination, flexion tests, diagnostic analgesia, and radiography. Horses with early osteoarthritis of the tarsocrural joint are managed with intra-articular injections of hyaluronan, with or without corticosteroids. Oral supplementation with glucosamine and chondroitin sulfate-containing compounds appears beneficial. Intramuscular and intravenous administration of polysulfated glycosaminoglycans and hyaluronan, respectively, are helpful. NSAIDs may be necessary. Horses with chronically boggy hocks usually respond well to routine intra-articular medications, but if they are refractory, I add 0.5 ml atropine sulfate.

Osteoarthritis and Osteochondrosis of the Fetlock Joint

Conditions of the fetlock joint in show horses are essentially the same as seen in other sport horses. The most common conditions are osteochondral fragments of the proximal dorsal aspect of the proximal phalanx and sesamoiditis. Plantar process osteochondritic fragments are uncommon but are recognized. During prepurchase examinations, I obtain lateromedial radiographic views of each metatarsophalangeal joint specifically to evaluate for plantar process fragments. Fractures of the proximal sesamoid bones occur infrequently. Osseous cyst-like lesions of the distal aspect of the third metacarpal bone and osteochondritis dissecans lesions of the sagittal ridge of the third metacarpal bone occur infrequently and are less devastating than in racing breeds. Mineralized proliferative synovitis lesions are often confused with osteochondral fragments, but show horses with this condition have a favorable prognosis. Primary osteoarthritis of the fetlock joint is seen commonly in show horses that wing the lower limb while moving, but radiographs are often negative.

Diagnosis of lameness of the fetlock joint is routine. Nuclear scintigraphy is most useful in diagnosing sesamoiditis.

Arthroscopic removal of osteochondral fragments of the proximal phalanx is not always necessary, because many show horses compete well and require little maintenance therapy when fragments involve the front fetlock joints. When fragments involve the hind fetlock joints, arthroscopic removal is recommended, because these horses have gait abnormalities characterized by a skipping motion that are accentuated at the rack and slow gait.

Horses with sesamoiditis respond well to corrective shoeing (lowering the heel), NSAIDs, isoxsuprine, and injection of hyaluronan and corticosteroids into the fetlock joint. Shock wave therapy appears to be effective in managing horses with chronic sesamoiditis and collateral ligament damage.

Osteoarthritis and Osteochondrosis of the Stifle Joint

Osteoarthritis of the stifle joints is a common lameness in young horses, particularly in horses being pushed to perform

at a young age. Normal weight distribution favoring the hindlimbs, coupled with learning the slow gait and rack at a young age, predispose the horses to stifle pain. Horses with stifle lameness usually have a shortened cranial phase of the stride and are said to be humping up in the hip at the beginning of the caudal phase of the stride. Lameness often is pronounced with the limb on the outside of the ring and is worse in the turns. Diagnosis is made using gait analysis, diagnostic analgesia, radiography and, if necessary, nuclear scintigraphy. Subchondral bone cysts of the medial femoral condyles and osteochondritis dissecans of the trochlear ridges of the femur are commonly diagnosed in young show horses with stifle effusion. Arthroscopic surgery, debridement, and fragment removal are recommended in those horses with effusion and lameness. Prognosis for a five-gaited horse with osteochondritis dissecans of the stifle is guarded, even with surgery. Many of these horses can compete successfully in other divisions such as harness, pleasure driving, and equitation.

Distal patellar fragmentation and cartilage damage is seen in older horses, and horses respond well to intra-articular injections of hyaluronan and corticosteroids. Arthroscopic surgery is recommended in horses refractory to this therapy.

Lateral luxation of the patella occasionally is diagnosed in foals and has a guarded prognosis. Upward fixation of the patella is seen in young horses with under-developed quadriceps muscles. Most improve with training in a jog cart to build up the hindlimb musculature in lieu of intense riding and training. Trainers must be advised to be patient. Medial patellar desmotomy should be used as a last resort and should be done only if radiographs of the stifle are negative.

Horses with early osteoarthritis and negative radiographs are managed by decreasing training intensity and implementing a jogging program to develop the hindlimb musculature. A weighted drag behind the cart is added later, before the normal riding program resumes. Internal blisters are used commonly. Intra-articular injection of hyaluronan and corticosteroids alleviates clinical signs, but without modification of exercise the results are short-lived.

Horses with chronic osteoarthritis are maintained using intra-articular injections, oral supplements, intramuscularly and intravenously administered polysulfated glycosaminoglycans, and NSAIDs.

Suspensory Desmitis

Show horses with long pasterns and high heels are prone to suspensory desmitis because the fetlock drops excessively and stretches the suspensory ligament. Show horses trained in deep footing (sand or mud) are at increased risk for suspensory desmitis. Avulsion fracture of the third metacarpal bone may occur in association with suspensory desmitis. Suspensory desmitis is more common in the forelimb than in the hindlimb. Treatment of show horses with suspensory desmitis involves rest, NSAIDs, periligamentous injection of corticosteroids, leg sweating, magnetic therapy, and support wraps. Rest includes hand walking, because horses with suspensory desmitis appear to respond better to limited, controlled exercise than to stall rest alone. The heels should be lowered and a palmar or plantar extension applied.

Shock wave therapy has been extremely beneficial in show horses with suspensory desmitis, particularly those with proximal suspensory desmitis. I used to recommend bone marrow injection in horses with refractory suspensory desmitis, but the advent of shock wave therapy has decreased the need for surgery dramatically.

Tendonitis

Performing in deep, muddy, outdoor show rings and a long-toe, high-heel hoof conformation predispose horses to tendonitis of the superficial digital flexor and deep digital flexor tendons and desmitis of the accessory ligament of the deep digital flexor tendon. As a rule the tendonitis is not nearly as devastating in show horses as in racehorses.

Horses with tendonitis respond well to sweats, peritendonous injection of corticosteroids, NSAIDs, magnetic therapy, and shock wave therapy. Rarely, show horses require tendon splitting and desmotomy of the accessory ligament of the deep digital flexor tendon or the accessory ligament of the superficial digital flexor tendon. Serial ultrasonographic examinations are important to monitor healing.

Splint Exostoses

Splint exostoses are caused by lunging young horses in tight circles too fast and too long. Direct trauma from interference can cause splint exostosis or fracture. Once splints become inactive, they rarely cause lameness, unless the mineralization impinges on the suspensory ligament or if the exostosis is so large it repeatedly becomes traumatized. Diagnosis is made by palpation and radiographic assessment for fracture. Treatment consists of leg sweats, subcutaneous injections of corticosteroids over the exostosis, and shock wave therapy in horses refractory to sweating and injections. Surgical removal of distal splint bone fracture fragments, large exostoses, or nonunions that impinge on the suspensory ligament should be performed.

OTHER LAMENESS CONDITIONS

Stringhalt

Stringhalt is common in the show horse and needs to be differentiated from other hindlimb lameness conditions. Local anesthetic solution (5 ml at each of three sites) is injected into the lateral digital extensor muscle, and the horse is observed while ridden 10 minutes later. If gait is improved, I recommend lateral digital myotenectomy. Previous trauma to the lateral or common digital extensor tendons can predispose horses to stringhalt (see Chapter 44).

Semimembranosus/Semitendinosus Myositis

Myositis of semimembranosus or semitendinosus sometimes can mimic stringhalt and cause bizarre gait abnormalities. Horses and ponies (particularly road ponies) show restriction of the cranial phase of the stride. The hindlimb appears to hang up in a flexed position or the horse is short-strided. Typically a trainer says the horse cannot get underneath himself. This occurs particularly in gaited horses that cannot perform the slow gait or rack. This condition may be an early form of fibrotic myopathy.

Diagnosis involves injecting 5 ml of local anesthetic solution in three to four sites each in the semimembranosus and semitendinosus muscles. The horse is evaluated ridden in 10 minutes, and often the change is dramatic.

Short-lived benefit is seen by injecting the involved muscles with Sarapin and a corticosteroid and using electrotherapy. The best solution appears to be tenotomy of the medial branch of the semitendinosus muscle, the same procedure described for surgical management of horses with fibrotic myopathy.

Tibial Stress Fractures

Young show horses, particularly young, talented five-gaited prospects, can become suddenly difficult to gait. Typically the trainer says, "The horse was one of the best young prospects I ever had and then suddenly I lost him," or "Everything came undone." Tibial stress fractures are the show horse counterpart to bucked shins in the Thoroughbred. Diagnosis is difficult and usually involves ruling out everything else first and proceeding to nuclear scintigraphy, the most consistent and best method of diagnosis. Horses are given 60 days of jogging and are re-assessed.

Hindlimb Extensor Tenosynovitis

The common digital extensor tendon sheath at the level of the hock can become inflamed and distended, a condition that is particularly prevalent in talented gaited horses. There is usually an indentation of the distended sheath just below the hock, caused by constriction by the retinaculum. Although lameness is unusual, severe distention of the sheath may cause a stiff gait, because the horse cannot flex the hock normally. Tenosynovitis may be confused with bog spavin. If left untreated, synovium becomes hypertrophic, and movement of synovium under constricting retinaculum causes a hitch in hindlimb gait. Treatment consists of draining synovial fluid and injecting a combination of hyaluronan, corticosteroids, and 0.5 ml of atropine sulfate. Massaging with dimethylsulfoxide and a corticosteroid is also beneficial.

Cervical Myositis

The degree of neck flexion required in show horses often causes pain, particularly in young horses. Older horses may develop osteoarthritis of the facet joints. Horses with cervical myositis and pain often are observed to be fighting the bit. Diagnosis is made by palpation. Injection of Sarapin and corticosteroids in the affected muscles, methocarbamol, electrical stimulation, and NSAIDs are used. Acupuncture and chiropractic procedures also may be beneficial. Shock wave therapy may be beneficial in horses with osteoarthritis of the facet joints.

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CHAPTER • 124

Lameness in the Arabian and Half-Arabian Show Horse

Jeffrey A. Williams and Bradley S. Root

HISTORY OF THE ARABIAN

The Arabian is one of the oldest breeds in the world. The horse originated in the deserts of the Middle East and was used by the Bedouins for transportation and in battle. The Arabian breed was noted for its speed and endurance. Three Arabian stallions (the Godolphin Barb, Byerley Turk, and Darley Arabian) imported to Europe during the late 1600s and early 1700s became the foundation of a new breed of horse, the Thoroughbred. Today, 93% of all modern Thoroughbreds can be traced to these three sires. In the 1800s many royal families of Europe established Arabian stud farms. Two of the most notable were the Polish National Arabian Stud in Poland and the Crabbet Arabian Stud in England. The Arabian is thought to have an influence on many of the light horse breeds that have developed throughout history. A typical Arabian ranges from 14.1 to 15.1 hands in height. The American Horse Show Association breed standards describe the Arabian as having a small, slightly dished face with large eyes set well apart, small ears, deep and wide jowls, a small muzzle, and large nostrils. The horse should have a long, arched neck; a long, sloping shoulder; well-sprung ribs; a short back with a relatively horizontal croup; and natural, high tail carriage. The limbs should have large, well-defined joints, short cannon bones, sloping pasterns of good length, and round feet of proportionate size.[1]

The Half-Arabian studbook originated with the U.S. Army Remount Service after World War II and was acquired by the International Arabian Horse Association in 1951. Half-Arabians must have a registered purebred Arabian sire or dam. The Anglo-Arabian is a cross between an Arabian and a Thoroughbred, whereas the more recently developed National Show Horse is a cross between an Arabian and a Saddlebred. Many Half-Arabians are double registered.

HISTORY OF THE SPORTING EVENT

The International Arabian Horse Association was created in 1950 to join the local and regional clubs across America into one united association. The International Arabian Horse Association promotes and coordinates all Arabian and Half-Arabian horse show activities and develops horse show rules. The International Arabian Horse Association also maintains the Half-Arabian and Anglo-Arabian registries, whereas the Arabian Horse Registry of America maintains the registry and pedigree records for purebred Arabian horses in the United States and Mexico.

The first U.S. National Arabian and Half-Arabian Show was held in 1966. The U.S. National Show is held in October and alternates yearly between Louisville, Kentucky, and Albuquerque, New Mexico. A separate Youth National show for riders under 18 years old is held in July of each year in Albuquerque. The Canadian National Horse Show is held in August in Regina, Saskatchewan. The United States and Canada are separated into 18 regions, each of which holds an annual show. Horses can qualify for the national show by placing in the top five at a regional show or by winning a championship or reserve championship at a Class A show. Some of the major shows other than the regionals and nationals are the Scottsdale Show (Arizona), the Buckeye Show (Ohio), the Pacific Slopes Show (California), the East Coast Championship (Pennsylvania), and the Pro-Am Challenge (Texas). Major international shows are held in England, France, South America, and Australia.

Performance classes for Arabian and Half-Arabian horses cover a broad spectrum and are listed in Box 124-1. Each of these classes is held separately for Arabians and Half-Arabians. They may be divided further into sections for junior owner, adult amateur owner, amateur owner, junior exhibitor, and

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Box • **124-1**

Performance Classes for Arabian and Half-Arabian Horses

Park Horse
English Pleasure
Country English Pleasure
Hunter Pleasure
English Show Hack
Hunter
Jumper
Dressage
Formal Driving
Pleasure Driving
Country Pleasure Driving
Roadster
Combination (Driving and Riding)
Mounted Nature Costume
Ladies Side Saddle
Western Pleasure
Reining
Working Cow Horse
Trail
Cutting
Equitation (Hunter Seat, Saddle Seat, and Stock Seat): youth classes only
Western Horsemanship (youth only)

amateur. The Park horse has a strong animated trot, with the forearm horizontal and the leg extending fully forward. The hock has a well-raised driving action. The walk and canter are animated and collected. The English Pleasure horse is shown at a walk, trot, strong trot (faster and more animated than the normal trot), canter, and hand gallop. Its gaits are less animated than the Park horse although the forearm, at the trot, is horizontal. The same gaits are used in the Country English Pleasure class, but horses have lower leg action, and high action is penalized. The Country English Pleasure horse must also halt, stand quietly, back, and walk off on a loose rein. With all pleasure classes the horse must give the appearance of being a pleasure to ride. Park and English Pleasure horses are shown with the head carried high and considerable flexion at the poll. Saddle seat attire is required.

In the English Show Hack class the horse must perform each gait (walk, trot, and canter) in a normal, collected, and extended manner. A transition between gaits should be noticeable and high knee action is not expected. Horses in the Hunter Pleasure division are shown under saddle at the walk, trot, canter, and hand gallop. The neck should be carried lower, the head should be carried with less bend at the poll and the horse should be in a generally longer frame than the English Pleasure or Show Hack horse. Working Hunters are shown over a course of fences set at levels of 0.6 to 1 m and are judged on performance, manners, and soundness. Jumpers are shown over courses of jumps that vary in height from 0.9 to 1.05 m. The maximum width (spread) is 1.5 m.

Horses in driving classes are shown pulling a four-wheeled (Formal and Pleasure) or two-wheeled (Pleasure and Country Pleasure) vehicle. The gaits judged in the Formal, Pleasure, and Country Pleasure driving classes correspond to the Park, English Pleasure, and Country English Pleasure classes under saddle. The Roadster is a driving class, which focuses on the trot at three different speeds.

The Western Pleasure horse is shown at the walk, jog (trot), lope (slow canter), and hand gallop. Ideally, contact with the reins is light, the head is carried low (approximately at the level of the withers), and the jog and lope are slow, easy gaits. The Working Western horse classes include reining, working cow horse, trail, cutting, and Western riding.

TRAINING: IMPACT OF INDUSTRY

The Arabian and Half–Arabian are versatile breeds, as shown by the many sports in which they compete. When appropriate, differences between the two breeds are specified. These include halter, endurance (see Chapter 119), pleasure, jumping, dressage, reining, cutting, and racing (see Chapter 112). Young performance horses are not shown under saddle until they are 3 years old. They then compete in futurity classes for horses 3 years of age or junior horse classes for horses 5 years of age or younger. Because these horses do not compete in performance classes until 3 years of age, this allows more time for adequate skeletal development compared with racehorses and Quarter Horses that start training before 2 years of age. This and the lack of speed work help minimize injuries such as fractures, early osteoarthritis, and severe soft tissue injuries, which are common in the racehorse. The reason that training of Arabian show horses is started later than some other breeds may be partly because of smaller size and late maturation, but it is also related to the fact that no performance classes are available for 2-year-olds, and therefore no economic incentives exist to start intensive training early.

Early training and conditioning typically involve a substantial amount of work in a round pen or by lunging. Excessive training in small circles causes increased torque on the distal interphalangeal joint, the navicular bone, and the distal phalanx. Seeing young horses trained in this manner and developing bilateral forelimb lameness localized to the distal interphalangeal joint is not uncommon. This type of training also causes increased stress on the other joints and support structures of the forelimbs and hindlimbs. These problems tend to be exacerbated by uneven and hard footing.

Rules govern the shoeing of the Arabian and Half-Arabian show horses. A maximum shoe weight of 14 oz (392 g) excluding pads and nails is allowed. The maximum toe length including the shoe and any pads is 4½ inches (11.43 cm). Pads are allowed, as are normal packing materials such as tar, oakum, and silicone. No weights are allowed under the pad. Foot length, shoe weight and shape, and pad usage are individualized for each horse to optimize the height and arc of flight of the forelimbs and hindlimbs. In English Pleasure horses, a common shoe is the toe-weighted shoe constructed by forging more steel in the toe of the shoe. The long foot and weighted shoes are used to enhance forelimb motion. Unfortunately this can contribute to strain on the suspensory ligament (SL) and joints of the lower limb.

In all performance divisions horses are shown in a collected frame. In each division the type of work performed, the body position required, and the conformational defects of the individual horse contribute to the common lameness conditions. Differences are apparent in gaits, degrees of collection, and head and neck position in the various divisions. In the English Pleasure division, for example, the degree of collection, neck elevation, and poll flexion required shift weight to the hindlimbs and increases the work of the back and abdominal muscles. These positional factors can cause lameness of the hindlimbs (especially involving the stifle and SL) and back pain. In the Western Pleasure and Reining divisions, similar problems are seen because of the amount of collection required. These horses also incur a variety of lameness conditions because they commonly are worked for longer periods of

time than English Pleasure horses. In the Western Pleasure and Reining classes, because any departure from a quiet, steady position is penalized, fatigue is part of the class preparation.

CONFORMATION AND LAMENESS

Mild to moderate carpus valgus and toed-out conformation commonly are seen and do not appear to have a major impact on soundness (Fig. 124-1). One reason is that the carpus is not a common location for lameness. If these conformational faults are severe, horses are at risk of suspensory desmitis. Horses with long sloping pasterns, back-at-the-knee conformation, or offset knees are also predisposed to suspensory desmitis. These conformational faults are more common in the Half-Arabian and National Show Horse than in purebred Arabians and are more common in certain pedigrees.

Horses with low, underrun heels certainly are prone to lameness from palmar heel pain. This fault can be difficult to correct, even with careful attention to shoeing and trimming. A small, upright, contracted foot (club foot) can be a source of lameness and appears to be increasing in incidence. Inflammation of the soft tissues such as the SL, accessory ligament of the deep digital flexor tendon, and distal sesamoidean ligaments tends to be more common in horses with club foot conformation.

Fig. 124-1 A 4-year-old Half-Arabian with toed-out, carpus valgus, back-at-the-knee conformation. These are common conformation faults in the Arabian and Half-Arabian breeds and may predispose the horses to suspensory desmitis.

A long, weak (sway) back and a short croup may predispose horses to soreness in the thoracolumbar, sacroiliac, and gluteal areas. Because problems in these areas are a common cause of poor performance, this type of conformation is a serious fault. Horses with cow-hocked conformation are the rule rather than the exception, but this conformation seems to have little effect on soundness.

TEN MOST COMMON LAMENESS CONDITIONS

The 10 most common lameness conditions of the Arabian and Half-Arabian show horse are as follows:

1. Bruised and inflamed feet
2. Osteoarthritis of the distal interphalangeal joint and palmar heel pain
3. Suspensory desmitis
4. Osteoarthritis of the stifle joints
5. Thoracolumbar, sacroiliac, and gluteal pain
6. Osteoarthritis of the metacarpophalangeal and metatarsophalangeal joints
7. Distal hock joint pain
8. Splint bone injuries
9. Osteoarthritis of the proximal interphalangeal joint
10. Desmitis of the accessory ligament of the deep digital flexor tendon

LAMENESS EXAMINATION

History

When describing lameness, trainers often comment that problems occur or are more noticeable during the first direction or the second direction. This refers to the directional order in which rail classes are run. In the first direction horses enter the ring and travel counterclockwise, and in the second direction horses travel clockwise. Important questions regarding history include the division in which the horse competes, onset, degree and progression of the lameness, previous or current treatment and response to therapy. Additional information that can be helpful includes knowing which direction is harder for the horse at the trot (jog) and canter (lope), whether the horse pulls unevenly on the reins, whether the horse tracks straight in each direction, whether the horse falls out of leads behind (in the hindlimbs) in corners, and whether the rider rides the correct or incorrect diagonal in each direction.

The age of the horse is important, because osteochondrosis is more likely to affect young horses recently started into training than older animals, but in older horses osteoarthritis is common. It is important to find out when the horse was last shod, and if any recent shoeing changes have been made. Altering medial to lateral hoof balance or hoof angle may increase pressure in certain areas and lead to bruising of the heel or sole. Increasing the hoof angle by raising the heel may increase load on the SL, which may lead to suspensory desmitis. The type and condition of the footing the horse has been working on is important to consider. Often footing at shows is less than ideal and in many cases is too hard, leading to the development of bruised feet. Conversely, if footing is too deep, it may lead to tendon and ligament injuries.

Visual Examination

Stepping back and visually examining the horse for overall symmetry of the limbs and upper body is helpful. Asymmetry of upper limb muscle groups may be a sign of atrophy from denervation, chronic lameness, or neurological disease. Asymmetry in the height and position of the point of shoulder, the tubera sacrale, the tubera coxae, or the tubera ischii

can indicate subluxations or fractures. The size, shape, and symmetry of the feet are important to note. Abnormalities such as club feet, underrun or sheared heels, growth rings, dished dorsal hoof walls, and contracted heels are some of the more common problems. Joint swelling, soft tissue swellings, poor conformation, scars, and abnormal structures such as splints are recognized quickly.

Palpation

Thorough palpation of the lower limbs is performed with the horse in weight-bearing and non–weight-bearing positions. Degree of joint filling; abnormal contours of bones, tendons, and ligaments; and intensity of the digital pulses are best evaluated while the horse is bearing weight. With the limb elevated, painful response to palpation of tendons, ligaments (origins and insertions), and splints; pain on joint flexion; and range of motion of joints are assessed. In the Arabian and Half-Arabian show horses, in contrast to the racehorse, moderate filling of the metacarpophalangeal/metatarsophalangeal joint capsules is common and should not be over-interpreted. Effusion of the distal interphalangeal and stifle joints (especially the medial femorotibial joint) tends to be clinically relevant. To evaluate filling in the medial femorotibial joint, it is helpful to have the horse bearing weight with the limb being palpated slightly ahead of the opposite limb and perpendicular to the ground.

Response of the Arabian and Half-Arabian to palpation of the SL varies greatly, depending on the horse. Differences between limbs should be considered important, and change in the response of an individual over time is noteworthy. Many horses have a painful response to palpation of the SL but have primary lameness localized to the foot. Possibly the SL is painful because of a compensatory gait caused by a primary foot problem. In contrast, absence of inducible pain, especially in the proximal suspensory region does not rule out this area as a source of pain causing lameness.

Careful palpation of the thoracolumbar spine and upper body is useful in diagnosing the reason for poor performance and lameness. Asymmetry, abnormal contours, and painful response to palpation are important to assess. Particular attention should be given to the thoracolumbar musculature, the spinous process, and the sacral tuberosities, because these areas commonly are involved in horses that perform poorly.

Hoof Tester Examination

Many Arabian and Half-Arabian show horses wear full pads in front during training and showing. Although sometimes inconvenient, especially at a competition, removing the shoe and pad for a complete hoof tester examination is helpful if the veterinarian suspects a foot problem. Some indication of painful areas may be obtained with the shoe and pad on, but many areas can be missed. Bruised heels and soles are common, especially at shows where footing may be too hard and horses are being worked longer than normal. Bruised heels and soles are two of the most common sources of lameness. Improvement in lameness can be dramatic if areas of bruising can be trimmed to reduce pressure or if the shoe is modified to eliminate weight bearing on a bruised area. Many horses are painful when the hoof testers are applied across both heels, but show no pain on the individual heel, soles, or bar of the hoof. These horses are just as painful with the shoe and pad on, and therefore this situation may relate to structures deeper in the heel of the foot, rather than simply bruising of the sole.

Medial to lateral hoof imbalances may contribute to lameness and should be addressed whenever lower limb lameness exists and when sore feet have been identified with the hoof testers. In general, pain is associated with the high side of the foot or that area making ground contact first.

Pain in the toe region occurs less commonly than in the heel, especially for horses in the English and Park divisions, and likely is related to the way the foot is trimmed. These horses usually have a long toe and thick sole that may protect sensitive structures from bruising and exaggerate heel first landing in the forelimbs. When pain over the middle of the frog is detected, bruising, palmar heel pain, or navicular-related pain should be considered. Diagnostic analgesia should be performed to confirm the relevance of hoof tester examination, because false-positive reactions occur.

Flexion Tests

Flexion tests are important, but responses should be interpreted carefully. A painful response to flexion of joints and the degree of lameness should be assessed. Many false-positive lower limb flexion tests occur in Arabian and Half-Arabian show horses. Many horses without lameness show pain to static flexion and a positive response when trotted. In a lame horse a lower limb flexion test is not specific. For example, in lame horses positive to a lower limb flexion test lameness may be localized anywhere from the foot to the distal metacarpal region. Carpal flexion usually is performed only in the static situation because we do not feel that substantial additional information is gained by trotting the horse. Horses that resent carpal flexion may have proximal suspensory desmitis, superficial digital flexor tendonitis, or carpal tenosynovitis, because bony injury of the carpus itself is unusual in Arabian and Half-Arabian show horses.

Initially the entire hindlimb is held in flexion and then individual lower limb flexion and upper limb flexion tests are performed. We attempt to flex the stifle independently of the tarsus by holding the distal tibia upward and behind the horse for 60 seconds. These flexion tests are not specific, but they may increase the index of suspicion in a certain area. For example, horses with hindlimb proximal suspensory desmitis or distal tarsitis respond positively to upper limb flexion and must be differentiated based on the results of other tests. Mildly positive hindlimb flexion tests are seen in sound horses that are actively training and showing. These mildly positive flexion tests may be related to subtle lameness or could be a normal response. For horses that have been competing successfully to have a moderate to severe positive response to a lower limb flexion test after a lengthy show is not unusual. This fact needs to be taken into account when prepurchase examinations are performed directly after horses have competed.

Examination on the Lunge Line and Under Saddle

If possible, examining the horse on the lunge line (or long line) and under saddle is helpful. Multiple-limb lameness, especially contralateral forelimb and hindlimb lameness and bilateral front foot lameness, is common and is important to keep in mind. The possibility of subtle neurological deficits should be considered during all phases of the lameness examination. In some horses lameness may not be apparent unless the horse is under saddle and working near the higher end of its performance capabilities. This type of lameness is more difficult to diagnose, and localization frequently requires diagnostic analgesia. Subtle lameness can be masked by a rider restricting free head movement or by controlling body position. The diagonal on which the rider sits can alter the appearance of lameness. When riding the *left* diagonal, the rider sits when the left forelimb and right hindlimb bear weight, and on the *right* diagonal the rider sits when the right forelimb and left hindlimb bear weight. The correct diagonal is the *left* when trotting clockwise and the *right* when trotting counterclockwise. In general a horse appears lamer behind when the rider sits when the lame limb is bearing weight, and the horse may try to throw the rider to the opposite diagonal. A horse with right hindlimb lameness appears lamer when the rider

sits on the left diagonal, that is, the rider sits when the left forelimb and right hindlimb are bearing weight. In Park, English, or Country English Pleasure classes, where riders are not penalized for riding the incorrect diagonal, horses often are ridden on the diagonal that minimizes lameness. Often a lameness is involved when a horse is seen being ridden on the incorrect diagonal when traveling in one direction and on the correct diagonal going the other way. These horses are referred as right or left diagonal horses. An inconsistent relationship exists between the degree of forelimb lameness and the diagonal on which the rider sits, but lameness may be modified by switching diagonals.

It is important to note whether the observed lameness is constant regardless of direction or pattern. It is important to note if lameness varies, whether the involved limb is on the inside or outside of a circle, whether the horse is on the straight or in a turn, and whether lameness is worse when the horse is on hard or soft ground. The only gait deficit visible in horses with subtle forelimb lameness may be a slight difference in the height or arc of flight of the involved limb. Often a head nod is absent, but the horse appears unsteady in the face (movement of the nose from a fixed position) because of the discomfort of the limb from landing or from pushing off the ground.

DIAGNOSTIC ANALGESIA

If possible, it is important to perform diagnostic analgesia to confirm the source of pain. For example, many horses that appear to have foot pain because of sensitivity of hoof testers or pain on palpation of the SL actually have pain elsewhere. Under American Horse Show Association rules with a properly filed medication report, local anesthetic solution such as mepivacaine can be administered up to 24 hours before a class.

If lameness is to be re-assessed with the horse under saddle or in a cart, intra-articular rather than perineural nerve blocks are preferred, because of possible loss of proprioception, which may lead to tripping or stumbling. After perineural blocks, horses may pull shoes or show an altered or awkward gait regardless of the lameness. If perineual blocks are performed in horses under saddle or in a cart, the rider or driver should be cautioned. Hindlimb nerve blocks can be difficult in uncooperative horses, and a tranquilizer (10 to 15 mg acepromazine intravenously) may be administered to fractious horses. Lameness also may be more obvious, but the risk of permanent penile prolapse with the use of acepromazine in stallions or geldings should be considered. In some horses, diagnostic analgesia of the hindlimbs may be impossible, and the response to intra-articular medication, administered under heavy sedation, must then be assessed.

NEUROLOGICAL EXAMINATION

An Arabian or Half-Arabian show horse with neurological disease may show subtle to overt clinical signs of weakness and ataxia, which may be mistaken easily for signs of musculoskeletal pain. Watching the horse pivot in tight circles, back up, and walk a figure of eight can be performed quickly and may pinpoint subtle deficits that were not noted or were less noticeable earlier in the examination.

Traumatic injuries caused by fractious horses falling during handling and training, and equine protozoal myelitis, are the most common causes of neurological disease and dysfunction in the Arabian and Half-Arabian show horses in North America. Two rare neurological conditions that may be confused with lameness and are much more common in Arabians than in any other breed are cerebellar abiotrophy and occipito-atlantoaxial malformation. Cerebellar abiotrophy is a congenital neurological abnormality that may have an inherited susceptibility and has been reported only in the Arabian horse and Gotland pony. Typical signs occur around 2 to 4 months of age and include head tremor, incoordination, hypermetria, and proprioceptive deficits. Horses may live into adulthood, and anecdotal evidence suggests that the condition may stabilize or improve, although for these horses to improve sufficiently to be useful performance horses is unlikely. Occipito-atlantoaxial malformation is an inherited congenital malformation that has been reported most frequently in Arabians. The severity of clinical signs varies greatly, from splinting of the neck to progressive ataxia and weakness to congenital tetraparesis. Reduced atlanto-occipital movement and abnormal head carriage with the neck extended are common findings.[2] Diagnosis is confirmed radiographically (see Fig. 55-4, *B*). Developmental cervical vertebral anomalies also may cause ataxia.

UNDIAGNOSED LAMENESS

When a diagnosis of the cause of lameness cannot be made, the veterinarian should consider the following:

1. Tack problems: A poorly fitting saddle may create abnormal pressure on the withers or lumbar region and cause poor performance or lameness. A poorly fitted bit or bridle that causes the horse to flip its head or lean into or away from the bit also can affect performance greatly.
2. Dental problems (causing behavior that may lead to poor performance): Sharp enamel points, wolf teeth, and fractured or infected tooth roots may all cause abnormal head carriage and poor performance.
3. Painful skin lesions in the area of the girth, saddle, or bridle may affect performance.
4. A poorly skilled rider pulling on the horse while posting, or with poor balance, or timing may affect the horse's performance.
5. Recurrent exertional rhabdomyolysis affects performance.
6. Subtle neurological deficits may affect performance.

If these problems are ruled out and a diagnosis still cannot be made, the veterinarian should try empirical treatment including a period of rest, a course of non-steroidal anti-inflammatory drugs (NSAIDs), systemic corticosteroids, intravenously administered hyaluronan, oral or systemic polysulfated glycosaminoglycans (PSGAGs), acupuncture, or chiropractic manipulation.

DIAGNOSIS AND MANAGEMENT OF LAMENESS

Bruised and Inflamed Feet

Differential diagnoses for lameness in the foot of an Arabian or Half-Arabian show horse are sole bruising, osteitis of the distal phalanx, foot abscess, penetrating wounds, fractures, laminitis, navicular syndrome, osteoarthritis of the distal interphalangeal joint, and undiagnosed palmar heel pain. The most common foot problems are bruising, osteoarthritis of the distal interphalangeal joint, and palmar heel pain.

Bruising is diagnosed by finding localized or generalized pain in the sole and by ruling out other lameness conditions. Digital pulse amplitudes are usually normal unless bruising is severe. Discoloration of the sole by hemorrhage usually occurs later and may not be seen with acute sole bruising. The most common causes of bruising are hard footing or poor medial to lateral hoof balance. The high side (longer when viewed from the palmar aspect) is usually the bruised side, and treatment

may be as simple as balancing the hoof. If hoof balance is judged to be adequate, then a pad can be cut out or the shoe can be beveled to reduce pressure on the bruised area. A common problem is a low, underslung heel. One foot may be affected, while the other tends to be upright or club footed. Horses with low, underslung hoof conformation commonly develop bruised heels. Although raising the heel angle with a degree pad may appear desirable, this correction actually may cause further heel bruising by concentrating the force on the heel. We recommend the use of a heart bar shoe, frog pads, or an egg bar shoe and a pad cut out over both heels.

Horses with club foot conformation also may develop contracted heels. Treatment involves the use of a heart bar shoe or a frog pad, or beveling the branches of the shoe. Some horses with the low, underslung heel or with club foot conformation may not be lame, and therefore attempts at correction should be tempered.

In a flat-footed horse, sole bruising may occur in areas underlying the shoe, where the sole is not concave enough to prevent contact with the shoe or pad. In these horses a concave inner surface shoe should be used. Pads and packing material are important in treating and preventing bruised feet, but bruising can still be a problem. Silicone, tar, and oakum and newer products such as advanced cushion support are commonly used packing materials. However, if the packing is too firm, it may create rather than prevent sole bruising.

Osteitis of the distal phalanx refers to a non-infectious inflammation of the distal phalanx, which in many horses appears to occur secondary to chronic sole bruising. The diagnosis is made radiographically by observing radiolucent changes and remodeling along the margins and proliferative new bone growth along the dorsal aspect of the distal phalanx. Horses that have these radiographic changes in the toe region also may have chronic bruising, laminitis, solar margin fractures, or club footed conformation. In the heel the margins of the distal phalanx are normally irregular and less well defined radiographically, and a distinction between bruising and osteitis is harder, if not impossible, to make. In most horses, however, the treatment is similar.

Treatment of horses with acutely bruised feet also includes the administration of NSAIDs (phenylbutazone, 4.4 mg/kg bid for 3 days and then 2.2 mg/kg bid for 3 days) and physical therapy. If treatment is necessary during or close to a show in North America, NSAIDs must be given according to the American Horse Show Association rules for therapeutic medication under which the Arabian and Half-Arabian divisions operate. Currently (2001) the guidelines suggest horses can receive phenylbutazone at a dose of 4.4 mg/kg once daily for 5 days in a row no closer than 12 hours before a class. If 2.2 mg/kg phenylbutazone is given by mouth every 12 hours, then the drug can be dosed at any time before a class. Under American Horse Show Association rules, two NSAIDs from a list of five (flunixin meglumine, ketoprofen, naproxen, meclofenamic acid, and phenylbutazone) are allowed to be given at the same time, except that phenylbutazone and flunixin meglumine may not be given to the same horse within 7 days of a class. If bruising is severe and a second NSAID is required, intravenously administered ketoprofen (2.2 mg/kg sid) is helpful. This can be given up to 4 hours before a class. Isoxsuprine (400 mg PO bid) used to increase blood flow to the foot, although controversial, appears to be helpful in many horses.

Physical therapy includes standing the horse in ice (15 minutes in and 15 minutes out for 3 repetitions, repeated several times a day) for the first 24 hours, followed by soaks in hot water and Epsom salts (15 to 20 minutes, 3 times daily). After soaking, the bottom and sides of the foot can be packed with a poultice and wrapped. Before the poultice is applied, dimethylsulfoxide painted on the affected areas is helpful. If areas of the sole are particularly soft, painting on a mixture of formaldehyde and tincture of iodine hardens the sole.

Osteoarthritis of the Distal Interphalangeal Joint and Palmar Heel Pain

Synovitis or acute inflammation of the distal interphalangeal joint is common and likely represents an early form of osteoarthritis. Lameness is usually mild to moderate and frequently is bilateral. Lameness tends to be more obvious in circles or tight turns, frequently with the affected leg on the *outside* of a circle. When horses are affected bilaterally, the only signs may be a shortened cranial phase of the stride and reluctance to go forward. Effusion of the distal interphalangeal joint can be palpated just proximal to the coronary band on the dorsal aspect of the limb. Horses may show pain when hoof testers are applied across the heels, but examination of the sole of the foot is unremarkable, unless other problems such as bruising or navicular syndrome are present. Many young horses develop distal interphalangeal joint synovitis early in training and lameness improves with a short period (14 to 28 days) of rest and NSAIDs. Synovitis of the distal interphalangeal joint is also common in older show horses when it may be associated with poor foot conformation or a heavy show schedule.

No radiographic abnormalities are detected unless the condition is chronic and advanced osteoarthritis develops. Lameness is abolished by analgesia of the distal interphalangeal joint or a basisesamoid nerve block. Three milliliters of mepivacaine deposited over the medial and lateral palmar digital nerves just above the bulbs of the heel also significantly alleviates pain originating from the distal interphalangeal joint.[3] Analgesia of the distal interphalangeal joint is not specific, but use of a small volume (6 ml) of local anesthetic solution and evaluation of the response within 6 to 8 minutes may minimize the effects of diffusion into the digital nerves and inadvertent misdiagnosis.

It is important to remember that several problems may coexist in the foot, and sorting out a single specific diagnosis can be difficult. Horses with low, underrun heels often have bruising, osteitis of the distal phalanx, and osteoarthritis of the distal interphalangeal joint. Horses with navicular syndrome may have bruising in the toe area related to decreased weight bearing on the heel. A bone scan may be useful in differentiating these potential causes of lameness.

