Plastic Surgery Emergencies

Principles and Techniques

Second Edition

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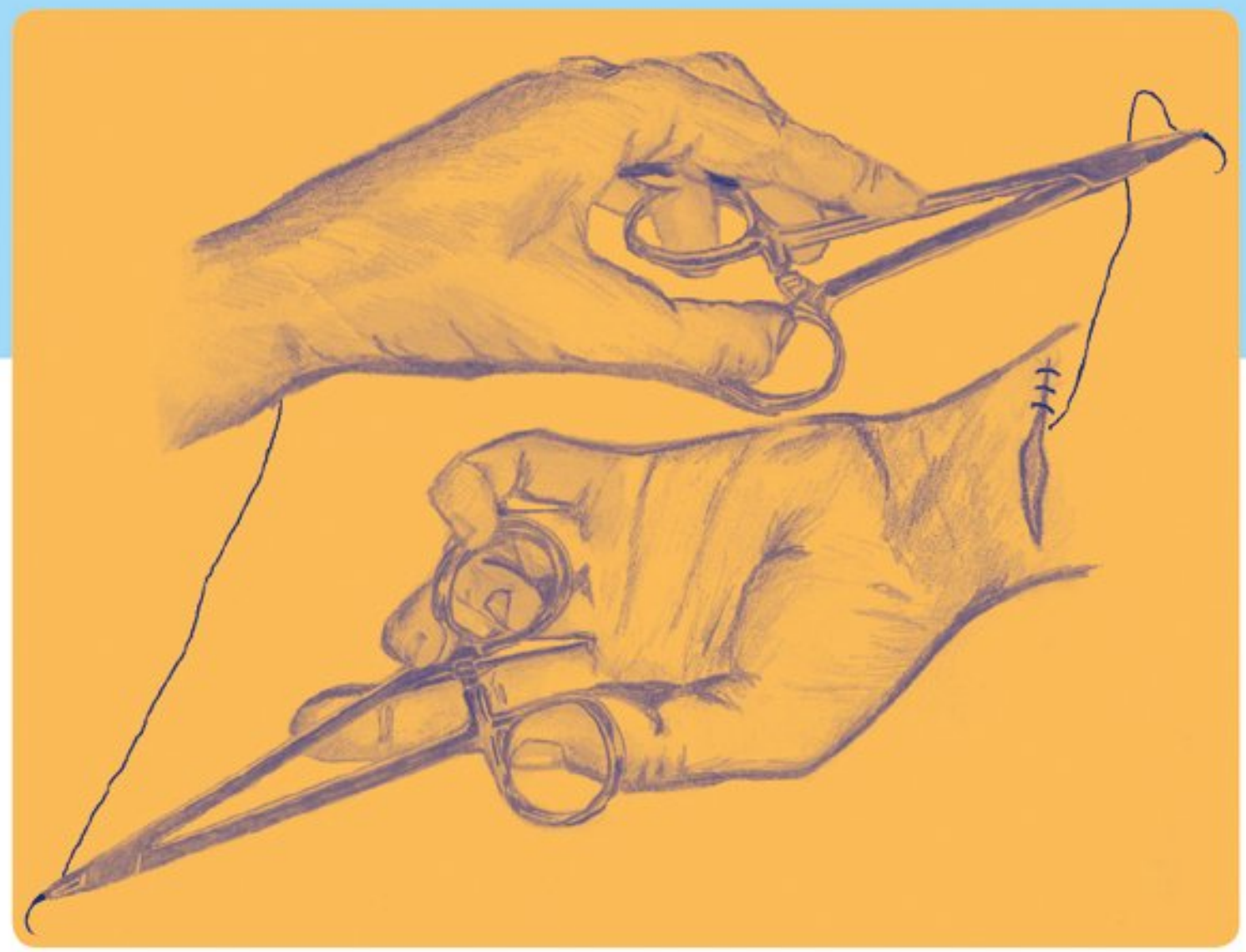

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150 illustrations

Thieme
New York • Stuttgart • Delhi • Rio de Janeiro

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Library of Congress Cataloging-in-Publication Data

Names: Bullocks, Jamal M., author.
Title: Plastic surgery emergencies / Jamal M. Bullocks, MD, FACS, Chief,
Plastic Surgery Department, Kelsey-Seybold Clinic, Clinical Assistant
Professor, Division of Plastic Surgery, Baylor College of Medicine,
Houston, TX [and three others].
Description: Second edition. | New York, NY : Thieme Medical Publishers,
Inc., [2016] | Revision of: Plastic surgery emergencies / Jamal M.
Bullocks ... [et al.]. c2008.
Identifiers: LCCN 2016016016 (print) | LCCN 2016016879 (ebook) | ISBN
9781626231153 (print) | ISBN 9781626231160 (eISBN) | ISBN 9781626231160
(e-book)
Subjects: LCSH: Surgery, Plastic. | Surgical emergencies.
Classification: LCC RD118 .P5363 2016 (print) | LCC RD118 (ebook) | DDC
617.9/52--dc23
LC record available at https://lccn.loc.gov/2016016016

Thieme Publishers New York
333 Seventh Avenue, New York, NY 10001 USA
+1 800 782 3488, customerservice@thieme.com

Thieme Publishers Stuttgart
Rüdigerstrasse 14, 70469 Stuttgart, Germany
+49 [0]711 8931 421, customerservice@thieme.de

Thieme Publishers Delhi
A-12, Second Floor, Sector-2, Noida-201301
Uttar Pradesh, India
+91 120 45 566 00, customerservice@thieme.in

Thieme Publishers Rio de Janeiro,
Thieme Publicações Ltda.
Edifício Rodolpho de Paoli, 25º andar
Av. Nilo Peçanha, 50 – Sala 2508
Rio de Janeiro 20020-906, Brasil
+55 21 3172-2297 / +55 21 3172-1896

Cover design: Thieme Publishing Group
Typesetting by DiTech Process Solutions

Printed in India by Replika Press Pvt. Ltd. 5 4 3 2 1

ISBN 978-1-62623-115-3

Also available as an e-book:
eISBN 978-1-62623-116-0

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It is with great pleasure that I dedicate the newest edition of our text to our senior author, Dr. Samuel Stal. Sam died several years ago after a lengthy illness. He would have been most pleased to see that this work has stood the test of time. Sam was a surgeon who dedicated his life to the treatment of children. He was passionate about teaching and passing on all that he had learned through the years. He was a mentor to all of us who worked with him on this book and he was instrumental in seeing the first edition through to publication. I know that I can speak for all of my coauthors, and thank Sam for being the person that he was—an excellent teacher, but first and foremost, a kindhearted and caring man.

—Larry H. Hollier, MD

Contents

Foreword from the First Edition

"The man who graduates today and stops learning tomorrow is uneducated the day after."

—*Newton D. Baker Jr.*

When I was asked to write a foreword for this book *Plastic Surgery Emergencies*, I must confess, my first thought was, "Is another book truly necessary?" But after reading it, I am both honored and flattered by the request. The browser might first question if this relatively small book fulfills a need, and second ask if it fulfills the need well. The answer to both questions is a resounding "Yes."

With the body of medical knowledge doubling every 5 years or so, the information that must either have been learned or be readily available and understandable to both the young as well as the experienced plastic surgeon continues to increase exponentially. This book distills present knowledge into an easily readable guide to almost any emergency a plastic surgeon might face who is on call in the emergency room, or responding to a late-night/early-morning call from the hospital relating to a postoperative patient.

The authors, who are general plastic surgeons and specialists from the Division of Plastic Surgery here at the Baylor College of Medicine, have culled information from their own surgical experiences, as well as a wide variety of outside sources. They have condensed this knowledge into a small, handy volume, which could easily be read either at one's leisure or immediately prior to assuming the care of a patient. It would be difficult to find an injury or complication from a plastic surgery operation whose emergency treatment is not covered in this book. The authors have detailed the specifics in terms of differential diagnosis and the corrective steps necessary to fulfill the responsibilities of a plastic surgeon answering emergency room call. There are many references to the general principles of treatments—those learned in residency training and in the early years of practice that have stood the test of time. The ability of the surgeon to present an organized treatment plan and then carry it out expeditiously will instill confidence in the patient and the health care personnel involved in the treatment of these patients. The format of the book is conducive to allowing readers to add both personal and technical notes, which will serve them well in the treatment of future patients with similar injuries.