Horses with early, acute osteoarthritis of the distal interphalangeal joint should be evaluated clinically for abnormalities of hoof balance and hoof angle. Shortening and rolling the toe of the shoe to ease breakover can be helpful. Shortening the toe in English show horses may not be well accepted by trainers because they think it decreases the desired forelimb action. If the lameness is moderate or severe, the horse should be allowed to rest for 30 days or the workload should be reduced drastically. Intra-articular medication with hyaluronan (20 mg) and a corticosteroid (80 to 120 mg methylprednisolone acetate) and systemic phenylbutazone (2.2 mg/kg PO bid for 5 days) are recommended. If lameness is mild, training can resume 2 to 3 days after the injections. Treatment with systemic hyaluronan (40 mg intravenously once a week for 3 weeks) or PSGAGs (500 mg intramuscularly once every 5 days for 4 to 7 treatments) sometimes is used also, but it should not be a substitute for intra-articular therapy. These products frequently are used as maintenance medications (one dose every 2 to 4 weeks) and for pre-show medication (one dose 24 to 72 hours before a class) for joint-related lameness.

Palmar heel pain is a frequent cause of lameness. Horses may or may not show pain on hoof tester examination, but lameness is eliminated by palmar digital analgesia. Causes include navicular syndrome, deep digital flexor tendonitis, impar desmitis, navicular suspensory desmitis, inflammation of the navicular bone, fragmentation of the distal border of the

navicular bone, and congenital bipartate or tripartate navicular bones. Reaching a specific diagnosis in some horses may be difficult. Diagnosis and management of the navicular syndrome in the Arabian and Half-Arabian show horses are similar to that described for other horses, although this condition is less common than in Quarter Horses or Thoroughbreds.

Although it is important to identify the source of lameness as specifically as possible, the treatment options in horses with palmar heel pain are somewhat limited and similar regardless of the cause. Rest, systemic steroidal and NSAID administration, therapeutic shoeing, medication of the distal interphalangeal joint or navicular bursa in horses with navicular involvement, and palmar digital neurectomy are the most common treatments. The natural balance shoes are not used routinely for English and Park horses because they decrease forelimb motion.

Suspensory Desmitis

Proximal suspensory desmitis is one of the most common lameness conditions of the metacarpal and metatarsal regions. Although more prevalent in the forelimb, proximal suspensory desmitis is a common hindlimb problem and typically is an insidious lameness but may have an acute onset. Lameness is more obvious when the affected limb is on the outside of a circle and is worse on turns than in straight lines. Usually no swelling is detectable, and the response to palpation is unreliable. Definitive diagnosis is based on the response to diagnostic analgesia, combined with radiography and ultrasonography. Many treatments are available, including the use of systemic anti-inflammatory agents; local injections of anti-inflammatory agents or counterirritants; topical agents with support wraps; shock wave, magnetic, laser, and ultrasound therapies; and various surgical procedures. The effectiveness of radial shock wave therapy currently is being investigated.

A decision must be made whether the horse is sound enough to remain in work, as in horses with a chronic low-grade proximal suspensory desmitis, or whether the horse should be allowed to rest. If the horse is to remain in work and local injections are to be part of the treatment regimen, the veterinarian has two options. An anti-inflammatory agent such as a short-acting corticosteroid, possibly mixed with hyaluronan or a PSGAG, may be used, followed by a few days of hand walking, before the horse resumes work. Alternatively, a counterirritant such as 1 to 2 ml of 2% iodine in almond oil is injected, followed by continued work. In both instances the work is limited to graduated periods of walking and low-speed trotting for several weeks. The walking and trotting generally are performed in escalating timed intervals. The canter or lope is not recommended initially, because the affected limb is the only limb on the ground for a portion of the gait sequence. The weight of the shoes and pads may be reduced during the initial portion of the recovery period. If the injury is too severe for the horse to remain in work, the horse is confined and hand walked until recovery is sufficient enough to begin a regimen of low-impact, controlled exercise. These horses are not turned out, because they tend to re-injure themselves with free exercise.

Proximal suspensory desmitis tends to recur with prolonged intense exercise. Horses with bilateral club feet, low underrun heels, one club foot and one low heel, or substantial rotational and angular deformities are predisposed to proximal suspensory desmitis. Horses with a chronic lameness in the opposing limb or the diagonal limb also are predisposed to lameness because of compensatory loading. It is important to identify other problems, because resolution of lameness in another limb may be crucial to the long-term resolution of proximal suspensory desmitis.

Suspensory branch desmitis also occurs frequently. With an acute injury there is pain, heat, and swelling over the affected branch and a positive response to lower limb flexion. Usually some distention of the fetlock joint capsule occurs. Diagnosis is confirmed by ultrasonography, and the proximal sesamoid bones should be evaluated radiographically. Horses with acute injuries are treated symptomatically with ice, poultice, NSAIDs, and systemic corticosteroids. If sesamoiditis is present, isoxsuprine or pentoxifylline treatment may be helpful. The shoeing should be evaluated and imbalance (frequently a low heel on the affected side) corrected. If possible, the toe should be shortened, or a rolled toe shoe should be used to ease breakover. The branches of the shoe should be extended, or an egg bar shoe should be applied to support the SL. Horses with chronic active suspensory branch desmitis may benefit from injection of corticosteroids (methylprednisolone acetate and isoflupredone acetate) subcutaneously in the affected area. Most horses are managed with medical treatment, although surgical splitting of the branch or bone marrow injections are alternative treatment options. Suspensory branch desmitis tends to recur, can be difficult to manage successfully, and may be a career-ending injury.

Osteoarthritis of the Stifle Joints

The stifle is a common source of lameness, and apart from occasional acute injuries most horses have chronic, often low-grade lameness that may not be evident when examined in hand. Riders may complain that horses are hard on one side of the mouth, tracking with a shoulder or hip in or out, falling out of a hind lead in the corners, and bending poorly. Horses with stifle pain also have difficulties in downward transitions, because they cannot maintain a collected frame and tend to fall out behind. If forced to maintain collection, the head may be raised. Horses exhibit increased discomfort going downhill. If the work area has even a slight grade, an affected horse appears more comfortable going up the grade than down. Horses also fatigue easily in deep footing, with loss of collection, diving forward in the bridle (English divisions), raising out of the bridle (Western and Hunter divisions), and loss of hindlimb cadence at the trot. Lameness is worse in turns and improves in the straight portions of the arena.

The medial femorotibial and femoropatellar joint capsules may be distended and fibrous thickening from a previous medial patellar desmotomy and other abnormalities from previous injuries may be apparent. Various flexion tests, such as pulling the limb caudally, may exacerbate lameness, but these tests are not reliable and may be dangerous to perform. Intra-articular analgesia usually is required. The medial femorotibial joint, the femoropatellar joint, and the lateral femorotibial joint are affected with decreasing frequency and generally are blocked individually in that order. Radiography should include caudocranial, lateromedial, and oblique views. Ultrasonographic examination, nuclear scintigraphy, and diagnostic arthroscopy are sometimes required.

Most commonly lameness is improved by intra-articular analgesia of the medial femorotibial joint, but radiography is negative or equivocal. Affected horses are thought to have chronic inflammation of the soft tissues of the joint and synovitis. Some of these horses eventually develop an enthesophyte on the proximal medial aspect of the tibia. Lameness often recurs, because lameness reflects the type of work the horse performs, the collected frame required, and conformational weaknesses, such as lack of angulation in the hip, stifle, and hock, camped-out conformation, and weak hindquarters in general. The condition is managed by adapting the work level between shows and the show schedule of the horse to accommodate for lameness, by corrective shoeing, and by using systemic anti-inflammatory agents and intra-articular medication. Corrective shoeing is individualized but involves trimming the foot short and using a light shoe that assists breakover. Many horses break over the dorsomedial aspect of

Fig. 124-2 A left hind shoe indicating natural breakover medial to the toe area (medial is right). Future shoes will be formed to allow breakover in this area rather than directly at the center of the toe.

the toe, and that portion of the shoe can be rolled or shaped to ease breakover. A worn shoe can be examined for breakover location (Fig. 124-2). Some horses are more sound barefooted behind and compete that way. To maintain soundness at shows, horses are maintained at a work level between shows that does not require intra-articular treatment. Systemic administration of hyaluronan and a PSGAG may help. Systemic and intra-articular anti-inflammatory therapy is begun before competition to minimize lameness during the competition. If a horse requires frequent therapy between shows, it has a poor chance of enduring a competition in good form.

Counterirritant therapy in the form of 2% iodine in oil commonly is injected in various patterns along the patellar and collateral ligaments of young horses and horses with loose stifles and in horses that have been rested as the intensity of training increases and the horse exhibits non-specific pain or weakness in the stifle area. Counterirritants are not used as a replacement for intra-articular therapy but may be used concurrently. Injections usually are limited to 5 to 10 ml to reduce postinjection inflammation and to decrease scarring from multiple injections. Work is continued, but the intensity is reduced for a short time as the horse recovers.

Osteochondrosis is the second most common problem seen in the stifle. Osteochondral fragments from the femoral trochleas can be removed arthroscopically, usually with a favorable prognosis. Horses with lameness caused by subchondral bone cysts in the medial femoral condyle have a poorer prognosis. Some horses respond to intra-articular corticosteroids. Surgical treatment may fail to resolve the lameness.

Thoracolumbar (Back), Sacroiliac, and Gluteal (Croup) Pain

Work-related back and croup pain is common in horses because of working in a collected frame frequently or for extended periods of time, compensatory altered carriage caused by lower limb injuries, poor saddle fit, poor balance and timing of a rider, injuries from falling and weakness from immaturity, lack of conditioning, or poor conformation (long, weak back).

Back pain should be suspected when behavioral changes such as kicking out during lead changes and uncharacteristic bucking occur. The horse is observed for muscle asymmetry while it stands with the body straight and the limbs squared on a level surface. The back and croup are palpated for areas of pain, using gradual pressure with the flat surface of the digits. The lumbosacral junction should be evaluated by placing the heel of one hand on the left side of the loin and, with the other hand over the base of the tail, rocking the area back and forth. A normal horse allows this, but a horse with pain stiffens and steps away from the veterinarian. Unilateral abnormalities may indicate concurrent lower limb injuries. Unilateral lumbar pain often results from compensatory carriage of the hindlimbs to that side. Unilateral sacroiliac inflammation or dorsal ligamentous thickening often results from over-bearing on that side from a chronic contralateral or diagonal limb injury. For example, horses with a chronic left front or left hind lameness may have swelling and pain in the area of the right tuber sacrale. Rectal examination is used to evaluate pelvic symmetry and ventral lumbar muscular pain. Other diagnostic modalities include radiography, ultrasonography, nuclear scintigraphy, and thermography, but physical examination remains the most important and reliable method of diagnosis. Myositis also occurs commonly and is diagnosed by history, observation of altered gait, palpation, and serum enzyme assay.

A variety of treatments is used, and these are discussed thoroughly elsewhere. Usually a period of rest is indicated, along with resolution of any associated lameness, followed by a period of reduced exercise. Systemic and local anti-inflammatory agents are administered. Other therapies include cold and heat therapy, acupuncture, chiropractic therapy, massage therapy, and magnetic, electromagnetic, laser, and ultrasound therapy.

Successful treatment of horses with back and croup injuries depends on identification of initiating causes and making appropriate changes to alleviate these causes if possible. Educating the rider is also helpful, because changes in training routines or tack may be helpful in preventing recurrence.

Osteoarthritis of the Metacarpophalangeal and Metatarsophalangeal Joints

Synovitis of the metacarpophalangeal and metatarsophalangeal joints, an early form of osteoarthritis, causes effusion, pain on fetlock flexion, and a positive response to a lower limb flexion test. The condition is frequently bilateral. Because mild effusion in sound Arabian and Half-Arabian show horses is seen commonly, it is important to perform intra-articular or perineural analgesia to confirm the source of pain when osteoarthritis of the metacarpophalangeal or metatarsophalangeal joint is suspected. Radiography is performed to determine the extent of osteoarthritic abnormalities. Many Arabian and Half-Arabian show horses perform well in spite of mild osteoarthritis of the fetlock (Fig. 124-3). Treatment is similar to that recommended for osteoarthritis of the distal interphalangeal joint, except that a shorter-acting corticosteroid such as triamcinolone or betamethasone is used for intra-articular medication.

Distal Hock Joint Pain

Osteoarthritis of the centrodistal and tarsometatarsal joints or distal tarsitis is common. Tarsal lameness is often bilateral, although one limb usually is affected more severely. Affected

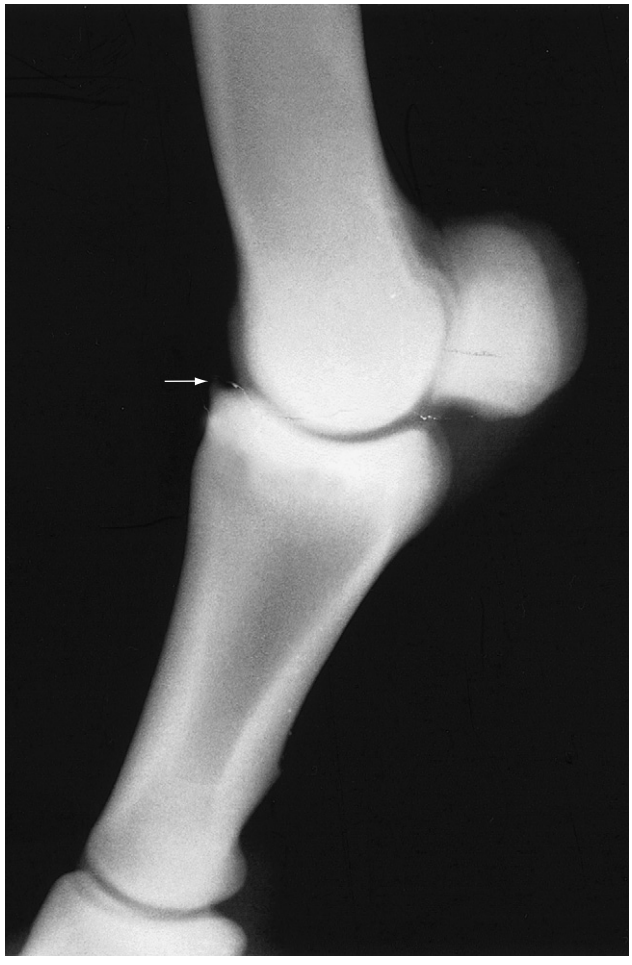

Fig. 124-3 Lateromedial radiographic view of a metacarpophalangeal joint of a sound Half-Arabian show horse. There is a moderately sized osteophyte *(arrow)* on the proximal dorsal aspect of the proximal phalanx, reflecting osteoarthritis.

horses have reduced hock flexion at the trot and tend to travel with a hip off to one side, especially in the corners (drift away from the lame limb). Rider complaints are similar to horses with stifle lameness. Western horses may tend to rate poorly and lope (canter) with a four-beat rather than a three-beat action. Reining horses tend to raise up in spins to the affected direction, bend poorly, stop unevenly or on the forehand, and change leads late behind. Diagnosis is based on palpation, including the Churchill test, flexion tests, diagnostic analgesia, radiography, and occasionally ultrasonography and nuclear scintigraphy. Treatment comprises systemic administration of anti-inflammatory agents and intra-articular medication with corticosteroids, possibly with hyaluronan (60 to 80 mg of methylprednisolone acetate and 10 mg of hyaluronan per joint), repeated as necessary, often 2 to 3 times during a show season. If medical therapy fails, chemical, laser, and surgical fusion techniques are available. Cunean tenectomy usually is not performed because the procedure commonly does not resolve lameness. Corrective shoeing involves inspecting the old shoe and squaring the new shoe to accommodate the breakover, setting the shoe back, shortening the toe, reducing the weight of the hind shoe, and occasionally using asymmetrical trailers.

Splint Bone Injuries

Proliferative periostitis, referred to as splints, and fractures of the second and fourth metacarpal bones and to a lesser extent fractures of the second and fourth metatarsal bones are common, especially in young horses. Diagnosis of splint-induced lameness is made using palpation, diagnostic analgesia, and radiography. Associated suspensory desmitis should be considered and assessed by ultrasonographic examination. Predisposing causes include conformational defects such as offset knees or base narrow and toed out. Heavy shoes and pads can exacerbate a tendency to wing in and cause interference. Poor footing that is excessively deep or hard can contribute to the development of splints. Immature horses in excessive work, or wearing shoes and pads that are too heavy for their level of fitness, are predisposed to splints. Shoeing with improper medial to lateral balance, obesity, and dietary imbalances can be contributory causes.

Proliferative periostitis originates from injury to the interosseous ligament, resulting in the development of proliferative fibrous connective tissue that subsequently mineralizes. Radiography is used to assess the injury and to determine the presence of fractures. Osteolysis of the third metacarpal or metatarsal bone and of the splint bone at the site of the injury is a sign of substantial inflammation and can indicate a prolonged recovery. Treatment includes rest (2 to 4 weeks), cold therapy, systemic and local administration of anti-inflammatory agents, topical agents with support wraps, and magnetic therapy. Large exostoses that interfere with surrounding soft tissue structures can be excised surgically.

Work-related fractures are predominantly distal, closed fractures and are thought to result from over-excursion of the SL during fetlock hyperextension. These fractures frequently are displaced, heal by non-union, and tend to irritate surrounding soft tissues. For these reasons they commonly are removed surgically. Mid-splint bone non-displaced fractures often resolve with conservative therapy. Initial therapy is similar to the treatment of a true splint, except that local administration of corticosteroids is not performed. If conservative therapy fails, surgical excision of the exostosis and distal portion of the splint bone may be performed. Horses with proximal fractures are evaluated on an individual basis, because some respond to conservative therapy, whereas others require surgical intervention.

Osteoarthritis of the Proximal Interphalangeal Joint

Incidental radiographic changes (lipping and spurring) indicating mild osteoarthritis of the proximal interphalangeal joint in Arabian and Half-Arabian show horses are not uncommon. Moderate to severe osteoarthritis, however, would be expected to cause chronic lameness. In most horses osteoarthritis of the proximal interphalangeal joint is caused by chronic repetitive trauma, although osteoarthritis secondary to a single severe injury is possible. In young horses osteochondrosis in the form of osseous cyst-like lesions and fragmentation may be involved. The importance of any radiographic changes should be validated using diagnostic analgesia. Lower limb flexion is usually positive, and lameness should improve with a basisesamoid or abaxial sesamoid block or analgesia of the proximal interphalangeal joint. Treatment is similar to that recommended for horses with osteoarthritis of the distal interphalangeal joint.

Desmitis of the Accessory Ligament of the Deep Digital Flexor Tendon

Desmitis of the accessory ligament of the deep digital flexor tendon occurs less frequently than does proximal suspensory desmitis, primarily in limbs with club feet or low, underrun heels. Footing that accumulates under the shoe, creating a rocking motion of the foot during the weight-bearing phase of the stride, may predispose horses to desmitis. Lameness is usually acute in onset, and palpation often reveals a painful

thickening along the length of the ligament that can be confused with the deep digital flexor tendon. If the condition is chronic and low grade, diagnostic analgesia may be required. Diagnosis is confirmed with ultrasonography. Treatment involves prolonged rest and systemic and local administration of anti-inflammatory agents. Adjuncts include cold therapy, topical agents with support wraps, and magnetic, laser, and ultrasound therapy. Because the predisposing conformational defect remains, the condition tends to recur.

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CHAPTER • 125

Lameness in the Driving Horse

Kevin Keane and Graham Munroe

DESCRIPTION OF THE SPORT

This chapter deals with horses used for competitive driving purposes, those driven privately for pleasure, and those used as beasts of burden as a mode of transportation. The sport of driving horses in competition is relatively new and has many variations, requiring different types of horses performing different tasks. Certain breeds or breed types are perfectly suitable for one form of driving sport but not another, and variation in the size, type, and breed of horse used is considerable. Horses and ponies are used, and the term *horse* is used in this chapter to refer to both, except when specific reference to a pony is required.

In the United Kingdom and Europe there is a much greater availability of a variety of driving competitions than currently exists in North America. Pleasure driving includes presentation classes in the show ring and general driving on roads and tracks. Presentation classes are grouped broadly into hackney and non-hackney types, the difference being based on the phenotype of the animal as opposed to a breed registry. The competitor pays close attention to the harness, attire of the driver, and an appropriate carriage to suit the horse, because judging is subjective and based on strict adherence to tradition, based on a suitable match of horses and carriage. The horses perform movements requested by the judges, and style and quality of the gaits are scored subjectively. These horses compete at the walk and trot (a park pace being roughly equal to a slow working trot). Pleasure driving events also can include drives at the walk or trot on roads and tracks of up to 5 to 10 miles.

Competitive driving includes combined driving or horse driving trials and scurry driving. Scurry driving is seldom seen in North America but has a strong following in the United Kingdom and other parts of Europe. Scurry driving consists of a single pair, or more usually pairs, of mainly ponies competing over a tight, coned course in a show ring against the clock. The horses and carriages are often small to allow the narrow gates (the gap between a pair of cones) and corners to be negotiated at speed. Horse driving trials are a driven form of horse trials, or eventing, and like its ridden counterpart, driving is a highly athletic, physically demanding sport for the horse and driver.

Driving trials as an international equestrian sport started in 1968, when the Federation Equestre Internationale (FEI) international rules were drawn up under the instigation of HRH Prince Philip, who was then the President of the Federation. The first international horse driving trials event took place in 1971 in Hungary. Initially the competition was only for horse teams (four-in-hand). A team is four horses, two before two, and those in front are called *leaders* and those closer to the coach, *wheelers*. As the sport developed and individuals of more modest means entered the fray, competitions for singles (one horse), tandems (two horses harnessed one behind the other), and pairs (two horses harnessed side by side) rapidly blossomed. These classes were further divided into those for ponies (less than 148 cm or 14.2 hands high) and horses. The sport is now structured at various levels depending on the ability of the driver and horse(s). The FEI is responsible for the international rules that cover the world and European championships and selected international events such as the Royal Windsor International Driving Grand Prix. The national associations liaise with the FEI and are responsible for running the national events and championships and producing national rules. Local driving clubs (found in Europe, the United Kingdom, and the United States) also run local events that may differ substantially in standards and requirements of competitors, but they essentially mimic FEI rules. Important events include the following: Royal Windsor (United Kingdom), Aachen and Riesenbeck (Germany), Breda (Holland), Saumur (France), Waregem (Belgium), St. Gallen (Switzerland), and Fair Hill (United States). World championships are held every 2 years in singles, pairs, and teams in horses, and a European championship is held in pony teams. The first World Pony Championships (singles, pairs, and teams) will be held in Saumur in 2003. Competitions in Europe run from April to September, with championships held toward the end of this period.

Horse driving trials consist of three phases—dressage, marathon, and cones—usually spread over 3 days, but in lower standard competition all three may take place over 1 to 2 days.

thickening along the length of the ligament that can be confused with the deep digital flexor tendon. If the condition is chronic and low grade, diagnostic analgesia may be required. Diagnosis is confirmed with ultrasonography. Treatment involves prolonged rest and systemic and local administration of anti-inflammatory agents. Adjuncts include cold therapy, topical agents with support wraps, and magnetic, laser, and ultrasound therapy. Because the predisposing conformational defect remains, the condition tends to recur.

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CHAPTER • 125

Lameness in the Driving Horse

Kevin Keane and Graham Munroe

DESCRIPTION OF THE SPORT

This chapter deals with horses used for competitive driving purposes, those driven privately for pleasure, and those used as beasts of burden as a mode of transportation. The sport of driving horses in competition is relatively new and has many variations, requiring different types of horses performing different tasks. Certain breeds or breed types are perfectly suitable for one form of driving sport but not another, and variation in the size, type, and breed of horse used is considerable. Horses and ponies are used, and the term *horse* is used in this chapter to refer to both, except when specific reference to a pony is required.

In the United Kingdom and Europe there is a much greater availability of a variety of driving competitions than currently exists in North America. Pleasure driving includes presentation classes in the show ring and general driving on roads and tracks. Presentation classes are grouped broadly into hackney and non-hackney types, the difference being based on the phenotype of the animal as opposed to a breed registry. The competitor pays close attention to the harness, attire of the driver, and an appropriate carriage to suit the horse, because judging is subjective and based on strict adherence to tradition, based on a suitable match of horses and carriage. The horses perform movements requested by the judges, and style and quality of the gaits are scored subjectively. These horses compete at the walk and trot (a park pace being roughly equal to a slow working trot). Pleasure driving events also can include drives at the walk or trot on roads and tracks of up to 5 to 10 miles.

Competitive driving includes combined driving or horse driving trials and scurry driving. Scurry driving is seldom seen in North America but has a strong following in the United Kingdom and other parts of Europe. Scurry driving consists of a single pair, or more usually pairs, of mainly ponies competing over a tight, coned course in a show ring against the clock. The horses and carriages are often small to allow the narrow gates (the gap between a pair of cones) and corners to be negotiated at speed. Horse driving trials are a driven form of horse trials, or eventing, and like its ridden counterpart, driving is a highly athletic, physically demanding sport for the horse and driver.

Driving trials as an international equestrian sport started in 1968, when the Federation Equestre Internationale (FEI) international rules were drawn up under the instigation of HRH Prince Philip, who was then the President of the Federation. The first international horse driving trials event took place in 1971 in Hungary. Initially the competition was only for horse teams (four-in-hand). A team is four horses, two before two, and those in front are called *leaders* and those closer to the coach, *wheelers*. As the sport developed and individuals of more modest means entered the fray, competitions for singles (one horse), tandems (two horses harnessed one behind the other), and pairs (two horses harnessed side by side) rapidly blossomed. These classes were further divided into those for ponies (less than 148 cm or 14.2 hands high) and horses. The sport is now structured at various levels depending on the ability of the driver and horse(s). The FEI is responsible for the international rules that cover the world and European championships and selected international events such as the Royal Windsor International Driving Grand Prix. The national associations liaise with the FEI and are responsible for running the national events and championships and producing national rules. Local driving clubs (found in Europe, the United Kingdom, and the United States) also run local events that may differ substantially in standards and requirements of competitors, but they essentially mimic FEI rules. Important events include the following: Royal Windsor (United Kingdom), Aachen and Riesenbeck (Germany), Breda (Holland), Saumur (France), Waregem (Belgium), St. Gallen (Switzerland), and Fair Hill (United States). World championships are held every 2 years in singles, pairs, and teams in horses, and a European championship is held in pony teams. The first World Pony Championships (singles, pairs, and teams) will be held in Saumur in 2003. Competitions in Europe run from April to September, with championships held toward the end of this period.

Horse driving trials consist of three phases—dressage, marathon, and cones—usually spread over 3 days, but in lower standard competition all three may take place over 1 to 2 days.

Table • **125-1**

Basic Format of the Five-Stage Marathon

	Length	Gait	Average Speed (kph)
Section A	≈ 10 km	Any type, usually trot	15
Section B	1 km	Walk	7
Section C	4 km	Fast trot	19
Section D	1 km	Walk	7
Section E	10 km	Trot/canter	14

The first phase is a driven dressage test, which consists of a set sequence of movements that are judged by a number of officials against a standard of absolute perfection. The test is designed to highlight the obedience, paces and suppleness of the horse(s), and the skill of the driver in handling of the reins. The second stage is the marathon, which tests the fitness and stamina of the horse(s) and the judgment of pace and horsemanship of the driver. The cross country marathon can be divided into three or five sections (depending on the level of competition) for which on each section a maximum and minimum time are allowed (Table 125-1).

The speeds and time allowances are adjusted for different classes, especially ponies. At the end of sections B and D are mandatory 10-minute halts. During the second of these, the animals are subjected to veterinary checks for lameness, injuries, and fitness (respiratory rate, pulse rate, dehydration, temperature, and speed of recovery). Section E has eight obstacles. Each obstacle is made up of a number of lettered gates. The aim is to drive through these gates (between white and red markers) within each obstacle in the correct alphabetical sequence in the shortest possible time. Most injuries occur on the marathon, although lameness may not become apparent until later, just before the third phase, the cones competition. The object of the cones phase is to test the fitness and suppleness of the horse after the marathon phase by driving through a course of narrowly spaced pairs of cones within an allotted time. Each cone has a ball balanced on top of it that is dislodged easily if the horse or carriage strikes the cone. The winner of the competition is the competitor with the fewest penalty points.

Veterinary inspections of horses taking part in an event also occur before the dressage day, at the end of the marathon, and at the beginning of the cones competition. Lameness at any of these inspections usually leads to elimination from the event, although considerable variation exists in the definition of working soundness among veternarians, particularly respecting hindlimb problems.

Horses used by Amish and Mennonite sects (United States) as beasts of burden are nearly always American Standardbreds or Saddlebred horses and are driven as singles. The horses are driven when needed, with no structured fitness training, and lameness is common.

TYPES OF HORSES USED

The type of horses used for driving varies considerably with the particular form of the sport or use to be undertaken. For pleasure and presentation driving the type of horse used is related mainly to the size and type of the carriage and the overall effect the driver is trying to convey to the judges (e.g., country or town turnout, meaning an informal or formal appearance of the coach, harness, and driver's attire). Horses used include Shires, Clydesdales, and Percherons in heavy horse turnouts such as drays; Hackneys (including crosses), Thoroughbreds, and Warmbloods in smart town turnouts; Cobs, larger pony types such as Welsh Cobs, Welsh Section C, Dales, Fells, Fiordlanders, and Friesians in country turnouts; and smaller pony types such as Welsh Mountain Section A, Shetlands, New Forest, and Dartmoor in small carriage turnouts. These aforementioned breeds primarily cover the variety seen in the United Kingdom and Europe. In the United States Warmblood, Warmblood crosses, Welsh, Hackney, Morgan, and Friesans are used most often for pleasure and driving trials. Most presentation and pleasure driving is undertaken with a single horse turnout. The horses range from 4 to 20 years of age and many have been or are used for other equestrian disciplines. Scurry driving usually involves single or pairs of small ponies or pony-type horses such as Shetlands, Welsh Mountain, New Forest, and Dartmoor. These ponies are often younger than pleasure-driving animals and are less likely to be used for other equestrian sports, except perhaps combined driving trials.

Driving trials horses must be older than 4 years before they can compete, and records show 19-year-old horses competing at world championship level. Many of the best driving trials horses are 12 to 19 years of age and have been used earlier for other purposes. This long working life and slow introduction to work at a young age, together with little high speed, more slow speed conditioning work, and regular winter breaks, has a considerable effect on the type of lameness seen in these animals. The types of horses and ponies used vary greatly. In continental Europe, Warmblood breeds are particularly popular; for example, Gelderlanders, Swedish Warmbloods, Dutch Warmbloods, Hanovarians, and Holsteiners, with a modern trait being an ever-increasing size. In the United Kingdom less uniformity occurs in the horses used; for example, Hackney crosses, Cobs, Welsh Cobs, Lipizzaners, Lusitanias, Orlovs, and some Warmbloods and Thoroughbred crosses. The most popular ponies used for driving trials in Europe are Welsh Sections A, B, and C, Haflingers, and New Forest crosses.

In the practice radius of one of the authors (K.K.) is a unique opportunity to observe, evaluate, and diagnose lameness conditions in driving horses used by members of the Mennonite and Amish religious sects as a mode of transportation. Several regions throughout North America have populations large enough to provide a reasonable number of horse owners for which to provide veterinary services. The incidence of lameness is influenced by the necessity of driving reasonably long distances on asphalt surfaces (up to 30 miles in one day), with sometimes a single horse pulling a Meadowbrook cart containing up to seven family members. Electricity is not used by the Amish sect and is consequently not available, so a veterinarian must be guided by the ability to palpate precisely and interpret the findings often without the adjunct diagnostic procedures relied on daily, such as radiography and ultrasonography. The veterinarian also faces great pressure because a lame horse may strand an Amish owner.

TRAINING

The training regimen for driving horses varies considerably depending on the type of driving to be undertaken and the level of competition to be attempted. Top-class horse driving trials horses require a regimented fitness program of up to several hours daily, with techniques for such fitness varying from trainer to trainer. In addition, some time is spent practicing cones and hazard training through schooling obstacles set up to mimic what is seen in competition. In pleasure or presentation driving, animals normally are worked intermittently, mainly on roads and tracks at the walk and trot, usually

in the summer months, with a rest or turnout to grass in the winter. In scurry driving the training required is more intense, with a combination of regular road work at the walk and trot to increase overall fitness, alongside school and field work concentrating on bending, suppleness, and turning at speed with accuracy. The scurry driving season can extend over longer periods of the year, when competition comes indoors. Amish horses attain a less quantitative level of fitness through irregular use and essentially no training.

Conditioning exercise tends to start in February, aiming for the first events in late April. Competitions are then available almost every weekend (Thursday to Sunday) throughout the summer, culminating in the national and international championships in August to October.

The type or breed of horse used in driving and the way the horse is trained have an important effect on the type and incidence of lameness. Differences in conformation, gait, and size have an influence. Generally, ponies are less likely to develop lameness, are easier to train to fitness, and are more agile. Pony conformation and foot shape are better and they carry less weight. Unfortunately, alongside this general toughness is all too frequently allied a cussed temperament. Cob types are similar to ponies in temperament and hardiness but are heavier and more powerful, often leading to low-grade osteoarthritis in later life. They often require considerable training to allow the necessary control and obedience to be obtained. Welsh Cobs and Hackney types and crosses have particularly exaggerated natural forelimb actions, which over long periods of use may increase wear and tear injuries in the forelimbs such as metacarpophalangeal joint problems. Some of the larger breeds, such as the Warmbloods, suffer from poor conformation, especially in the hindlimbs, such as straight in the stifle and hock, which has a major effect on the incidence of lameness. Most driving animals are not broken to harness until they are 3 to 4 years old and are not worked until they are 5 to 6 years old. Therefore conditions prominent at an earlier age, such as osteochondrosis of the tarsus and stifle, are uncommon.

The incidence of lameness in driving horses is much influenced by a long working life; the training, which is mainly flat work at the walk and trot; the different stresses and strains placed on them by the carriage (increased pressure on the hindlimbs, especially distally); the regular rest periods during the driving career; slow start in life; and whether they have been used for other purposes, previously or concurrently. All of these factors contribute to a low incidence of lameness, especially from fractures or acute soft tissue injuries seen in racing animals, but an increased incidence of low-grade wear and tear injuries, particularly of the hindlimb joints. In recent years the increasing competition and prestige at the top end of the sport, particularly internationally, has led to increased demands on horses, less patience to wait for horses to mature or to become seasoned, and consequently more lameness.

GROUND CONDITIONS

Training is usually carried out on roads or tracks of varying surfaces, depending on the region and country involved. In the United Kingdom this is mainly tarmacadam roads and firm tracks, whereas in the eastern United States, tarmacadam, dirt, and gravel roads and precut paths through agricultural fields predominate. Training for dressage and cones is usually in grass paddocks, although some drivers have access to all-weather areas. The variable, often hilly terrain in the United Kingdom and United States helps develop fitness. In Europe some of the competitions and areas for training are flat and have a sandy soil, which gives an even, absorbent, good surface for exercise. The firm surfaces on which many driving horses train and work tend to increase concussion to the feet and limbs, but they strengthen tendons and ligaments. Repeated concussion over many years may contribute to low-grade joint disease and does mean that good foot conformation and shoeing are imperative to help offset some of this constant trauma. The variable and unpredictable ground conditions present on marathon courses contributes to injuries to joints, such as the fetlock, and to ligaments and the digital flexor tendon sheath.

CONFORMATION

The huge variety of breeds and types of animals used in driving means that no particular traits of conformation have been established as representative of this type of work. Many of the Warmbloods, which are the most common breeds on the continent of Europe and are now becoming so popular elsewhere in the world for riding and driving, have a conformation that appears to predispose them to an increasing incidence of osteoarthritis of the distal hock joints. The hindlimbs are often straight through the hock and stifle, with increased body weight on small-boned legs. Some are also sickle hocked and cow hocked. Many Warmblood breeds have a high incidence of osteochondrosis, especially of the tarsocrural and stifle joints, which may manifest itself later in the working life of the horse. The headlong dash in recent years for bigger and stronger Warmblood driving horses has in our opinion lead to a heavier, less agile animal, often with small feet and limited bone, which cannot help the animal cope with work over the many years that the horse is driven. Foot conformation in some of the carriage breeds, such as the Hackney, Orlov, Gelderlander, and Lipizzaner, can be upright and boxy, which may decrease concussion protection by the foot and increase trauma reflected up the limb. Many of the native breeds or crosses have good conformation and inherent limb soundness, which is reflected in their low level of lameness. The exception to this statement is the pleasure or presentation pony that is worked irregularly and kept at grass in the summer. The overweight (show condition) nature of these ponies and the access to large amounts of grass predisposes them to laminitis, which reflects more on owner management than inherent unsoundness.

TEN MOST COMMON LAMENESS CONDITIONS

A 15-year retrospective study of the clinical practice of one of the authors (K.K.) led to the formation of the following list of common lameness conditions, in order of incidence. Some variation in incidence occurs from leaders to wheelers in four-in-hand teams and in other countries, such as the United Kingdom.

1. Suspensory desmitis
2. Foot lameness: palmar heel pain, corns, and navicular syndrome
3. Distal hock joint pain
4. Exertional rhabdomyolysis
5. Tenosynovitis of the digital flexor tendon sheath (DFTS)
6. Osteoarthritis of the proximal and distal interphalangeal and fetlock joints
7. Stifle lameness
8. Lesions associated with turnout: solar abscesses and bruising, mud fever, rain scald, cracked heels, and kick wounds
9. Interference and traumatic injuries of forelimbs and hindlimbs
10. Desmitis of the accessory ligament of the deep digital flexor tendon (ALDDFT) and superficial digital flexor tendonitis

LAMENESS EXAMINATION

Examination of lameness in the driving horse differs little from that of a standard approach. The major objectives are to decide if the animal is lame, which limb or limbs are affected, and which portion or portions of the limb(s) are affected and to determine a pathological process. This process is complicated in a driving horse, particularly as the horse ages, because of the possibility of old or insignificant lesions and the low-grade and often bilateral nature of some causes of lameness. Further problems are related to intermittent or variable lameness. Identification of lameness is more difficult in a driving animal when being driven, especially in the hindlimb, than in a ridden horse.

The history should consist of typical questions asked before any standard lameness evaluation: for example, how the lameness initially presented; what the duration of lameness is; whether lameness is worse on hard or soft ground; and whether the horse has responded to any treatments used. In addition, because driving horses are exercising between two poles that are parallel to the ground, straightness is generally fairly easy for the driver to observe. Is the horse resting one hip on the right or left shaft? Is the horse leaning more on one rein than the other? With pairs and four-in-hand is one horse taking more of the workload, indicating unwillingness of the other horse to pull an equal load? Have any changes in tack or harness been made? The veterinarian should observe the stance and attitude of the horse, areas of muscle atrophy, limb and foot conformation, and the presence of swellings before assessing the horse moving in hand at the walk and trot on a hard, flat surface. Examination in harness and carriage is rarely useful, because lameness is sometimes less evident while the horse is pulling, rather than moving freely and without restriction on a lunge line. When a driver or trainer feels that the demonstration of the lameness for which the horse is being presented is observed only when in work, that is, driven, seeing the horse in harness then may be necessary. Standard flexion tests of the forelimbs and hindlimbs are useful, but as a horse ages the likelihood of a positive result in an otherwise working, sound horse increases. Exercise on the lunge or in hand on a circle is helpful in bilateral or mild lameness. Exercise on different surfaces (hard and soft) and when possible on a slight incline up and down hill can be useful.