I would be remiss if I didn't call special attention to the lead author, Dr. Jamal M. Bullocks, whose ability and youthful enthusiasm has amalgamated the thoughts and experience of the other authors into a volume that will find great value for all plastic surgeons as well as general surgeons and emergency room physicians.

To those older plastic surgeons who may believe that they have already learned the answers to most of the problems presenting to the plastic surgeon on call, I respectfully suggest that although the problems that presented a decade or two ago may be the same, the answers (i.e., treatment) today may be different. It is to that difference that we are indebted to the authors of this book for their effort and time in providing us with concise and practical answers.

Melvin Spira, MD, DDS
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Preface

The goal in creating *Plastic Surgery Emergencies* is to provide a quick reference guide for health care providers to rapidly assess, triage, and treat patients with problems that are commonly referred to the plastic surgeon. The first version targeted acute care scenarios commonly seen in the emergency department of acutely injured facial and hand trauma patients, as well as patients sustaining a variety of soft-tissue injuries from varying mechanisms of trauma, including burns. This new edition provides the reader with additional content, including added chapters, photos, and sections which will expand the book's audience to outpatient and hospital-based physicians caring for chronically ill patients with wounds. The information presented will prove significantly beneficial to plastic surgeons, otolaryngologists, dermatologists, pediatricians, family practice, and hospitalist and emergency room physicians for treating and triaging patients in the acute and chronic disease setting. Ultimately, the aim is to demystify simple problems that present to these providers and elucidate scenarios that require a higher level of care or follow-up with a plastic surgeon. Our intended audience additionally extends to residents and students training in these fields who experience these encounters as consultations and during on-call activity.

The outline format was preserved with truncated introductory vernacular to confer direct mechanisms for instructions on how to work up, categorize, and initiate the first level of treatment. We hope that this focused and simplified presentation with instructive illustrations, charts, and diagrams will provide a single-source reference in a convenient pocket-sized format.

Acknowledgments

Plastic Surgery Emergencies is a collection of the collaborative knowledge and experience of all the affiliated and full-time faculty of the Division of Plastic Surgery in the Michael E. Debakey Department of Surgery at Baylor College of Medicine. The authors would also like to express their gratitude to the residents, staff, and institutions of the Texas Medical Center for their support in the completion of this work.

Illustrator: Mike de la Flor

Cover Illustrator: Cara Ryan Downey, MD

List of Abbreviations

3D	three-dimensional
ABCs	airway, breathing, and circulation
ABGs	arterial blood gases
ACON	acute compressive optic neuropathy
ACS	abdominal compartment syndrome
AFib	atrial fibrillation
AP	anteroposterior
APB	abductor pollicis brevis
APD	afferent pupillary defect
APL	abductor pollicis longus
APTT	activated PTT
ASA	aspirin
BP	blood pressure
BSA	body surface area
BSAB	body surface area burned
CBC	complete blood count
Chem-7	a basic metabolic panel
CK	creatine kinase
CMC	carpometacarpal
CML	carpometacarpal ligament
CN	cranial nerve
COPD	chronic obstructive pulmonary disease
CRP	C-reactive protein
CSF	cerebrospinal fluid
C-spine	cervical spine
CT	computed tomography
CVP	central venous pressure
CXR	chest X-ray
D5 1/2NS	5%dextrose in 0.45%normal saline
DIC	disseminated intravascular coagulation
DIEAP	deep inferior epigastric artery perforator flap
DIP	distal interphalangeal
DISI	dorsal intercalated segment instability
DJD	degenerative joint disease
DRU	distal radioulnar
DVT	deep venous thrombosis
EBL	estimated blood loss
ECRB	extensor carpi radialis brevis
ECRL	extensor carpi radialis longus
ECU	extensor carpi ulnaris
EDC	extensor digitorum communis
EDM	extensor digiti minimi
EIP	extensor indicis proprius
EMG	electromyogram
EMLA	eutectic mixture of local anesthetics
ENoG	electroneuronography
ENT	ear, nose, and throat
EPB	extensor pollicis brevis
EPL	extensor pollicis longus
ER	emergency room
ESR	erythrocyte sedimentation rate (or sed rate)
FCR	flexor carpi radialis
FCU	flexor carpi ulnaris
FDA	Food and Drug Administration
FDM	flexor digiti minimi
FDP	flexor digitorum profundus
FDS	flexor digitorum superficialis
FFP	fresh frozen plasma
FPB	flexor pollicis brevis
FPL	flexor pollicis longus

GCS	Glasgow Coma Scale
Hct	hematocrit
HDCV	human diploid cell rabies vaccine
Hgb	hemoglobin
I&D	incision and drainage
ICU	intensive care unit
IM	intramuscular or intramuscularly
INR	international normalized ratio
I/Os	intakes/outputs
IP	interphalangeal
IRV	inverse ratio ventilation
IV	intravenous or intravenously
IVF	intravenous fluid
JP	Jackson-Pratt
JVD	jugular venous distention
LDH	L-lactate dehydrogenase
LET	lidocaine-epinephrine-tetracaine
LFT	liver function test
LR	lactated Ringer's
MAP	mean arterial pressure
MCP	metacarpophalangeal
MMF	maxillomandibular fixation
MRI	magnetic resonance imaging
MRSA	methicillin-resistant *Staphylococcus aureus*
MVA	motor vehicle accident
NCS	nerve conduction studies
NOE	naso-orbital-ethmoid
NPO	nothing by mouth
NS	normal saline
NSAID	nonsteroidal anti-inflammatory drug
OOB	out of bed
OR	operating room
ORIF	open reduction and internal fixation
OTC	over-the-counter
PA	posteroanterior
PDS	polydioxanone suture
PEEP	positive end-expiratory pressure
PIP	proximal interphalangeal
PL	palmaris longus
PNM	polynuclear monocyte
POD	postoperative day
PRBCs	packed red blood cells
PT	prothrombin time
PTT	partial thromboplastin time
RBBB	right bundle branch block
RBC	red blood cell
RIG	rabies immunoglobulin
ROM	range of motion
RR	respiratory rate
SBP	systolic blood pressure
SC	subcutaneous or subcutaneously
SCM	sternocleidomastoid
SSEP	somatosensory evoked potential
SMAS	superficial musculoaponeurotic system
SOF	superior orbital fissure
STAT	at once, immediately
TBSA	total body surface area
Tc 99m MDP	technetium 99m methylene disphosphonate
Td	tetanus toxoid
TFCC	triangular fibrocartilage complex
TIG	tetanus immunoglobulin
TMJ	temporomandibular joint
TON	traumatic optic neuropathy
TPA	tissue plasminogen activator

TPN	total parenteral nutrition
TRAM	transverse rectus abdominis myocutaneous flap
TSST-1	toxic shock syndrome toxin-1
UC	ulcerative colitis
VDR	volume diffusive respirator
VISI	volar intercalated segment instability
WBC	white blood cell
ZMC	zygomaticomaxillary complex

1 Wound Management

Evaluation

Accurate assessment of the characteristics of and circumstances surrounding the presentation of wounds is critical to guiding treatment strategies. Therefore, before wound management is planned, a full evaluation of the wound must be undertaken with the following considerations.

Acute Wounds

- Assess size, shape, and location.
- Determine the timing of the wound—acute (time elapsed since injury) versus chronic (persistent > 3 months).
- Establish laceration, avulsion, or chronic open wound.
- Evaluate the wound for odor, exudate, purulent drainage, bleeding, and debris.
- Determine if there is exposure of vessels, tendons, nerves, joint, muscle, or bone.
- Evaluate for foreign bodies in the wound; consider X-ray evaluation—if the history is inconsistent with clinical evaluation.

Chronic Wounds

Chronic wounds require investigation into reasons why proper wound healing is not accomplished (**Table 1.1**).

Therefore, chronic wounds warrant serologic evaluation to include

- White blood count.
- Hct/Hgb.
- Albumin.
- Prealbumin.
- ESR, or sed rate.
- C-reactive protein.
- LFTs, hepatitis panel.
- Blood glucose.
- Biopsy of wound.
- Culture of wound.

Table 1.1 Contributors to poor wound healing

Local factors that contribute to poor wound healing	Patient comorbid conditions that contribute to poor wound healing
Tissue ischemia	Anemia
Venous hypertension	Hypoxia
Edema	Advanced age
Infection	Malignancy
Microbial contamination	Poor nutrition
Bacterial > 10^5 or 10^4 group B Streptococcus species	Vitamin deficiencies
Fungus	History of radiation
Atypical mycobacteria	Severe systemic disease (e.g., diabetes, hepatic disease)
	Collagen vascular diseases
Wound tension or pressure > 30 mm Hg	Immunosuppression
	Smoking
Presence of foreign bodies	Obesity

Treatment

Irrigation

Acute Wounds

Irrigation in the acute wound setting is designed to remove blood, foreign bodies, debris, and bacteria from a wound. This can easily be accomplished with a 1-L bottle of normal saline with two or three holes punched into the cap with an 18-gauge needle. When squeezed forcefully, it serves as an effective pressurized irrigator. The wound should be irrigated until all visible debris is washed away. Anesthetizing the wound prior to irrigation and débridement provides for greater patient comfort and allows for aggressive decontamination of the wound.

Chronic Wounds

Simple surface irrigation of a chronic wound is usually only marginal and minimally effective. It can be useful at the bedside if there is debris grossly evident in the wound. Studies have shown that pressure irrigation at approximately 70 psi is needed to reduce bacteria count and particulate

matter. This is best done in the operating room with a pulse lavage or a jet lavage system. If needed, a thorough débridement of devitalized tissue can also be done in the operating room. Tangential hydrotherapy via the Versajet (Smith & Nephew) device is often useful for irrigation and mechanical débridement. A chronic wound may benefit from biopsy and tissue culture as clinically indicated.

Débridement and Hemostasis

Adequate débridement of devitalized tissue and skin edges is important in preparing the contaminated wound for closure. The skin is highly vascular and excessive skin removal is usually not necessary. Jagged skin edges should be trimmed to facilitate an easier closure. Hemostasis can be achieved with pressure, silver nitrate, topical fibrin, Surgicel (Johnson & Johnson), topical thrombin or epinephrine (1:100,000), suture ligature (absorbable for small vessels and nonabsorbable for larger vessels), or cautery (**Fig. 1.1**).

If there is any question as to the viability of the tissue, it is better to allow the tissue to demarcate rather than to débride it initially. Tissue of questionable viability can often undergo necrosis after débridement due to retrograde thrombosis. Once demarcated, the tissue can be débrided to healthy bleeding tissue. This approach allows conservative preservation of the tissues without causing additional tissue loss and disfiguration.

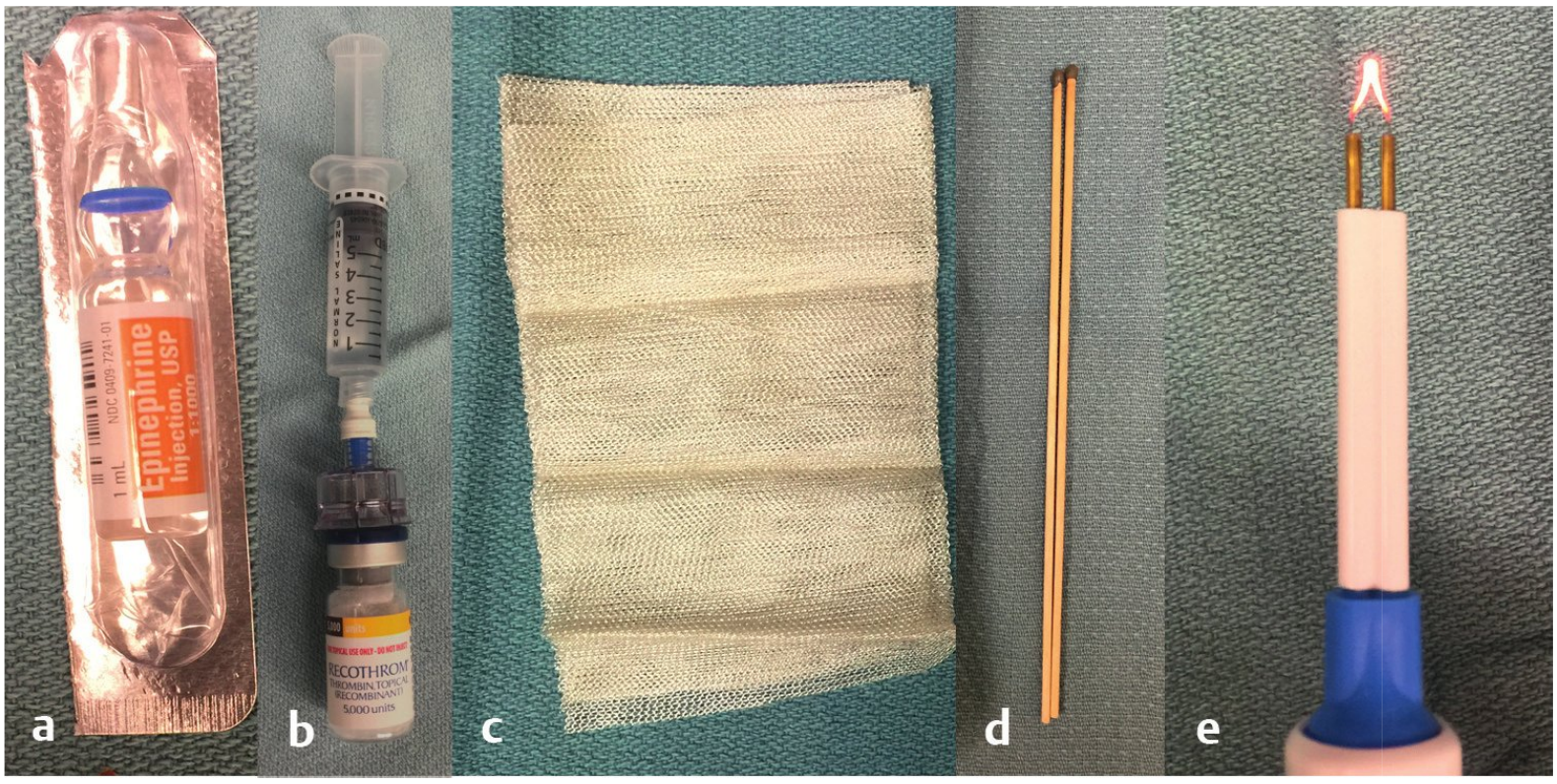


Fig. 1.1 Useful tools for establishing hemostasis in acute wound management: **(a)** topical epinephrine diluted to 1:100,000, applied with gauze, **(b)** topical thrombin spray, **(c)** oxidized methylcellulose (Surgicel), **(d)** coagulation with silver nitrate, or **(e)** disposable portable cautery device.

Closure and Antibiotics

Prior to closure, irrigation, débridement, hemostasis, and trimming of the skin's jagged edges should be performed. A tension-free closure will help to ensure healing with an optimal scar.

Most clean lacerations, if addressed in < 8 hours, have minimal contamination and can be closed primarily without the need for antibiotics. Clean wounds presenting after 8 hours can be closed after débridement of the entire wound and sharp débridement of edges. This would include stab wounds, lacerations by window or glass, and clean avulsions. In effect, sharp débridement and decontamination of these late presenting wounds converts them into fresh wounds that are more appropriate for closure. On the other hand, contaminated wounds, such as wounds with dirt and debris, should be treated with systemic antibiotics with additional consideration for tetanus prophylaxis.

Choice of antibiotics should usually cover gram-positive organisms (cefazolin 1 g IV). Due to the increase in methicillin-resistant *Staphylococcus aureus* (MRSA), certain wounds may require other antibiotics for coverage (clindamycin 600 mg IV or vancomycin 1 g IV). The astute caregiver should take advantage of administration of a single IV dose of antibiotics to wounds at risk for contamination while the patient is in a health care setting undergoing evaluation.