Identification of the affected lame limb(s) should be followed by detailed palpation and manipulation of the limb(s), with the limb bearing weight and in a flexed position. Assessment of any swelling by digital palpation and manipulation of the limb(s) to determine whether pain can be elicited can help to differentiate old, clinically insignificant lesions. In older horses distention of fetlock and tarsocrural joint capsules and DFTSs is a common, insignificant finding. Skin scars and areas of fibrosis from previous trauma may further complicate the localization of the site of lameness. Careful examination of the foot with hoof testers is important. The use of diagnostic analgesia is considered vital to rule out incidental lesions or previously managed chronic problems.

DIAGNOSTIC ANALGESIA

Many of the causes of lameness in driving horses are related to chronic wear and tear injuries in one or more joints. Distention of a joint capsule or DFTS can be an incidental finding. Localization of pain by a logical system of regional analgesia is central to our approach to lameness diagnosis and is particularly useful in horses with bilateral or multiple limb lameness, because such an approach allows the effect on the gait of one or more of the lame limbs to be removed, allowing identification of additional lame limb(s). In most horses localization of pain is initially undertaken by a sequence of perineural analgesic techniques beginning distally and moving proximally in a logical manner. In the forelimbs the sequence is palmar digital; abaxial sesamoid; low palmar and palmar metacarpal (four point); high palmar (subcarpal); and median/ulnar/musculocutaneous. In the hindlimb the sequence is abaxial sesamoid; low six point; and tibial/fibular. Nerve blocks of the antebrachium or crus are useful when the veterinarian is uncertain if the lesion is above or below mid-limb. Once that has been determined, more specific blocks may be planned proximally or distally as needed. If the veterinarian suspects a particular site, performing an intra-articular block at that site first may be more efficient. It may be necessary to perform intra-articular blocks after perineural analgesia. Sites for intrasynovial analgesia are prepared aseptically (with or with out clipping, at the discretion of the veterinarian), and fresh bottles of mepivacaine or bupivacaine, needles, and syringes are used. Gloves are mandatory for intrasynovial techniques. Assessment of response can be difficult in horses with mild or intermittent lameness or when several painful sites exist. The veterinarian should be as certain as possible of the degree of improvement in the lameness, but unfortunately this is sometimes equivocal. In a driving horse the most common intrasynovial structures injected are, in the forelimb, the distal interphalangeal, proximal interphalangeal, and metacarpophalangeal joints and the DFTS; and, in the hindlimb, the proximal interphalangeal, metatarsophalangeal, tarsometatarsal, centrodistal, and tarsocrural joints and the DFTS. Local infiltration of local anesthetic solution around areas of possible damage, such as periosteal reactions and ligament insertions, may be helpful in horses with specific injuries.

IMAGING CONSIDERATIONS

Many causes of lameness in the driving horse, especially in the older horses, are related to problems in the distal limb and the hock joints. Considerable variation in the radiological appearance of these structures can occur, representing normal anatomical variability, old injuries, incidental findings, or wear and tear. Only by localizing the site of pain to a particular joint by intra-articular analgesia is interpreting these findings possible. Standard radiographic views are obtained. Upper limb injuries are often induced by trauma, are not localized easily by diagnostic analgesia, and are difficult to examine with small x-ray machines. Ultrasonography is particularly useful in examining some of these injuries, not only to differentiate the soft tissue components (e.g., hematoma, muscle injury, edema, and wounds), but also to examine the cortical outline of the proximal limb bones in the shoulder and stifle regions for evidence of fractures or other damage. Comparison with the opposite limb is helpful in determining the normal in these areas.

Ultrasonography is essential for examining the DFTS and associated structures and for definitive diagnosis and monitoring of desmitis of the suspensory ligament (SL) or ALDDFT.

DIFFICULTIES IN DIAGNOSIS

The most difficult problem in diagnosing lameness in a driving horse is an intermittent, often low-grade lameness that may flare up with increased exercise or competition. Many lamenesses are bilateral hindlimb problems, often with several sites of pain contributing to lameness. If making progress in these horses by standard examination techniques is impossible, scintigraphic examination may be useful. The advantages of scintigraphy include the ability to cover many areas of the body (forelimbs and hindlimbs, back and pelvis) at one session,

the non-invasive nature of the technique, and its use to monitor healing. The disadvantages include cost, availability, and the lower sensitivity of the technique in localizing chronic sites of inflammation. Several sites may be detected as having increased radiopharmaceutical uptake, and other techniques, such as local analgesia, are necessary to assess clinical significance. Making a definitive diagnosis is not always possible, but it is important to treat what is diagnosable or visible and to monitor the horse's progress carefully during convalescence. A re-assessment of the horse at regular stages may cast further light on the problem. Rest may be easy to accomplish in driving horses because they have a long working life, and in pairs and teams a spare horse may be available to take the lame horse's place, allowing competition to be continued.

A problem occasionally encountered in lameness from harness and carriage use is an abnormal gait, often intermittent, seen only while the horse is working in harness, usually with the carriage. Lameness often is manifested at the collected trot in the forelimb, and an extra lift in the shoulder movement during protraction is apparent. Usually only one limb is affected. The horse appears normal at the walk and extended trot, and the problem is not exacerbated by exercise. The actual diagnosis of this condition remains obscure, but one possible theory revolves around the effect of the neck collar on the action of the scapula while the animal is in harness: a mechanical interference. In some horses, changing from a neck collar to breast band harness appears to stop the problem.

SHOEING

Most driving horses are shod conventionally, with few special techniques or shoes. Many are shod with flat, fullered shoes set rather short and tight at the heels to minimize interference injuries to the horse and other members in the pair or team. Other reasons for such shoeing given by farriers and owners include minimizing shoe loss in the varying surfaces of the competition and preventing inadvertent removal by the wearer or another horse by standing on the shoe. Short-shoeing a horse, with a tight fit in the heel, exacerbates poor foot conformation, whether the horse has upright, boxy feet or long-toed and low, weak-heeled feet.

The use of studs and occasionally calks, especially in the hind feet, is common in an attempt to increase grip in the marathon phase of competition. In the teams, the wheelers, where the power is mainly delivered, often have studs or calks. If they have to be used, we prefer to see studs only put in for the marathon and, if possible, only for the obstacles. The extra grip studs can give may lead to ligament or joint damage, particularly in the lower limb. The use of studs or calks all the time, especially on hard tracks and roads, increases heel trauma and changes the forces transmitted up the limbs. Unilateral studs and calks are not acceptable and should be actively discouraged.

DIAGNOSIS AND MANAGEMENT OF LAMENESS

Suspensory Desmitis

Desmitis of the SL, both body and branches, is common, particularly in Amish and Mennonite carriage horses. Seventy percent of the Amish carriage horses are Standardbreds, nearly all of which have raced previously and have pre-existing chronic, healed, or healing desmitis. Forelimbs (80%) and hindlimbs (20%) are affected. Body and branch lesions occur with similar prevalence in forelimbs, but branch injuries predominate in hindlimbs.

Suspensory desmitis may cause an acute onset, moderate lameness, but often a low-grade insidious lameness is reported by the owner or driver. There is often considerable swelling and inflammation associated with the SL. Horses with branch lesions especially are positive to a lower limb flexion test. Diagnosis usually is based on clinical examination and is confirmed by ultrasonography, but in some horses local analgesia is required to confirm that the SL is the source of pain.

With branch lesions radiographic examination of the proximal sesamoid bones and splint bones is required to identify concurrent fractures or sesamoiditis, which influence prognosis. Proximal sesamoid bone fractures usually are seen in Standardbred horses that have raced previously and were retired with a fracture that becomes apparent after a second career as an Amish driving horse. Amish and Mennonite horses with a less than good prognosis may be culled.

Rest is essential. The duration is determined by improvement in lameness, reduced sensitivity on palpation, and improvement in fiber pattern assessed by ultrasonography. In the initial, acute stages, cold hosing, icing for a few days, and support wrapping are recommended. The combined oral use of dexamethasone and a diuretic is helpful in decreasing inflammation and filling without masking pain. It is important to provide appropriate palmar support, and if necessary, an egg bar shoe is used to increase the weight-bearing surface.

In horses with the most severe injuries, and in those where economics does not play a role in selection of treatment, intralesional β-aminoproprionitrile fumarate injections or percutaneous ligament splitting can be performed. We currently have insufficient data to determine whether intralesional β-aminoproprionitrile fumarate improves the prognosis over conventional therapies and surgery and the drug has been taken off the market. Very good results have been obtained with surgical treatment of horses with large core lesions in the body of the SL. After perineural analgesia and localization of the core lesion by ultrasonography, we use a sharpened teat bistoury to enter the core lesion with the limb bearing weight.

Some clients prefer to turn out a horse with suspensory desmitis rather than adhere to a structured rehabilitation. Our impression is that lesions heal more slowly, often with less acceptable cosmetic results, compared with horses in which controlled exercise is used. Horses with branch injuries are treated conservatively with a long, slow progressive exercise schedule, with periodic ultrasonographic monitoring. Therapeutic ultrasound treatment also is recommended.

Prevention of suspensory desmitis is primarily by selecting well-conformed horses with a good hoof-pastern axis and good feet with continual re-assessment of hoof balance, the adequacy of heel support, and the angle of the pastern relative to the limb conformation.

Foot Problems

Foot lameness is common in driving horses and is traumatic, degenerative, or inflammatory. Lameness varies from simple trauma (puncture wounds and bruises) to infectious conditions such as thrush, degenerative conditions such as navicular syndrome, and a host of lesions caused or affected by quality of shoeing.

Carriage horses receive a moderate amount of direct trauma resulting in wounds of the digit. The lack of available prepared and manicured training surfaces often necessitates fitness work on gravel roads and through streams and wooded areas, and solar punctures by stumps or sharp objects are not uncommon. Injuries to the coronary region of horses in pairs and four-in-hand occur when driving conditions are difficult, as in the hazards, when one or more horses may be stepped on by an adjacent horse.

Although wounds of the coronary band are generally obvious, a puncture of the sole may not be readily apparent. A horse with a puncture may or may not be lame at the time

of injury. The excitement of competition allows horses to continue without overt signs of discomfort, even with frank injury to the sole. Once back in the stable or sometimes the following day, lameness becomes apparent. Many wounds of the sole and frog are difficult to see, and they can be missed easily. Frog and sole tissue tends to close over the entry site, particularly if the offending object was sharp. If foreign material remains in the foot, severe lameness is usually present. Most horses respond well to curettage and soaking in antiseptic solutions, but protracted lameness is usually a sign of infection. Contrast radiography is useful to detect direction and depth of a tract. When a veterinarian cannot determine if vital structures are involved, surgical exploration may be indicated.

Bruises and corns are diagnosed by localization of pain to the sole or heel, history, ruling out other causes of foot lameness, and identification of solar discoloration. Treatment for bruising is directed at protecting the sole. In the case of susceptible horses with a chronic tendency toward bruising, foot soaks or preferably packing gel with dimethylsulfoxide with an overwrap frequently are indicated for flare-ups. Horses with corns, in our experience, require changes in shoeing to relieve pressure on the heel and constant monitoring of the heel growth to discourage underrun, rolled in heels.

Synovitis of the distal interphalangeal joint occurs as an acute or, less commonly, as a recurrent condition. Effusion of the distal interphalangeal joint and lameness generally are exacerbated by distal limb flexion. Diagnosis is confirmed by intra-articular analgesia. Treatment comprises phenylbutazone (2 g sid) or flunixin meglumine (500 mg sid) for several days, icing of the digit, and intramuscular administration of a polysulfated glycosaminoglycan (PSGAG), with or without intra-articular administration of hyaluronan or corticosteroids.

Navicular syndrome may be more prevalent in carriage horses of certain breeds. The European Warmbloods, particularly the heavier horses, appear to be at risk. Navicular syndrome is nearly non-existent in driving ponies. Horses with navicular syndrome have an insidious lameness that usually resolves with palmar digital analgesia. Occasionally a horse has sudden onset of moderate to severe lameness without any history of a chronic problem. These horses may have dramatic lesions apparent radiographically without a reasonable explanation for why the lameness occurred suddenly rather than gradually. A critical evaluation of hoof balance is important, and we recommend using bar or egg bar shoes and oral isoxsuprine (400 mg bid). Horses with more pronounced lameness are also treated with phenylbutazone (1 to 2 gm PO sid).

The greatest number of foot lamenesses seen in carriage horses fit into a broad category of palmar heel pain. Lengthy drives on hard surfaces result in chronic contusions to the hoof capsule and subsequent lameness. Palmar heel pain often is correlated with the degree of work the horse is undertaking currently and may occur seasonally in susceptible individuals. Lameness varies from slight to moderate and is exacerbated by circling on hard ground with the affected leg on the inside of the circle. Hoof tester examination may reveal pain from the medial or lateral sulcus across to that quarter, but a large number of horses have no reaction. Lameness is eliminated by perineural analgesia of the palmar digital nerves. Intra-articular analgesia of the distal interphalangeal joint and intrathecal analgesia of the navicular bursa ideally should be performed to rule out these structures as sources of pain. Radiographic examination usually is unrewarding.

Assessment of hoof balance and shoeing is necessary, and until any imbalance has been corrected, recommending other therapies is pointless. We have had good results from increasing the length of support or ground surface in the dorsal/palmar direction of the hoof, without any other therapy. Many horse owners do not understand when instructed to move the weight bearing surface farther palmarly, and they simply raise the heel. Talking directly with the farrier and providing explanatory diagrams to the client is worthwhile. If the heels are too weak or inadequate to allow for corrective trimming, using egg bar shoes until heel growth is adequate may be necessary.

Distal Hock Joint Pain

Osteoarthritis of the distal tarsal joints is common, particularly in older and larger horses (>10 years old). Many of these horses are not presented by the owner or driver as overtly lame but with a history referring to lack of performance or action, stiffness, back problems, or poor bending. Some horses with osteoarthritis, especially from teams, are not identified until veterinary inspections at events. Many older driving horses, particularly those in horse driving trials at a high level, have an uneven hindlimb gait, especially when trotted in hand, and have positive upper limb flexion tests. The horse often is said to warm or work into its work (warm out of lameness), and owners do not request investigation. A high percentage of these horses have low-grade osteoarthritis of the distal tarsal joints. In some horses acute lameness or gradual worsening of lameness (often described as unilateral lameness by the driver) leads to lameness investigation. In the Warmblood breeds an earlier incidence of this problem has become apparent in recent years, with horses of 5 to 8 years old showing severe osteoarthritis and even ankylosis of affected joints. Many have poorly conformed and small hocks, and in some this osteoarthritis appears to be a sequel to developmental orthopedic disease in a young, growing animal.

Diagnosis is based on clinical examination, intra-articular analgesia, and radiography. Many horses have bilateral lameness, with one side worse than the other, and evidence of gluteal muscle atrophy. Poor hock conformation (sickle-hocked, straight, or cow-hocked) predisposes some horses to this condition. Shoe and hoof wear may indicate toe dragging or abnormal lateral breakover. Swelling may be palpable or visible at the seat of spavin. Tarsocrural joint distention is a common incidental finding, but it may indicate proximal intertarsal (talocalcaneal-centroquatral) joint involvement. The gait often is characterized by reduced foot flight arc and hock flexion, leading to toe dragging, and adduction of the limb medially underneath the body to land and then breakover laterally. On the lunge, lameness of the inside hindlimb may be worse, with shortening of the cranial phase of the stride and a tendency to fall toward the handler. Bilateral lameness is common, although one leg is often worse, leading to a choppy or stilted gait.

Flexion tests of the upper hindlimb are often positive bilaterally, but the response varies enormously depending on the stage and extent of the disease and the individual horse. Positive flexion tests in the hindlimb of older driving horses are common, even in the absence of lameness.

Initial localization of the lameness to the hock region can be accomplished by perineural analgesia, although intra-articular anesthesia is more specific Previously the centrodistal and tarsometatarsal joints were blocked separately, but more recently, only the tarsometatarsal is blocked, on the basis of recent research confirming the high rate of diffusion of mepivacaine between the two joints.

A poor correlation exists between the degree of lameness and the extent of radiological abnormalities. Scintigraphic examination may be indicated in the absence of radiological abnormalities.

Treatment depends on a variety of factors including the degree of lameness, other concurrent causes of lameness (e.g., back problems), the type and extent of radiological abnormalities, the age of the animal, the level of work, the competition undertaken, the time and cost constraints, and the response to previous treatment. Conservative treatments often are used when financial constraints exist, when lameness

is mild, when radiographically the disease is advanced, or simply to assess the effects of treatment before considering more radical therapy. Surgical treatments involve the willingness of the owner to make the financial investment and subject the horse to the risk of general anesthesia. We usually assess all aspects on an individual basis before embarking on a standard treatment plan, involving three monthly clinical and radiographic examinations.

In general, because of the low-grade and chronic nature of the complaint in driving horses, the horse initially is treated conservatively with corrective shoeing (graduated toe heel shoe, rolled at the toe, with or without lateral extension), a controlled graduated exercise program (up to 90 minutes) placing an emphasis on walking and trotting in a straight line (ridden or driven), and oral medication with variable levels of NSAIDs (usually phenylbutazone) to control pain and encourage a more normal action. Many driving horses make considerable clinical progress with this regimen, but few high-level driving trial horses are able to return to competition quickly. Healing may take up to 18 months, and in some healing never happens using conservative treatments. Predicting the course of the disease and ultimate result in any one horse is difficult early on and requires consideration of all the clinical and radiographic findings. Many horses improve, but they do not become sound, and have minimal progression of radiological abnormalities over 6 to 12 months. Horses with more obvious lameness, or those that are unable to cope with exercise with orally administered phenylbutazone, may be candidates for intra-articular injections with long-acting corticosteroid preparations (40 mg methylprednisolone acetate per joint). This may result in improvement for 3 to 6 months, and repeated injection usually is required. The use of NSAIDs or corticosteroids is not allowed in official competitions, and drugs must be withdrawn according to the manufacturers' recommendations before any competition.

Arthrodesis is used in horses that fail to respond to conservative treatment, are too painful to achieve ankylosis by exercise, and where time to return to performance soundness is important. In horses with minimal radiographic changes, intra-articular injection of sodium monoiodoacetate is performed under general anesthesia. In horses with more advanced radiological abnormalities or those in which monoiodoacetate has failed to achieve ankylosis, surgical treatment using three drill holes is used in each joint. Controlled exercise postoperatively is essential for success with either treatment. Although many driving horses with osteoarthritis of the distal tarsal joints are useable with treatment, complete resolution of lameness is unusual.

Exertional Rhabdomyolysis

Carriage horses have isolated episodes of tying up during competition or strenuous exercise, and this is a true exertional rhabdomyolysis. Competition horses may develop mild signs of muscle stiffness affecting the hindquarters, during or at the conclusion of an exertional session, such as the marathon. The condition may become apparent in the 10-minute box, during the formal veterinary examination section, or before the marathon course. Clinical signs are invariably subtle and may be interpreted as fatigue by an inexperienced competitor. Affected horses should be withdrawn from the competition to avoid further skeletal muscle damage. Treatment should include administration of anti-inflammatory drugs and low doses of acepromazine (5 to 25 mg total dose) and insuring adequate hydration. Horses should not be walked out of the stiffness and should be moved by trailer from the 10-minute box to a treatment stall.

To prevent further episodes, the feeding up to and including the competition and the horse's fitness program should be evaluated and amended as necessary. Teaching drivers how to assess the horse's fitness by measurement of temperature and pulse and respiratory rates on the stress days of training sessions is particularly valuable.

Acute, severe rhabdomyolysis (tying up) also occurs in horses driven by the Amish and Mennonites. These are frequently emergency situations. Horses have true, severe exertional rhabdomyolysis and are often in recumbency when examined. The clinical signs in non-recumbent horses are extreme muscle stiffness and swelling over the topline and gluteal muscle groups, increased heart rate, sweating, and colic-like symptoms. The history is consistent: the horse becomes stiff while being driven, but the driver needed to continue to a destination and may have been driven another 1 to 15 miles. The condition in colloquial Amish terminology is referred to as *kidney shot*.

Rehydration and controlling pain and shock are imperative. Tranquilizers are particularly helpful, and measurement of packed cell volume and total protein concentration is useful for monitoring hydration. Measurement of creatine kinase may be useful prognostically in recumbent horses, although the initial response to treatment and the ability to get the animal on its feet are primary factors in determining whether the horse can be saved. Creatine kinase concentrations are often 400,000 to 800,000 IU/L (normal 130 to 400 IU/L), and we have treated horses successfully with levels up to 600,000 IU/L. Without prompt emergency attention, nearly all of the more severely affected horses die or are ultimately humanely destroyed. Most horses that remain recumbent after 24 hours are lost.

Horses are viewed by Amish farmers as utilitarian and necessary for transportation, and prevention of fatal exertional rhabdomyolysis is not necessarily easy. Owners should be advised of the need to match exercise with the intake of concentrates. If possible, the horse's entire body can be clipped if the onset of summer or warm weather is sudden or the winter coat is still present. Clipping can be done using a gas-powered generator for the clippers. Clipped horses have less risk of severe dehydration, and a clipped horse apparently is less prone to rhabdomyolysis than a hirsute horse.

Tenosynovitis of the Digital Flexor Tendon Sheath

Tenosynovitis of the DFTS is a common cause of acute hindlimb lameness. Often extreme swelling of the DFTS occurs and is turgid and firm, which contrasts to chronic, benign distention of the DFTS in which fluid in an overly stretched sheath can be balloted from side to side.

With horses with acute tenosynovitis there is a substantial response to lower limb flexion, and in some horses (5%) a non–weight-bearing lameness develops. Using local analgesia is unnecessary, but intrathecal analgesia using 6 to 10 ml of mepivacaine can be used to confirm the source of pain. Synoviocentesis is best performed distal to the fetlock and is facilitated by applying a disposable elastic bandage around the distal metatarsal region to push the fluid into the distal projections of the sheath on the plantar aspect of the pastern (Fig. 125-1). Once the sheath has been entered, an assistant cuts off the bandage, and local anesthetic solution is injected.

Radiography is usually negative, but it is necessary to be certain that no unexpected lesion is missed. Ultrasonographic examination is essential to determine if tenosynovitis is associated with a lesion of the deep digital flexor tendon, which requires a longer convalescence and horses have a more guarded prognosis than those with primary tenosynovitis.

Treatment is based on reducing inflammation and controlling exercise. Uncontrolled turnout results in prolonged healing, and stall rest followed by walking in hand is preferable. A horse with an acute injury is treated with icing for 40 minutes twice a day, for 2 to 3 days, and orally administered NSAIDs. Intrathecally administered hyaluronan is beneficial. Caution should

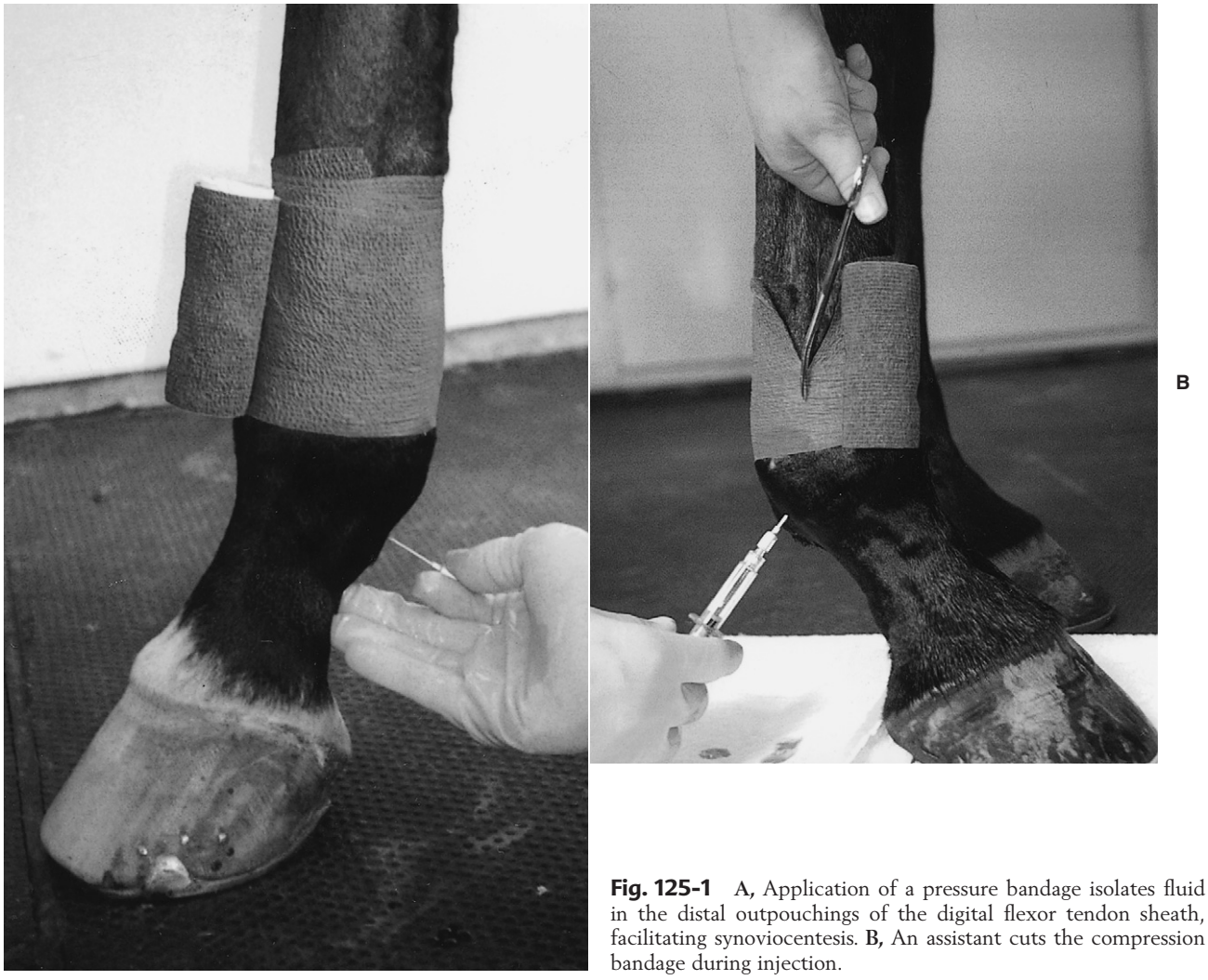


Fig. 125-1 **A,** Application of a pressure bandage isolates fluid in the distal outpouchings of the digital flexor tendon sheath, facilitating synoviocentesis. **B,** An assistant cuts the compression bandage during injection.

be exercised in the use of corticosteroids, which may provide temporary relief and often give the client a false sense of security regarding the rate of healing. Isoflupredone acetate is recommended, is short-acting, and may control the acute flare-up without any long-term affect that makes monitoring progress difficult. Methylprednisolone acetate lasts at least twice as long and may predispose the horse to develop dystrophic mineralization in the sheath. We have had better long-term results with conservative medical management than with surgical treatment by desmotomy of the plantar annular ligament.

Prevention of tenosynovitis and tears of the deep digital flexor tendon is difficult, because most injuries are acute and unforeseeable, occurring during strenuous exercise. A team horse previously used as a wheeler may be better placed as a leader. The foot should be kept well balanced, with a full-fitting shoe providing adequate plantar support.

Chronic Osteoarthritis of the Lower Limb Joints

Many driving horses develop chronic osteoarthritis of the distal interphalangeal, proximal interphalangeal, and metacarpophalangeal joints as a result of wear and tear because of the work they undertake, the length of time they are used, and other factors such as conformation, foot shape, and shoeing. Radiographic examination of these joints, especially in the hindlimbs and in larger animals, often reveals a variety of intra-articular and peri-articular abnormalities that may be associated with lameness. Older driving horses often live with low-grade hindlimb gait abnormalities associated with low-grade joint pain. This low-grade, chronic, and often multiple leg or multiple joint pain can be exacerbated by excessive or different exercise regimens, leading to acute flare-ups, increased lameness (often unilateral), joint flexion pain, peri-articular heat, and joint swelling. Radiographs obtained at this time reveal little more than the pre-existing abnormalities. Intra-articular analgesia may be essential to localize lameness, but multiple limb and joint involvement can make localization difficult. Nuclear scintigraphy may be helpful, but it is expensive, and often is used only after early treatment has proved unsuccessful.

Treatment of horses with acute flare-ups of osteoarthritis involves cold therapy such as cold hosing or ice packs, compression bandaging, systemic NSAIDs, topical applications of various anti-inflammatory substances and intra-articular or systemic medications such as corticosteroids, hyaluronan, and PSGAGs. Rest and a controlled, graduated return to exercise are important. Management of horses with chronic osteoarthritis involves a careful review and possible modification of the horse's work program, judicious use of NSAIDs, physiotherapy techniques such as laser or ultrasound treatment, and systemic medication with hyaluronan or PSGAGs. The prognosis varies enormously depending on the severity and extent of the problem, the way in which the individual horse responds to

pain and the level of work required to keep the horse sound and competing. In many horses, if the hindlimbs are affected and especially if the condition is bilateral and the horse is part of a pair or team, then the chronic low-level lameness is not observed, is undiagnosed, or is ignored. An acute flare-up may lead to recognition of the problem and diagnosis. Prognosis for horses with the first acute flare-up is reasonable, but those with further occurrences warrant a more guarded prognosis.

Stifle Lameness

A definitive diagnosis of the cause of stifle pain in driving horses is often difficult to determine. Most horses with stifle lameness are wheelers of four-in-hand or one of a pair. It is generally accepted that wheelers supply 60% of the workload in pulling, which may affect the incidence of stifle lameness. Lameness is usually acute in onset and unilateral, with no stifle effusion or other localizing clinical signs. Stifle flexion may cause more obvious accentuation of lameness than other hindlimb flexion tests. Lameness is improved by intra-articular analgesia.

Radiographs uniformly are negative. Ultrasonographic examination may sometimes reveal a lesion, but in more than 80% of horses no cause of pain can be identified. Arthroscopic exploration may be warranted, but this is expensive, and only a limited part of the stifle can be assessed. The prognosis generally is guarded for horses with a known meniscal or cruciate ligament injury and for those without a specific diagnosis. The best results have been achieved after intra-articular treatment with hyaluronan, combined with systemic administration of PSGAG and rest or rigidly controlled exercise, walking in hand.

We recommend that horses should not resume work until 60 days after complete resolution of lameness *and* when they do not respond to flexion of the stifle.

Direct Trauma

Traumatic injuries to the limbs and body of the driving animal are common.

Interference Injuries

Distal limb lacerations of varying severity are common, especially in horses that are part of a team, particularly in combined driving, and are caused by over-reaching (forelimbs) or by interference from other horses. The most common lesions involve the heels and coronary band (Fig. 125-2), although wounds to the mid-pastern and distal palmar (plantar) aspect of the metacarpal (metatarsal) region occur frequently. Many wounds are superficial and consist only of contaminated skin, but it is essential to check for injury to the flexor tendons and the DFTSs, joint integrity, and damage to the coronary band. The horse should be restrained properly, the affected area clipped and vigorously cleaned, and the damage assessed before medical or surgical treatment is initiated. In some horses detailed examination using digital palpation with sterile gloves, radiography, ultrasonography, and even surgical exploration may be necessary. Early and aggressive treatment may improve the prognosis considerably in the short and long term.

Injuries to the Brisket, Lower Neck, Antebrachium, Stifle, and Crus

Injuries usually occur during the marathon phase of driving trials, especially in the obstacles, or after runaways and accidents in any driven animal. The tendency of some drivers in combined driving in recent years to drive the carriage as a battering ram through the obstacles has increased the incidence of these injuries. Skin lesions occur, especially in the brisket and lower neck and upper forearm regions, and can range from full-thickness lacerations to mild hair loss and deeper bruising (Fig. 125-3). In some horses little is visible externally except mild swelling to suggest the site of impact. Stiffness or lameness often develops several hours later. Severe injuries, particularly involving broken shafts or obstacles, can lead to life-threatening injuries to the chest, abdomen, or vital vascular structures.

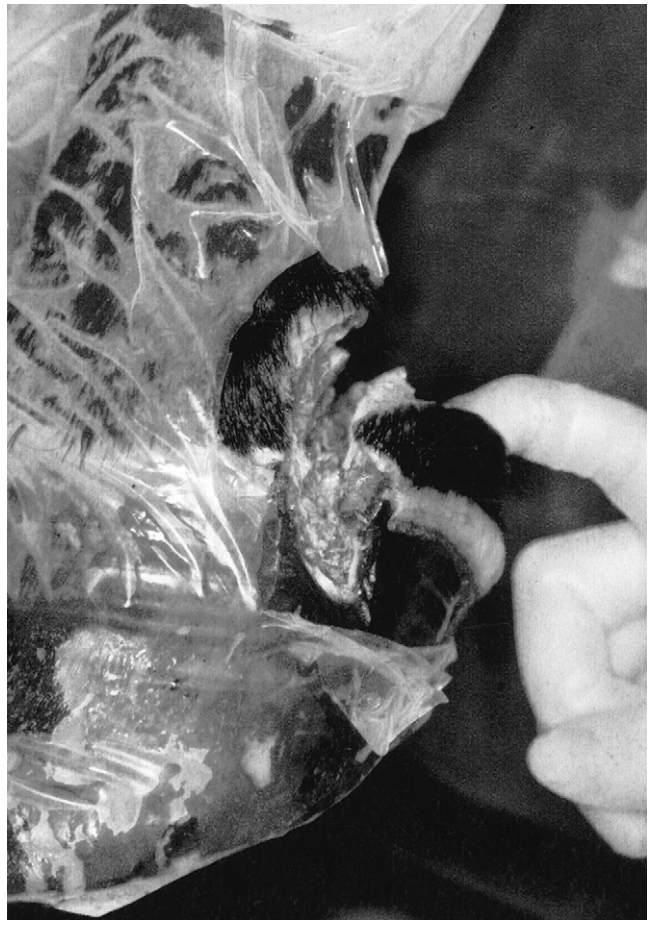


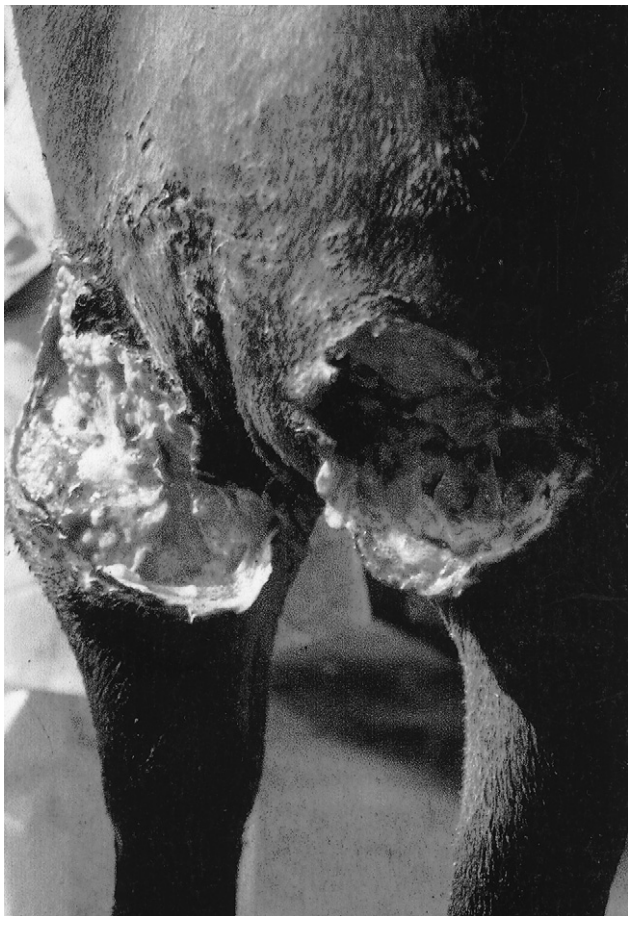
Fig. 125-2 A severe heel bulb laceration sustained by a driving horse during a team competition, probably caused by interference from another horse.

The injured area should be examined carefully, particularly when lacerations are present. In these, thorough lavage and cleaning, followed by digital exploration using sterile gloves to assess for additional deeper damage is essential. Surgical repair may be necessary. Horses with blunt soft tissue damage and bruising benefit from light walking in hand, cold therapy, topical and systemic anti-inflammatory medication, and physiotherapy techniques.

Desmitis of the Accessory Ligament of the Deep Digital Flexor Tendon

Desmitis of the ALDDFT is a common cause of forelimb lameness. Older horses, including ponies, are more likely to be affected, and injury can occur at exercise or during turnout. Onset is usually acute, with unilateral forelimb lameness occurring during or immediately after exercise. Swelling of the ALDDFT occurs with edematous swelling of the surrounding soft tissues. In horses with severe desmitis, periligamentous hemorrhage can occur. Palpable pain is rare, but heat usually is apparent.

Ultrasonographic examination is used to determine the extent of injury. Diffuse lesions are more common than focal core lesions. Damage to other tendon structures is uncommon, but adhesions to the SDFT or deep digital flexor tendon do occur, particularly in horses with chronic recurrent injuries.



Treatment of horses with acute injuries includes cold therapy, compression bandaging, box rest, and topical or systemic anti-inflammatory medications. We regularly recommend the use of systemic PSGAG therapy and therapeutic ultrasound physiotherapy from 7 days after injury. Graduated walking in hand is encouraged from 7 days after injury, increasing in duration up until 12 weeks, when limited free exercise is allowed. Ultrasonography is essential to determine when healing is sufficient to allow exercise to begin. A total convalescent period of 6 to 15 months may be required. Some injuries, particularly in older horses, do not heal satisfactorily. Occasionally horses with chronic injuries with adhesions have been treated by desmotomy of the ALDDFT, but the prognosis for return to full soundness is guarded.

Superficial Digital Flexor Tendonitis

Superficial digital flexor tendonitis is not common in the driving horse, although the condition is seen occasionally as a flare-up of a lesion sustained previously in another discipline, or in wheelers when the going during the marathon phase has been soft and deep. Most lesions are in the distal half of the metacarpal region and are generally mild. Diagnosis is based on clinical and ultrasonographic examinations. The prognosis for return to full work as a driving horse is generally good, because most of the injuries are mild and the type of work to be undertaken is usually slow.

Fig. 125-3 A severe traumatic injury to the upper forelimb and brisket regions caused by a collision with an obstacle in the marathon.