If the wound is grossly contaminated with debris or if the patient is diabetic, broader-spectrum antibiotics should be considered, for example, Avelox (Bristol-Myers Squibb) 400 mg IV or by mouth daily, Zosyn (Wyeth Pharmaceuticals) 3.375 g IV every 6 hours, imipenem 1 g IV every 8 hours, or combination therapy.

Contaminated wounds should be left open except for those on the face. Wet to dry dressing changes should be done at least twice a day. In addition, the patient should shower frequently and wash the wound with soap and water.

A 5- to 7-day course of outpatient antibiotics may also be warranted. Coverage should include gram-positive and MRSA coverage (oral clindamycin 450 mg by mouth four times a day, or oral trimethoprim/sulfamethoxazole twice a day). Cephalexin is not effective in treating a contaminated wound. Rarely, acute wounds will require inpatient treatment with IV antibiotics. Usually débridement and prophylactic oral antibiotics should suffice. In the case of more subacute or chronic wounds with gross contamination or purulence, consideration should be given to admission, IV antibiotics, and formal débridement.

Skin-Flap Wound Closure

If the patient has an avulsed skin flap, the flap should be tacked down where it lies (**Fig. 1.2**). **Do not put tension on the skin flap for complete closure**. Tension will lead to total flap loss. First, débride all devitalized tissue and then inset the flap so that no tension is present. Distal margins of the flap will usually undergo necrosis. Plan on additional débridement as the flap demarcates.

Tetanus Prophylaxis

Tetanus-prone wounds are those that are old (> 6 hours), deep (> 1 cm), and/or contaminated, especially those that involve rusty metal, feces, or soil. Depending on the degree of contamination, tetanus toxoid, tetanus immunoglobulin, or complete immunization may be required. Specific recommendations for tetanus prophylaxis are included in **Table 1.2** through **Table 1.4**.

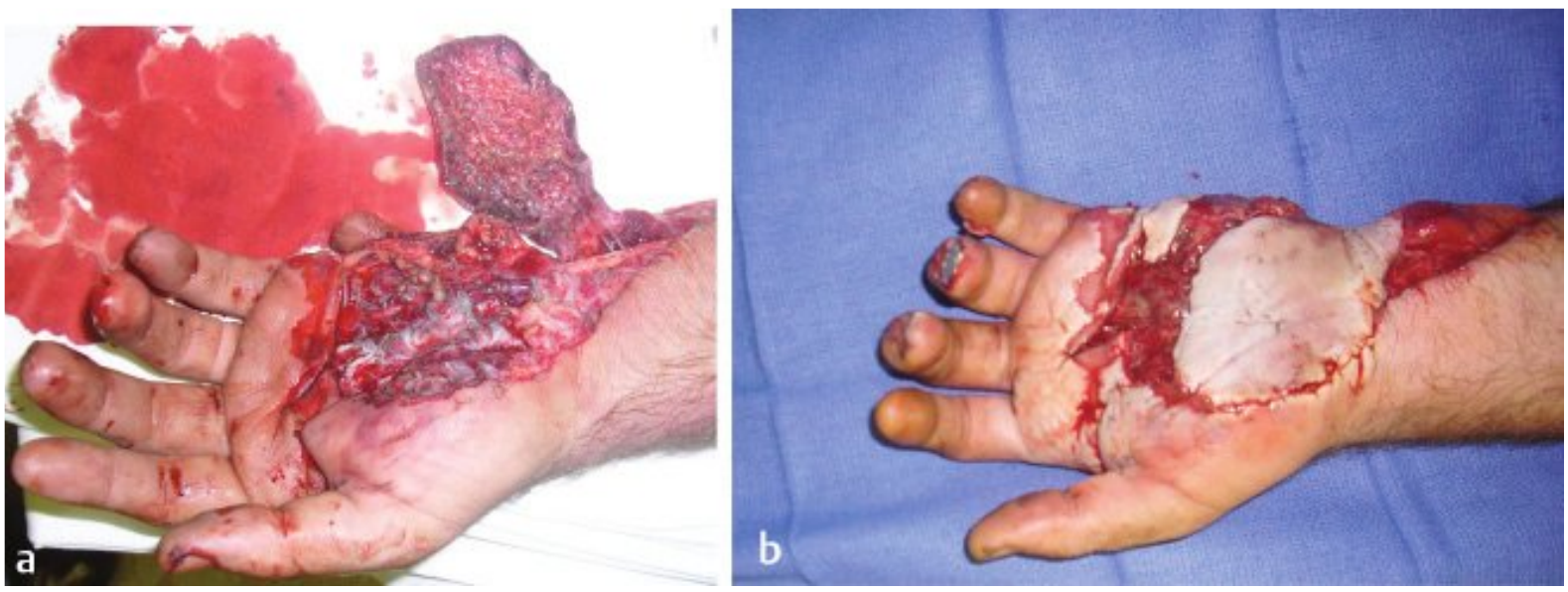


Fig. 1.2 **(a)** Avulsed skin flap. **(b)** Avulsed skin flap tacked down without tension.

Table 1.2 Tetanus-prone wounds

Clean (low risk)	Tetanus prone (high risk)
Clean incised wound	Any wound or burn > 6 h old
Superficial graze	Contact with soil, feces, compost, or saliva
Scalded skin	Puncture-type wound Avulsion wounds Crush open wounds
	Infected wound
	Compound fracture
	Large amount of devitalized tissue
	Animal or human bite Burns and frostbite

Table 1.3 Immunization status and tetanus risk

Immunization status	Low risk	Moderate risk	High risk
Fully immunized, < 5 y since booster	None	None	None
Fully immunized, 5–10 y since booster	None	Td	Td
Fully immunized, > 10 y since booster	Td	Td	Td + TIG
Incompletely immunized or uncertain	Full tetanus vaccine	Full tetanus vaccine + TIG	Full tetanus vaccine + TIG

Abbreviations: Td, tetanus toxoid; TIG, tetanus immunoglobulin.

Table 1.4 Recommendations for vaccination with tetanus immunoglobulin

Patient	Dosage	Treatment
Adult	250–500 U	For both patient groups, the vaccine should be given IM in the opposite upper extremity (arm) to the tetanus toxoid
Pediatric	250 U	

Follow-up

Careful and frequent follow-up is imperative for all wounds. Patients should be asked to return to the clinic or general practitioner within 3 days if possible and educated on all the signs and symptoms of an infection. Specific instructions on wound care and antibiotic therapy are crucial to guaranteeing patient compliance and ultimately a favorable prognosis.

2 Anesthesia and Wound Closure

All wounds should be clean of foreign bodies and adequately irrigated (see Chapter 1). Hemostasis is achieved with pressure, silver nitrate, fibrin, Surgicel, thrombin, or suture ligature (absorbable for small vessels and nonabsorbable for larger vessels) to prevent hematoma formation. Any devitalized tissue, as well as jagged edges, should be trimmed for optimal cosmesis.

Wounds can be closed with sutures, staples, skin tapes, or wound adhesives. Generally, wounds should be closed in layers using appropriate sutures and the epidermis reapproximated so that it is relatively tension free and everted if possible. Everted skin edges eventually flatten out and produce a level wound surface, whereas inverted skin edges have a tendency to produce a depressed scar.

To guarantee a successful wound closure, a comfortable environment should be created for both the practitioner and the patient. The use of analgesics, local anesthesia, and even sedation are helpful adjuncts in reducing patient anxiety. This will ultimately increase the likelihood of more precise closure.

Anesthesia

Local Anesthetics

Local anesthetics work by affecting the sodium (Na^+) channels on afferent sensory nerves. Local anesthetic enters the cell membranes and reversibly binds to Na^+ channels. This reversible binding incapacitates the cells so that they are then unable to depolarize. Lidocaine is the most commonly used and easily accessible local anesthetic agent in the emergency room (ER). Epinephrine should routinely be used with any local anesthetic to assist in hemostasis and to prolong duration. The vasoconstrictive properties lead to decreased absorption so that larger doses of anesthetic can also be used without systemic toxicities.

The maximum safe dose for lidocaine is 4 mg/kg. With the addition of epinephrine (usually at 1:100,000 concentration), the maximum dose increases to 7 mg/kg. A 1% solution of lidocaine is defined as

1 g/100 mL = 10 g/1,000 mL = 10,000 mg/1,000 mL = 10 mg/1 mL

Example Maximum dose of lidocaine with epinephrine in a 70-kg (154-lb) man

70 kg × max dose (7 mg/kg) = 490 mg of lidocaine
490 mg × 1 mL/10 mg (concentration of 1% lidocaine) = 49 mL of 1% lidocaine with epinephrine

Although many textbooks have cited the contraindication for using epinephrine in end arteries such as those in digits and the nose, recent studies have exonerated epinephrine as the culprit in causing tissue necrosis. Therefore, it is safe to use lidocaine with epinephrine virtually anywhere on the body. For the maximum effects of epinephrine to take place, the practitioner should wait 10 to 15 minutes. **Table 2.1** provides other local anesthetics that may be used, with their maximum dosages and duration of action.

Once you have chosen your local anesthetic, it is useful to add bicarbonate to the solution, particularly when the patient is awake. The pH of local anesthetic solutions is generally buffered to between 4 and 5 to prolong shelf life. This acidity routinely leads to a burning pain upon injection. Adding a base such as bicarbonate to the local anesthetic not only alleviates the pain but also accelerates the action because the higher pH favors the nonionized form of the anesthetic, which crosses the cell membrane more easily. The addition of 1 mL of a 1-mEq/mL solution of bicarbonate for every 9 mL of local anesthetic can alleviate the burning and improve patient comfort. Warming the anesthetic, using a smaller-caliber needle (25 gauge or higher), and injecting by inserting the needle within the wound (instead of through the skin) all help in reducing pain felt by the patient.

Table 2.1 Local anesthetics for wound closure

Drug	Onset	Maximum dose mg/kg (with epinephrine mg/kg)	Duration (with epinephrine)
Lidocaine	Rapid	4.5 (7)	120 min (240 min)
Mepivacaine	Rapid	5 (7)	180 min (360 min)
Bupivacaine	Slow	2.5 (3)	4 h (8 h)
Procaine	Slow	8 (10)	45 min (90 min)
Chloroprocaine	Rapid	10 (15)	30 min (90 min)
Etidocaine	Rapid	2.5 (4)	4 h (8 h)
Prilocaine	Medium	5 (7.5)	90 min (360 min)
Tetracaine	Slow	1.5 (2.5)	3 h (10 h)

Topical Anesthetics

- *Eutectic mixture of local anesthetics (EMLA):* 2.5%prilocaine and 2.5% lidocaine cream.
- *Lidocaine-epinephrine-tetracaine (LET) gel:* 4%lidocaine, 1:2,000 epinephrine, 1%tetracaine.

Topical anesthetics are more commonly used in the pediatric patient to alleviate the pain associated with local injections. Although effective, they are not nearly as effective as local anesthetic infiltration in providing anesthesia. The duration and depth of the blockade is dependent on the amount of time the cream is in contact with the skin. Apply to the wound and then cover with a Tegaderm (3M) or other occlusive dressing. The cream or gel will usually need to be in place for at least 45 minutes before *any* anesthetic effect is achieved.

Digital and Facial Nerve Blocks

Please see respective chapters: Upper Extremity Injuries (Chapter 18) and Facial Trauma (Chapter 8).

Conscious Sedation

Most simple and even moderately complex lacerations can be repaired with relative ease with local anesthetics alone in the adult patient. However, fear and anxiety are common in the pediatric patient. Therefore, it may be difficult to repair a laceration in the understandably uncooperative pediatric patient. Conscious sedation may be used if conditions are appropriate and the necessary precautions followed. A well-trained pediatrician or anesthesiologist should be consulted for administration of conscious sedation, especially if the surgeon's experience is limited in this field. Full monitoring by a nurse is required throughout the procedure.

Prior to administering conscious sedation, a complete history and physical examination should be obtained, including

- Age.
- Weight (measured, not estimated, whenever possible).
- Vital signs.
- Oxygen saturation.
- Absence of head injury (document).
- Heart, lung, neurologic, and mental status.
- Complexity and location of injury.

Prior to sedation, there should be

- No oral liquids for 2 hours prior to procedure in children < 2 years of age—3 hours if > 3 years.
- No milk or solid food for 8 hours prior to the procedure.

During the procedure,

- Maintain continuous oxygen saturation and heart rate monitoring.
- Record vital signs and blood pressure every 15 minutes for conscious sedation and every 5 minutes for deep sedation.
- Record drug dose and time administered.
- Record state of consciousness and response to stimulation.

As precautionary measures, ensure that

- Nasal cannula and intubation tray are available during the procedure.
- Reversal agents are ready, prepared in syringe (Narcan [DuPont Pharma] 0.4 mg IV push every 2 to 3 minutes as needed, flumazenil 0.2 mg IV push given over 30 seconds, then 0.3 mg IV push given over 30 seconds as needed, maximum total dose 3 mg).
- Suctioning apparatus and canister are available.
- Nursing staff is in the room during the procedure to assist.
- Drug combinations that include amnestic and analgesic effects are used.

The drugs commonly used are as follows (Table 2.2):

- For adults.
  - Short procedure: Versed (Hoffman LaRoche) + fentanyl.
  - Moderate interval procedure: Morphine + Ativan (Biovail Pharmaceuticals, Inc.).
- For pediatric patients.
  - Ketamine + Versed.

For all patients, start with a subtherapeutic dose, then rebolus in small intervals to titrate sedative effect.

Sutures

A variety of suture materials are available, and, in general, they can be differentiated based on the following categories (**Table 2.3**):

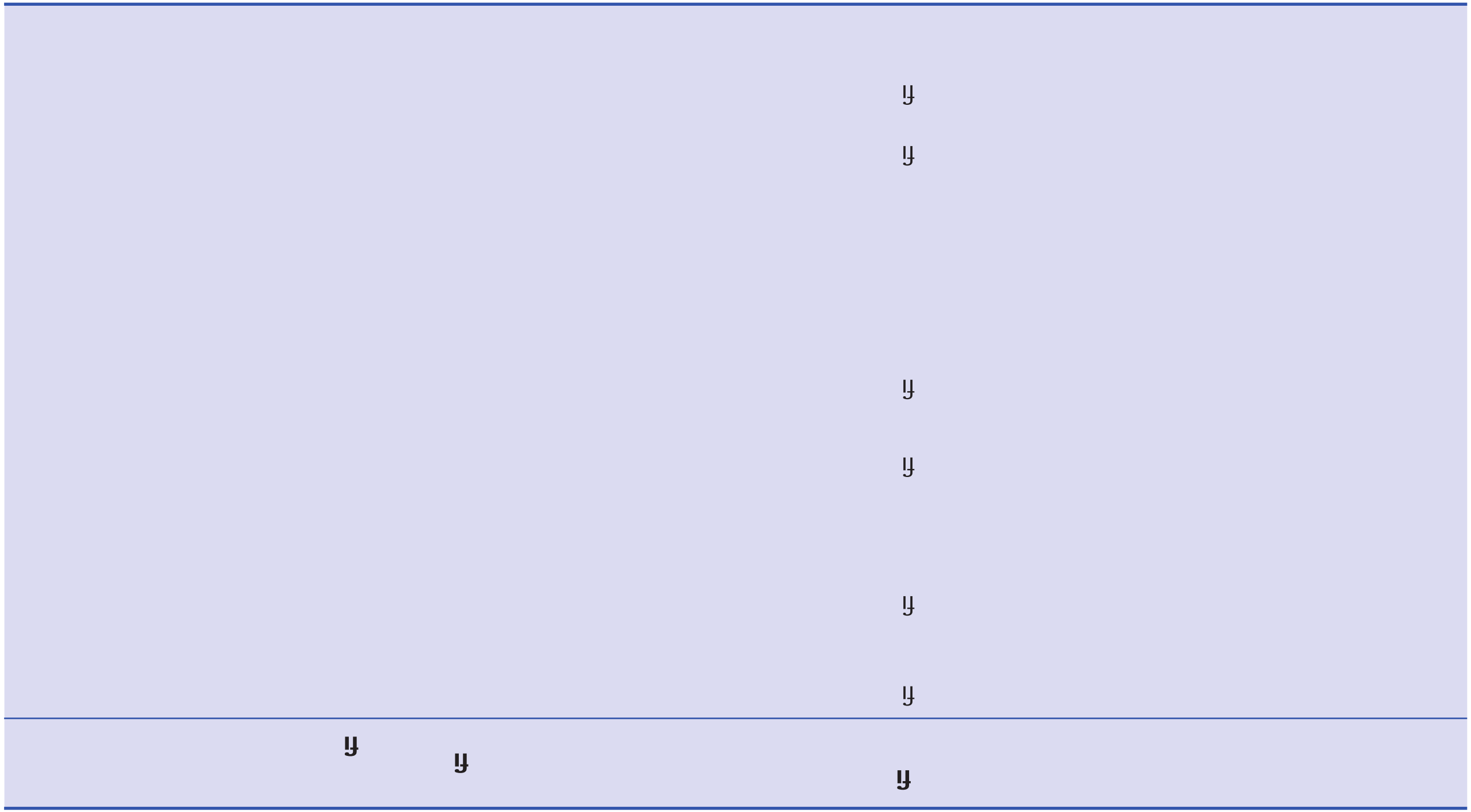
- Absorbable versus nonabsorbable.
- Braided versus nonbraided.
- Tensile strength.
- Half-life.