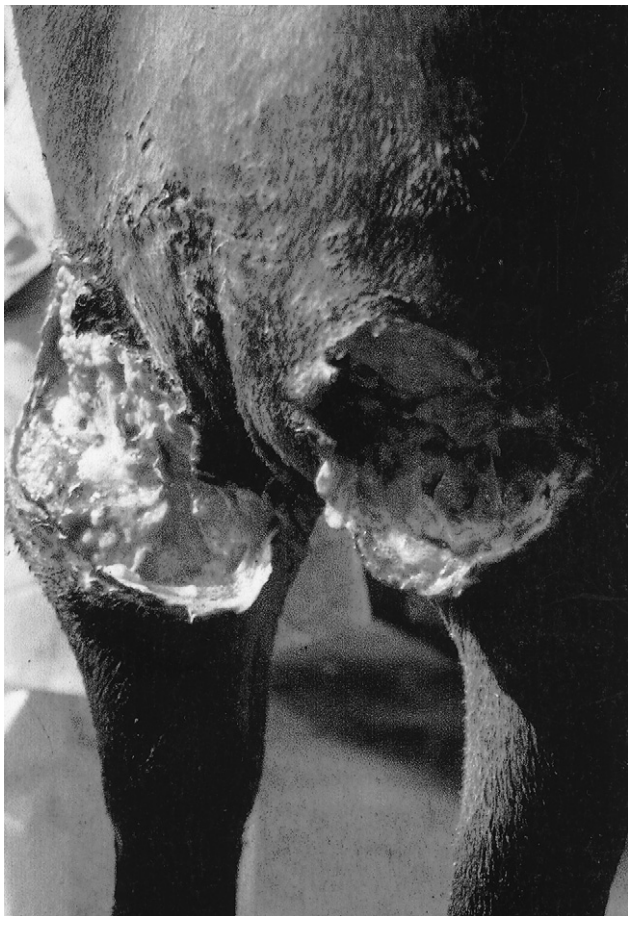
CHAPTER • 126

Lameness in Draft Horses

Dallas O. Goble

The Draft horse today enjoys a position in the world far different from when it was considered primarily a work animal. Draft horses supplied power for farming, transportation of commodities, lumbering, road building, and all such tasks until the 1930s, when the gasoline engine essentially replaced the horse. Isolated areas still continued to use the Draft horse for farming and some lumbering activities until the early 1940s. After this period, Draft horses were used primarily for specialty areas such as movie production, theme parks, parades, and frontier celebrations. But in some communities, religion and family tradition mandated the continual use of Draft horses as a power source for farming. During the 1950s and 1960s, Draft horses decreased in numbers in the United States and subsequently, the genetic pool was decreased. Draft horses started gaining popularity in the late 1970s and early 1980s and have once again become a popular member of the horse industry.

Draft horses today are different from those of the early 1900s. They perform different functions and are owned for a different purpose. A Draft horse is far more likely to be simply a hobby than an economic part of the family income. Many horses are used strictly for show purposes in classes such as halter, fine harness, and equitation, or for parades and advertising. Draft horses are used for trail riding, pleasure riding, fox hunting (often crossbred with Thoroughbreds), and other pleasure use. In some mountain areas, where selective lumbering of individual trees is undertaken, Draft horses are still used today. With a resurgence of interest in mules, Draft horse mares often are used in the breeding programs. Draft horses frequently are crossed with Thoroughbreds to change the genetic pool for the breeding of large hunter, jumper, and Three Day Event horses. Pulling contests are popular in certain areas and represent an additional use of the modern-day Draft horse.



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MODERN-DAY DRAFT HORSES

The most important physical change of the modern-day Draft horse is its size. In many modern breeding operations, size is the main criterion used for selection. When Draft horses were used predominantly as working horses, the average weight was 680 to 775 kg (1500 to 1700 lb) and the average height was 1.6 to 1.7 m (15.5 to 17 hands). This size was optimal for the multipurpose activities these horses performed on most farms and ranches. They pulled the plow, mowed and racked hay, helped round up cattle, and often took the family to church on Sunday. Draft horses today are more frequently in the range of 820 to 1000 kg (1800 to 2200 lb) and measure 1.8 to 2 m (17.5 to 19.5 hands) in height. As a result of selection based on size as the dominant characteristic, conformation and quality have suffered in some respects. Distribution of lameness also has changed. Foot size and quality have not increased proportionately with body size and weight. Osteoarthritis is common in Draft horses and likely is related to body size rather than the use of the horse. Hybrid vigor, once thought to be advantageous, has decreased by using the practice of line breeding for selected traits. The large size of a Draft horse sometimes misleads one to think the horse can withstand greater stress and disease than can light horses, but I have not found this to be true. Draft horses may not recover as well as light horses with the same injury or disease. Because Draft horses have a tendency to be stoic, recognizing a severe problem early in the disease process may be difficult. This tendency may cause a costly delay in diagnosis and management. The physical size of Draft horses today has hindered veterinary care and treatment. Veterinarians tend to be unsure of drug dosage and appropriate treatment schedules because of the large body size. Many hobby horse owners do not have facilities adequate to handle a 1000-kg horse and may not be knowledgeable in Draft horse care. Draft horses are not always well trained or easy to handle, and owners are sometimes unable to lend assistance when needed. These facts have curtailed the interest of many practicing veterinarians who have difficulty rationalizing being stepped on by a l000-kg Draft horse as fun. In addition, finding quality foot care for a Draft horse is sometimes difficult, because most farriers are experienced with light horses. Farriers feel similar to veterinarians: "Why hold up a 1000-kg horse twice as long as needed to hold up a 500-kg horse?" In addition, farriers who shoe Draft horses have to stock nails, shoes, and bar stock often on special order or low-volume items, a fact that dramatically increases overhead expenses. Thankfully, some veterinarians and farriers are willing to treat and specialize in Draft horse care.

The following discussions related to Draft horse lameness are my personal clinical experiences and do not necessarily reflect what may be in the equine literature. Lameness distribution may vary from my observations depending on the use, location, individual draft breed studied, or other factors associated with different populations of horses.

TEN MOST COMMON LAMENESS PROBLEMS

Over the last several years I have evaluated 745 Draft horses admitted with a chief complaint of lameness. The following list shows the top 10 lameness conditions that I have observed in order of decreasing frequency (33 horses had other problems):

1. Foot lameness (abscess, hoof cracks, laminitis, and sidebone) 260
2. Tarsal lameness (osteoarthritis, bog spavin, and osteochondrosis) 207
3. Splints 84
4. Tendonitis and suspensory desmitis 45
5. Osteoarthritis of the distal interphalangeal or proximal interphalangeal joints 44
6. Fetlock lameness (sesamoiditis, osteoarthritis, and osteochondrosis) 23
7. Thoroughpin 18
8. Carpal lameness (traumatic or infectious carpitis) 13
9. Stifle lameness (traumatic, upper fixation of the patella, and osteochondrosis) 10
10. Myopathy 8

LAMENESS EXAMINATION

Diagnosis and management of lameness in a Draft horse may seem more intimidating simply because of the large size of the horse and the infrequency with which requests are made for examination compared with light horses. In reality, Draft horses suffer from the same problems as do light horses, although the distribution is different, and the principles of diagnosis and management are the same. The lameness examination is the same as in light horses, and any deficiency of hands-on experience can be overcome by a systematic and thorough examination. Palpation of peripheral nerves (palmar and plantar digital nerve, in particular) can be difficult because of thick skin and hair, and veterinarians are sometimes reluctant to attempt perineural or intra-articular blocks. The anatomy is the same, but the ability to palpate the nerves is diminished by these factors and also by subcutaneous thickening that some Draft horses develop in the lower limb.

Draft horse lameness diagnosis and management lagged behind that of light horses for many years. First, as long as the horse could still accomplish farm work, less concern was shown for a horse that limped slightly. Possibly the person behind the plow or cultivator also limped and accepted it as part of doing the job. Second, economic considerations were a major factor in the farming operation. This does not reflect necessarily a lack of care, but it was simply an accepted part of working in that day and time. But today, Draft horses are afforded the same concerns and care given to the light horse, and only modification of most treatment protocols needs to be made.

Detailed description of the lameness examination can be found in earlier chapters. Special attention should be paid to several critical points, however. Draft horses are more stoical than light horses and, as a result, lameness may be advanced when first recognized. For example, Draft horses with osteoarthritis of the proximal interphalangeal or distal interphalangeal joints may have severe radiographic changes, but the owner may report that the horse only recently showed signs of lameness. Granted, many owners are inexperienced, but even the experienced owner may not recognize a problem until it is well advanced. This can be explained partially by the fact that a Draft horse often is used at a gait (walk) that makes lameness less obvious, and the horse frequently is hitched with one to seven additional horses, and an individual horse's problems are less discernible in a group.

When possible, observing the horse at a walk and trot on soft and hard surfaces is useful. Hoof tester examination, in my experience, is less reliable in Draft horses than in light horses. Small hoof testers are of questionable value. Even with long-handled hoof testers, it is difficult to apply enough pressure to produce a positive response in many horses. Foot lameness should be suspected if a horse shows grade 1 of 5 lameness on soft footing but grade 3 of 5 lameness on a hard surface. The examination always should include backing the horse at least two or three times and observing the horse at the walk and trot in a circle and in tight turns. Sometimes conditions such as shivers, stringhalt, or intermittent upward fixation of the patella are seen only during these maneuvers.

Lower limb flexion tests are less rewarding in Draft horses than in light horses. It is difficult to apply sufficient pressure during the flexion test to accentuate pain in this region. A Draft horse may be less willing to trot after a flexion test, making it difficult to determine a true positive response. Most light horses will trot after flexion, even when the reaction is highly positive, but a Draft horse will not trot off as readily without strong urging. In addition, Draft horses often are not accustomed to being trotted in hand routinely. Hindlimb upper limb flexion tests in a Draft horse are a more reliable indicator of hindlimb lameness than in a light horse.

I prefer to perform diagnostic nerve blocks with the horse in a standing position, rather than having an assistant elevate the limb. This is especially true when attempting the palmar digital nerve block or abaxial sesamoid block. Palpating anatomical landmarks in this area when a Draft horse is weight bearing is easier than when the limb is elevated. To maintain the limb in an elevated position or to restrain the limb in this position can be difficult. Adequate restraint usually can be achieved by the application of a nose twitch.

LAMENESS COMMON TO THE FORELIMB AND HINDLIMB

Foot

The foot is the most common source of pain causing lameness in Draft horses. A thorough and complete examination of the foot is paramount to diagnosis and management of lameness. Hoof quality and conformation have suffered in modern-day breeding selection, and as a result we tend to have large horses that are supported on feet that lack hoof size and quality. I suggest that breeders of Draft horses give strong consideration to hoof conformation when making critical selections for breeding programs.

Subsolar Abscess

The most common cause of foot lameness in a Draft horse is a subsolar abscess, a problem most frequently encountered in a forelimb. The high incidence of subsolar abscess formation in Draft horses can be related to several factors. First, obtaining consistent farrier care may be difficult in many locations, and foot care may be neglected. Second, Draft horses often have poor hoof quality and easily develop hoof cracks or severely chipped and broken hoof walls. Clydesdales often have poor hoof quality. Many Draft horses have dropped soles, predisposing them to bruising and subsolar abscess formation.

A pair of large, good quality hoof testers provides the simplest method of determining the location of the abscess. Tapping the hoof wall or sole with a hammer (or the hoof testers) can help locate the abscessed area. In horses that recently have been shod or reset, each nail should be examined. An abscess associated with a nail usually develops 5 to 11 days after shoeing. Hoof cracks causing instability of the hoof capsule can cause lameness even though the area of abscessation may have resolved. If a foreign body is lodged in the hoof (nail, glass, or other penetrating object), a fistulogram (contrast radiograph) should be performed. A subsolar abscess can become a life-threatening problem if osteitis of the distal phalanx develops or penetration of the distal interphalangeal joint, deep digital flexor tendon, or navicular bursa occurs. Proper diagnosis is mandatory; otherwise, the long-term prognosis becomes worse.

Once the abscess is located, the sole should be pared with caution, especially if the horse has a dropped sole or has a concomitant full-thickness hoof wall crack. Overzealous sole paring may result in extensive mechanical damage to laminae and loss of hoof wall strength. Adequate drainage is paramount, but removing a large amount of sole is not necessary, even when substantial undermining has occurred. In Draft horses, it is important to err toward a conservative approach, at least initially. Thorough flushing of the foot with povidone-iodine or chlorhexidine diacetate solutions should be done at least once a day for 3 days or until the drainage has stopped. The foot can be soaked in a saturated solution of magnesium sulfate for 3 to 5 days to reduce inflammation and to aid in drainage. Finding a soak boot large enough for Draft horse feet at a reasonable cost is difficult, and I have found an easy solution by using a 1 m length of truck tire inner tubing. The tubing is slipped half its length over the foot, and then up the leg, with the remaining half doubled back up the leg and secured in place by a wrap of choice (Fig. 126-1). The tube then can be filled with the soak solution. Draft horses with an uncomplicated subsolar abscess do not need to be treated with systemic antibiotics. However, if cellulitis of the coronary band and pastern region is present, the administration of antibiotics is indicated. Trimethoprim-sulfadiazine (15 mg/kg PO bid) or ceftiofur sodium (1 mg/kg IV bid or IM) is my usual choice. Judicious use of non-steroidal anti-inflammatory drugs (NSAIDs) is indicated, but theses drugs should not used for extended periods of time or at levels that may mask a more serious problem. Phenylbutazone, 4 g PO or 2 g IV, on the first day is sufficient. Thereafter, 3 g and then 1 g is given orally on the second and third days, respectively. A tetanus booster should be administered if the horse's vaccination status is not current or is unknown.

If the horse's condition is not improved in 3 days, the horse should be re-examined. Plain radiographs and positive contrast fistulography should be performed. A fistulogram is performed using contrast material administered through a Foley catheter. The foot should be held off the ground when contrast media is infused and held up for 2 minutes thereafter. The foot is then placed in a weight-bearing position, and radiographs are obtained immediately. During weight bearing, a fistulous tract

Fig. 126-1 Draft horse with rubber inner tube in place and used as a soak boot for a foot abscess.

often is closed by soft tissue compression. If deeper structures are involved (distal interphalangeal joint, deep digital flexor tendon, distal phalanx, or navicular bursa), an extensive treatment program is initiated, including bacterial cultures, surgical drainage or curettage, lavage of the affected area, and the administration of broad-spectrum intravenous antibiotics. The initial antibiotic treatment usually includes 20,000 IU/kg of potassium penicillin 4 times a day and 6.6 mg/kg of gentamicin once a day. Bacterial culture and sensitivity results may require changing the antibiotic regimen.

Hoof Wall Cracks

Forelimb hoof wall cracks were found in 67% of the 165 Draft horses examined for lameness, although not all were the primary cause of the horse's current problem. Full-thickness hoof wall cracks need to be stabilized when they cause lameness. The crack should be cleaned carefully and curetted, and normal hoof wall should be present on each side of the defect. The technique of dovetailing may provide additional support if hoof repair material is used. Dovetailing is accomplished most easily using a 1-cm (⅜-inch) round burr on an electric drill and undermining the hoof wall at about a 45° angle, leaving a shelf of hoof wall down to the white line on each side of the defect for the full length of the crack. This provides additional surface area for a stronger repair and reduces the likelihood that the repair material will come out. Care must be exercised to avoid damage to the sensitive laminae or to create bleeding during this procedure because these may predispose to abscess formation beneath the repair material. If the horse is initially unshod, a shoe with clips may be sufficient to provide support and to immobilize the defect. Radiator hose clamps and 1-cm long, No. 8 metal screws can be used to stabilize the crack. Because infection is often a problem, the radiator clamp method can be used initially, when filling the defect with repair material is contraindicated. Antibiotic-impregnated repair material has been used, but my success with this material in Draft horses has been limited, and my preferred method is a shoe and the radiator clamp/ screw combination. It is important to have at least two screws through each piece of clamp and on each side of the defect to add stability. The top clamp should be placed at the proximal limit of the crack. I generally space the clamps 1.9 cm (¾ inch) apart, and the number of clamps needed depends on the length, depth, and the amount of instability in the crack and on the size of radiator clamp used. The clamps are removed as the defect grows and replaced if broken. The clamps must be tightened carefully, because lameness from laminar pain will be worse if the clamps are too tight. This problem is corrected by adjusting the clamp with a screwdriver.

Laminitis

Laminitis in Draft horses is a serious lameness condition, and prognosis for complete resolution often is guarded to unfavorable. Regardless of the cause (grain overload, colitis, metritis, retained placenta, toxemia, and so on), once the pathophysiological process of laminitis is in motion, the end results are similar. The solution to the primary cause often is solved more easily than the secondary problem of laminitis. This is especially true in mares with retained placenta in which the retained placenta and metritis are solved easily, but secondary complications may be devastating. Many of these mares develop severe laminitis with distal displacement (sinking) of the distal phalanx.

I would like to contrast my observations of Draft horses with laminitis to similar conditions in light horses. Laminitis carries a more guarded or unfavorable prognosis in Draft horses for many reasons. Our ability to manage secondary problems is less satisfactory in Draft horses. Size, when we consider a 1000-kg as opposed to a 400- to 500-kg horse, is the most important factor. Slings are seldom big enough, and hoists or hoist support systems may not be available to lift a Draft horse safely. Locating a farrier who will work on a chronically lame Draft horse and forge therapeutic shoes on a consistent basis is often difficult. Management of Draft horses with myositis, decubital ulcers, infections, pneumonia, and other secondary complications is more difficult and costly. The owner must be informed clearly of cost, and a dedicated team (owner, farrier, and veterinarian) must be assembled to manage these complications. I generally tell clients that at least 1 year will pass before the horse's level of function can be assessed reasonably.

Classification of laminitis is confusing, and I make little attempt to classify laminitis based on chronicity or by using the Obel grading system (see Chapter 35). Regardless of classification used, prognosis for return to function is poor if lameness is severe and persists for longer than 10 days with intensive treatment. In my experience, two major differences exist between Draft horses and the light horses. First, Draft horses develop laminitis more frequently and severely in the hindlimbs. Second, Draft horses are more likely to develop distal displacement (sinking) of the distal phalanx once laminitis occurs (Fig. 126-2). This latter difference may be related to hoof quality or hoof care in general and the important role that body weight plays in causing distal displacement. In addition,

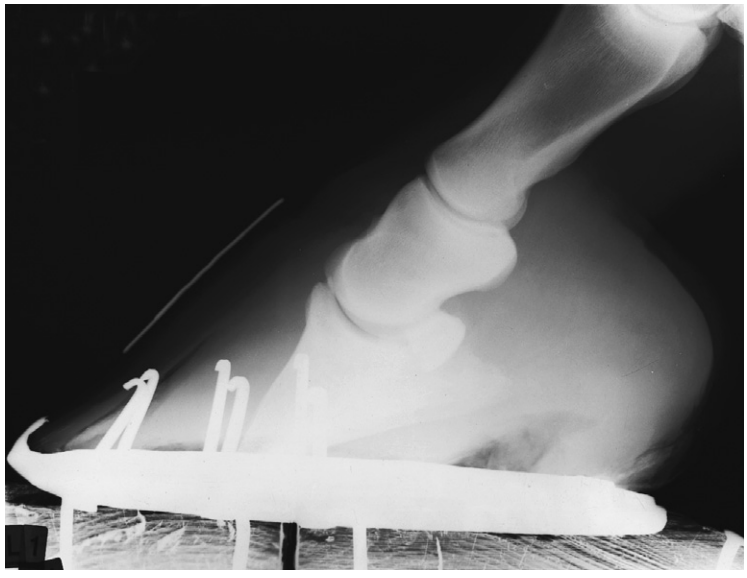

Fig. 126-2 Lateromedial radiographic view of the front foot of a Draft horse mare that had developed laminitis in all four limbs within 24 hours of foaling and retention of the placenta. The radiograph waas obtained 10 days after foaling. The proximal end of the radiopaque marker on the dorsal hoof wall is at the level of the coronary band. There is both extensive sinking and rotation of the distal phalanx. Note also the broad radiolucent line in the dorsal hoof wall, the result of laminar necrosis. The mare was managed until the foal was weaned but humane destruction was ultimately necessary.

shoeing methods that flair the hoof wall simply to give the impression of a large foot weaken laminar support. Distal displacement can occur in horses with traumatic laminitis without a traditional laminitic episode. Traumatic laminitis and sinking can occur unilaterally, only to then occur weeks to months later in the opposite foot. Traumatic laminitis caused by incorrect shoeing can be reduced greatly or eliminated when proper shoeing is provided on a regular basis.

Diagnosis of laminitis is not difficult except in Draft horses with traumatic laminitis that is slowly progressive, without an acute episode typically seen with laminitis in a light horse. These horses often have a dropped sole (including the frog in many horses), with the entire sole at a level below the hoof wall. Increased digital pulse amplitudes may be missed easily in a Draft horse, but hoof tester sensitivity and abnormal stance are important clinical signs. Initial and follow-up radiographs should always be obtained.

Management of Draft horses with laminitis is similar to that in light horses, but some precautions need to be taken. Pain amelioration is important but often difficult to achieve. Clydesdales, for instance, are susceptible to gastric and colonic ulcers when treated with phenylbutazone. The Belgian horses that I have treated with phenylbutazone seem less susceptible to ulcers than do Clydesdales. In Clydesdales I seldom if ever administer more than 4 g phenylbutazone orally daily, or do not exceed 3 g intravenously daily, unless no other alternative is available. The dose should be lowered as quickly as possible, and the high dose should be maintained for a maximum of 5 to 7 days. Although this dose is low based on milligrams per kilogram, Clydesdales have many gastrointestinal complications with higher doses. Flunixin meglumine, meclofenamic acid, and other NSAIDs can be used, but phenylbutazone is the most frequently used and cost-effective drug. Butorphanol tartrate (0.01 to 0.02 mg/kg) can be used with phenylbutazone to lower the necessary dose of NSAIDs. Butorphanol tartrate alone is not a satisfactory analgesic for Draft horses with laminitis, but it is useful with NSAIDs, and can be repeated 3 to 4 times each day.

To provide frog support initially, I use pieces of rubber stall matting, 1.9 cm (¾ inch) thick. The matting is cut to fit the foot and frog and then secured with fiberglass cast material. To increase thickness, two rubber pads can be glued together. To raise the heels in Draft horses with acute laminitis, a hand grinder can be used to make a wedge pad from the rubber mat. I believe raising the heels helps to reduce pain and rotation of the distal phalanx. Heels should be elevated 10° to 14°, however. The single most important factor in Draft horses with laminitis is to avoid excessive paring of the sole. Paring the sole away to make it look like a normal sole is putting the curse of death on Draft horses with laminitis. This practice removes natural protection from rotation or sinking, and the sole supports the foot better than anything we can apply externally.

Radical hoof wall resection should be avoided. I recall two horses that were referred after complete dorsal hoof wall resection. In both horses the remaining hoof wall lacked strength, and although one horse was shod and the other was unshod, the distal phalanx rotated and sank through the remaining hoof wall in both horses, which were euthanized. If hoof wall resection is indicated, a shoe with side or quarter clips is applied *before* the procedure is performed. The entire hoof wall should not be removed at one time, but resection should be staged over 2 to 3 weeks. If the hoof wall spreads and crowding of the side clips occurs or if further rotation or sinking of the distal phalanx is observed, any further resection is delayed. The coronary band should be assessed each day for signs of sinking (depression at the top of the coronary band). Radiography is helpful but does not replace careful physical examination. Horses with subsolar abscesses are treated by drilling small holes (3 to 5 mm) through the hoof rather than by resection of large portions of hoof wall and sole. Two or more holes may need to be placed in the hoof wall or sole to provide adequate drainage and to flush the site. Drilling small holes does not reduce hoof wall strength compared with paring or removing large portions using conventional methods.

Sidebone

Mineralization of the cartilages of the foot (sidebone) is a common radiographic finding in Draft horses, particularly in the forelimbs, but is an infrequent cause of lameness. Of 113 Draft horses with sidebones, 80 had the condition bilaterally in the forelimbs, and 28 horses had it bilaterally in the hindlimbs. Five horses had unilateral involvement. Draft horses with angular limb deformities are more likely to develop sidebone than are horses with normal bone structure. Sidebone is more common in Draft horses with poor hoof quality than in horses with normal hooves. Trauma to the heels and quarters and reduced palmar support are contributing factors. If sidebone is the cause of lameness, lameness grade is usually mild (1 to 2 of 5). Lameness is most common in horses 4 to 7 year of age. Sidebone fractures, occurring in older horses, can cause acute lameness. Lameness is usually most apparent when the horse is working on pavement or other hard surfaces. On soft surfaces the hoof can tip or angle, whereas on hard surfaces the bony column cannot move. Lameness is most obvious when the horses are working in circles or tight turns.

Lameness associated with sidebone can be difficult to confirm. Palmar or plantar digital nerve blocks usually greatly improved clinical signs, and abaxial sesamoid blocks abolish signs. Both nerve blocks provide analgesia to numerous other structures that are more frequent causes of lameness, however. Physical examination is more helpful than is diagnostic analgesia. Tapping on the upper one fourth of the hoof wall (while avoiding hitting the coronary band) with hoof testers or a hammer may elicit pain. A lateral or medial wedge test often causes lameness (see Chapter 8). Thermography is valuable in some horses, showing increased temperature in the area of the sidebone. Radiography is of limited value, because the mere presence of mineralization is not conclusive evidence for lameness diagnosis.

Treatment includes rest and hoof care. The hoof should be trimmed level. Hoof strike should be evaluated dynamically (how it strikes the ground) rather than by just viewing the hoof in a static position. Vertical and parallel grooving may allow the hoof wall to expand and may reduce pressure, but I have not found grooving highly rewarding. The horse should have stall rest or small paddock confinement for 4 to 8 weeks and NSAID therapy. The foot must be balanced properly before the horse resumes a normal exercise program. Fractures associated with large sidebones may be accompanied by osteoarthritis of the distal interphalangeal joint. Rarely the proximal interphalangeal joint is involved. Surgical management of Draft horses with sidebone fractures often is unrewarding, and I do not recommend it unless conservative management efforts have failed. Conservative management includes an extended period of stall rest (8 to 12 weeks) and then small, level paddock exercise for 6 to 8 months. Healing as shown on radiographs may require extensive time, and even then the fracture still may be evident, surrounded by proliferative exostosis. Unilateral palmar digital neurectomy also can be performed. This will provide relief in most horses unless there is concurrent osteoarthritis of the distal interphalangeal or proximal interphalangeal joints.

Quittor

Quittor is defined as a chronic infectious condition associated with one of the cartilages of the foot. A mixed bacterial infection causing chronic or recurrent drainage at or near the coronary band is most common (Fig. 126-3). Trauma is the most common cause of the condition referred to as necrosis of the

cartilage. Wire cuts and wounds incurred from large calks on the shoes when horses are working in harness or being transported are common histories. Quittor is considerably less common today than it once was.

Surgical management is the only option, because scar tissue and limited circulation preclude successful conservative management with local or systemic antibiotics. Excision of necrotic cartilage and scar tissue is best. Samples should be submitted for aerobic and anaerobic bacterial culture. Standing surgery can be performed, but I prefer to use general anesthesia, which reduces the chance of the distal interphalangeal joint being penetrated, improves the ability to provide hemostasis, and provides maximal restraint during surgery. A tourniquet at the level of the fetlock joint provides excellent surgical hemostasis, and as a precaution I always administer prophylactic broad-spectrum antibiotics before surgery. The distal interphalangeal joint occasionally is opened, and although penetration does not necessarily indicate surgical failure, an open joint alters the post-operative management protocol. If necrotic material (cartilage or scar tissue) extends distal to the coronary band, adequate distal drainage and flushing requires drilling a hole in the hoof wall.[1]

Osteitis of the Distal Phalanx

Many Draft horses have flat soles and bear significant weight on the sole, and thus are prone to sole bruising and osteitis of the distal phalanx. The condition is most prevalent in the forelimbs and often is associated with improper shoeing or lack of proper hoof care. Draft horses with osteitis of the distal phalanx may assume a stance similar to that seen with traumatic laminitis. Hoof tester examination may be helpful to differentiate these conditions. A laminitic horse is most sensitive immediately dorsal to the apex of the frog in line with the dorsal one third of the toe. With osteitis of the distal phalanx, hoof tester pain response usually is less severe and more likely involves the sole in general. Soles may be pink or red, showing indications of bruising, and lameness is worse on hard surfaces or gravel. Mildly affected horses warm out of the lameness, but in horses with severe pain lameness increases as exercise continues. Radiographs can be difficult to interpret, and a definitive diagnosis is best reached using nuclear scintigraphy, observing increased radiopharmaceutical uptake in the distal phalanx. Management includes the judicious administration of NSAIDs and corrective shoeing. The sole should be protected and minimally pared. Pads frequently are used initially but may be counterproductive in the long run, because the sole has a tendency to become soft and lose thickness. Pads are needed for the first few shoe changes but then should be removed, and the sole should be hardened. To harden the sole, a common mixture referred to as sole paint (comprised of equal parts of 7% iodine, buffered formalin, and liquid phenol) is applied. This solution is applied to the sole once each day for 3 to 5 days or until the sole becomes hard. Overzealous application can cause the sole to become too hard. Paddock rest for 45 to 60 days is given. If shorter rest periods are given, recurrence is common. Hard, frozen, or rough surfaces should be avoided.

Canker

Canker is not common but is a difficult problem to solve. Canker is proliferative pododermatitis of the frog that may extend to undermine the sole and heel bulbs. The condition can occur in one or all feet and has no predilection for forelimbs or hindlimbs. Often horses are thought to have nonresponsive thrush, but later when the problem persists, canker is diagnosed (Fig. 126-4). Canker is seen more commonly in Draft horses than in light horses, a fact that may reflect differences in hoof care and environment or simply may represent a breed predisposition. Two clinical signs that differentiate canker from thrush are a foul odor (necrotic) and the presence of granulation-like tissue that bleeds easily when manipulated (Fig. 126-5). Lameness is highly variable depending on the severity and number of feet involved. Once the superficial layer of tissue is removed, bleeding is often profuse. Creamy exudate is typical initially, especially if the owner has initiated treatment with caustic preparations.

Successful treatment of canker requires patience on the part of the owner and the veterinarian because recurrence is common. The application of a hospital plate shoe is critical,

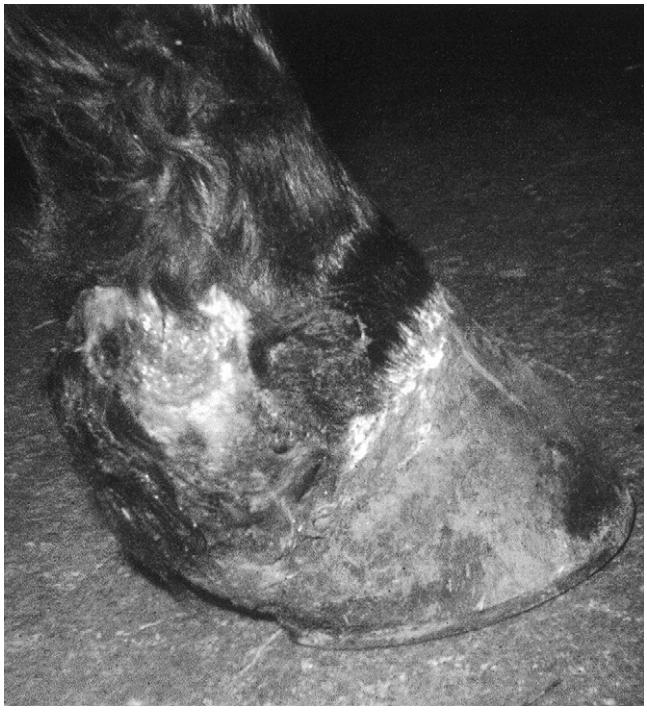

Fig. 126-3 Draft horse foot showing chronic scar and granulation tissue associated with quittor.

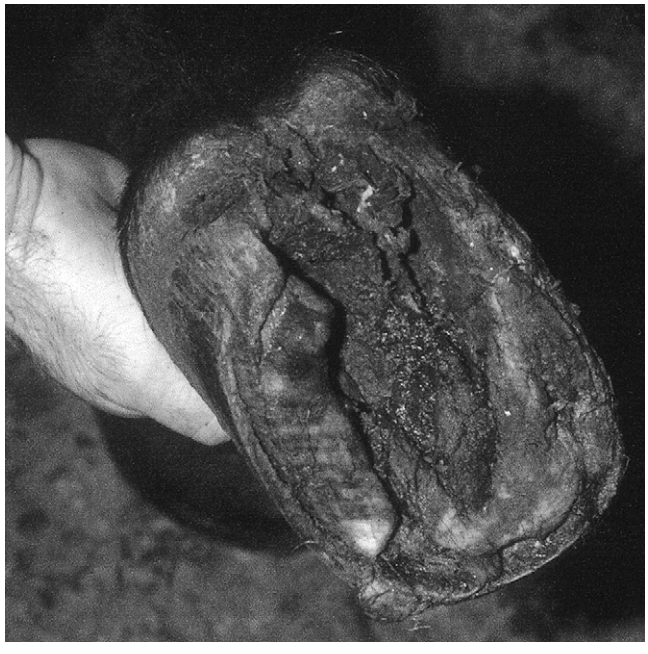

Fig. 126-4 Draft horse foot showing the typical appearance of thrush complicated by canker beneath the superficial layer of the frog.

Fig. 126-5 Chronic canker with proliferative granulation tissue. Infected granulation tissue has undermined the sole and heels.

because this shoe allows for long-term treatment and protects the sole. Importantly, pressure on the sole appears beneficial to healing, similar to that seen with granulation tissue at any site. Hospital plate shoes reduce the cost associated with daily care and management of a bandage. The shoe allows the horse to be exercised. The hospital plate shoe is made and fitted to the foot, and then the foot is blocked. A tourniquet is placed at the level of the fetlock joint, because bleeding during debridement is often profuse. Complete debridement of all abnormal proliferative tissue is often not possible and in fact may be counterproductive, because aggressive debridement may expose uninvolved deep tissue. Dry gauze sponges are packed to apply pressure on the frog and sole when the plate is replaced. Metronidazole appears to be the best topical agent, but I have used tetracycline and sulfapyridine powder successfully. The dressing should be changed daily for the first 10 to 14 days and then as needed. Debridement often needs to be repeated several times. Long-term treatment is necessary, and the owner needs to be prepared to provide it. Caustic compounds are not effective and in fact may worsen the condition. In the final stages of healing, when cornification of the frog is complete, caustic agents may be applied. Sole paint is applied to the sole for 8 to 10 days before the hospital plate shoe is removed. Horses are administered trimethoprim-sulfamethoxazole (15 mg/kg) for 3 weeks starting the day before initial debridement. Penicillin (20,000 IU/kg IM) is also effective, but oral antimicrobial agents are administered more easily.

Osteoarthritis of the Proximal and Distal Interphalangeal Joints, Ringbone

Osteoarthritis of the proximal interphalangeal or distal interphalangeal joint was diagnosed in 26 Draft horses in my series.

Ringbone has been described classically as being peri-articular or articular in nature. The prognosis for Draft horses with articular ringbone is worse than for those with the peri-articular form. Pulling horses (heavy loads) are affected most commonly, especially in the hindlimbs. Osteoarthritis of the proximal interphalangeal and distal interphalangeal joints appears to occur with similar frequency, because in 26 horses, 11 had proximal interphalangeal, 10 had distal interphalangeal, and 5 had proximal interphalangeal and distal interphalangeal involvement. Horses with osteoarthritis of the proximal interphalangeal joint remained serviceably sound for 2.6 years, whereas horses with osteoarthritis of the distal interphalangeal joint or in both joints were serviceably sound for only 11 months. Early diagnosis appears to be an important criterion for an improved prognosis, and reducing workload, instituting corrective shoeing, and providing medical management can make a substantial difference in delaying the progression of osteoarthritis.

Draft horses with short, upright pasterns are predisposed to osteoarthritis of the proximal interphalangeal and distal interphalangeal joints, and other factors such as angular limb deformities, toed-in or toed-out conformation, hoof imbalance, and trauma may play a substantial role. Horses required to work on pavement are predisposed to osteoarthritis of the proximal interphalangeal and distal interphalangeal joints. Proliferative peri-articular changes often result from lacerations or wire cuts. Horses with moderate to severe lameness have obvious enlargement of the pastern or coronary band area and heat. The degree of lameness varies from grade 1 to 5, and it worsens with lower limb flexion or after rotation of the digit. Rotation causes a substantial positive response if collateral ligaments are involved in horses with the peri-articular form. In horses with osteoarthritis of the distal interphalangeal joint, a palmar digital nerve block generally results in substantial improvement in lameness score. Thorough examination of the foot must be done to eliminate other potential causes of lameness. If the diagnosis is unclear after radiography, intra-articular analgesia should be performed. In horses with osteoarthritis of the proximal interphalangeal joint, a palmar digital nerve block may provide some improvement in lameness score, but an abaxial sesamoid block is needed to improve lameness substantially. Intra-articular analgesia can also be used.

Management depends on when the diagnosis is first made. Horses with osteoarthritis and mild lameness should be given rest in a small level paddock for 4 months. I do not use stall rest unless horses are very active, because I have had greater success allowing the horse to walk in a confined area. Hoof balance should be corrected, and the horse should be shod with flat shoes without calks, toe grabs, or borium. I recommend oral glucosamine or chondroitin sulfate supplementation or intramuscular injection of a polysulfated glycosaminoglycan. I also inject the joints with a corticosteroid and hyaluronan. Initial and follow-up radiographic examination is recommended. If the horse is sound and radiographic changes do not progress, the horse is put back into a light exercise program, whereas if radiographic changes progress, additional rest and injections are given. Many horses with osteoarthritis perform for extended periods of time without recurrence of lameness. Horses with severe osteoarthritis of the proximal interphalangeal or distal interphalangeal joints have an unfavorable prognosis for athletic use. Medical management is of little benefit, and although spontaneous fusion of the proximal interphalangeal joint may occur, giving an accurate time estimate of when this may occur is difficult. Spontaneous fusion of the distal interphalangeal joint seldom occurs. Surgical arthrodesis of the proximal interphalangeal joint can be performed by using bone plates or the three-screw technique with 5.5-mm bone screws (see Chapter 36). A half-limb cast is placed for at least 4 to 6 weeks after surgery. In Draft horses the surgical procedure is difficult compared with light horses

because the joints are large, removing articular cartilage is difficult, and horses may have difficulty with anesthetic recovery. Draft horses in general do not recover well from extended periods (>2 hour) of general anesthesia. The surgical procedure can be long, in particular if there is substantial peri-articular bone formation and fibrosis. Implants can break because of the large size of a Draft horse. Because of this, I have used an alternative technique of drilling across the joint with a 4.5- or 5.5-mm drill bit and then applying a half-limb cast without using implants. In horses with substantial peri-articular bone proliferation or fibrosis, this technique is my method of choice. Intraoperative radiographs are mandatory to assure proper placement of the drill bit within the joint. I drill at three to five sites in a fan-shape pattern from each side of the joint to destroy as much articular cartilage as possible. Breaking a drill bit in the joint is possible, and if this happens, removing the bit generally is not worth the extra time required. The palmar digital artery, vein, and nerve must be avoided during this procedure. A half-limb cast encompassing the foot is applied and maintained for 12 to 16 weeks. Advantages of this technique over those involving implants are that this technique is faster, requires shorter anesthesia periods, and requires no major skin incisions, and implant failure is not a concern. Pain control must be used to maintain reasonable comfort to avoid contralateral laminitis, which is always a concern.

Metacarpophalangeal (Fetlock) Joint Lameness

Problems associated with the fetlock joint are less frequent in Draft horses than in other breeds, and most result from direct trauma. Fetlock joint problems plague horses that perform at speed, and therefore Draft horses seldom have lameness in this region. Sesamoiditis does occur in Draft horses and usually results from suspensory desmitis and insertional injury at the level of the proximal sesamoid bone. Sesamoiditis is more common in the hindlimbs. An unusual problem of the fetlock joint in young Draft horses is osteochondritis dissecans.