A variety of needles are also available and can generally be classified as taper or cutting.

- Taper/round needle: Use in muscle, cartilage, and mucosa.
- Cutting needle: For skin.
  - Use a half-circle cutting needle for subcutaneous tissue.
  - Use a 3/8-circle cutting needle for skin.

Suture Techniques (Fig. 2.1)

- **Simple interrupted:** General tissue approximation.
- **Continuous running or running baseball:** An effective and fast continuous suture for long lacerations.
- **Vertical mattress:** Most effective stitch for everting skin edges. Be careful to not set tension too tight to prevent tissue necrosis.
- **Horizontal mattress:** Effective in everting skin edges. Be careful to not set tension too tight to prevent tissue necrosis.
- **Running subcuticular:** A buried dermal suture for closing skin in clean wounds without jagged edges.
- **Staples:** Simple and fast closure commonly used in the scalp or dirty wounds to be closed loosely to allow drainage. Staples should be removed in 5 days to avoid epithelialization and a poor cosmetic result.
- **Adhesive skin tape:** Used to reapproximate small lacerations with very little tension.
- **Dermabond (Ethicon):** Skin adhesive that can be used for clean lacerations without jagged edges. After the wound is adequately prepared, reapproximate skin edges with a finger and apply the first coat, let it dry for 20 seconds, and then apply a second coat.
- In conclusion, the astute practitioner will repair lacerations and wounds in the following order:



1. Anesthetize the patient's wound.
2. Débride it meticulously with removal of jagged edges, devitalized tissue, and any foreign bodies.
3. Properly irrigate the wound in preparation for closure.
4. Obtain hemostasis.
5. Repair the wound in layers with care to reapproximate the dermal and epidermal layers of the skin to provide the patient with the best cosmetic result.

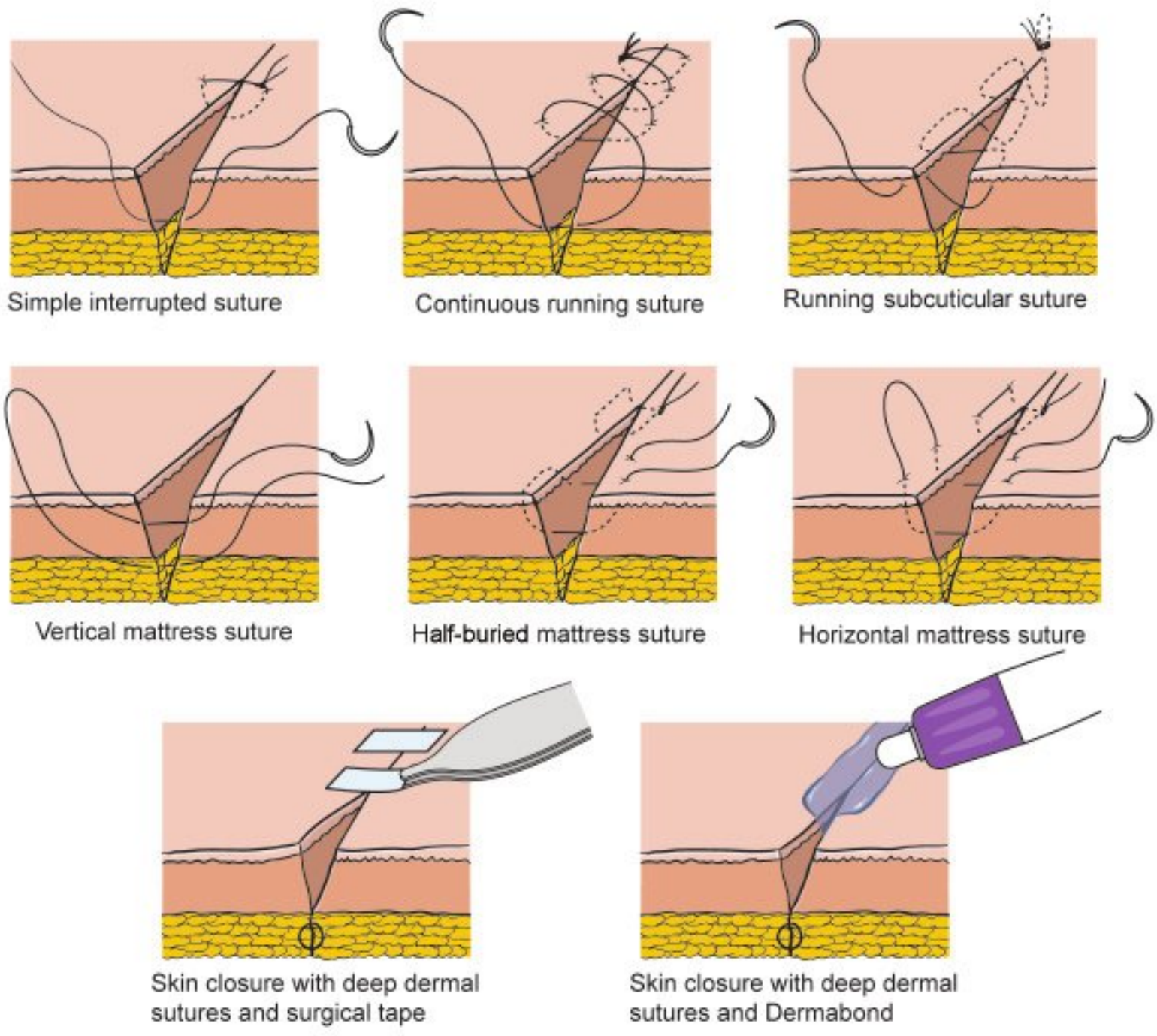


Fig. 2.1 Suture techniques.

3 Pressure Sores

The treatment of pressure sores can be a long and difficult challenge. Pressure sores are frequently a secondary sequela to the sedentary patient with a more complicated primary issue. Commonly, patients with pressure sores present with multiple comorbidities. It is essential to keep in mind that the likely source of fever and infection is oftentimes not the sore itself, since most sores are open to drain. Each case warrants a complete evaluation by the examiner to rule out the pressure sore as the likely cause of an infection.

Pressure Sore Staging System (Fig. 3.1)

- **Stage 1:** Intact skin with nonblanchable erythema.
- **Stage 2:** Superficial ulcer involving partial thickness of the epidermis and dermis; usually presents as an abrasion, blister, or very shallow ulcer.

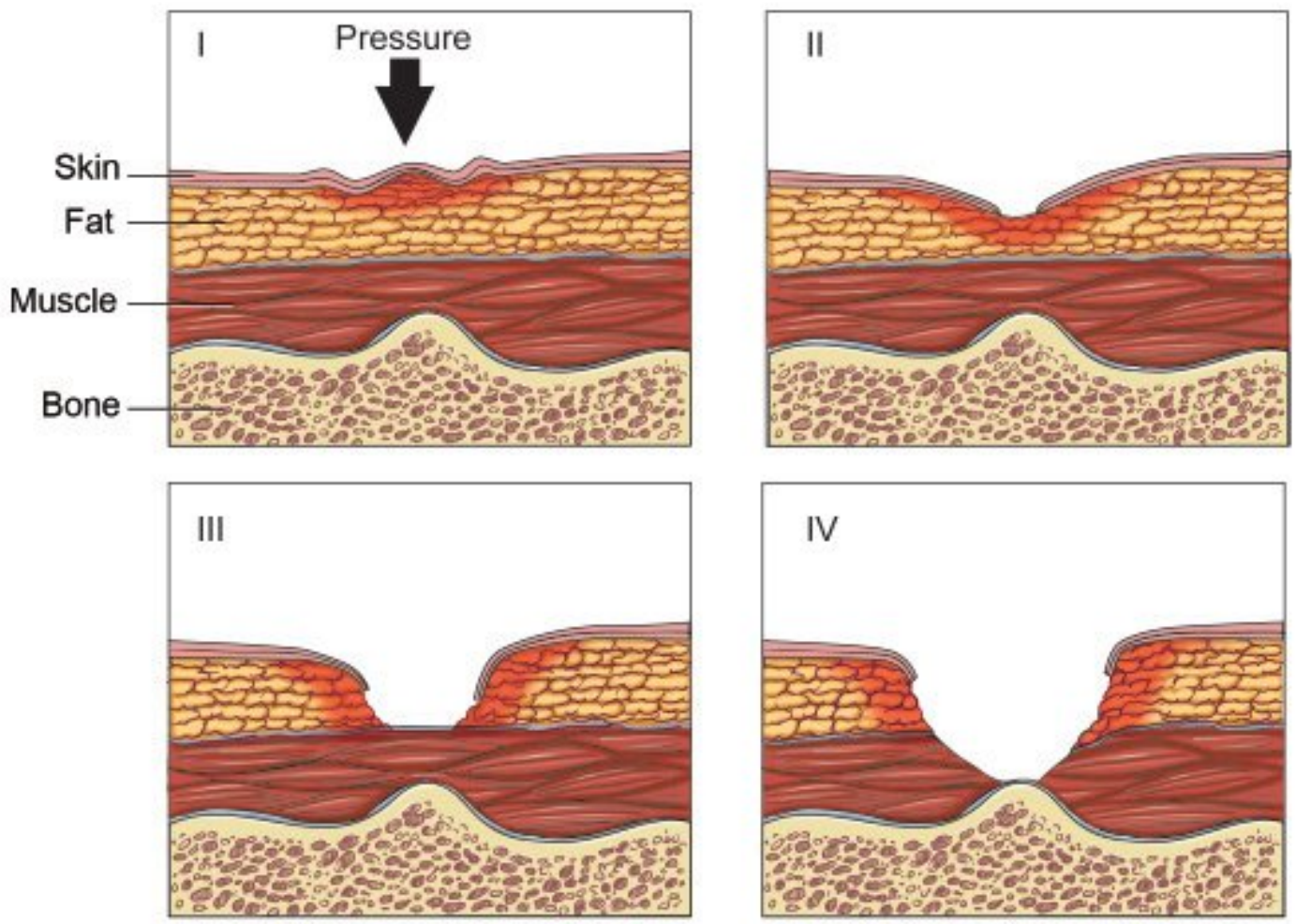


Fig. 3.1 Pressure sore staging system.

- **Stage 3:** Full-thickness skin loss down to the subcutaneous tissue, which does not extend beyond underlying fascia.
- **Stage 4:** Full-thickness skin loss down through subcutaneous tissue with involvement of muscle, bone, tendon, ligament, or joint capsule.

Evaluation

Position the patient in a well-lit area to facilitate visualization of the ulcer. Gently probe the wound and assess for fluid collection or purulent drainage. If pus is present, incision and drainage (I&D) should be performed and the wound irrigated copiously and packed wet to dry (see below). Obtain a culture and samples of the purulent material. Necrotic soft tissue is common. If it is devoid of purulent drainage, it is unlikely to be the source of sepsis. Copious drainage may be indicative of a much larger wound beneath the skin.

Subcutaneous fat and muscle are more prone to ischemia than skin. Therefore, intact skin (possibly with small eschar) may harbor a large area of necrotic tissue below, making the wound unstageable. Often, an eschar at or above the adjacent skin layer is indicative of partial skin thickness loss. An eschar that is depressed may represent full-thickness skin loss.

Larger sores may warrant radiographic evaluation to assess the extent of soft tissue involvement and possible bony involvement. CT scan or MRI will provide more information versus traditional X-ray. An MRI should be performed to rule out osteomyelitis if bone cultures are not available. Check routine studies—CBC, blood cultures, CXR, blood sugar, albumin and prealbumin, ESR, CRP, and urinalysis. Rule out other possible systemic causes of fever—pneumonia, central lines, and urinary tract infections. Check for incontinence.

Treatment

General Treatment for All Ulcers

- Alleviation of pressure—place patient on an air-fluid mattress; use pillows, egg cartons, donuts.
- Avoidance of shearing forces.
- Frequent turning of the patient, hourly if possible.
- Cleaning or diverting away incontinence—use of Foley and suprapubic catheter, rectal tube, or colostomy.
- Maximizing nutrition (albumin > 3.0, prealbumin > 18).

For Staged Ulcers

- *Stage 1:* Use moisturizers to prevent dryness.
- *Stage 2:* No débridement is necessary; use occlusive dressings such as polyurethane film (Duoderm [Convatec Inc.]) or hydrocolloids.
- *Stages 3 and 4:* Sharp débridement is often necessary with the addition of pulse lavage irrigation. Wounds are packed wet to dry with Kerlix (Kendall Co.). Consider bone biopsy to assess for osteomyelitis and to obtain bone cultures to guide antibiotic therapy.

The initial treatment and management of a pressure sore should include trying to alleviate the pressure that initially caused the wound. Even a clean wound will have trouble healing if the inciting event is still present. Proper débridement and washout is paramount. A minimal to moderate amount of necrotic tissue can often be débrided at bedside with local anesthesia, scissors, and a scalpel. Larger areas should be done in the operating room because of pain and the potential for uncontrolled bleeding. Sharp débridement should be performed down to healthy vitalized tissue that bleeds. Properly anesthetizing the patient will help with pain control and hemostasis. Refer to Chapter 2 for a list of local anesthetics to use.

Wet to dry dressings can be done with normal saline, 0.25%Dakin's solution, 0.25%acetic acid, and gauze. Kerlix gauze is commonly used because of the large surface area it can cover. Kerlix gauze is soaked in one of the preferred solutions and then squeezed dry. The moist gauze is then inserted and packed directly into the wound to cover the entire surface of the wound. Do not place moist Kerlix directly on the skin, since this can lead to skin maceration. The dressing should be changed two to three times per day depending on how dirty the wound is. Wet to dry dressings keep the wound moist and allow for mechanical débridement of devitalized tissue once the gauze dries, hence the name "wet to dry." Dakin's 0.25%or acetic acid 0.25%solution should be used in the infected purulent wound; however, it should be discontinued once the wound is clean, since both solutions can inhibit tissue growth and healing. Most hospitals have a wound care team that can assist with bedside pulse lavage and a small amount of sharp débridement by the wound ostomy nurse. Consult the wound care team for assistance with dressing changes, bedside pulse lavage, and minor débridements. Duoderm may be placed on either side of the wound, thereby avoiding frequent tape contact directly with the skin.

Enzymatic débridement is a common adjunct to the management of pressure ulcers. Agents such as Santyl (Smith and Nephew) are frequently applied to the wound to provide constant proteolytic débridement.