Splints

Exostoses of the second and fourth metacarpal or metatarsal bones (splints) most commonly affect the second metacarpal bone, similar to light horses. In 84 Draft horses diagnosed with splints, 71 horses had forelimb involvement. Lameness associated with true splint (tearing of the interosseous ligament) is not common in Draft horses. Lameness caused by splints is most prominent in the first 2 to 3 weeks after the condition is first recognized and usually resolves with 6 to 8 weeks of rest. Because Draft horses do not perform at speed or change direction quickly, splints do not cause long-term lameness. Splints can result from interference injury from the contralateral limb, a problem that usually is corrected by proper shoeing and trimming. Local infiltration of local anesthetic solution in the region of the splint may be performed if the diagnosis is in question. Although a splint may be painful during palpation, missing a more distal limb problem is easy; therefore diagnostic analgesia should be performed distal to the splint before local infiltration. Radiographs should be obtained to check for a fracture.

To manage a Draft horse with lameness resulting from a splint, I administer NSAIDs for 5 to 7 days and recommend stall rest. If the condition is acute, I use cold water therapy or ice boots for 20 to 30 minutes twice daily and apply support wraps. The horse should be hand walked for 10 minutes twice daily. In horses with chronic splints, I recommend a sweat (50/50 mixture of glycerine and alcohol) for 7 to 10 days. Paddock exercise can be given for 12 hours each day. A total rest period of 60 to 90 days is usually adequate. I avoid overzealous treatment with topical or internal blisters.

Fractures of the splint bones are rare but usually involve the fourth metatarsal bone, and they often occur from kick wounds. These fractures often are comminuted and usually infected, because owners do not seek veterinary attention until 2 to 3 weeks after injury. Severe lameness often is not seen or is short lived, so the owner usually is not concerned until drainage starts. If the tarsometatarsal joint is involved and infected, the prognosis for return to original use is unfavorable. Many of these fractures heal if horses are given rest and treated with antibiotics. If the articular surface of the tarsometatarsal joint is not involved, displacement of fragments is minimal, and if the horse is not an athlete, many times this injury can be treated conservatively. Standing curettage of a draining tract and removal of sequestra can be accomplished in many patients using sedation and local analgesia. A culture is performed, and the horse is placed on appropriate antibiotics for 3 to 4 weeks, and the outcome is often satisfactory. For economic reasons this method also is chosen by some owners for horses intended for athletic use. However, I believe the problem is best treated surgically in those horses that are expected to be athletes. Surgical removal of the distal fractured segment of the fourth metacarpal bone is recommended occasionally, and horses usually have a good prognosis. If greater than 80% of the total length of the fourth metacarpal bone is removed, I use a screw to stabilize the proximal fragment. I do not like placing a screw into an area of known infection if I can avoid doing so. In those horses with substantial displacement or a fracture involving the articular surface, internal fixation to realign the fracture is indicated, if the injury is less than 3 weeks old. Fractures older than 3 weeks can be difficult to realign because callus formation occurs quickly.

Tendonitis and Suspensory Desmitis

Flexor tendonitis and suspensory desmitis are not as common in the Draft horse as one might expect. Of 45 horses examined for these soft tissue problems, the distribution of these combined soft tissue injuries between the forelimbs and hindlimbs was similar, in contrast to most sport horses. Suspensory desmitis is more common in the hindlimb and often is seen with sesamoiditis. Tendonitis and suspensory desmitis are more common in horses that pull heavy loads or weighted sleds. Clinical signs include heat, pain, swelling, and lameness, and diagnosis is usually straightforward. Horses with hindlimb proximal suspensory desmitis may have more subtle clinical signs, and diagnostic analgesia is usually necessary. In horses with early forelimb superficial digital flexor tendonitis, the anastomosing branch of the palmar nerves may be enlarged and sensitive to palpation before the tendon itself shows clinical abnormalities. Ultrasonographic examination should be performed.

Management of Draft horses with tendonitis and suspensory desmitis is similar to that in light horses. Ultrasonographic examination, rest, and a staged return to work are important. I am an advocate of percutaneous tendon or suspensory ligament splitting under ultrasonographic guidance in Draft horses. The surgery can be accomplished in a standing horse, using local analgesia and sedation, thus avoiding the need for general anesthesia. Prognosis is guarded to favorable in Draft horses with forelimb tendonitis and suspensory desmitis. Draft horses with hindlimb injury, however, have a guarded to unfavorable prognosis, especially if horses are used for pulling heavy loads. Broodmares with chronic, severe suspensory desmitis must be given special attention during late pregnancy because of weight gain. These mares should be housed on level surfaces and alone. Situations that require the mare to move quickly to avoid an aggressive pasture mate, or to slip while going over rough terrain, should be avoided. Muddy or slick footing also should be avoided. Stall rest, while advantageous for healing of the suspensory ligament, is avoided, because these broodmares usually develop substantial ventral edema.

Other Forelimb Lameness

Other causes of forelimb lameness are unusual. Upper limb lameness usually is caused by direct trauma and is not the result of athletic use. Carpal, elbow, or shoulder joint lameness is not common. I have examined only 13 Draft horses with carpal lameness. Various manifestations of osteochondrosis are seen in the elbow and shoulder joints.

Sweeny, a neuromuscular disorder thought to be associated with injury of the suprascapular nerve, does occur with some frequency in Draft horses. Muscle atrophy may not be obvious for weeks to months, depending on the extent of the injury. Sweeny may result from acute direct trauma, but in Draft horses insidious trauma from a collar is the most common cause. This is especially true of poorly fitting collars, or old and worn collars in which the padding is no longer adequate. Collars of inadequate size often are used on today's large-sized Draft horses. Some Draft horses with sweeny continue to perform adequately despite muscle atrophy. Diagnosis is made by observing muscle atrophy over the scapula, especially affecting the supraspinatus muscle. Lameness is most likely functional and not related to pain, because horses cannot extend the shoulder joint normally. In some horses, however, the shoulder joint actually may subluxate, when the horse is walking or especially while turning in a circle. Subluxation results more frequently from external trauma from being kicked or from running into a solid object rather than from collar injury and may involve additional nerve injury.

Management of horses with acute trauma of the shoulder area includes the administration of NSAIDs, corticosteroids, and dimethylsulfoxide and by performing physical therapy. A water hose with a hard stream of water can be useful for physical therapy. Liniments, massage, handheld muscle stimulators, and heating pads can be useful physical therapeutic modalities as well. The administration of dimethylsulfoxide (0.3 g/kg sid IV for 3 to 5 days) is recommended in the acute phase. Prognosis is difficult to assess, but if improvement is not seen in the first 6 to 8 weeks after diagnosis, prognosis becomes less favorable. I have not attempted scapular notch resection in Draft horses, but knowing the complications associated with the procedure in light horses leads me to hesitate recommending it.

Unusual Signs Consistent with Lameness Caused by Mange Mites

In the United Kingdom a significant incidence of mange occurs in draft breed horses.[2] Draft horses often are presented for evaluation of presumed lameness because of a stiff, stilted gait and with abnormal stamping of the limbs to the ground. In these horses careful lameness evaluation reveals areas of dermatitis, but this condition can be confused easily with musculoskeletal pain. I have not seen this to be a problem in Draft horses in the United States.

Hindlimb Lameness

Hindlimb lameness occurs frequently in Draft horses, and the most common cause is lameness associated with the tarsus (hock joint). Draft horses in general have a predisposition to develop tarsitis, and lameness does not seem to be related to use. The custom of lowering the inside wall of the hoof and attempting to turn the hocks, a shoeing change thought to improve the horse's pulling ability, may predispose a Draft horse to tarsitis. To my knowledge, however, this has never been proved in a controlled study. Draft horse shoes often have a large calk on the heel of the lateral branch, increasing torque and shear stress on the entire limb. Heel calks are especially detrimental in horses that work on hard surfaces, because the calk does not sink into the surface, thus putting even greater stress on the limb. This method of shoeing causes a flaring of the lateral hoof wall and predisposes the hooves to separation and breakdown of laminae. Even if this method of shoeing is abandoned, at least 12 to 18 months are required for the foot to return to normal.

Tarsus

Draft horses with hock lameness show a variety of clinical signs. The perceived problems by the owner also vary considerably. The horse may stop pulling or may rest its butt on the stall wall when in the barn. Breeding stallions may be reluctant to mount a mare, and when mounted, they may be unable to maintain erection. Horses may be reluctant to back up, back in crooked fashion, and have difficulty going up and down inclines. The horse may take short strides in harness or may have a change in attitude, sometimes biting other horses. Because the prevalence of hock joint lameness is high, carefully considering this region when evaluating a Draft horse with hindlimb lameness is always wise.

Diagnosis may be straightforward, based on clinical signs, or may require diagnostic analgesia to pinpoint the problem. Upper limb flexion tests in Draft horses appear to be slightly more accurate as an indicator of hock lameness than the same test is in light horses, but false-negative results do occur. Pulling the medial splint bone with the fingers (Churchill test) with the limb in flexion also may be of value. The horse may show a positive response while standing, or lameness may be exacerbated after performing this maneuver. When the horse is trotting, careful attention is given to the vertical movement of the wings of the ilium (hip or pelvic hike), but if the problem is bilateral, a pelvic hike may not be obvious. Stride length may be shortened when viewed from the side, and when evaluated from behind or in front, horses have a tendency to swing the limb toward the midline and then place the limb laterally as it strikes the ground. Horses may scuff the toes because of the low arc of limb flight. Bony exostosis on the dorsal medial aspect of the hock joint may be obvious or absent.

Tarsocrural effusion, or bog spavin, is observed commonly in Draft horses. This clinical observation often does not correlate with the source of pain. Even if osteochondrosis is suspected or confirmed, intra-articular analgesia always should be performed (Fig. 126-6). Although present, osteochondritis dissecans fragments may not be causing the major source of pain, and surgical removal of these fragments may not resolve lameness. Arthroscopic removal of osteochondral fragments is often less rewarding in adult horses. In Draft horses less than 2 years of age, arthroscopic removal of osteochondritis dissecans fragments is more likely to resolve lameness, however. The decision to perform surgery is based on the level of performance, cosmetic considerations related to joint distention, location of the lesion, and most importantly, whether lameness resolves with tarsocrural analgesia. Horses with osteochondritis dissecans lesions located more distally on the trochlear ridges have a more favorable prognosis that those with lesions located on the proximal aspect. Horses with osteochondritis dissecans lesions associated with the cranial intermediate ridge of the distal tibia have a favorable prognosis if lesions are removed early before chronic synovitis or capsulitis occurs. With only a limited number of horses from which to draw experience, lesions seemingly occur more frequently on the lateral trochlear ridge of the talus (tibial tarsal bone) than in any other location in a Draft horse.

Lameness associated with osteoarthritis of the centrodistal and the tarsometatarsal joints is common. Intra-articular analgesia is often necessary, although some veterinarians medicate these joints and assess the clinical response to this treatment. I prefer to perform intra-articular analgesia and consider management options once a definitive diagnosis is made, however. In my opinion, conservative management should be attempted before using corticosteroids. I block the tarsometatarsal and the centrodistal joints first with the horse standing squarely on

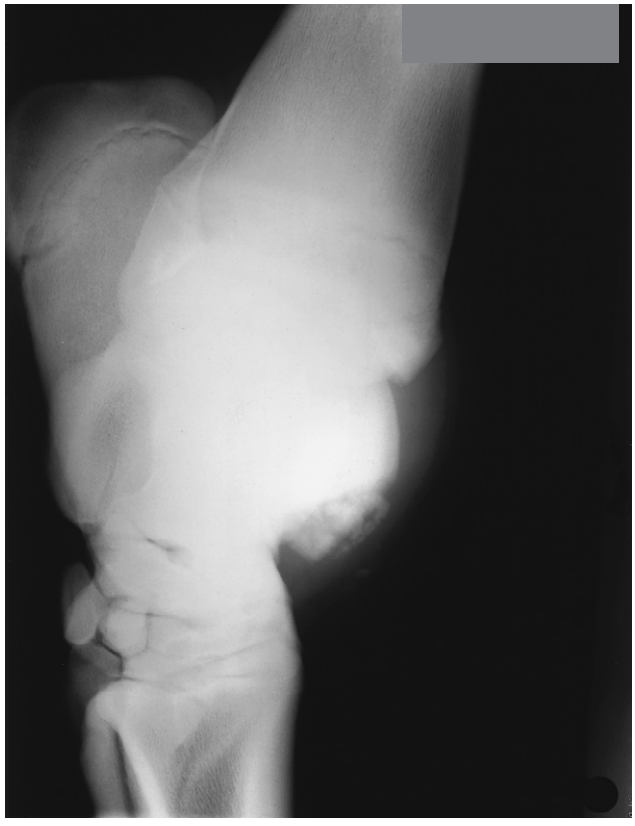

Fig. 126-6 Dorsomedial-plantarolateral oblique radiographic view of a hock of a yearling Draft horse. The radiograph was deliberately underexposed to highlight the extensive osteochondritic lesion involving the distal half of the lateral trochlear ridge of the talus.

each limb. In Draft horses with exostoses on the medial aspect, confidently blocking the centrodistal joint may be difficult. Seven to 12 ml 2% mepivacaine hydrochloride is injected into each joint using a 20- to 22-gauge, 2.5- to 4-cm needle in the standard locations (see Chapter 10). In most Draft horses a twitch is adequate for restraint. Radiographs should be obtained, but unfortunately they are unreliable in confirming the diagnosis of osteoarthritis. In some Draft horses with substantial radiographic changes, lameness may originate elsewhere, whereas in some horses with minor or equivocal radiographic changes, severe lameness from early osteoarthritis of these joints is diagnosed. Radiographic examination helps to select management options and to determine prognosis, however.

Management of the Draft horse with osteoarthritis of the distal hock joints includes physical and medical and surgical options. Correction of any shoeing or trimming problems should be performed before any other treatment. The foot is balanced in a medial to lateral direction according to conformation to achieve a level foot strike. My definition of a level foot strike means that the foot lands evenly when contacting the ground during movement. I remove the large calks and trailers that often are placed on the lateral branch of the shoes. If the horse is not going to be worked for a period of time, I prefer to shoe the horse without any special trim (calks, toe grabs, or inside rims). The dorsal aspect of the shoe is placed at the white line, and the toe is rasped back to the level of the shoe. Squared-toe shoes also can be used. The heels of the shoe should extend far enough behind to support the plantar aspect of the heel completely. In some horses slight heel elevation improves breakover and comfort. If the lateral hoof wall is flared, it must be rasped at each shoeing, and the lateral branch of the shoe gradually must be adjusted to conform properly to a more normal hoof shape. If borium is needed for traction on hard surfaces, it should be placed so that it provides a level surface on the bottom of the shoe. Uneven stress from borium points may cause hoof wall cracks. Usually, using six to 10 spots of borium on the shoe adequately supports the foot and also provides adequate traction.

Medical management may include oral supplementation, intramuscular administration of polysulfated glycosaminoglycan, and intravenous administration of hyaluronan. NSAIDs are recommended at the time of shoeing changes, but long-term use of NSAIDs is usually not a solution in the management of chronic distal hock joint pain. Chronic administration often results in gastrointestinal ulceration and additional problems such as colic or colitis. Horses with distal hock joint pain can be given 2 to 3 g of phenylbutazone daily. Intra-articular injection with hyaluronan alone has limited value in Draft horses with osteoarthritis of the distal hock joints, and I prefer to use hyaluronan with methylprednisolone acetate. If treatment involves economic considerations, the corticosteroid is most important and can be injected alone. The dose of these compounds is similar to that used in the light horse and is not based necessarily on body weight. I increase the corticosteroid dose by about 25% using methylprednisolone acetate (100 mg) in each of the centrodistal and the tarsometatarsal joints. If treating bog spavin, I use methylprednisolone acetate (120 mg) and hyaluronan (40 mg). I seldom if ever use triamcinolone acetonide in Draft horses, because of a concern about laminitis induction and the fact that I often am injecting several joints simultaneously.

Cunean tenectomy is used in some horses, especially those that have obvious enlargement on the medial aspect of the hock (Fig. 126-7). Cunean tenectomy is accomplished in a standing horse using local analgesia and sedation. Horses often are put back into work once the skin sutures are removed, 12 to 14 days after surgery.

Stifle Joint

Draft horses have long been thought to have more stifle joint lameness than do light horses, but when reviewing the records of 745 Draft horse lameness examinations, only 10 horses had primary lameness of the stifle joint. Perhaps long ago, when Draft horses were used daily to pull heavy loads, the frequency of stifle lameness may have been far greater than it is today. Of these 10 horses, three had bilateral idiopathic effusion of the femoropatellar joint. One horse had bilateral subchondral bone cysts of the medial femoral condyles, one had upward fixation of the patella, and five had trauma from kick injuries. Poor conformation and extended stall confinement predispose some Draft horses to upward fixation of the patella. Emaciated horses or those with significant weight loss also are predisposed to the condition. I make every attempt to correct upward fixation of the patella by using conservative management including foot care, improving muscle tone or conditioning, and improving nutritional intake. Raising the heels often alleviates the problem, and block wedges to raise the angle of the foot 6° to 10° are needed. Heel elevation is reduced gradually as the problem is rectified. Improved muscle tone and exercise is beneficial, as is avoiding prolonged periods of stall confinement. Backing exercise strengthens the quadriceps muscles and is a useful form of physical therapy. Internal blisters, injected at the origin and insertion of the patellar ligaments, may cause mild fibrosis and thus tighten the joint. This treatment is used less frequently now than in the past because the preparations need to be special ordered. I seldom if ever elect medial patellar desmotomy as my initial treatment. If conservative management is unsuccessful, desmotomy needs to be performed. The procedure should be

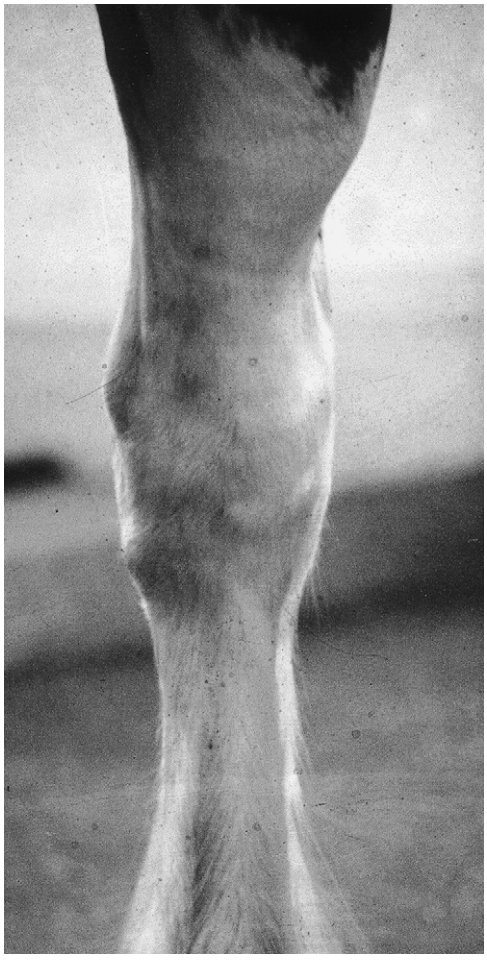

Fig. 126-7 The dorsal aspect of the left hock of Draft horse showing bony exostosis of the dorsal medial surface. If lameness were confirmed to originate from the lower hock joint region, this horse would be a candidate for cunean tenectomy.

done in the standing horse and not under general anesthesia and should be done with a sharp, strong-backed bistoury. This procedure should not be attempted in a Draft horse with an ordinary scalpel handle and blade, because the risk of the blade breaking during the surgery is real.

Shivers

Shivers is considered a progressive, degenerative neuromuscular disease predominantly affecting Draft horses or other large breed horses (see Chapter 49). Various causes have been suggested, including immune-mediated disease following viral infection or strangles and exposure to organophosphates. Presently, polysaccharide storage myopathy has been put forth as a possible cause, but like previous proposed causes, this one may go by the wayside. Clinical signs include an involuntary jerking or twitching most frequently affecting the hindlimbs and the tail. A reflex-like maximum degree of flexion can be seen. Clinical signs often are noticed first by the farrier, because manually picking up the hindlimb frequently stimulates the jerking movements. Signs may be most obvious when the horse first starts to walk, or is backed, or is turned sharply. On occasion, twitching of the muzzle, lips, and ears and forelimb involvement occurs. The condition may improve with rest but usually returns when the horse is put back into work.

Currently, no effective treatment is available. Shivers has been confused with stringhalt in some horses, and the surgical procedure, lateral digital flexor tenectomy, has been performed erroneously. Muscle biopsy to check for polysaccharide storage myopathy and treatment with high-fat, low-carbohydrate diets may be warranted, but definitive proof that polysaccharide storage myopathy is the cause of shivers may be difficult to substantiate. Horses still can be worked, but often owners elect to use them sparingly.

LAMENESS OF FOALS, WEANLINGS, AND YEARLINGS

Young Draft horses require careful monitoring and early attention to lameness conditions. Lameness is often multifactorial and is related to nutrition, genetics, and environment. Early diagnosis and management of lameness appears more critical in Draft horse foals than in light horse foals. Often the size and strength of Draft horse foals gives the erroneous impression that they can overcome many problems.

Infectious Arthritis

Infectious arthritis is common in Draft horse foals and may be related to a high frequency of umbilical problems. Umbilical hernias, umbilical infections, and patent urachus are more common in Draft horse foals than in light horses. Careful examination and treatment of the umbilicus at birth is mandatory, and the umbilicus should be monitored closely for a minimum of 3 weeks. Compared with light horse foals, Draft horse foals are often slower to stand and nurse after birth, leading to many infectious processes. Owner education is important, not only in assisting slow foals up to nurse but also in careful, daily evaluation of joints. Early and aggressive management of infectious arthritis and umbilical remnant infections should be performed (see Chapter 66).

Developmental Orthopedic Disease

Draft horses are affected by all of the various manifestations of developmental orthopedic disease, including flexural deformities, epiphysitis/physitis, osteochondrosis, angular limb deformities, and vertebral malformations (wobbler syndrome). A multifactorial cause is suspected, including nutrition (calcium/phosphorous ratios and trace mineral levels) and hereditary factors (certain bloodlines have a high prevalence). Hereditary factors are complex and include high growth rate, feed deficiency conversion, milk production, and individual horse size.

Physitis/Epiphysitis and Flexural Deformities

These conditions are closely related and seldom if ever a single problem. These conditions are seen over a wide age range from 6 weeks to 24 months, but peak occurrence is in foals 4 to 12 months of age. When raising large horses, owners have a tendency to overfeed, resulting in high-energy rations, fast growth, and nutritional stress, all of which include the possibility of developmental orthopedic disease. My experience with some 300 Draft horse foals suggests a distinct correlation between the frequency of developmental orthopedic disease and nutritional management. Foals having the highest frequency of developmental orthopedic disease were fed free choice high-quality alfalfa hay, approximately 0.015 kg feed/kg body weight/day, of a 16% protein grain ration and two vitamin/mineral supplements. Foals were fed in groups of six to 10 in 5- to 6-acre paddocks. Group feeding allowed the aggressive eater to consume far more than the prescribed amount of grain, and of course hay was fed free choice. None of the foals was thin or malnourished, but numerous foals were obese. Overweight foals, yearlings, or 2-year-olds do not exercise properly, a fact that exacerbates problems associated with excess ration. Under these conditions, two to four foals per group were affected with developmental orthopedic disease each year.

To correct this problem, the nutritional program was revamped. The owner accepted the fact that yearling size may

be slightly less than previously attained, but that developmental orthopedic disease would occur less frequently. Anecdotally, buyers of yearlings from this group had complained in previous years that 2-year-olds had become lame when training had started. The nutrition was changed as follows. The hay ration was given at a 50/50 mixture of alfalfa to timothy. Hay was fed at a rate of 0.015 kg/kg body weight/day, or the amount the foals could consume in about 16 hours. The grain ration was reduced to approximately 0.01 kg/kg body weight/day of a 14% protein ration. Only one vitamin/mineral supplement was given. Foals were grouped according to size, age, and disposition and were monitored closely and checked every 2 weeks by the farm veterinarian. Changes in conformation (becoming upright in the pastern and fetlock) or enlargement (heat and pain) of the growth plates were noted.

Any foal with a change in the pastern or fetlock angle (developing more upright conformation), joint effusion, or physitis was treated immediately. Stall rest was given for 7 to 10 days, the feed intake was reduced, and the foal subsequently was given controlled exercise in a small paddock. Foals were fed only timothy hay, and the grain ration was reduced to 0.002 kg/kg body weight/day. They were placed on phenylbutazone (2.2 mg/kg bid for 3 consecutive days and then every other day for 3 more days). They were maintained in this environment until the problem resolved. Any hoof imbalance or growth abnormality was treated. A 60% decrease in developmental orthopedic disease was noted the first year, and now that farm management has become more attentive to noticing early signs of developmental orthopedic disease, the frequency of this complex of diseases is now less than 10%.

Osteochondritis Dissecans and Osteochondrosis

In my experience, osteochondrosis is observed more commonly in Draft horse foals than in light horse foals. In Draft horse foals the tarsocrural and femoropatellar joints are affected most commonly. Any joint can be affected, including the cervical vertebrae. Horses affected with osteochondrosis show varying degrees of lameness from 1 to 4 of 5 degrees lame. Lameness is less frequent with osteochondrosis lesions of the tarsocrural joint than with osteochondrosis of the stifle. Loose osteochondral fragments in the femoropatellar joint should be removed as soon as possible. Arthroscopic surgery does not guarantee success but improves the prognosis for athletic use. In my experience, Draft horses have a more guarded prognosis for future soundness with osteochondrosis of the stifle than do light horses. I am unsure if this is related simply to the size of the horse or the amount of pulling many of these Draft horses are expected to do, but only about 50% of Draft horses are sound after surgery. Early diagnosis and surgical management improves prognosis considerably. In some foals, without loose fragments, conservative management has resulted in a favorable outcome. Large osteochondritis dissecans defects involving the lateral trochlear ridges have healed based on radiographic examination, but this process may take up to 6 months to occur.

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CHAPTER • 127

Lameness in the Pony

Andrew McDiarmid

Horses under the height of 14 hands, 2 inches (148 cm) at the withers generally are recognized as ponies. Despite this classification some pony breeds are referred to as horses; for example, Icelandic (toelter) *horses*. Many native pony breeds occur throughout the world; for example, Asturian (Spain), Connemara (Ireland), Fell (England), Gerrano (Portugal), Highland (Scotland), M'Bayar (Senegal), Merens (Pyrenees), Ob (Russia), Pindos (Greece), and Welsh ponies. Several small island breeds also occur; for example, Barearic, Eriskay, Faeroe island *horse*, and Chincoteague ponies. The size and conformation of most pony breeds has evolved over centuries because of specific work requirements and geographical isolation. Several breeds are endangered, including the Yonaguni and Noma in Japan and Sorraia in Spain, and some have become extinct; for example, the Fen, Galloway, and Tarpan. Attempts are being made actively to prevent extinction for several breeds; for example, the Kerry bog pony in Ireland and the Taishuh in Japan. In recent times new breeds of pony have evolved, including the Welara and Pony of the Americas. Despite this, most ponies are crossbred.

Ponies have a considerable range of height, weight, and conformation and are involved in most spheres of equine work, including show jumping, eventing, dressage, driving, and general pleasure activities. Many ponies show great athletic ability and, when used for show jumping, they often jump heights similar to their own height at the withers. Ponies have a long life expectancy and can remain in athletic work well into the twenties. Although a small number of ponies are used for high-level competition work such as eventing and show jumping, most are used for general purpose work, when they may be ridden only in the summer months. General purpose ponies tend to be much less valuable than horses and are ridden predominantly by children, which may create emotional and financial conflicts for owners (parents) when deciding the appropriate treatment of a seriously injured pony. Many general purpose ponies remain at grass all year round and do not have access to a stable. Competition ponies often change ownership, at high prices, every 2 or 3 years, as children outgrow them.

Few reports in the literature describe orthopedic conditions specifically affecting the pony. In this chapter I hope to

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give an overview of some of the conditions that are recognized as affecting the pony.

LAMENESS AFFECTING THE PONY

Ponies suffer from orthopedic problems similar to those horses suffer, but the overall prevalence of lameness in the pony is less than in the horse. This may be because of differences in temperament and body weight. With a relatively older population the incidence of some specific orthopedic conditions affecting older ponies may appear higher than in horses, but the prevalence is often unclear and may, for some conditions, be similar; for example, Cushing's disease.

LAMENESS EXAMINATION

Gait assessment in ponies can be challenging because they can have a higher limb speed than horses, and the larger, heavier pony breeds (e.g., Highland) often have a base-wide gait behind, which may make assessment of hindlimb lameness difficult. Several breeds also have gaits additional to walk, trot, and canter; these include the South African, Basotho pony, and Icelandic *horses*. In small ponies the handler may have to walk when leading a trotting pony, rather than run, to keep the limb speed to a rate at which the observer can detect low-grade lameness.

The low height of some ponies compared with most veterinary surgeons may create difficulties with assessment of the lower limb, and excessive force or torsion can be applied easily to the distal joints while performing a flexion test.

In small ponies, care should be taken not to apply excessive pressure with hoof testers and, if the veterinary surgeon is tall, not to flex or twist the lower limb excessively in an effort to raise the foot to a convenient height to use the testers. Paradoxically, in comparison with horses many ponies have harder hoof horn, making eliciting foot pain difficult, so care always should be taken when assessing the response to hoof testers. The use of small or adjustable hoof testers is advisable for ponies.

Ponies can have a positive response to lower limb flexion tests despite being sound and performing to the owner's expectations.

DIAGNOSTIC ANALGESIA

Some ponies are strong-willed, which can limit a conventional lameness evaluation using perineural or intrasynovial analgesia. Using a nose twitch or a low dose of an α_2-sedative may aid this (e.g., 40 to 60 µg/kg romifidine or 0.6 to 0.9 mg/kg xylazine intravenously). In some ponies the lower limbs may have thick skin and be very hairy, making accurate palpation of the structures difficult, particularly finding the site for a palmar digital nerve block. I am conscious that because of the relatively shorter limbs in ponies, local anesthetic solution may diffuse farther than in the horse, and a reduced amount of local anesthetic solution is used for perineural analgesia (e.g., 1 ml of mepivacaine hydrochloride for each nerve when performing a palmar digital or palmar [abaxial sesamoid] analgesia).

IMAGING CONSIDERATIONS

For most clinical situations a similar number of radiographic projections are required to examine the lower limb of a pony as for a horse. Although obtaining good-quality radiographs of the lower limb is generally easier, some important lesions may be more subtle than in the horse, particularly involving the distal interphalangeal joint and navicular bone. Conversely, one should note that a substantial amount of joint degeneration can be present in some sound ponies (Fig. 127-1). It is therefore important in the pony to undertake intrasynovial analgesia to confirm that any peri-articular and intra-articular abnormalities are associated with joint pain.

In some ponies, particularly those kept at grass, a prominent increased thickness of skin in the distal limbs, pelvis, and thoracolumbar areas can be present and reduces skin penetration by ultrasound waves, resulting in poor ultrasonographic images. I have found that shaving the hair and hosing the area to be scanned with warm water for 15 minutes often substantially improves the quality of the image.

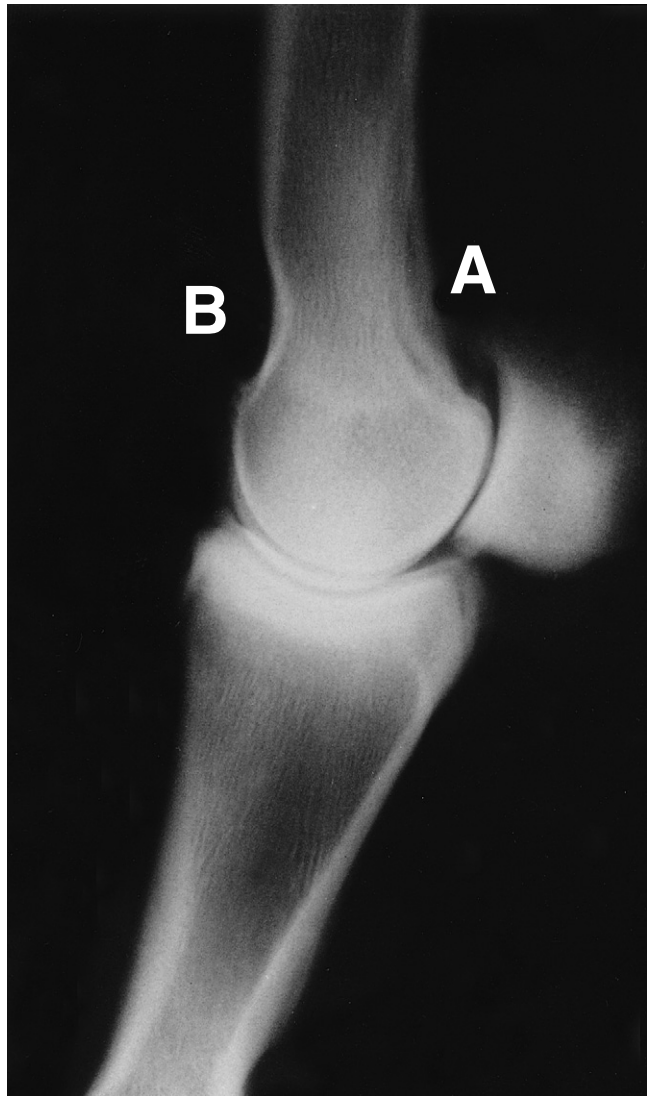

Fig. 127-1 Lateromedial radiographic view of the metacarpophalangeal joint of a 12-year-old jumping pony. Peri-articular osteophytes on the articular margins of the proximal and distal aspect of the proximal sesamoid bones reflect osteoarthritis. There is also evidence of supracondylar lysis (*A*) on the palmar aspect of the third metacarpal bone and villonodular (proliferative) synovitis (*B*) on the dorsal border. This pony had met the owners' expectation until 5 weeks before this examination.

MOST COMMON CONDITIONS AFFECTING COMPETITION PONIES

Competition ponies appear to have a reduced prevalence of lameness in comparison with horses, but the conditions that they suffer tend to mimic competition horses.

- Laminitis
- Distal hock joint pain—osteoarthritis of the distal tarsal joints (bone spavin)
- Desmitis of the accessory ligament of the deep digital flexor tendon (ALDDFT)
- Palmar and plantar annular desmitis
- Osteoarthritis of the metacarpophalangeal joint
- Superficial digital flexor tendonitis
- Exertional rhabdomyolysis
- Osteoarthritis of the carpus

LIMB DEFORMITIES

Mild angular limb deformities in ponies are common, but few affect performance or require medical or surgical correction. In many native breeds in the United Kingdom a small degree of carpus and fetlock valgus deviation is common. The persistence of a full-length ulna and fibula has been associated with the development of severe angular limb deformities in Shetland ponies.[1] This may be a form of atavism, the inheritance of a characteristic from remote rather than recent ancestors.

Congenital laxity of the flexor tendons in the hindlimb of Shetland ponies is not uncommon and can be associated with hyperextension of the distal interphalangeal joint. Treatment is similar to that in the horse.

Adactyly (absence of all or part of a normal digit) and polydactyly (duplication of all or part of a digit beyond the normal number) and other congenital musculoskeletal defects have been recorded in ponies.[2]

JOINT DISEASE

Osteochondrosis

Rarely do ponies have lameness associated with osteochondritis dissecans. Histopathological evidence of osteochondrosis specifically in ponies has been reported only in the lateral trochlear ridge of the femur.[3] Based on a limited number of ponies, in my opinion the most commonly affected joints are the tarsocrural and femoropatellar joints, and lesions are found at the same recognized sites as in the horse, that is, the cranial aspect of the distal intermediate ridge of the tibia, trochlear ridges of the talus, medial and lateral malleoli of the tibia, and lateral trochlear ridge of the femur. Osteochondrosis has been seen particularly in ponies bred for showing,[4] and this may be because these animals were receiving high feed intakes in an effort to improve their show ring appearance.

Lameness associated with a subchondral bone cyst in the medial femoral condyle in the stifle is not uncommon in the pony.

Osteoarthritis

Ponies suffer from osteoarthritis in similar joints as do horses. However, many ponies have mild to moderate joint degeneration, are clinically sound, and perform to the level of the owner's expectation (Fig. 127-1). Osteoarthritis in ponies tends to be primary rather than secondary to a previous intra-articular condition, such as intra-articular chip fractures, osteochondritis dissecans, osseous cyst-like lesions, or ligamentous damage.

Scapulohumeral Joint

Shetland ponies have a higher prevalence of osteoarthritis of the scapulohumeral joint than do other equine breeds. This typically occurs in young ponies; mean age in one survey was 5.2 years. The lameness is usually unilateral but can be bilateral and is sudden and severe (grade 4 to 5 of 5) in onset. Lameness is characterized by a reduction in the cranial phase of the stride and a low arc of foot flight. Radiographic abnormalities may not be present in the acute stage.[5] No reported treatment has resulted in successful resolution of the lameness. Shetland ponies have a flatter and shallower glenoid cavity of the scapula than other equine breeds, and this may be caused by a primary joint dysplasia that could predispose Shetland ponies to osteoarthritis of the scapulohumeral joint.[6]

Carpus

Older ponies appear to have an increased incidence of osteoarthritis of the carpometacarpal joint, often referred to as *carpal spavin*. The radiographic abnormalities found within the carpometacarpal joint are similar to osteoarthritis of the distal tarsal joints, and periosteal and enthesophyte formation on the abaxial margins of the second and fourth metacarpal bones also may be present.[7]

I have observed several lame ponies with restricted flexion of the carpus and radiographic evidence of carpal joint degeneration, but the lameness was abolished by perineural or intrasynovial analgesia in the more distal limb. Restricted carpal flexion, without overt lameness, may result in a pony being presented for a history of tripping and concern for the safety of a child rider.

Stifle

In a retrospective study of stifle lameness, a higher incidence of osteoarthritis of the femorotibial joints was noted in Highland ponies compared with the normal referral horse population at the University of Edinburgh.[8] The condition presents as sudden-onset, severe (grade 4 to 5 of 5) lameness associated with substantial damage to the menisci and collateral or cruciate ligaments. Ultrasonography and arthroscopy are useful to assess the extent of the soft tissue damage. In some of these ponies concurrent abnormalities in the surface of the medial femoral condyle, as described in horses, were present.[9] The extent of the meniscal and ligament damage was such that treatment was generally unsuccessful. Osteoarthritis of the femoropatellar joint can occur in Shetland and Miniature Shetland ponies secondary to chronic lateral luxation of the patella[10] (see following discussion).

Hock

Osteoarthritis of the distal tarsal joints (bone spavin) is a common cause of hindlimb lameness in ponies, but as in other joints, intrasynovial or perineural analgesia is advisable to confirm the relevance of any radiographic changes. Icelandic *horses* appear to be predisposed to osteoarthritis of the distal hock joints.[11] An epidemiological study showed that 23% of Icelandic *horses* in Sweden had radiographic signs of bone spavin.[12] A causal relationship has been found between hindlimb lameness and radiographic evidence of bone spavin and the animals' ages and hock angles. No relationship was found with environmental factors such as training and showing.[13] The lameness found in Icelandic *horses* is often mild, despite moderate to severe radiographic changes.[11]

Other Specific Joint Conditions

Luxation of the Coxofemoral Joint

Rupture of the coxofemoral ligaments and secondary luxation of the coxofemoral (hip) joint has a higher prevalence in the pony than in the horse (Fig. 127-2).[14] This may be because in the horse, unlike in ponies, the ilium tends to fracture before luxation of the hip occurs.[15] In the pony, ligament rupture usually occurs after trauma, and the head of the femur is often displaced in a craniodorsal direction. Affected ponies adopt a characteristic posture with outward rotation of the foot and stifle, inward rotation of the tarsus, and a pronounced shortening of the affected limb. Open or closed reduction has been

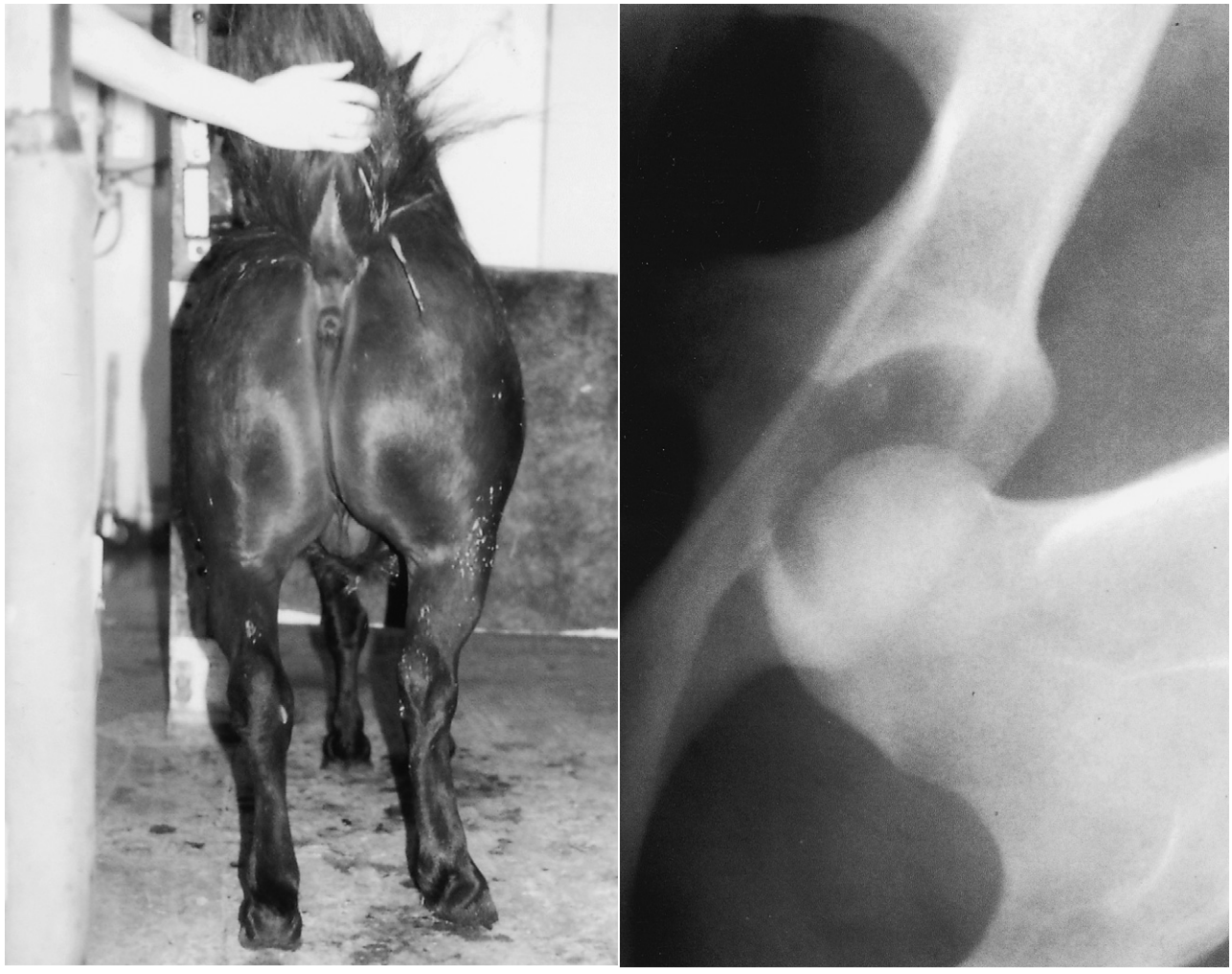

Fig. 127-2 **A,** Typical appearance of luxation of the coxofemoral joint in a pony. Note the outward rotation of the stifle and foot, inward rotation of the tarsus, and shortening of the right hindlimb. **B,** Ventrodorsal radiographic view of the right coxofemoral joint demonstrating the caudodorsal luxation of the head of the femur.

used to treat the luxation, but the prognosis for long-term soundness is poor.[14] Ponies with unsuccessful reduction or irreducible luxation can be salvaged for breeding or companion purposes by excision arthroplasty.[16] Coxofemoral luxation often is complicated by upward fixation of the patella.[15,16] Ligament rupture may occur without luxation of the coxofemoral joint, and the clinical signs are similar to luxation except for the absence of limb shortening.[15]

Dysplasia of the Coxofemoral Joint

Hip dysplasia has been reported in the Norwegian Dole and a Shetland pony colt.[17] In the Shetland pony, hip dysplasia was associated with the development of osteoarthritis in the coxofemoral joint.

Luxation of the Patella

Lateral (sub)luxation of the patella in Shetland ponies is common.[18] The condition is usually congenital, but can be acquired, and affects one or both hindlimbs. A distinction should be made between (sub)luxation and congenital permanent lateral luxation (see the following discussion). The cause of the condition is thought to be malformation of the trochlear ridges and groove, but rupture of the medial femoropatellar ligament has been reported in one pony.[19] Post-mortem examination usually reveals that (sub)luxation is associated with a broadening and flattening of the medial and lateral trochlear ridges, particularly the distal aspect of the medial trochlear ridge. Whether these changes are congenital or are caused by bone modeling is unknown. The clinical signs vary, but in most ponies the patella can be easily manipulated laterally and then replaced in the trochlear groove, and in some ponies the (sub)luxation can be observed during motion. The degree of (sub)luxation varies between ponies and can vary during an individual pony's life. Patellar luxation can be observed on lateromedial and caudocranial radiographs, but to confirm (sub)luxation a cranioproximal-craniodistal oblique (skyline) projection of the patella is required. In ponies with (sub)luxation the patella may appear an abnormal shape on the skyline projection, but this is caused by rotation and abnormal positioning and not by bone modeling.[18] Lateral luxation has been treated successfully by medial imbrication and lateral release incision,[10] or imbrication and recession sulcoplasty.[19] Hermans et al.,[18] using a limited breeding experiment with a group of Shetland ponies, found evidence to suggest monogenic autosomal recessive hereditary transmission of this defect.

Congenital permanent lateral luxation of the patella also is seen in Shetland ponies. Affected newborn foals are unable to extend the stifle fully and therefore have a crouched, squat-

ting position when standing. The lateral trochlear ridge in affected foals is often flat rather than convex.[18] The prognosis for successful treatment is poor.

Upward Fixation of the Patella

Intermittent upward fixation of the patella is a not uncommon condition in ponies, affecting young ponies (typically 2 to 3 years old), and older ponies secondary to an orthopedic problem in the limb. The clinical presentation is similar to that in the horse.

Hemarthrosis

A higher incidence of hemarthrosis has been noted in ponies than horses,[20] and this is often secondary to a systemic disease such as a blood clotting disorder or hepatopathy. Affected ponies have acute-onset lameness with a severely painful joint effusion(s).

Subluxation of the Proximal Interphalangeal Joint

Non-traumatic dorsal subluxation of the proximal interphalangeal (pastern) joint has been recorded in the hindlimbs of ponies.[21] The condition usually occurs bilaterally in young ponies (Fig. 127-3). Dorsal proximal interphalangeal joint subluxation is observed when the affected limb is not bearing weight. The subluxation is reduced as weight is borne on the affected limb, and an audible clink often accompanies joint reduction. Tenotomy of the medial head of the deep digital flexor tendon in the proximal metatarsal region has produced good results in treating this condition.[21] Unilateral proximal interphalangeal joint dorsal subluxation has been recorded in a 3-year-old pony secondary to infectious arthritis of the tarsocrural joint.[22] The subluxation resolved after successful treatment of the infectious arthritis.

Treatment of Joint Disease

Non-steroidal anti-inflammatory drugs should be used with care in lightweight ponies, because they may be more susceptible to phenylbutazone toxicity.[24] (Recommendations by some phenylbutazone-supplying drug companies in the United Kingdom state the maximum level of phenylbutazone in ponies is 4.4 mg/kg orally on alternate days compared with a maximum level of 4.4 mg/kg twice daily for horses.) The ulcerogenic properties of orally administered phenylbutazone are possibly more pronounced in ponies because of less efficient gut absorption. Systemic and intra-articular corticosteroid use in ponies also has to be undertaken with caution because of the potential for development of laminitis.[25]

If arthroscopy or arthrotomy is undertaken in the diagnosis or treatment of joint disease, one should note before giving a prognosis that ponies appear to accommodate greater cartilage degeneration than horses.

A Fell pony foal with infectious arthritis should be checked for immunodeficiency syndrome before treatment is initiated.[23]

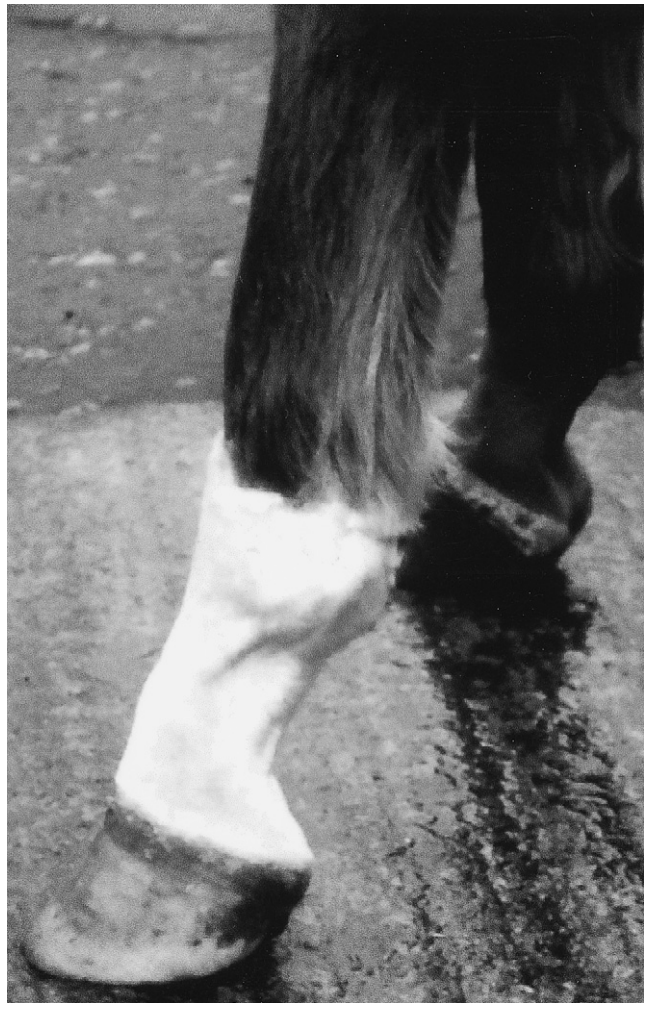

Fig. 127-3 Bilateral non-traumatic dorsal subluxation of the proximal interphalangeal joints in a 3-year-old Welsh pony. The condition resolved after tenotomy of the medial head of the deep digital flexor tendon.

FRACTURES

Common fractures seen in horses (i.e., third metacarpal/metatarsal bone condylar, proximal phalanx, and third carpal bone slab fractures) are rare in ponies. Most fractures are secondary to trauma. Hindlimb fractures, particularly involving the lateral splint bone (fourth metatarsal bone), often result from kicks from other horses. Because of the reduced value of ponies, owners may not opt for surgical intervention to repair fractures.

In some ponies, closed fracture reduction and using a cast alone may result in successful bony union in fractures that would require internal fixation in a horse. The lighter weight of most ponies also means that the use of internal or external fixation of long bone fractures is more successful than in a horse. Repair of some complete fractures of the humerus, tibia, and radius that would require euthanasia in a horse can be attempted in lightweight ponies (<300 kg).[26] Treatment may involve using intramedullary implants such as a single Steinmann pin, stacked pin fixation, or intramedullary interlocking nails. External fixation for comminuted lower limb fractures (e.g., proximal phalanx) is also more successful in the pony.[27] External fixation using a transfixation cast, with or without a U-bar,[28] or an equine modified type II external skeletal device may be successful.[29]

Many fractures of the splint bones (second and fourth metacarpal and metatarsal bones), including proximal fractures, heal satisfactorily with conservative management in the pony. I also have found in the pony that complete excision of the fourth metatarsal bone, as previously described,[30] to be successful in treating fractures of the proximal third of the fourth metatarsal bone.

In ponies with severe or multiple fractures of the carpus, pan-carpal or partial carpal arthrodesis also can be considered.[31] Arthrodesis of the scapulohumeral joint of a Miniature *horse* also has been described.[32]

It may be important when undertaking internal (or external) fixation in older ponies to assess whether the pony may be compromised systemically with Cushing's disease, because this potentially could result in delayed fracture healing and problems with implant failure.

Stress fractures in ponies are rare but have been recorded.[33]

FOOT-RELATED PROBLEMS

Ponies generally tend to have better foot conformation than do horses, and the prevalence of long-toe, low-heel foot conformation appears to be lower than in the Thoroughbred. Navicular disease does occur in ponies, but the incidence is considerably lower than in the horse.[34] Intrasynovial analgesia of the distal interphalangeal joint or navicular bursa should be undertaken to confirm any suspect radiographic changes in the flexor surface of the navicular bone.

Ossification of the cartilages of the foot (sidebone) commonly is observed radiographically in ponies.[35] As in horses, this rarely is associated with lameness.

Laminitis

The incidence of laminitis in ponies is high. Ponies may be more susceptible to laminitis because of an innate insulin insensitivity.[36] Reduction in insulin effectiveness may lead to elevation in thromboxane A_2 activity, causing vascular changes within the hoof. The incidence may be higher than in horses because ponies have a longer life span and insulin resistance may increase with age. In my opinion, the incidence of laminitis in fit, athletic ponies is considerably lower than in ponies that undertake minimal athletic work. This may be due to an improvement in insulin sensitivity.[37] In the United Kingdom many ponies used for showing are excessively overweight, and these animals have a high incidence of laminitis. Ponies often have repetitive annual bouts of laminitis, particularly in the spring, and many respond quickly to acute therapy within a matter of days. The quick response often leads to the feet of chronically laminitic ponies not receiving sufficient corrective farriery (Fig. 127-4), and in the subsequent months many secondary complications can occur, including recurrent subsolar hemorrhage, onychomycosis-induced white line disease,[38] and abscess formation within the dorsal wall.

Dorsal wall resection is indicated in treating some ponies with laminitis, and this can reduce the incidence of chronic problems.[39] Repetitive bouts of laminitis also can lead to hoof and distal phalanx deformation resulting in modified shoes having to be constructed for each foot.[40] The deformation also can result in substantial gait alterations. In a survey of horses and ponies suffering from laminitis, the prognosis for horses was found to be no different than for ponies.[41] Restricted access to grass may need to be enforced to prevent laminitis in general purpose ponies that are never stabled. This can involve mowing the field, strip grazing, or muzzling the pony for part of the day. Care should be taken, however, not to undernourish ponies to prevent laminitis because of the potential development of hyperlipemia.[42]

The incidence of Cushing's disease is higher in ponies than in horses, although no evidence suggests that prevalence is higher.[43] Cushing's disease always should be considered in an older pony with laminitis.

Fig. 127-4 Foot of pony with chronic laminitis that has not received any corrective farriery. Note the extremely long toe and laminitic rings.

SOFT TISSUE INJURIES

Superficial Digital Flexor Tendonitis

Superficial digital flexor tendonitis has a lower incidence in ponies than in horses. Tendonitis does occur in older animals, however, often associated with desmitis of the ALDDFT (inferior or distal check ligament).[44] Tendonitis may not occur necessarily after strenuous exercise, and the site of the superficial digital flexor tendon damage can be in the proximal metacarpal region or within the carpal canal.

Deep Digital Flexor Tendonitis

In my experience, deep digital flexor tendonitis within the digital sheath is also not an uncommon soft tissue injury in old ponies. Tendonitis presents as acute-onset lameness, with a painful digital sheath effusion. Diagnosis is confirmed by ultrasonography. Often a focal area of reduced echogenicity is seen. Dystrophic mineralization also can be evident. The lameness is often slow to resolve (8 to 12 months), treatment is prolonged (often 15 months), and the rate of re-injury is high after return to normal work.[45]

Desmitis of the Accessory Ligament of the Deep Digital Flexor Tendon

Desmitis of the ALDDFT has a higher incidence in ponies than horses.[46,47] An in vitro study has found that ALDDFT rupture occurs at lower forces in older horses and ponies, and this suggests that an age-related degeneration occurs within the ligament.[48] Because of the older pony population the prevalence of ALDDFT desmitis therefore may be similar to that in the horse. Clinically, desmitis presents as an acute-onset lameness and swelling in the proximopalmar metacarpal region. Ultrasonography may show a focal area of reduced echogenicity or disruption of the entire cross-sectional area of the ligament. The damage may occur for part or the entire length of the ALDDFT. Prompt aggressive anti-inflammatory therapy is required in the acute stage to prevent adhesion formation linking the ALDDFT to the superficial digital flexor tendon around the parietal layer of the carpal sheath.[47] Treatment recommended for ponites with desmitis of the ALDDFT is similar to that recommended for superficial digital flexor tendonitis in the horse.[45] If extensive adhesion formation occurs, an acquired flexural deformity of the metacarpophalangeal joint may develop, which is difficult to treat.[47]

Palmar (Plantar) Annular Desmitis

Desmitis of the palmar (or plantar) annular ligament of the fetlock joint is not uncommon in ponies and may be associated with tenosynovitis of the digital flexor tendon sheath and can cause secondary stenosis of the fetlock canal. I have noted a higher incidence of plantar annular desmitis in ponies with chronic or repetitive laminitis. The condition may develop because of the postural and gait changes occurring with laminitis that put greater strain on the plantar structures of the fetlock joint. In such a situation, plantar annular desmitis could be described as a repetitive strain injury. Treatment initially should include anti-inflammatory therapy and treatment of the laminitis. In ponies that are unresponsive or have chronic desmitis, annular desmotomy may be required to relieve fetlock canal stenosis. One should note that chronic enlargement of the annular ligament can occur without signs of lameness.[49]

WOUNDS

In ponies, second intention (contraction and epithelialization) wound healing has a reduced incidence of excessive granulation tissue (*proud flesh*), and wound contraction is faster compared with horses.[50] This appears to be because of better myofibroblast organization and a more effective acute inflammation phase.[51] An in vitro study also has found that horse limb fibroblasts have significantly less growth than in ponies.[52]

OTHER CONDITIONS

The results of nuclear scintigraphic examinations of a limited number of athletic jumping ponies I have treated are similar to those described in horses[53] (i.e., increased ^{99m}Tc uptake in the proximal and distal aspects of the proximal phalanx, dorsal spinous processes, and the distal hock joints). Similarly to the aforementioned study, not all of these lesions were associated with lameness and may represent bone adaptation to the act of jumping. In all ponies the results of scintigraphy should be confirmed by diagnostic analgesia.

Back Pain

Back injuries in ponies, particularly involving the sacroiliac joint, appear to have a lower prevalence than in horses.[54] Ponies, however, often demonstrate abnormal behavioral problems, such as bucking, secondary to hindlimb and occasionally forelimb lameness, that can be perceived by the owner as signs of primary back pain.

Recurrent Exertional Rhabdomyolysis

Recurrent exertional rhabdomyolysis (*azoturia* or *tying up*) does affect ponies, especially excitable types and ponies used for eventing. General purpose ponies often suffer from recurrent exertional rhabdomyolysis when the owner exercises an unfit pony. In one survey in the United Kingdom, ponies were the third most commonly affected group of horses suffering from recurrent exertional rhabdomyolysis.[55] A raised blood level of creatine kinase and aspartate aminotransferase confirms the diagnosis, and treatment is similar to that for the horse. Polysaccharide storage myopathy also has been reported in Welsh-cross ponies and in larger breeds of horses.[56]

Cerebellar Abiotrophy

Cerebellar abiotrophy has been recorded in Gotland[57] and Eriskay ponies.[58] The disease is inherited in the Gotland pony as an autosomal recessive gene.[57]

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CHAPTER • 128

Lameness in Breeding Stallions and Broodmares

Benson B. Martin and Sue M. McDonnell*

Although maximal athletic fitness is typically not the goal for breeding stock, maintenance of musculoskeletal soundness is an important concern for breeding stallions and broodmares, especially because longevity is the usual expectation and a high percentage of these horses have existing musculoskeletal disease related to previous training and performance.

THE STALLION

Musculoskeletal fitness is critical to breeding efficiency of a stallion. The principal goals are to maintain adequate fitness for normal breeding behavior and to avoid conditions or treatments that may affect spermatogenesis or sperm viability adversely. Libido, mounting, thrusting, and ejaculatory dysfunction represent major causes of poor breeding performance in stallions.[1] Musculoskeletal and neurological disease have been estimated to account for as much as 50% of these problems.[1,2] The goal of managing a lame breeding stallion includes maintaining significant comfort and fitness to sustain libido and adequate copulatory agility.

Examination

Breeding stallions require skillful handling and should be examined in an area away from other stallions and mares, a secluded area if possible. An experienced breeding stallion handler with appropriate restraint aids is ideal. Sedation can be used, but care must be taken not to confound the examination or jeopardize fertility. Phenothiazine tranquilizers, although useful for lameness examinations, have been associated with an increased risk of paralysis of the retractor penis muscle. If acepromazine is used, we recommend a small dose (5 to 10 mg intravenously 30 minutes before the examination) well before the horse gets excited and diligent observation of the stallion for several hours to monitor its ability to retract the penis normally. Should the penis not retract with tactile stimulation once the horse is again otherwise normally alert and reactive, steps can be taken immediately for physical support and management to minimize loss of erectile function.[3] For further diagnostic procedures (e.g., radiography), xylazine or detomidine and butorphanol may be necessary.

History should include signalment, information about the performance career, duration and details of breeding experience, presenting problem and associated details, medical history, medication, nutrition, and environment including housing, breeding facilities (e.g., flooring and dummy), and breeding protocol (semen collection technique and stallion handling). Knowledge of old performance-related problems is useful. During the breeding season, the veterinarian should determine the breeding schedule, because this will influence the diagnostic and therapeutic strategy.

Complete musculoskeletal evaluation in a breeding stallion ideally begins with a routine breeding soundness examination, including a general physical examination, examination of the internal and external genitalia, evaluation of at least two ejaculates collected 1 hour apart, and evaluation of penile microflora, as outlined by the Society of Theriogenology.[4]

The veterinarian should observe the stallion at a walk and trot in hand, preferably on an even, hard, flat surface or on grass; perform upper and lower limb flexion tests of the forelimbs and hindlimbs, because osteoarthritis is common; examine the feet with hoof testers; and palpate the back. Manifestations of back pain include abnormal sinking, prolonged muscle fasciculation or guarding, or contraction of the epaxial muscles without touching the back. Back pain is common during the breeding season because of the work or secondary to lameness.

A simple neurological examination should include observation of the horse moving in small circles to the left and right, walking in a serpentine pattern with the head elevated, and assessment of hindlimb strength by pulling the tail as the horse walks. The veterinarian should look carefully for unusual patterns of muscle asymmetry, possibly indicative of equine protozoal myelitis (EPM).

A stallion often is examined because of difficulty in breeding and should be observed during teasing, mounting, thrusting, and dismounting. Specific findings suggestive of a musculoskeletal or neurological problem include failure to couple squarely and to thrust with smooth, rhythmic pelvic action; asymmetric hindlimb weight bearing, particularly one hindlimb dangling while thrusting with the other; failure to properly flex or use the neck or back; abnormal tail posture (spiked high) and anal tone (relaxed; voiding gas or feces) during thrusting; an anxious look in the eye or atypical ear postures suggesting discomfort or distraction; failure to grasp securely with the forelimbs; lateral instability; falling during thrusting or dismount; weak, thready, or irregular ejaculatory pulses (often variable from day to day); and lameness after breeding.[1] Other common manifestations of musculoskeletal pain are reluctance to mount or dismount, early dismount, squealing during dismount, or savaging the mare or handler during or immediately after mounting. Frame-by-frame videotape analysis may help to identify handling factors that enhance or impair performance. Diagnostic analgesia can be difficult to perform and is often unnecessary because of the obvious signs of osteoarthritis. Further investigation in selected stallions may include ultrasonography, nuclear scintigraphy, and measurement of creatine kinase before and 1 hour after breeding.

Specific Diagnostic Considerations and Therapy

Sore Back

A sore back is associated with inadequate coupling and thrusting during breeding. A stallion may fail to wrap the pelvis or neck around the mare or dummy and may tend to hold its head off the neck of the mare and paddle with the forelimbs. Thrusting is often of irregular depth and rhythm. An acutely sore back may become a chronic condition, exacerbated by

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breeding and an increased number of unsuccessful mounts, unless treated aggressively.

It is important to determine the primary cause of lameness and initiate the appropriate treatment. Pain can be secondary to another musculoskeletal cause such as osteoarthritis, a heavy breeding schedule, poor footing, poor shoeing, poor fitting of the breeding dummy to the stallion, or allowing the stallion to advance up the side of a dummy mount so that it is thrusting with a curved back. Chronic back pain may be treated using acupuncture once weekly for 8 weeks, and repeated every 3 to 4 weeks, or as needed.[5] Acupuncture therapy may be combined with non-steroidal anti-inflammatory drug (NSAID) therapy, or NSAIDs may be used alone.[5,6]

Prognosis for normal breeding performance is guarded to good and varies with management factors. One key factor is ejaculatory efficiency. Once a stallion needs more than one or two mounts per breeding, expected performance diminishes rapidly.

Osteoarthritis

A stallion with osteoarthritis appears stiff and painful during breeding; stiffness is often worse after rest and improves with exercise. The carpus, front fetlocks, and hocks are affected most commonly. Diagnosis may be obvious on physical examination and is confirmed radiographically.

Pain control is essential to maintain adequate breeding performance. We recommend phenylbutazone (1.5 to 2 g orally bid for 4 to 5 days and then 1 g bid for 5 to 7 days)[7] combined with a period of sexual and general rest if possible. Significant improvement may not be evident for up to 5 to 10 days. Long-term, low-dose phenylbutazone treatment has no measurable effect on sperm production or testicular size,[8] but horses should be monitored for signs of toxicity including colic, loss of appetite, diarrhea, dependent edema, and mucosal ulceration or renal disease.[9] Other NSAIDs such as flunixin meglumine and ketoprofen, and intra-articular or systemic medication with corticosteroids, polysulfated glycosoaminoglycans, and hyaluronan may be useful in selected horses.

Arthrodesis may be warranted for severe osteoarthritis of the fetlock joint, but post-operative pain may persist, requiring long-term management. Pan-carpal arthrodesis may be considered for stallions with severe carpal osteoarthritis or after acute, severe carpal injury. With any lameness pain management must be used to prevent increased recumbency, which can compromise thermal regulation of the testes, especially in hot conditions, and reduce sperm viability and fertility.

Neurological Disease

Neurological disease (see also Chapter 11) is usually evident during mounting, intromission, and thrusting, with a stallion stepping on its hind feet, bearing weight on a toe rather than the sole, and sometimes knuckling over. The stallion may bear weight unevenly during thrusting, sometimes becoming high sided on the mare or dummy mount, or falling. The stallion may have reduced proprioceptive control of the penis in seeking and intromission and may have poor anal tone during thrusting. The horse may require extraordinary effort to ejaculate.

Treatment of cervical vertebral malformation in adult horses is challenging, and management methods are important, including good footing, proper shoeing, proper positioning of the mare or dummy mount, ground collection of semen, and pharmacological aids to ejaculation when necessary (Box 128-1).[1,10] Special precautions must be taken during collection of semen or breeding to protect the stallion, the mare, and the personnel working around the stallion. The mare or dummy mount should be positioned to minimize the risk of a stallion with poor lateral stability falling. Lateral support can be provided at the hips. For semen collection, a mare that does not wiggle from side to side should be used. A dummy mount may be better, but usually elicits less vigorous thrusting than a live mount and cannot be moved forward to assist the stallion with dismounting. Surgery occasionally is performed, with recovery taking many months.[11]

The management of horses with EPM is similar, together with daily pyrimethamine and sulfadiazine or sulfamethoxazole for at least 60 to 90 days (Table 128-1). To prevent anemia, supplementation with folate and vitamin E is advised.[12] This treatment does not affect sperm production adversely.[13]

Sore Feet

Signs of sore hind feet include shallow thrusting, failure to plant the feet securely during thrusting, thrusting on one leg, and early dismount. Signs of sore front feet or other forelimb pain include hesitancy to mount and dismount. If not resolved quickly, sore feet can lead to long-lasting psychogenic sexual behavior dysfunction.

Many breeding stallions that come from the racetrack have poor hoof quality, underrun heels, long toes, and thin soles, predisposing them to sore feet. The feet should be observed daily and trimmed every 4 to 6 weeks. Leaving stallions barefoot is best, but some may require front shoes to remain comfortable. These horses may be left barefoot during the off season to allow the feet to grow more normally. Provision of soft footing in the breeding shed and weight control are important. Pain control using NSAIDs may be necessary.

Fractures of the Distal Phalanx

Fractures of the distal phalanx can be acute or chronic injuries. Most are palmar process fractures and horses can be managed with a bar shoe with side clips reset every 6 weeks, stall rest, and NSAIDs. Sagittal fractures of the distal phalanx are more serious, but horses may be managed in the same way.

Laminitis

Common primary causes of laminitis include contralateral orthopedic injuries, colitis, Potomac fever, *Salmonella*, and other infectious diseases that predispose to the release of endotoxins. The treatment of horses with acute laminitis includes treating the primary disease, supporting the distal phalanx, decreasing blood pressure, treating inflammation, and managing pain (see Chapter 35).[14-16]

Chronic laminitis, with rotation of the distal phalanx, can be an important problem in breeding stallions, presenting as a stiff gait or more overt lameness in one or both front feet. Trimming, shoeing, and providing analgesia are important (see Chapter 35).

Rhabdomyolysis

Rhabdomyolysis is usually evident during or after thrusting. Inefficient thrusting on early mounts is followed by increasing discomfort, leading to rapid mounting and dismounting. The horse may sweat profusely and have an increased respiratory rate and obvious pain. Treatment of an acute episode includes administering acepromazine (10 to 20 mg IV) to control anxiety and 10 to 20 L balanced electrolyte solution intravenously, keeping the horse confined to a stall, monitoring progress, and evaluating the stallion closely for paraphimosis. In horses with chronic rhabdomyolysis, the diet should be altered to primarily hay and high-quality, low-protein, low-carbohydrate feed.[17] Methocarbamol (Table 128-1) is useful in some horses.

Injuries

Injuries in stallions, including lacerations, punctures, abrasions, and fractures or dislocations, are managed similarly as in other horses. A common injury is abrasion or contusion of the carpus during mounting, especially with dummy mounts. A stallion with a sore carpus may fail to grasp a mare with the forelimb and may stand up on the mare or dummy. Sexual rest until the wound heals is usually best. If continued breeding is necessary, emoliative topical analgesic ointment and skillful bandaging from the coronet to several centimeters above the carpus can relieve apparent discomfort, with the administration of NSAIDs if required.

Hyperextension injuries of a forelimb may occur at dismount but are usually not serious and resolve with NSAID treatment.

Box • **128-1**

Management and Pharmacological Aids to Enhance Libido and Facilitate Ejaculation

Management aids

To enhance sexual arousal

Prolonged teasing under conditions that yield the highest safe level of arousal
Breeding schedule for maximum arousal
Natural estrus stimulus and mount mare
Stable (no side to side movement) mount mare, or dummy is necessary
Minimal distractions in the breeding area
Established breeding routine rich with conditioned stimuli for maximum arousal
Encouragement and positive reinforcement

To reduce back and hindlimb pain and accommodate musculoskeletal deficiencies

Mount mare or dummy of appropriate height and conformation
Mare or dummy down grade from stallion to reduce weight on hindlimbs
Semen collection on the ground (artificial vagina or manual stimulation)
Weight loss to reduce work of hindlimbs, particularly during breeding
Pain treatment
Lateral support at the hips during mount
Good footing (grass or dry non-slip, synthetic surface)

To increase positive stimulation of the penis

Pressure and temperature of artificial vagina that yields best response
Hot compresses applied to the base of the penis

Pharmacological aids

To enhance sexual arousal

Gonadotropin-releasing hormone: 50 µg Cystorelin SC 1 and 2 hours before breeding[20]
Diazepam: 0.05 mg/kg slow IV administration[21,22]

To lower ejaculatory threshold (in copula)

Imipramine: 500 to 1000 mg PO in grain[28,29]

To induce ejaculation (ex copula)

Imipramine: 2.2 mg/kg PO followed 2 hours later by xylazine: 0.4 to 0.6 mg/kg IV[30,31]

Aortoiliac Thrombosis

Signs suggesting aortoiliac disease include delayed ejaculation in spite of good libido (multiple mounts and more than 12 thrusts); progressive hindlimb weakness or pain during thrusting resulting in camping under the mare or dummy; and difficulty backing up to dismount.[18] Erection aberrations are not as common as ejaculatory dysfunction, but they can include delayed or rapid tumescence or detumescence, or loss of erection during thrusting.

Comprehensive Management

The useful breeding career of a stallion with lameness problems often can be prolonged substantially with coordinated veterinary care and breeding management. Particular concerns and challenges include the following:

1. Controlling pain without adversely affecting libido or fertility
2. Maintaining a level of fitness adequate for the breeding work without exacerbating lameness
3. Using available management and veterinary techniques to enhance libido and ejaculatory function, to reduce the work of each breeding, and to reduce the total number of breedings required over the season
4. Managing the breeding book to maximize breeding value while not exacerbating the lameness

Table • **128-1**

Medications

DRUG	DOSE	ROUTE	FREQUENCY
Phenylbutazone	2.2 mg/kg	PO, IV	BID
Flunixin meglumine	1.1 mg/kg	IV, IM	BID
Ketoprofen	1.1 mg/kg	IV	SID
Aspirin	15-100 mg/kg	PO	SID
Isoxsuprine	1.2 mg/kg	PO	BID
Methocarbamol	40 mg/kg	PO	BID
Sulfamethoxazole	12.5 mg/kg	PO	BID
Sulfadiazine	12.5 mg/kg	PO	BID
Pyrimethamine	1 mg/kg	PO	SID
Folate	0.5 mg/kg	PO	SID
Vitamin E	5000 IU	PO	SID
Xylazine	150 mg	IV	AN
Detomidine	5-10 mg	IV	AN
Butorphanol	10-25 mg	IV	AN
Acepromazine	10-25 mg	IV	AN

PO, Orally; *IV*, intravenously; *IM*, intramuscularly; *BID*, twice daily; *SID*, once a day; *AN*, as needed.

Pain Management

Phenylbutazone is the most common NSAID used for breeding stallions. A 4-week oral treatment course (2.2 mg/kg twice daily) has no adverse effects on semen quality at the time of ejaculation, or after 24 and 48 hours stored at 4° C.[8] Although fertility trials have not been done, clinical experience suggests no adverse effects of chronic treatment with phenylbutazone at levels tolerated well by a stallion. We recommend orally administered phenylbutazone daily throughout the breeding season to keep the stallion comfortable and functioning with as little effort and pain as possible to avoid a potential downward spiral of delayed ejaculation, additional effort, and exacerbated lameness.

Complimentary pain management including acupuncture,[5,6] electrostimulation, and massage therapy may provide additional comfort, with no adverse effects on semen quality or fertility.

Fitness and Weight Management

Routine exercise is one of the most important management factors for maintaining cardiovascular and musculoskeletal physical and mental fitness of any breeding stallion. A general recommendation for exercise for a sound breeding stallion is daily paddock exercise for a minimum of 8 hours or a minimum of 30 minutes of supervised exercise or light work. Although the common tendency is to rest a lame horse, controlled exercise is probably even more important for breeding efficiency of a lame stallion. A daily exercise program, including turnout or supervised light work, appears to improve the breeding efficiency of a lame stallion significantly. The type of exercise is influenced by the degree of lameness and the temperament of the stallion. Controlled exercise may include daily riding, lunging, jogging, swimming, or exercise on a treadmill or mechanical walker.

Libido and breeding efficiency almost always improve with a stallion carrying less rather than more body weight, particularly for hindlimb lameness or incoordination. We have seen remarkable improvement in breeding efficiency and stamina after reduction in body weight in Quarter Horses with small sore feet or with aortoiliac thrombosis.

Breeding Management and Handling

Breeding schedule For any hand-bred stallion, and particularly for a stallion with lameness, an important goal of breeding management is to minimize the work of each breeding and the cumulative work of the season. For heavily booked stallions, total seasonal work and efficiency of each breeding often can be achieved by limiting services to one per mare cycle. This forces upgraded mare management so that she is close to ovulation when covered. This type of mare tends to be more stimulating to the stallion and to stand more solidly for breeding. For certain stallions with smaller books, availability for service often can be limited to one mare, 3 or 4 days weekly. In our experience, by shifting effort toward mare management and effectively increasing the sperm numbers per service, seasonal pregnancy rates are often as good or better than with more frequent services.

For stallions that are breeding by artificial insemination, with good to excellent longevity of sperm motility, a reasonable book of mares can be served with collection of semen limited to 2 or 3 times weekly. For stallions in registries permitting frozen semen, it is helpful to bank frozen semen for consideration for use during busy periods or to reduce pressure for obtaining fresh ejaculates when problems arise.

Maintaining ideal levels of libido It is important to maintain good to high libido to help a stallion to ejaculate more efficiently in the face of pain, neurological dysfunction, or musculoskeletal disability. Well-tested management schemes and pharmacological aids are available to enhance libido (Box 128-1). The most useful management tools in a stabled stallion are housing near mares and away from stallions, or ample daily teasing exposure to mares.[19] Treatment with gonadotropin-releasing hormone can be useful to boost libido via increased circulating testicular steroids.[20] Diazepam, by inhibiting the memory of pain associated with breeding, may be helpful.[21,22]

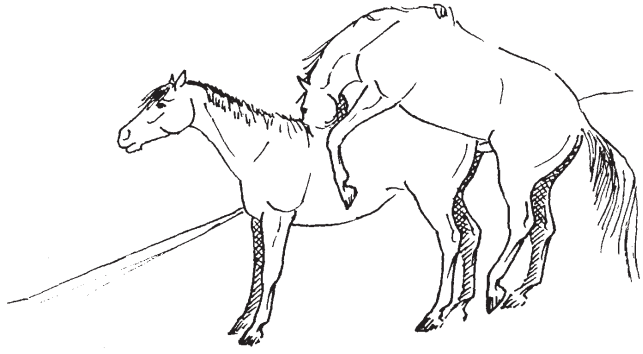

Fig. 128-1 Mare positioned down grade for breeding.

Occasionally, rowdy or over-enthusiastic breeding behavior complicates management and breeding of a lame stallion. The stallion handler should use judicious correction and guidance to establish and maintain organized control, without overcorrection that will diminish libido.[23] Medication is not recommended, because tranquilization at a level effective to control behavior makes the stallion unstable for breeding, and using progesterone or other endocrine-mediated treatments adversely affect spermatogenesis.

Breeding shed handling considerations Most disabled stallions benefit from having the mare or dummy mount slightly down grade. This can reduce the work of the hindlimbs in supporting the body weight. For a live mount mare, a down grade is achieved best with a sloping ramp extending at least 3 m behind, in front, and to each side of the mare (Fig. 128-1). A height differential between the stallion and the mare can be built with mats or by placing the mare in a pit. These work well if the mare remains stationary, but if the mare moves back, the grade may be reversed for the stallion. If the mare moves forward, the stallion can become trapped between the mare and the front edge of mats, or in the pit with the mare. When breeding a stallion with the mare downhill, it is important to avoid the peak of a knoll that slopes off to the sides, or behind the stallion, or in front of the stallion. This sets the stallion up to fall sideways should the mare move sideways, or to be at further disadvantage if the mare moves backward. Once the stallion mounts, restraint of the mare should be optimized to reduce movement in any direction. Disabled stallions that are tentative to mount also can be deterred by low ceilings, cornered mares, or dummy mounts.

When using a dummy mount for stallions with hindlimb, back, or neck problems, we find that greater heights are better. Most disabled stallions do better when guided to remain mounted squarely from the rear, as opposed to across the dummy, or traveling up the side. This is best accomplished by securing the artificial vagina at a physiological angle against the dummy for the stallion to serve, rather than taking the artificial vagina to the horse.

Excellent footing in the breeding area, that is customized to a stallion's particular needs, can improve breeding efficiency greatly (Table 128-2). The surface should be non-slip, even when wet; seamless and even, to avoid stumbling; and provide moderate cushion. Stallions with hindlimb weakness or incoordination tend to camp under the mare. For these stallions the footing behind the mare is critical to maximize the ability to correct the stance. Although some cushion is good, it should not be so spongy or deep that the stallion gets caught

Table • **128-2**

Flooring Materials for Breeding Sheds*

MATERIAL	FOOTING (NON-SLIP)	CUSHION	CLEAN (NO DUST OR KICK-UP)	EASE OF CARE AND CLEANING	COST	COMMENTS
Rubber composite bricks or mats	+++	+++	+++	+++	$$$$	Stable, highly durable
Shredded rubber	+ Shallow — If deep	+++	—	++	$$$$	Highly durable
Poured rubber composite	— If dry ——— If wet	++	+++	+++	$$$$	Durable
Hard rubber mats	— If dry ——— If wet	+	+++	++	$$$	Shift, wrinkle
Fiber	+++	+++	——	—	$$$$	Erodes
Cocoa mats	+++	++	++	—	$$	Shift
Wood chips	++	+	———	Replace	$	Erodes
Sawdust	+	+	———	Replace	$	Erodes
Wood shavings	+	+	———	Replace	$	Erodes
Brick pavers	———	———	+	+++	$$$$	
Concrete	———	———	+++	+++	$$	
Asphalt	—	———	+++	++	$$	
Dirt	+ At best ——— If wet	+ At best ——— If hard	——	Replace	$	Erodes
Sand	+	++	———	Replace	$$	Erodes
Gravel fines	———	——	——	Replace	$$	Erodes

$*, Inexpensive; *$$*, moderately inexpensive; *$$$*, moderately expensive; *$$$$, expensive; +, positive; –, negative.
*Data reflect our opinion.

up or buried in the substrate. Stallions with sore feet, especially front feet, benefit from a softer footing, particularly a soft landing during dismount. Heavy sod on well-drained soil is often the simplest and best outdoor footing. Composite rubber athletic surfaces, particularly if poured and seamless, make ideal breeding surfaces.

A stallion should be given ample freedom about the head, particularly during mounting and thrusting, to allow postural adjustments. Enforcing routines that work well for sound stallions, such as always keeping the head on the left side of the mare or dummy mount, may discourage a disabled horse. For severely ataxic, weak, or uncomfortable stallions, thrusting and ejaculation can be facilitated with gentle stabilizing assistance or spotting during breeding. We recommend an assistant applying gentle pressure with the hands on one or both hips to support the stallion from falling (Fig. 128-2). Some stallions tolerate support from the rear, for example, with a wide strap held from either side behind the buttocks. Some resent this type of support, appearing to become trapped on the mare.

Gentle, guiding handling during dismount is important for all stallions but particularly for a disabled horse. For stallions with hindlimb weakness, pain, or incoordination, special consideration should be given to allowing the stallion as much recovery time as possible before dismount and to moving the mare slowly out from under the stallion rather than forcing the stallion to back off.

Ataxic stallions that tend to step on themselves and the mare while breeding can benefit from simple, well-secured protective boots or wraps for themselves and the mare.

Monitoring breeding performance Stallions with chronic musculoskeletal discomfort or disability usually deteriorate, and breeding performance should be monitored systematically so that adjustments can be made to maximize performance. A sensitive measure of improved comfort is the number, rhythmic pattern, and strength of pelvic thrusts from insertion to ejaculation. Normal, sound stallions typically require seven to nine thrusts at about 1 second intervals, with even sweep and strength, to ejaculate. The normal range of mount duration is 20 to 30 seconds, with insertion time 12 to 20 seconds. With discomfort, horses tend to require more thrusts, the sweeping pattern of the thrusts becomes less organized (irregular depth, rhythm, and strength), and the hindlimbs become more dancy as opposed to being planted with equal weight bearing on each. The head and ear positions of an uncomfortable stallion also suggest distraction (as if about to quit, fall, or dismount) as opposed to commitment. Horses with sore backs or necks tend to fail to couple closely. Thrusts may be jerky and shallow.

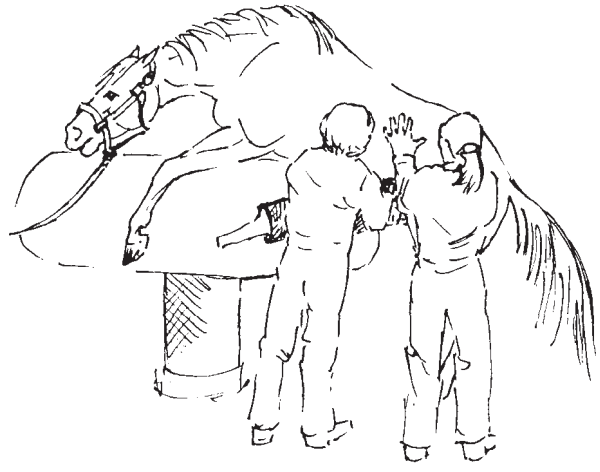

Fig. 128-2 Spotting a stallion on the left side during breeding.

Stallions with hindlimb neurological deficits may bear weight unevenly, become camped under, or become tipped toward the stronger side, with the weaker hindlimb dangling. They seem particularly unable to maintain lateral stability. We recommend developing a customized record sheet for daily grading of each abnormality. Videotaping each breeding from a direct lateral view that spans the entire mount and stallion and from the rear provides the most complete record.

One mount rule For longevity of a disabled stallion through a season or through a breeding career, we recommend that ejaculation should be achieved with one mount. If a horse does not ejaculate in one or at most two mounts, the horse should be taken back to the stall or paddock to rest and return fresh. This strategy often effectively yields a greater number of successful mounts, provided that conditions are optimized for the first mount. The horse should be at the highest level of arousal possible for safe handling, the mount well positioned and optimally restrained if it is a live mare, the artificial vagina at optimum pressure and temperature for semen collection, and the handling team in top form and focused. If particular procedures or medications (see Box 128-1) are known to make the horse more comfortable, enhance libido, or reduce the ejaculatory threshold, we recommend using these from the start.

Ground semen collections For stallions that breed by semen collection, a valuable alternative to mounting is ground semen collection. Most stallions with good to excellent libido are excellent candidates for semen collection while standing on the ground or even while supported in a sling. This technique is particularly useful in horses with aortoiliac thrombosis, cervical vertebral malformation, EPM, and other conditions with hindlimb instability or weakness.

Pharmacologically induced ex copula ejaculation For stallions that breed by artificial insemination, pharmacologically induced (or chemical) ejaculation can be used with the stallion at rest in his stall. Currently the generally most effective treatment (50% to 70% of attempts) is a combination of imipramine hydrochloride administered orally about 2 hours before intravenously administered xylazine hydrochloride (Box 128-1). For a particular stallion, best results can be achieved by titrating through a range of doses for each component drug.

BROODMARE

Soundness of a broodmare is important for the demands of pregnancy and lactation and for ongoing comfort for normal expression of estrus behavior, willingness to stand for natural service when necessary, conception, and normal interactive maternal behavior.[24] Diagnosis and treatment should not adversely affect fertility or the safety of the developing fetus, or nursing foal. As a population, broodmares tend to have a high rate of husbandry-related feet and leg problems. They may have old training or racing injuries. Broodmares coming and going tend to have herd social aggression and related injuries. Broodmares also suffer lameness secondary to foaling.

Specific Diagnostic Considerations and Therapy

Sore Feet

It is recommended that maiden mares should arrive on a breeding farm at least 8 to 10 weeks before the beginning of the breeding season. This allows time to assess and monitor condition of the feet and time for trimming as needed. Early arrival also affords time to acclimate the horse to the farm routine and social environment and for advanced photoperiod manipulation (under lights). Simple sore feet are a common problem in a maiden mare coming from the racetrack. The mare may have thin-walled feet and soles, underrun heels, long toes, bruised feet, quarter cracks, or corns. The mare may have been shod for a long time and not be accustomed to being barefoot. If sore feet are suspected as a cause of lameness, the feet should be examined with hoof testers. Baseline radiography may be indicated. Soaking, poulticing, sole painting, and the administration of phenylbutazone or flunixin meglumine (see Table 128-1) may be required for effective management of mares with foot soreness. Feet of broodmares should be trimmed every 6 to 8 weeks and ideally should be barefoot on all limbs and certainly for the hindlimbs.

A subsolar abscess is the most common cause of acute foot pain (see Chapter 28). Other causes include a sequestrum, laminitis, or a fracture of a palmar process of the distal phalanx. A sequestrum of the distal phalanx may be removed in a standing horse,[25] eliminating the risks of general anesthesia for a developing fetus.

Laminitis

Acute or chronic laminitis can be a management problem in mares and may be secondary to pleuritis, diarrhea, severe lameness, or most commonly after foaling followed by a retained placenta (more common in Draft breeds; see Chapter 126) or infectious metritis. Chronic laminitis also occurs in overweight mares, mares with a previous history of laminitis, or those that have ingested large amounts of feed or fresh pasture. Acute or chronic laminitis can have a negative effect on fertility secondary to pain.[24]

The treatment goals in broodmares with acute laminitis and chronic laminitis are similar to that described for the stallion. In our experience, some mares with chronic laminitis can be salvaged as successful broodmares with careful foot care, pain medication, good footing, separation from other broodmares and competition, and management similar to that described for the breeding stallion. In addition, some can conceive and deliver a normal foal, if these management techniques are successful. However, reproductive lives are often limited by laminitis.

Osteoarthritis

A broodmare with osteoarthritis may need confinement to a stall or small paddock and analgesic medication (see Table 128-1). With severe osteoarthritis of the fetlock or carpus, arthrodesis may be considered as a last resort in a broodmare that can no longer conceive or maintain a foal because of chronic pain.

Enlarged or Swollen Limbs

Palmar or plantar dermatitis of the pastern (scratches or mud fever) secondary to wet or muddy footing may cause lameness, particularly in hindlimbs. The mare should be removed from the wet area. Occasionally, local therapy may be necessary, including washing, drying, and clipping the affected area and local application of a steroid-triple antibiotic topical ointment.

Lymphangitis is seen most commonly in the hindlimbs, is worse in later pregnancy, and causes a reluctance to move, painful, pitting edema, and fever. Treatment includes stall rest, antibiotic therapy, and phenylbutazone.

A puncture wound and secondary cellulitis may cause lameness. Locating the original wound is often difficult. Aggressive antibiotic and anti-inflammatory therapy is necessary. Additional treatment may include surgical drainage, hot packs, warm water hosing, and bandaging.

Hindlimb lameness may be associated with mastitis. The udder is painful to touch, the mare may not allow the foal to nurse, her appetite may decrease, and she may be febrile. Lameness resolves with symptomatic treatment of the mastitis.

Rhabdomyolysis

Acute rhabdomyolysis occurs occasionally in late gestation after particularly vigorous exercise. The mare may not come in for normal feeding or night stables and is often anxious, has a stiff walk, and may have dark-colored urine. Diagnosis is confirmed by measurement of serum creatine kinase. Treatment includes stall rest and NSAIDs. A mare with a foal at foot can be treated with the foal present.

Foaling-Related Injuries

Obturator nerve paralysis secondary to dystocia can result in mild to severe lameness. The mare may show difficulty in abducting the hindlimb and standing immediately after foaling. Femoral nerve paralysis secondary to dystocia results in inability to fix the patella and therefore the hindlimb. This is seen immediately after foaling. Symptomatic therapy for either condition includes NSAIDs, supportive therapy, nursing care slinging if necessary, and adequate bedding and footing. The prognosis for full recovery is guarded, and a nurse mare may be needed for the foal.

Prepubic tendon rupture or ventral body wall hernias may occur during late gestation, apparently without reason. Ventral edema cranial to the udder is severe, unilaterally or bilaterally. An affected mare may have difficulty rising. It is difficult to obtain an accurate diagnosis before cessation of the edema after foaling. The body wall enlarges in the ventral flank. Diagnostic tests include palpation per rectum and diagnostic ultrasonography. Occasionally a tear in the ventral body wall can be palpated per rectum. Supportive treatment consists of NSAIDs, physical support of the enlarged area using large bandages, restricted exercise, close monitoring before and during foaling, a laxative diet, and assisted early induction of foaling. Successful surgical repair of ruptured prepubic tendons and large ventral body wall hernias is often unrewarding, but smaller defects can be repaired successfully. The prognosis for mares with large tears of a ruptured prepubic tendon and future use as broodmares is guarded to poor.[26] Embryo transfer may be a reasonable alternative in breeds that allow this form of management (the Editors).

Neurological Disease

The most common neurological disease of adult broodmares in the United States is EPM. Cervical vertebral malformation also occurs and should be distinguished because treatment is different. Treatment is as for a stallion, but folate supplementation is *not* recommended in broodmares receiving pyrimethamine because of the potential alteration in organogenesis.[27]

Old Injuries

Many broodmares have old superficial digital flexor tendon or suspensory ligament injuries that may deteriorate or sustain acute re-injury. Aged broodmares may have gradual thickening and lengthening of the hindlimb suspensory ligaments. Broodmares with acute re-injuries are treated by restricted exercise, analgesia if needed, local therapy, and ultrasonographic monitoring. Stall confinement with daily hand walking may be needed for 3 to 4 months, depending on ultrasonographic evidence of healing. Treatment of mares with hindlimb suspensory desmitis with a dropped fetlock consists of strict stall confinement, pain medication if needed, and application of an extended heel, egg bar shoe. This shoe should be removed just before foaling for safety.

Occasionally older broodmares develop extensive new bone, especially around the carpus, causing lameness. The mare should be separated from the normal herd to reduce competition for food and water, with restricted exercise and analgesia provided.

General Husbandry and Breeding Management Considerations

Simple management and handling may improve the comfort of an acutely lame broodmare and prolong the useful life of a chronically unsound broodmare. Attention to social groupings can reduce the chance of new injury and provide greater comfort for an older or lame broodmare. Many farms separate broodmares into compatible groups, including maidens, seasoned broodmares, barren mares, and older broodmares. The goals should be to limit competition for food and water and to minimize risk of encountering manufactured obstacles during aggressive interactions. Some farms follow a buddy system, in which compatible pairs are kept together as much as possible. Convalescent broodmares often do well grouped together for rehabilitation or special medical attention. Good footing, including breeding areas, pastures, alleyways, areas around hay bunks, loading ramps, and in stalls can prevent injury and provide comfort for a lame broodmare. Broodmares benefit from as much exercise as possible to maintain overall good musculoskeletal fitness, and obesity should be avoided.

Phenylbutazone is the most commonly used drug for the effective control of pain and has limited or no effect on organogenesis. Its use may be associated with gastric ulceration and concurrent treatment with omeprazole (4 mg/kg curative, or 2 mg/kg preventative) may be of value, although any effects on organogenesis have not been reported.

Emergency surgery to repair a fracture may be necessary to save the life of the mare and fetus. Ideally a pre-formed plan should be in place, especially close to gestation. Is the decision to save the mare and the foal or to consider a non-survival cesarean section to deliver the foal? If the decision is made to proceed to save the mare and fetus, administration of a synthetic progesterone (30 mg/kg orally bid) may be helpful, especially in the first trimester of pregnancy. Elective surgery should be delayed until the mare has foaled whenever possible.

For lame or otherwise disabled mares, artificial insemination should be considered whenever possible. For natural cover, we recommend excellent footing, a gentle approach, reduced restraint, and acute pain control. To reduce the stress in painful broodmares, maiden mares, or broodmares with a known volatile live cover breeding history, the use of 25 mg of acepromazine and 10 to 25 mg of butorphanol given together intravenously 15 to 20 minutes before breeding may increase safety without the ataxia or sudden outbursts seen with xylazine alone. When possible, medication including nutritional supplements should be avoided during gestation and early lactation, including detomidine and xylazine used alone.

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CHAPTER • 129

Lameness in Foals

Robert J. Hunt

Like all horse, foals are prone to injury and lameness; however, considering the immature and fragile musculoskeletal system, the incidence of serious injury is surprisingly low. Exposure of a foal's naive immune system to the array of pathogens places the foal at high risk of infectious causes of lameness. Degree of lameness in foals ranges from subtle and nearly imperceptible to non–weight bearing. Although no published studies document the incidence of lameness in foals, the economic impact on the horse industry is substantial when factors such as cost of diagnosis and treatment, long-term morbidity and mortality, and the effect on athletic potential are considered. Economic and emotional losses may be substantial for horse owners and breeders, enough so that they may abandon the horse industry. Most lameness is self-limiting, however, and in many instances early recognition and prompt treatment makes a substantial difference in costly medical bills and improves the long-term soundness or survival. It is imperative that veterinarians learn to recognize the early clinical signs of these serious disorders and to initiate prompt treatment. Clients should be made aware of the potential danger of a wait-and-see approach and a delay in treatment.

EVALUATION OF A LAME FOAL

Foals often resist handling and limb manipulation, making assessing responses difficult and potentially confusing. It is imperative that the veterinarian is patient when evaluating foals. The basics of lameness localization are the same as in adult horses. The stance and any obvious swelling or sensitive areas on palpation should be noted. It is important to palpate the contralateral limb to gauge the foal's response to manipulation of that limb. Hoof and limb temperature (hot or cold) and amplitude of the digital pulses should be evaluated. Detailed assessment with hoof testers is invaluable in differentiating causes of hoof lameness. Any aberration in hoof development (club foot, deformed foot, unbalanced feet, or

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mismatched sizes of feet) should be noted. Careful palpation of all structures of the skeletal system including all palpable surfaces of joints, bones, digital flexor tendons, and ligaments in a standing and flexed position is important. Swelling should be differentiated as intra-articular, peri-articular, or both.

Gait deficits in foals manifest like those in adult horses in that lameness is expressed as reduced time or weight on the lame limb.[1] The phase of the stride in which the gait is altered is the result of reducing loading of the limb during the painful portion of locomotion. Foals are more likely to show lameness in dramatic fashion by carrying the leg or exaggerating efforts to reduce load on the limb. It is important to observe the foal at a walk to discern subtle differences in stride and foot placement that may be missed at a trot.

Routine ancillary diagnostic tests used in foal lameness evaluation include diagnostic analgesia; radiographic, ultrasonographic, and nuclear scintigraphic examinations; and arthrocentesis and aspiration of other fluid pockets.[1-4] Diagnostic analgesia should be a routine component of lameness localization in foals just as in adults, unless a fracture is suspected. Any areas of suspect pathological bone conditions should be evaluated by radiography. Follow-up radiographic evaluation on a weekly basis may be required to recognize bony changes. Soft tissue evaluation is aided by ultrasonography, especially when the veterinarian is looking for fluid pockets or alterations in soft tissue architecture. Nuclear scintigraphy has limited applications for skeletal evaluation in foals, because normal radiopharmaceutical uptake in the physes can confuse or mask subtle abnormalities.

Fig. 129-1 Typical crouched stance of a foal with rupture of the left gastrocnemius muscle subsequent to overextension while chasing a mare. The foal is unable to extend the limb or support full weight on it. Note the excessive flexion of the hock.

NON-INFECTIOUS CAUSES OF LAMENESS

Non-infectious causes of lameness include external and internal traumatic injuries and developmental or metabolic musculoskeletal diseases. Lameness also may result as a sequella to vascular disorders or may be immune mediated secondary to an infectious problem.

Lameness resulting from external trauma varies with the area involved and the severity of the injury. Essentially any portion of the skeletal system is prone to injury. Traumatic injuries resulting in lameness are common but fortunately self-limiting in most instances. Boisterous activity, coupled with a hazardous environment places a foal at an increased risk of sustaining injury compared with an adult horse. Trauma from other horses (a mare kicking a foal or collision injuries with other foals) is a common cause of lameness. Impact with stationary objects while a foal is being chased may result in serious injury. Internal trauma includes over-exertion injuries, such as a foal running excessively with a mare or being chased, resulting in fracture of the proximal sesamoid bones (PSBs), or rupture of the suspensory apparatus or other soft tissue structures (Fig. 129-1). Common fractures in foals include those involving the long bones, physes, PSBs, carpal and tarsal cuboidal bones, and the distal phalanx.[1,2,5,6]

Fractures of Long Bones

Clinical evaluation may be diagnostic in foals with lameness of traumatic origin. Unstable fractures or severe soft tissue injuries cause obvious, immediate clinical signs. If a fracture is suspected, radiographs are necessary to confirm the diagnosis and determine the severity of the injury (Fig. 129-2).

Fractures of long bones most often result from external trauma such as a kick or having the limb pinned beneath an object while rolling. A limb mis-loaded while rearing may result in fracture of the tibia, femur, or pelvis. If a limb is unstable distal to the femur or humerus because of a fracture, the limb should be immobilized before transport. Surgical approaches and repair techniques for individual fracture types are described elsewhere.

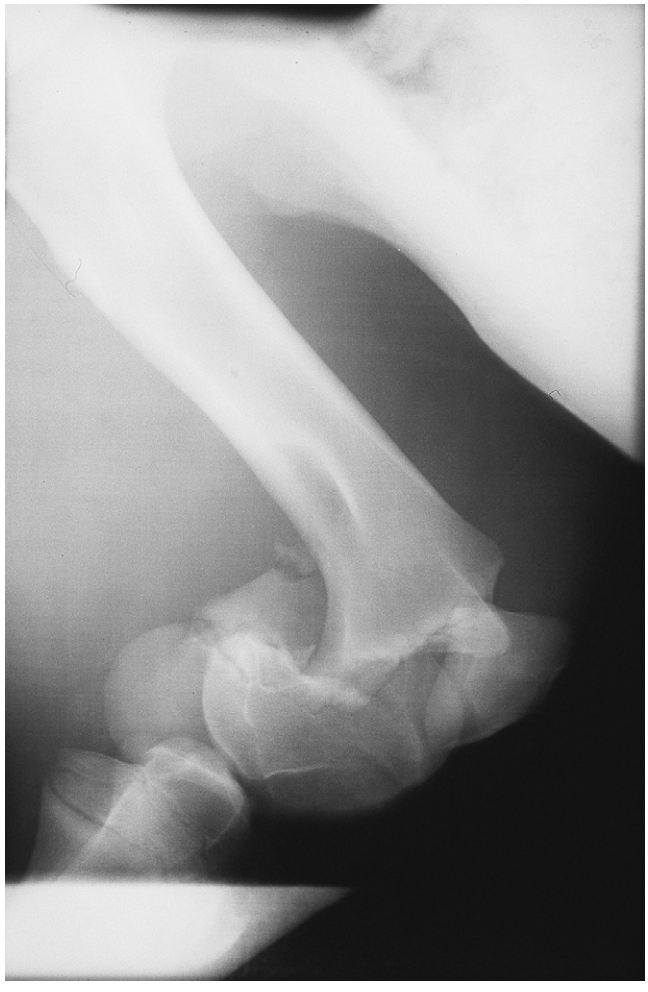

Fig. 129-2 Slightly oblique lateromedial radiographic view of the femur of a foal. There is an unstable, displaced fracture of the distal femur, the result of trauma. This is a Salter-Harris type II fracture (see page 1086).

Prognosis for a foal after fracture depends on anatomical location of the fracture; the complexity, orientation, and degree of soft tissue injury around the fracture; and whether the fracture is open. Secondary problems associated with prolonged lameness, or confinement and immobilization, often dictate the degree of long-term soundness. These problems include excessive laxity of the metacarpophalangeal or metatarsophalangeal joint, varus deviation, or deformity of the foot such as underslung or crushed heels and overgrowth of the toe. Mechanical laminitis is not as common as in adult horses, but it does occur. Contracture or excessive laxity of the fractured limb associated with disuse may prevent future soundness.

Fractures of the radius, ulna, and tibia are generally more amenable to surgical repair than are femoral and humeral fractures. In my experience, conservative management of foals with femoral and humeral fractures by stall confinement is a feasible alternative to surgical intervention, if economics do not allow surgery. Complications encountered following surgery, such as implant failure or infection, are usually fatal. Without post-operative complications, however, the foal has a greater chance of soundness for light athletic use with surgical management. Fractures of the radius and tibia require surgical stabilization if any displacement of the fracture is present. Most foals have a fair to good prognosis for light to medium athletic use, or for breeding purposes. Foals with fractures of the ulna that have any displacement should undergo surgery, but those with non-displaced fissure fractures do not require surgical fixation. Foals with non-displaced fractures of the ulna should have stall confinement for a minimum of 8 to 10 weeks and undergo radiographic monitoring every other day for the first 2 weeks and then weekly to ensure displacement does not occur. Foals with displaced, unstable fractures of the metacarpal and metatarsal bones generally have a guarded to poor prognosis for future athletic use, with or without surgery. Foals with simple, stable fractures not requiring surgery have a good prognosis.

Fractures of the Pelvis

The occurrence of pelvic fractures is high and usually results from the foal mis-loading a hindlimb when rearing or from flipping over and landing on one side. Gait and degree of lameness depend on the portion of the pelvis involved and the amount of trauma to the surrounding tissue. Common sites of injury include the tuber coxae, tuber ischium, acetabulum, and the shaft of the ilium. Deep palpation of the area may elicit pain.

The characteristics of the lameness may reflect the site of injury. A foal with a fracture of the tuber ischium is reluctant to advance the limb and has a prolonged caudal phase of the stride. As with many pelvic injuries, the tail often is elevated and held to the opposite side, and the foal may carry the limb when running but bear full weight while walking. Foals with fractures of a tuber coxae have a shortened caudal phase of the stride, and the injured tuber coxae is often lower than the contralateral side.

Treatment for foals with pelvic fractures includes stall confinement for 8 to 12 weeks followed by a gradual return to controlled exercise. Improvement in the degree of lameness should be seen within 2 to 3 weeks, depending on the region of the pelvis injured. Foals with acetabular injuries may require a longer duration of confinement before improvement is seen.

Prognosis for foals with pelvic fracture not involving the acetabulum is fair to good for light use but guarded for competitive athletics. Some horses are able to race following healing of pelvic fractures. Most foals with pelvic fractures involving the acetabulum have a poor prognosis for an athletic future; however, they generally survive to make breeding animals. In the Editors' experience, the prognosis for athletic function is not invariably poor, because in contrast to adults, foals have a remarkable capacity for bone remodeling, and even severe articular fractures can heal satisfactorily, allowing normal athletic function.

Fractures of the Physes

Physeal fractures have been categorized and defined using the Salter-Harris classification scheme.[2,7] A type I fracture occurs through the physis and involves only the zone of hypertrophied chondrocytes; the adjacent epiphysis and the metaphysis are not involved. A type II fracture occurs across the physis and extends into a portion of the metaphysis. A type III fracture involves the physis and epiphysis and extends into the joint. A type IV fracture includes involvement of the physis, epiphysis, and metaphysis. A compression injury to the physis is referred to as a type V fracture. This classification scheme is applicable to pressure physes that contribute to the longitudinal growth of bone and are subject to compressive forces. Traction physes, such as the olecranon growth plate, are subject to tensile forces and are not included in this classification scheme.

Physeal fractures are common because physeal bone is weak compared with diaphyseal bone. Diagnosis of physeal injuries is made on clinical and radiographic findings (Fig. 129-3). Clinical signs include varying degree of lameness, pain on palpation of the injured area, swelling, and possibly instability of the limb or angular deviation of the limb distal to the fracture. The primary differential diagnosis for a stable physeal fracture is infectious physitis.

Surgical repair is necessary in foals with displaced, unstable physeal fractures. The major difficulty in surgical stabilization is the short section of epiphysis available for purchase. This shortcoming generally is overcome with the use of a form of T-plate, cross pins, transfixation pins, or tension-band wiring. If adequate reduction and stabilization are achieved, the prognosis for future soundness is good.

Cuboidal Bone Injury

Injury to the cuboidal bones of the tarsus or carpus often is associated with failure or delay of endochondral ossification in premature or dysmature neonates.[5,6] Crushing and malformation of the soft cartilaginous precursors of the cuboidal bones results in osteoarthritis and varying degrees of lameness (Fig. 129-4). Outwardly the limb may appear normal or have an angular deviation or, if a hindlimb is involved, it may be sickle hocked or curbed. Attempts at prevention of this condition are more effective than treatment after its onset, although these are limited in effectiveness. Prolonged stall confinement of premature neonates is recommended until signs of radiographic ossification of the cuboidal bones occur. Ossification generally requires 4 to 8 weeks but may vary. Foals affected with crushing of cuboidal bones rarely make competitive athletes, although most are suitable for light use, breeding, or as comfortable pasture animals.

Fractures of the Distal Phalanx

Non-articular fractures of the palmar processes of the distal phalanx occur with high frequency in Thoroughbred foals (Fig. 129-5). Other types of fractures of the distal phalanx occur but are not common. The forelimbs frequently are involved, and the lateral palmar process is more commonly fractured; bilateral and biaxial fractures occur occasionally. In the hindlimb, medial and lateral wing fractures occur evenly. Palmar process fractures may be detected on radiographs as incidental findings and may not necessarily cause lameness.

Salient clinical findings associated with a fracture of the palmar process of the distal phalanx include acute onset of lameness, which may be intermittent in nature, and lameness that is almost always more noticeable on sharp turns.

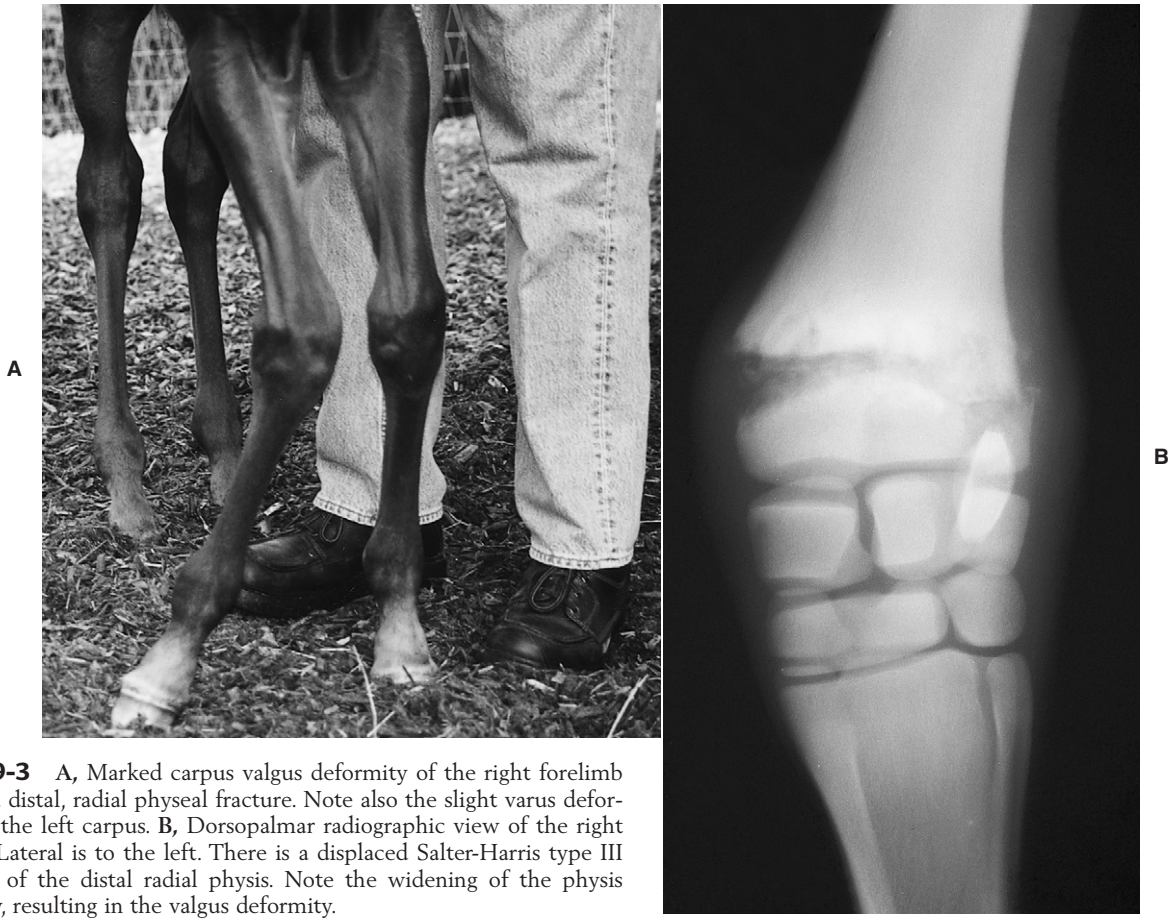

Fig. 129-3 **A,** Marked carpus valgus deformity of the right forelimb due to a distal, radial physeal fracture. Note also the slight varus deformity of the left carpus. **B,** Dorsopalmar radiographic view of the right carpus. Lateral is to the left. There is a displaced Salter-Harris type III fracture of the distal radial physis. Note the widening of the physis medially, resulting in the valgus deformity.

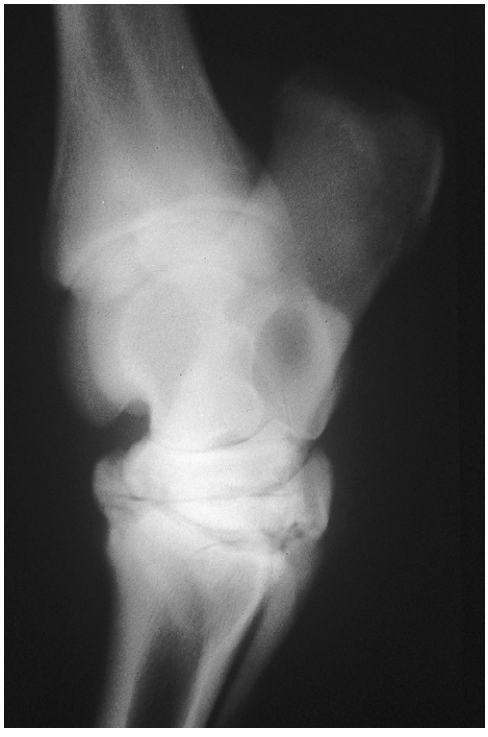

Fig. 129-4 Slightly oblique lateromedial view of the hock of a mature horse with crushing of the third tarsal bone and secondary osteoarthritis of the centrodistal and tarsometatarsal joints, the result of incomplete ossification of the third tarsal bone as a neonate. Note the wedge shape of the third tarsal bone and the abnormal dorsal contour.

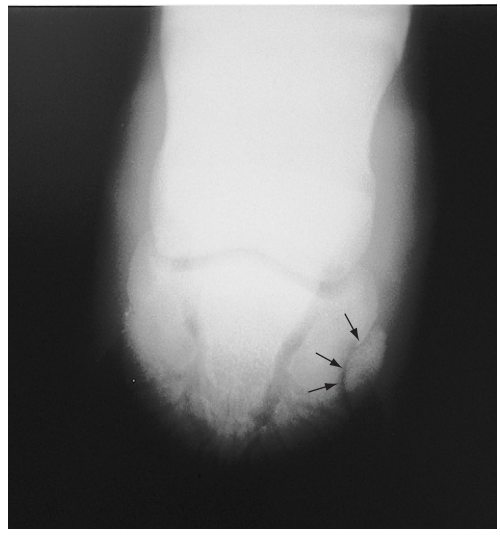

Fig. 129-5 Dorsoproximal-palmarodistal oblique radiographic view of the foot of a foal. Lateral is to the right. There is a non-articular fracture of the lateral palmar process of the distal phalanx *(arrows)*.

Response to hoof tester evaluation is consistently painful when testers are applied across the heels. Atypically a response may be noticed with pressure at the toe. When diffuse pain is noted during hoof tester evaluation along, with an increased temperature of the foot, sub-solar abscessation should be considered.

Treatment includes confinement to a stall or small pen for 4 to 6 weeks. Lameness should improve dramatically in the first week. External coaptation such as a foot cast or glue-on shoe is contraindicated and may potentiate development of clubfoot conformation. Follow-up radiographs are not mandatory unless lameness persists or recurs. The prognosis is good. Differential diagnosis in foals suspect of fracture of the distal phalanx include subsolar bruising, subsolar abscessation, infectious osteitis of the distal phalanx, or infectious arthritis of the distal interphalangeal joint.

Fractures of the Proximal Sesamoid Bones

Fractures of the PSBs are common in young foals. Fractures usually are confined to one limb, but several limbs may be involved. Uniaxial (one) or biaxial (both sesamoids) fractures in a single limb can be found. PSB fractures occur most commonly in foals between 2 weeks and 2 months of age. Fractures most often involve the base or apex of the sesamoid (Fig. 129-6). Mid-body PSB fractures occur less commonly. Hindlimb PSB fractures are less common, but they generally occur in conjunction with PSB fractures in a forelimb.

Lameness varies from moderate to severe and is exaggerated on turns. Often an increased intensity in the digital pulse amplitudes is apparent, and palpation of the involved bone elicits a painful response. Depending on the fracture type and location, the fetlock joint capsule may be distended or regional swelling may occur over the fractured proximal sesamoid bone. Diagnosis is confirmed radiographically.

Conservative management is recommended and involves confinement to a small area for 6 to 8 weeks. If swelling is noticed, a lower limb bandage should be applied. Surgical repair is not beneficial in foals because of the lack of substance of the PSB. Prognosis depends on the type of fracture, number of PSBs involved, and the number of limbs involved. Most PSB fractures have an elongated appearance (megasesamoid) after healing, but foals may be suitable for athletic use if healing is complete (Fig. 129-7). The prognosis for athletic use is worse if both PSBs in one limb are affected, or if fractures occur in more than one limb, compared with a uniaxial fracture in a single limb.

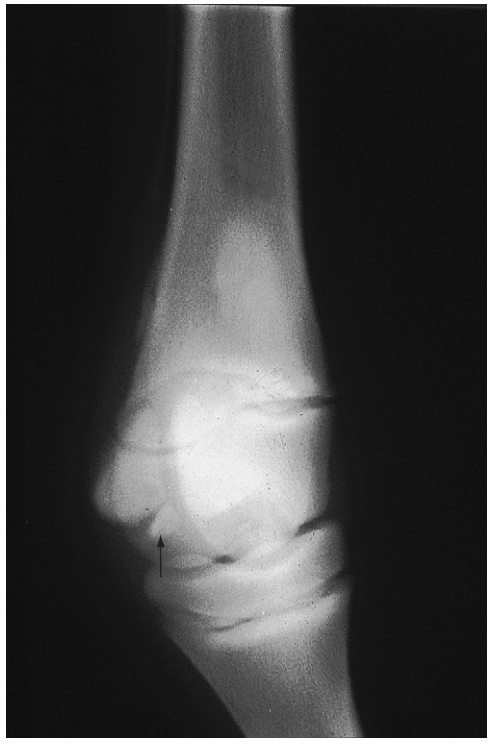

Fig. 129-6 Dorsolateral-palmaromedial oblique radiographic view of the metacarpophalangeal joint of a foal. There is a wedge-shaped basilar fracture *(arrow)* of the lateral proximal sesamoid bone.

Developmental Orthopedic Disease

Developmental orthopedic disease is an uncommon cause of lameness in foals less than 2 to 4 months old.[3,8] However, developmental orthopedic disease is a common skeletal disease in weanlings and yearlings and generally is considered self-limiting. Developmental orthopedic disease is complex and includes flexural deformity, osteochondrosis, physeal dysplasia (physitis), and cervical vertebral malformation. The pathogenesis, clinical manifestations, and management are discussed in Chapters 56, 58, 59, and 62.

Diagnosis of osteochondrosis is based on clinical evaluation and radiographic findings.[3,8] Clinical findings include synovial distention and varying degrees of lameness in the acute stage. Most young foals (less than 2 months old) afflicted with

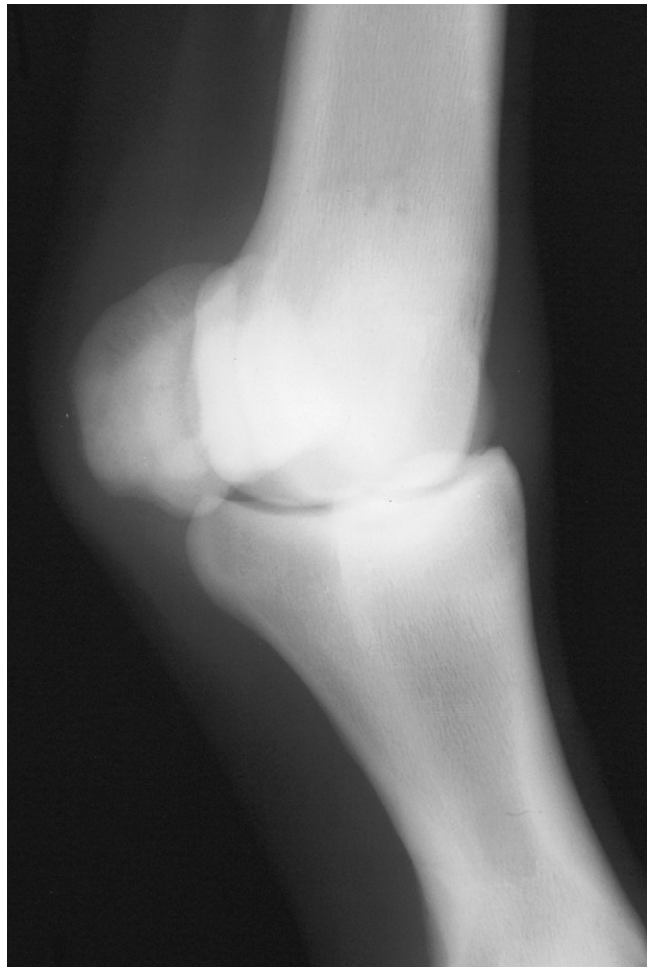

Fig. 129-7 Dorsolateral-palmaromedial oblique radiographic view of the metacarpophalangeal joint seen in Fig. 129-6, obtained when the horse was a yearling. The basilar fracture of the lateral proximal sesamoid bone has healed, resulting in an elongated, distorted appearance of the bone.

osteochondrosis have several joints or bones involved. These foals are subsequently lamer than older foals and usually have severe osteochondrosis. The cause often is not found because individual foals usually are involved, although heredity likely plays a primary role. Radiographic findings may initially include osteochondral fragmentation or erosive changes in subchondral bone, or may be normal. Therefore, it is important to plan on serial radiographic studies to monitor the progression of changes.

Conservative management for young foals with osteochondrosis almost always is recommended and includes rest, anti-inflammatory drugs, systemic chondroprotective drugs, and occasionally joint lavage and injection with hyaluronan. If a free fragment is evident, surgical retrieval may be beneficial; however, one should keep in mind that surgery for osteochondrosis is an elective procedure that should be performed at a time optimal for the foal. If a non-displaced fragment occurs in a weanling or younger age foal, surgery may be delayed until the yearling period and monitored radiographically (Fig. 129-8). Time will allow maturation of the underlying bone so minimal debridement and curettage will be necessary. In many instances the extra time allows these fragments to remineralize, and surgical removal is not necessary. In a longitudinal study of 43 Dutch Warmblood foals osteochondritic lesions were found at the distal intermediate ridge of the tibia in 68% at 1 month of age, but at 11 months of age abnormalities were detected in only 18%.[9] Lesions in the lateral trochlear ridge of the femur developed later, with an incidence of 20% at 5 months of age, but by 11 months of age abnormalities were detected in only 3%.

Vascular Thrombosis

Vascular thrombosis is an unusual cause of lameness in foals. Usually a central focus provides emboli, the most common being a vegetative lesion on a heart valve. Clinical signs include a variable degree of lameness involving one or more limbs. The limb may feel cold during periods of lameness but normal at other times. Response to diagnostic analgesia is inconsistent. Ultrasonographic examination of the heart usually reveals a lesion on a heart valve. Although any valve may be involved, most commonly the aortic or mitral valves are affected. Bacterial blood cultures should be obtained, especially during febrile episodes, if present.

Treatment consists of long-term antimicrobial drugs, based on bacterial cultures if available, and optionally rheological agents such as pentoxifylline. Follow-up ultrasonographic examination is important to determine the effectiveness and duration of treatment. The prognosis is guarded and depends on the degree of ischemia in the distal extremity.

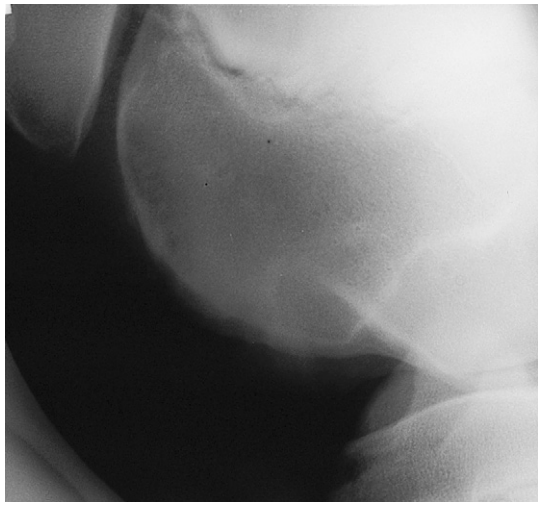

Fig. 129-8 Lateromedial radiographic view of a stifle. There is irregularity in outline of the cortex of the lateral trochlear ridge of the femur with subtle alteration of the subchondral bone opacity. This is the result of osteochondrosis. There are no displaced fragments, so surgery as a weanling is not required, although the foal should be monitored both clinically and radiographically.

Traumatic Nerve Injury

Collision injuries involving the proximal forelimb of foals may result in injury to the radial nerve or components of the brachial plexus. Most commonly a foal has a dropped shoulder, is unable to advance the limb, and drags the limb during ambulation. Differential diagnosis includes fracture of the humerus or scapula, and radiographic evaluation should be performed to differentiate these conditions. The condition may be transient, lasting only a few hours if attributable to neuropraxia, or may be permanent.

Treatment for nerve injury consists of anti-inflammatory drugs, including corticosteroids and non-steroidal drugs, and dimethylsulfoxide. Supportive care, such as physical manipulation of the limb and splinting, may aid in preventing flexural limb deformity. Acupuncture may be beneficial in some foals with nerve injury. In general, if no improvement is seen within 3 to 5 days of treatment, only 25% of foals recover.

INFECTIOUS CAUSES OF LAMENESS

Determining the location and cause of lameness in foals attributable to infection is crucial in deciding a course of therapy and assessing prognosis. As a general rule, assuming that all foal lameness is infectious in origin until proved otherwise is safest. In this instance, prompt appropriate treatment uniformly results in a more favorable outcome than if treatment is delayed.[10]

The clinical complex of infectious arthritis, tenosynovitis, infectious osteitis, or osteomyelitis occurs in foals commonly,[10-15] especially in foals less than 4 months of age, but occasionally occurs in older foals. The different forms of infection can be discussed together, because the pathogenesis is common. Bacteria may gain entry through the umbilicus, respiratory tract, or gastrointestinal tract, although direct penetration of a synovial structure from external trauma may result in synovial infection. Hematogenous dissemination of bacteria allows localization into metaphyseal, physeal, or epiphyseal cartilage and is believed to be associated with the rich vascular network comprised of metaphyseal loops, sinusoidal veins, and epiphyseal and transphyseal vessels that supply the end of the long bones and synovium (Fig. 129-9).[2,12] The sluggish blood flow and vascular stasis of nutrient vessels approaching a cartilage interface allows bacteria to proliferate and colonize. Bacterial colonization incites an acute inflammatory response associated with kinin, complement, and coagulation system activation, eventually leading to cartilage destruction. Vasoactive amines and prostaglandins acting as chemotactic agents result in rapid accumulation of inflammatory cells. Inflammatory products released from synoviocytes, fibroblasts, chondrocytes, neutrophils, and monocytes result in the degradation of articular cartilage proteoglycan, collagen, and hyaluronan. Mediators involved include collagenases, elastases, lysosomal enzymes, plasmin, prostaglandins, metalloproteases, monokines and free radicals. Extrinsic mediators include interleukins, bacterial lipopolysaccharides, and physical forces placed on the damaged cartilage, all of which further compound collagen degradation. Further compromise of cartilage occurs because of poor nutrition of the chondrocytes, caused primarily by fibrin clot formation over the

articular surface, and by thrombosis in the synovial membrane vasculature. The primary mediators of infectious arthritis and eventual cartilage destruction are toxic metabolites released from chondrocytes. Cartilage matrix degradation and proteoglycan loss may occur within 2 days and collagen loss by 9 days after bacterial inoculation.[2,16,17]

No explanation is apparent for disease localization in the epiphysis, metaphysis, physis, or joint. Certain joints do appear, however, to be affected more frequently than others. For example, the stifle, hock, metacarpophalangeal, and metatarsophalangeal joints are involved more often than the carpus or pastern. Infectious arthritis and osteomyelitis of hematogenous origin have been classified into five types based on location of the structure involved (Fig. 129-10).[12,18]

Infectious synovitis (S-type) affects foals usually within the first 10 days of life. Several joints are usually involved, and

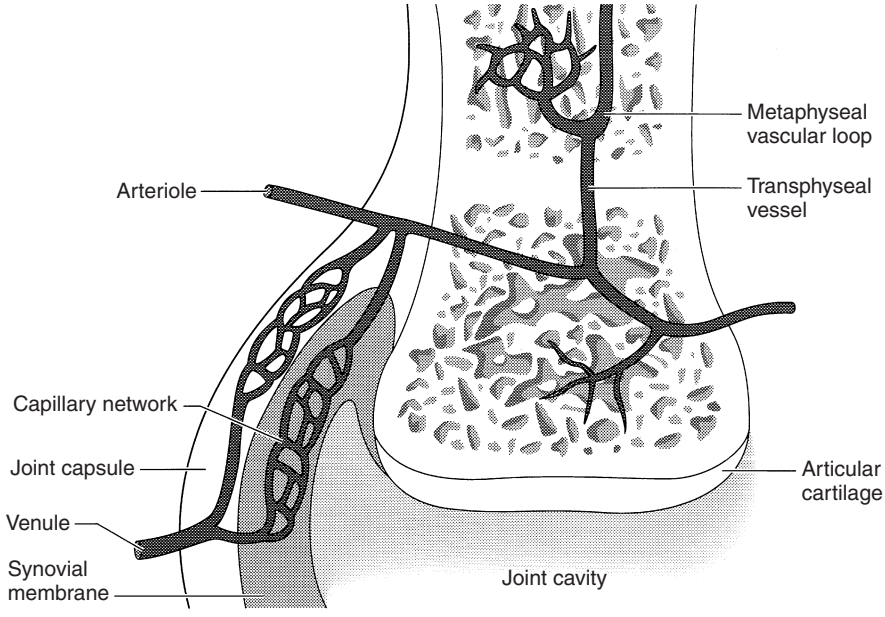

Fig. 129-9 Vascular supply to long bone and articular soft tissue. Blood-borne bacteria may colonize at the metaphyseal vascular loop and disseminate to the physis, epiphysis, or synovial membrane. (Modified from Kobluk CN, Ames TR, Geor RJ, editors: *The horse: diseases & clinical management*, Philadelphia, 1995, WB Saunders.)

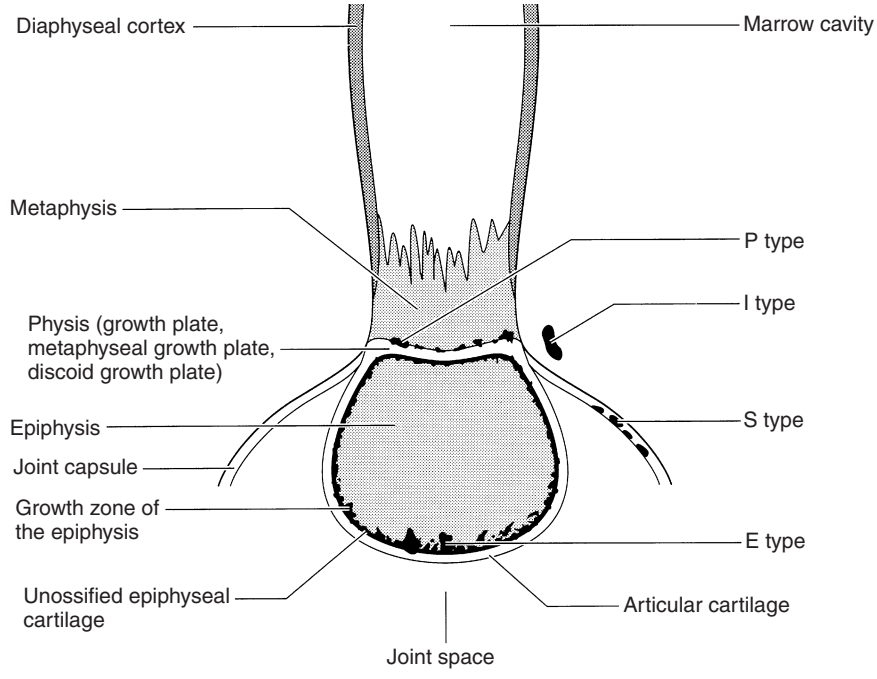

Fig. 129-10 Morphology of the long bone and types of S, E, P, and I infections. *P type,* Physeal-type infectious arthritis; *I type,* bacterial invasion into a physis or joint from a peri-articular abscess; *S type,* infectious synovitis; *E type,* epiphyseal-type infectious arthritis. (Modified from Kobluk CN, Ames TR, Geor RJ, editors: *The horse: diseases & clinical management,* Philadelphia, 1995, WB Saunders.)

few if any radiographic changes are seen. Predominant clinical signs include peri-articular soft tissue swelling, effusion, and lameness. Arthrocentesis reveals an elevation in nucleated cell count (usually >50,000/µl), and degenerate neutrophils predominate.

Epiphyseal type (E-type) infectious arthritis involves the joint and adjacent epiphysis. Foals are generally several weeks of age or older. One or more joints may be infected, and other systemic illnesses may occur concomitantly. Radiographic evidence of epiphyseal involvement commonly is seen.

Physeal type (P-type) may occur in foals from 1 week to 4 months of age. Degrees of soft tissue swelling and lameness may vary, and generally only one site is involved. Sympathetic effusion eventually may occur in a nearby joint (articular structure in close proximity). Lesions may be seen radiographically in the metaphysis, physis, or epiphysis (Fig. 129-11). Pathological fracture may result because of weakening of the involved structure.

Small tarsal or carpal cuboidal bone (T-type) osteomyelitis may result in collapse of the central or third tarsal bones (Fig. 129-12). Several joints commonly are affected.

Invasion into a physis or joint from a peri-articular soft tissue abscess is called I-type infection. Most commonly, joints of the upper limb such as the hip or stifle are involved. If soft tissue abscessation is detected early, infectious arthritis often can be avoided.

Most commonly foals with osteomyelitis have acute onset lameness, diffuse regional swelling, heat, and detectable pain of the involved area. Fever is generally present before and at the onset of lameness but may go undetected. Degree of lameness depends on what bone is involved, the time frame, and stage of detection. Owners often erroneously assume lameness results from trauma because signs occur acutely. A common assumption is that the mare stepped on the foal (the Editors).

Diagnosis of complex infection is based on physical examination findings and the results of arthrocentesis (if intrasynovial involvement occurs) and radiographic examination.[2,10,12,14,15] Foals with infectious osteitis or early osteomyelitis may have no or subtle radiographic changes, but within 1 to 2 weeks radiographic changes consist of erosive, radiolucent changes with sclerosis and eventual proliferation of new bone. It is therefore important to perform follow-up radiographic evaluation to monitor the progression of the disease.

Other imaging modalities are of limited value. Sensitivity of nuclear scintigraphy alone is questionable in young foals because of the high metabolic activity of developing bone.[10] White cell imaging using nuclear medicine techniques is useful, but adds expense and radiation exposure, and has not gained widespread acceptance in private practice.

Ultrasonographic examination is most helpful in identifying soft tissue injury or inflammatory processes of the upper limb. Abscessation of the soft tissue adjacent to the infected bone is a common finding in foals with physeal infection. Using ultrasonographic guidance, abscesses can be aspirated for culture and susceptibility testing and opened, drained, and lavaged.

Arthrocentesis is the key in diagnosing infectious arthritis, but a negative culture does not rule out infection.[4,10-14,19] Joint fluid should be obtained in EDTA tubes to evaluate the differential and total nucleated white blood cell count and protein concentration and for cytological evaluation. Anaerobic and aerobic culture and susceptibility tests should be performed. Using an antimicrobial removal device may be of benefit to potentiate a positive culture if the foal is receiving antimicrobial drugs. Blood cultures should be collected.

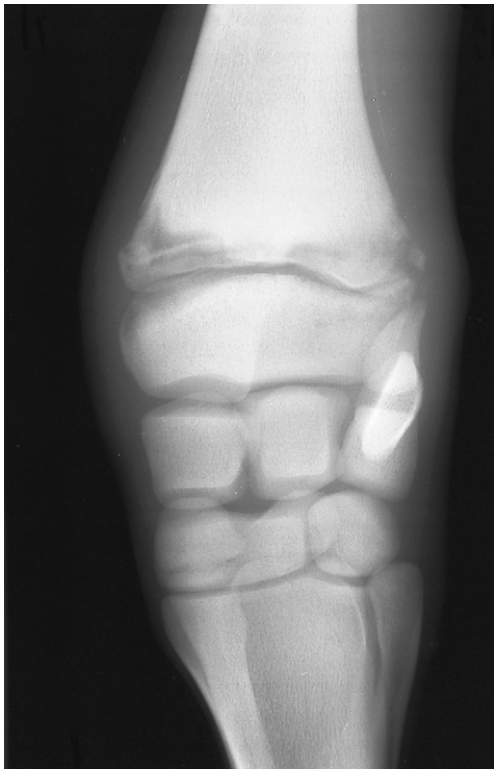

Fig. 129-11 Dorsopalmar radiographic view of the carpus of a foal with P-type osteomyelitis of the distal radial physis. Lateral is to the right. Note the soft tissue swelling and the extensive lytic areas in the physis, metaphysis, and epiphysis. Infection has caused fragmentation of the lateral aspect of the metaphysis.

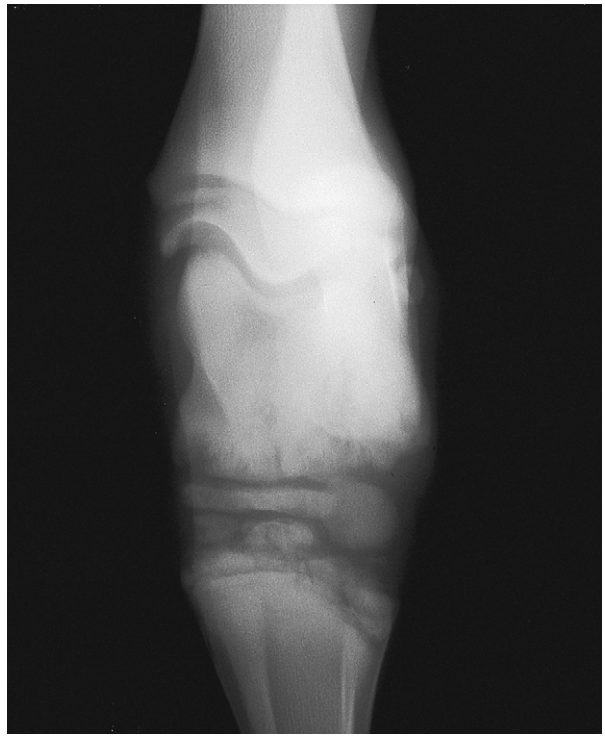

Fig. 129-12 Dorsoplantar radiographic view of a hock of a young foal with type T osteomyelitis involving the cuboidal bone of the tarsus. Lateral is to the right. Note the mottled opacity of the central and third tarsal bones and the proximolateral aspect of the third and fourth metatarsal bones. These extensive lesions warrant a grave prognosis.

In horses suspected of having infectious arthritis, synovial fluid cultures are positive 64% of the time.[11,13,16,17,19] In our practice, of 158 synovial samples submitted in 1 year, there were only 64 (40%) positive isolates. Reasons for low positive culture results include previous administration of antimicrobial agents, partial success of the immune system, the intrinsic bacteriocidal properties of infected synovial fluid, poor storage, or no bacteria present in the synovial fluid.

If there is radiographic evidence of osteomyelitis well away from a joint, a sample of bone and debris may be obtained using a Michelle's trephine. An alternative technique is to use a 2.5- or 3.2-mm drill bit and collect the shavings for culture and sensitivity testing.[10] This tract then may be used for intraosseous infusion of an antimicrobial agent.

The results of hematological testing can be confusing. Complete blood count and fibrinogen level should be assessed, but results are often within normal limits initially because infection is localized. Serial hemograms should be performed in this instance. Any elevation in the white blood cell count or fibrinogen level in a lame foal with fever should be assumed to reflect bone or joint infection until proved otherwise. A normal hemogram does not rule out infectious arthritis or osteomyelitis. Foals less than 1 month of age should have immunoglobulin G levels checked to evaluate for failure of passive transfer.

Treatment of foals with infectious arthritis consists of joint lavage with sterile polyionic fluids through large-bore needles or by using an arthroscope. If a large amount of fibrin and cellular debris is in the synovial fluid, arthrotomy and lavage with or without primary closure of the joint may be indicated. Joint lavage may be repeated every 1 to 3 days depending on the clinical response. A long-term closed suction drainage system may be used as well.[10] Broad-spectrum systemic antimicrobial drugs should be started immediately and adjusted accordingly based on the clinical progression and culture and sensitivity testing.

Other treatments for joint and bone infection include local intraosseous injection or regional hyperperfusion with antimicrobial agents. To perform intraosseous injection, an 18- or 20-gauge needle is inserted into the physis or adjacent bone. An aminoglycoside such as amikacin (250 to 500 mg) may be used. To perform regional hyperperfusion, a site over the abaxial digital neurovascular bundle is prepared aseptically and a rubber tourniquet is placed in the proximal metacarpal region. A 23-gauge butterfly catheter is inserted in the digital vein. An antimicrobial solution (500 mg amikacin diluted in 10 ml sterile water) is infused into the digital vein (Fig. 129-13). The tourniquet is removed after 15 minutes, and a lower-limb bandage is applied. The procedure may be repeated as necessary. Regional digital hyperperfusion is especially useful in treating infectious osteitis of the distal phalanx and distal limb infectious arthritis or osteomyelitis. I have not encountered complications from tourniquet application such as distal limb ischemia.

Foals with infectious arthritis should be allowed to rest for a minimum of 3 to 4 weeks to prevent further traumatic cartilage damage.[2] Systemic administration of chondroprotective agents or intra-articular administration of hyaluronan may be of benefit.

Severe erosive lesions of the physis and metaphysis may result in pathological fracture and collapse of the bony column, resulting in an unstable fracture or severe angular deviation. Surgery may be indicated, but prognosis is poor for soundness if this occurs.

The prognosis for foals with infectious arthritis and osteomyelitis depends on the antimicrobial susceptibility of the organism and how early effective treatment is instituted.[11-19] In general, foals treated early for articular infections have a good prognosis for full recovery. Foals with articular infection with subchondral bone involvement have a poor prognosis, and the clinical course of disease and treatment are prolonged. Foals with focal bony lesions, involving only the physis without bone instability, have a good prognosis. Those with a pathological fracture or severe angular limb deformities have a poor prognosis and may be candidates for humane destruction.

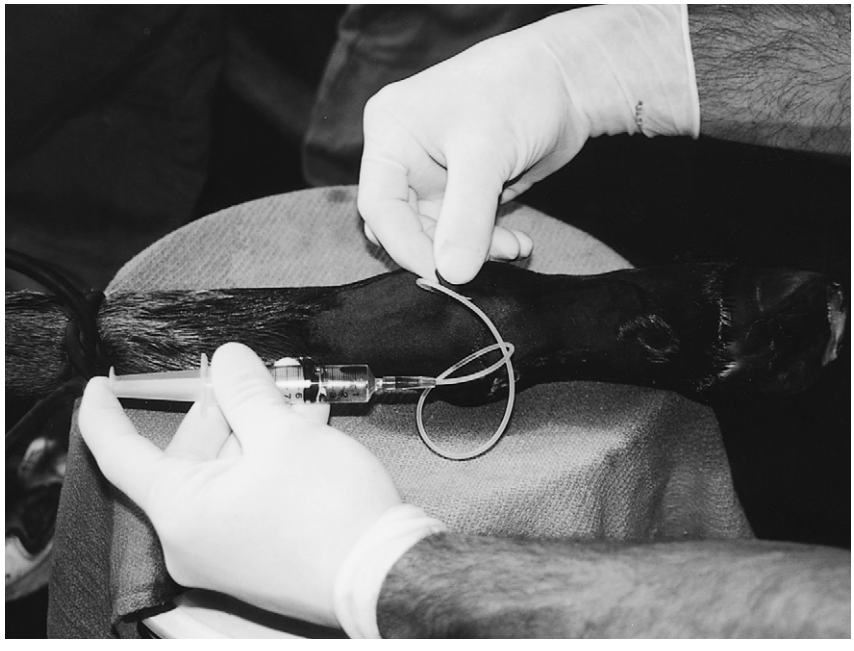

Fig. 129-13 Regional intravenous hyperperfusion of an antimicrobial solution for treatment of infection of the digit. A needle has been placed into the lateral digital vein. There is a tourniquet around the proximal metacarpal region.

Immune-Mediated Synovitis

Immune-mediated synovitis is a sequella to a primary inflammatory focus such as pneumonia, umbilical remnant infection, or a peripheral abscess.[10,20] *Rhodococcus equi* pneumonia is the most common primary focus of infection. The syndrome results from the deposition of immune complexes in the synovial lining and complement activation, resulting in synovitis. The condition usually involves more than one synovial structure. Foals usually have a stiff gait resulting from the synovial distention, and severe lameness is unusual.

The primary rule out for immune-mediated synovitis is early infectious polyarthritis, causing only moderate lameness and mild synovial fluid distention. Cytological evaluation of joint fluid in foals with immune-mediated synovitis commonly reveals nucleated cell counts of less than 20,000 nucleated cells/µl. The cells are well-preserved neutrophils and large mononuclear cells. Foals with infectious arthritis generally have nucleated cell counts greater than 50,000 nucleated cells/µl, and neutrophils are degenerative.

Therapy for foals with immune-mediated synovitis is based on identifying and resolving the underlying disease. Synovitis is self-limiting once the offending cause is removed. Systemic chondroprotective agents may be of benefit to the health of the joint, because prolonged inflammation may cause cartilage damage.

Infection of the Digit

Infection of the digit is a frequent cause of substantial lameness in foals. Infection may be contained to the subsolar or hoof wall regions, or it may involve the distal phalanx or the distal interphalangeal joint. It is critical to differentiate between these sites as soon as possible. Infection at all sites causes increased intensity of the digital pulse amplitudes and, at some stage, increased temperature of the hoof capsule. With subsolar or wall abscessation, pressure applied with hoof testers usually produces a painful response along the toe and sole, unless overlying horn is separated from the sensitive tissue caused by accumulation of purulent material. Radiographs in these foals reveal gas accumulation in the involved area. If the distal phalanx is infected, radiolucency or sequestration may be evident. Diffuse swelling at the coronary band usually means involvement of the distal interphalangeal joint, and arthrocentesis should be performed in an area remote from the swelling.

Subsolar abscesses should be drained early to prevent involvement of deeper tissues. To facilitate drainage, the foot can be soaked in hot water and bandaged with an Animalintex pack (3M Animal Care Products, St. Paul, MN). Both procedures soften the horn. Surgical curettage of the distal phalanx and lavage of the infected bone should be performed in foals with sequestration. A sterile, antiseptic bandage should be maintained after surgery for a minimum of 3 to 4 weeks or until a healthy keratin covering has grown over the exposed bone. Ancillary treatment includes regional digital hyperinfusion using a dilute antimicrobial solution. A good prognosis is warranted in foals with infection of the distal phalanx if the disease is detected and treated early. If more than 25% of the distal phalanx is involved, prognosis for future soundness worsens. Recurrence of infection after initial curettage requires additional surgery. Because of the limited surface area and porous nature of the distal phalanx in the foal, recurrence worsens the prognosis considerably.

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CHAPTER • 130

Pleasure Riding Horse

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Pleasure riding horses are a vital part of the equine industry and many owners devote great amounts of time, money, and emotion to them. Although pleasure riding horses are of far less monetary value than competition horses, the overall importance to the owner should never be underestimated.

Pleasure riding horses often tend to be used seasonally and somewhat episodically. Uses include trail riding, hunting, gymkhana-type activities, showing, lesson horses (teaching horses), the pasture pet, and eating grass. Pleasure riding horses can be divided loosely into two categories, ex-professionals and others. The ex-professionals are horses that formerly were used for a specific athletic use such as racing, but are no longer able to perform in the sport, and have been demoted. The others include potential athletes that were unable to be used for the intended purpose because of lack of ability, poor conformation, or temperament and horses and ponies that were bred casually as pets. The common causes of lameness in these two groups differ to some extent.

It is vital to be able to relate to the owners of pleasure riding horses, who often are inexperienced with horses, have little veterinary knowledge, but have tremendous emotional involvement with the horse and are often anxious. It is vital to first establish communication with the owner and try to relieve any anxieties. The veterinarian should explain the intended examination and treatment and why in straightforward, non-technical language. Facilities for examination may be far from ideal and may complicate the examination. The veterinarian should not over-interpret a short striding gait shown by a horse trotting on an uneven paddock or a rocky driveway. The clinician should start from the basics and evaluate left-right symmetry, with the horse standing on a flat, level surface, if available, and then assess the horse moving in straight lines and circles. The veterinarian should be aware that the horse may have been living with a low-grade lameness, unrecognized by the owner, for a considerable time. If the owner is concerned about a severe left forelimb lameness, the veterinarian would be prudent to avoid mentioning a low-grade left hindlimb lameness observed concurrently, because the lameness is probably not of material relevance and will only further worry the owner.

Interpretation of findings is in part dictated by the age and previous occupation of the horse. Many ex-professional horses have previous soft tissue injuries or show lameness after flexion of a variety of joints, and it is important to try to establish which is the current active disease process causing lameness. Many clinical observations reflect previous injuries and are unrelated to the current lameness.

Local analgesic techniques are useful in some situations, but the temperament of the horse, difficulties in adequate restraint, or examination facilities available may mitigate against them. Owners of pleasure riding horses may resist techniques that are invasive or potentially painful to the horse, whereas they may be fully prepared to pay large sums for advanced diagnostic techniques. The clinician should bear in mind that a twitch in the inexperienced hands of an owner may be dangerous and should consider using tranquilization (e.g., 1 ml of acepromazine) in the horse to facilitate local analgesic techniques.

In some situations an owner prefers a step-by-step diagnosis reached by assessing the response to treatment, even without a definitive diagnosis. This can provide the slow acquisition of useful information. For example, assessment of the response to non-steroidal anti-inflammatory drug treatment can be helpful. Lameness associated with a subsolar abscess is likely to deteriorate, whereas lameness from navicular disease or osteoarthritis of the proximal interphalangeal joint is likely to improve.

In ex-professional horses, osteoarthritis and previous tendon and ligament injuries are common. Minor trauma to a joint in a horse with pre-existing osteoarthritis may result in severe, persistent lameness, whereas similar trauma to a normal joint probably would not result in chronic lameness. Commonly injured joints include the carpus, fetlock, hock, and stifle. Superficial digital flexor tendonitis, suspensory desmitis, and distal sesamoidean ligament injuries are common chronic injuries in ex-professional horses, but re-injury is comparatively unusual unless the horse is subjected to a sudden and substantial increase in exercise intensity; for example, a horse is ridden hard for 2 hours after 3 months of little or no exercise. Previous sites of chronic inflammation may become a long-term problem. For example, a horse may lose a shoe and then gallop about on hard ground, resulting in solar bruising. However, lameness may persist and radiographs may reveal osteitis of the palmar processes of the distal phalanx. A horse with toed-out conformation may traumatize the medial proximal sesamoid bone, resulting in acute lameness, but radiographic examination may reveal pre-existing abnormalities.

Many causes of lameness in pleasure riding horses relate to the lifestyle of pampered pets, and the environment in which they are kept. Subsolar abscesses are common and often result in a severe lameness, creating panic for an owner, who assumes the horse must have sustained a fracture. Managing the owner is equally as important as treating the horse. Subsolar abscesses also may be sequelae to previous laminitis.

Creation of effective drainage is essential for successful management of subsolar abscesses. Without drainage the use of systemic antimicrobial drugs is contraindicated in my experience, because such drugs may prolong the course of the infection. In some pleasure riding horses with a hard hoof capsule, determining accurately the site of abscess may not be possible initially. I recommend intensive soaking with Epsom salts and warm water and poultice at night. Periodic survey radiographs can be useful. Up to 30 days may pass before the abscess can be located accurately and drainage can be established. Once the abscess has opened or been opened, systemic antimicrobial drugs may be useful, especially if a large area of the foot is damaged.

Pleasure riding horses are at high risk for sustaining lacerations or puncture wounds, often resulting from impact with less than ideal fencing such as barbed wire or a jagged post. Injuries vary from minor to severe, resulting in long-term lame-

ness. Injuries may go unrecognized for several days because not all pleasure riding horses are inspected carefully and regularly. Many pleasure riding horses are kept in groups at pasture, and the introduction of a new horse can result in disruption of the hierarchy and the risk of horse-induced injury.

The veterinarian also should recognize that many pleasure riding horses have major conformational abnormalities that predispose them to the early development of osteoarthritis. Many pleasure riding horses live to an old age, and age-related osteoarthritis is not uncommon. Farrier care may be less than ideal and may predispose the horses to chronic foot pain and osteoarthritis of the distal limb joints. Nail bind and excessive shortening of the toe are common farriery-related causes of lameness. It is important to establish the time of onset of lameness relative to when the horse was last shod. These problems are usually apparent within 48 hours. If a nail was driven inside the white line and immediately removed, this may predispose the horse to a subsolar abscess, which usually causes lameness within 7 to 10 days. If foot lameness develops more than 2 weeks after trimming and shoeing, the lameness is unlikely to be related to the farrier. Other primary foot problems include solar bruising, sheared heels, puncture injuries, and thrush. The relative incidence of these problems may be related to the ground conditions if the horse lives out. Early, wet springs increase the incidence of thrush. Long, dry summers resulting in hard ground are associated with an increased incidence of bruising and sore feet.

Laminitis is a problem seen most commonly in two types of pleasure riding horse, obese ponies and older horses and ponies with a pituitary adenoma. Laminitis is often seasonal, occurring most in the spring and early summer and also in the autumn, if a flush of grass occurs late. Navicular disease is not uncommon, and an irregular exercise history may be a predisposing factor. Navicular disease is less common in horses that have never been shod.

Cellulitis can create an acute-onset, non–weight-bearing lameness associated with pyrexia and inappetence. In some horses minor skin abrasions can be identified through which infection was initiated, but in others the primary cause may not be identified.

The incidence of tendon and ligament injuries is low and may be related to the weather and environmental conditions, or to the age of the horse. Excessively deep, muddy pastures and extremely icy conditions may predispose horses to tendonitis or desmitis. Age-related degenerative changes take place in some tendons and ligaments, and in older pleasure riding horses, even those receiving no ridden exercise, sudden onset of a severe, progressive tendonitis of the forelimb superficial digital flexor tendons may develop. Progressive stretching of the hindlimb suspensory apparatus, resulting in dropping of the fetlocks, also occurs in older horses.

Neurological problems such as radial nerve paralysis may result from trauma induced by a kick from another horse in the same field or from a collision with another horse or a static object such as a gate post. In older horses neoplastic lesions may result in secondary lameness. For example, a large melanoma in the gluteal region in a horse I examined created pressure on the sciatic nerve and thus lameness.

Pleasure riding horses are often kept at pasture in a group, with access to field shelters of variable design. Long bone fractures, the result of a kick from a companion horse, are not uncommon, especially during the winter months. Splint bone fractures are also common.

Treatment of many of these conditions is no different than in other athletic sport horses; however, certain constraints may apply that must be considered. No facilities may be available to restrict the horse's exercise or to keep it on its own. Some facilities are completely inadequate for performing clean procedures, such as intra-articular medication, in a safe manner. However, owners often are prepared to spend a disproportionate amount of their income on treating a condition in their much loved horse, despite a guarded prognosis, so it is important to describe all available treatment options. It is also critically important to explain carefully the treatment protocol, and writing it down can be useful to ensure owner compliance. The veterinarian can create a chart that the owner should complete as medication is administered.

In managing osteoarthritis, the fact that a pleasure riding horse lives out and is constantly exercising actually may be of benefit. I prefer to start with the least invasive therapy first and only use alternative methods if that does not work. Some oral nutraceutical agents may be of benefit, as may be intramuscular administration of polysulfated glycosaminoglycans.

Management of wounds can be difficult because client compliance is often poor. I treat wounds in anticipation of the worst-case scenario, using non-steroidal anti-inflammatory drugs, broad-spectrum antimicrobial drugs, and confinement. Complete stall rest is often impossible, and restricted area turnout is a frequent compromise. If a penetrating injury possibly may have entered a joint or tendon sheath, I recommend referral for intensive therapy, stressing to the owner that this injury is potentially life threatening.

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