Although frequently used, they are by no means a substitute for sharp mechanical débridement of necrotic tissue. These agents have low morbidity and assist in cleaning up the small amounts of necrotic tissue that are often hard to completely remove during bedside débridements.

If the patient is seen in the emergency room and there is no cellulitis, no elevation in white blood cell count, and no purulent drainage, then the wound can be débrided as necessary and the patient can be seen as an outpatient. In these cases, instruct the family on (1) dressing changes every 8 hours, (2) the importance of keeping the wound clean, and (3) the need for frequent turning of the patient. If the patient presents with cellulitis and purulent drainage of the wound, then admission to the hospital should be considered, especially if the patient has other comorbidities.

Silvadene (Sanofi-Aventis Pharmaceuticals), Mafenide Acetate Cream, or Betadine (Purdue Products LP) can be used in selected cases of wounds with superficial eschars. These are usually small sores that involve only the dermis. These antimicrobial agents can help decrease the chance of an infection while the wound heals by secondary intention. Culture swabs are of little use because all wounds are colonized, even in the clean granulating wound. A quantitative tissue biopsy can be obtained to evaluate tissue bacterial counts ($> 10^5$ per gram of tissue), and bone biopsy to rule out related osteomyelitis.

4 Bite Wounds

Bite wounds from animals, insects, and humans are commonly present in the emergency setting. More severe and violent bites can be associated with complex composite tissue defects with devitalized tissue.
General management includes:

1. Infiltrate local anesthesia to anesthetize the wound to allow thorough evaluation and débridement.
2. Removal of foreign bodies (teeth) and débridement of devitalized tissue.
3. Copious irrigation with NS.
4. Determine if tetanus or rabies prophylaxis is indicated.
5. Repair of wound/laceration—consider loose closure or leaving open if infected or contaminated.
6. Postclosure antibiotics and monitoring.

Human Bites

Human mouths contain some of the most concentrated and varied bacteria. Organisms include *Eikenella, Staphylococcus,* viridans streptococci, and *Bacteroides*. The general principles of contaminated wound management, as mentioned above, apply to all human bite wounds. In the acute bite, the wound must be assessed fully and irrigated copiously. The patient should be placed on appropriate prophylactic antibiotics and followed closely for any signs of infection.

The initial injury often appears minor to the patient; thus no care is sought until an infection develops. It is important to fully assess the patient in the urgent care setting and triage for possible hospital admission, IV antibiotics, and operative management. Bite injuries require careful evaluation for a deep infection because of the relatively benign presentation of their appearance. At times, due to the close proximity of the skin and underlying structures, nerve and tendon injuries may also be present. Also, due to the inherent depth of penetration by the teeth, microorganisms easily seed the depth of wounds, allowing rapid dissemination along the deep planes of the fascia and subcutaneous tissue. Therefore, rule out a deep injury even when the presentation is a minor wound such as an abrasion.

Assessment and Treatment

1. Evaluate wound for depth, foreign body, drainage, and cellulitis.
2. Assess for crepitus (subcutaneous emphysema), which would indicate gas-forming organisms along the deep planes.
3. Débride devitalized tissue and copiously irrigate.
4. Loose closure versus pack wound—facial bites should be closed for the best cosmetic results.
5. Treat with antibiotics.

Closed-Fist Injury (Fight Bite)

With closed-fist injuries, the force of the blow to the mouth will often penetrate the skin over the metacarpophalangeal joint to lacerate or infect the extensor tendon and contaminate the underlying joint, such as the metacarpophalangeal joint, with bacteria from the mouth. When the hand is placed back into a neutral position, the bacteria can be displaced, resulting in more proximal contamination. Fight bite wounds not only involve soft tissues but also can infect joints and tendons. Aggressive incision and drainage, irrigation, and débridement in the operating room should be considered for grossly contaminated wounds and those that present late.

1. Evaluate wound for depth, foreign body, drainage, and cellulitis.
2. Assess for crepitus (subcutaneous emphysema), which would indicate gas-forming organisms along the deep planes.
3. Evaluate the integrity of the extensor and flexor tendons (flexor tenosynovitis).
4. Assess for loss of joint height, which would indicate metacarpal head fracture.
5. Obtain hand series (rule out metacarpal head fracture, osteomyelitis, and dental foreign body).
6. Débride devitalized tissue and copiously irrigate.
7. Close the wound loosely or leave the wound open and perform daily dressing changes with gauze.
8. Treat with antibiotics.

See Chapters 14 and 16 for management of fractures of the metacarpal head and extensor tendon injuries, respectively, associated with fight bites.

Antibiotics

All patients seen in the emergency setting should receive a single dose of IV antibiotics. IV antibiotics should be continued in those who require admission for more complicated infections. Those who can be discharged home are released on the appropriate oral regimen with close follow-up within 1 week.

- *First-line IV:* Unasyn (Pfizer Pharmaceuticals) 1.5 g IV every 6 hours or clindamycin 600 mg IV every 6 hours + Levofloxacin 500 mg IV daily.
- *First-line oral:* Augmentin (GlaxoSmithKline).
  - *Adult:* 875/125 mg twice a day × 10 days.
  - *Pediatric:* 45 kg/day twice a day × 10 days.
  - *Alternatives:* Moxifloxacin 400 mg daily × 10 days or clindamycin 450 mg four times a day + Bactrim DS twice a day × 10 days.

Cat

Cat bites are deeply penetrating wounds that are heavily contaminated, and approximately 80% of these wounds become infected. Organisms include *Pasteurella multocida* and *Staphylococcus* species. Irrigate heavily, wash daily, treat with antibiotics, and see below for rabies vaccination criteria. Evaluate for tetanus prophylaxis. Do not close the wound.

Antibiotics

- *First-line oral:* Augmentin.
  - *Adult:* 875/125 mg twice a day × 10 days.
  - *Pediatric:* 45 kg/day twice a day × 10 days.
  - *Alternatives:* Doxycycline 100 mg twice a day × 10 days or cefuroxime 0.5 g twice a day × 10 days.

Dog

Dog bites constitute 80 to 90% of all animal bites. Organisms *include P. multocida, Bacteroides,* viridans streptococci, *Fusobacterium,* and *Capnocytophaga*. Massive force can often cause significant avulsion injuries; however, due to the lower bacterial count, infection is not seen as frequently as in cat bites. Large avulsion injuries can be reapproximated loosely as long as the wound can be packed and allowed to drain should an infection ensue. Elevate and treat with antibiotics. See below for rabies vaccination criteria. Evaluate for tetanus prophylaxis.

Antibiotics

- *First-line oral:* Augmentin.
  - *Adult:* 875/125 mg twice a day × 10 days.
  - *Pediatric:* 45 kg/day twice a day × 10 days.
  - *Alternatives:* Unasyn 1.5 g IV every 6 hours or clindamycin 450 mg four times a day + Bactrim DS twice a day × 10 days.

Rabies

Rabies is a viral infection of the central and peripheral nervous system that causes encephalitis with or without paralysis. If left untreated, it has close to 100% mortality. In the United States, rabies is most common in bats, raccoons, skunks, foxes, coyotes, ferrets, cats, and dogs. Bats are the most common wild animals to carry rabies. Cats are the most common domestic animals to carry rabies because of the high number of unvaccinated strays and their contact with raccoons, bats, and other wild animals.

Transmission is through the mucous membranes and saliva through breaks in the skin. The virus then replicates locally in the muscle and eventually travels through peripheral nerves to the spinal cord, then to the brain. Incubation times have ranged from as short as 5 days to as long as 7 years; however, the average incubation time is approximately 1 to 3 months. Common signs and symptoms of rabies are detailed in the box below.

The most common signs and symptoms of rabies infection

Paresthesias at the site of the bite
Hypersalivation
Hydrophobia
Altered mental status
Anxiety
Hyperactivity
Bizarre behaviors
Hypertension
Hyperthermia
Hyperventilation
Spasms and contractions of the neck muscles
Pharyngeal and respiratory muscle paralysis
Seizures

Treatment

The wound should be copiously irrigated with normal saline. Devitalized tissue should be adequately débrided, with all wounds left open to heal by secondary intention. Tetanus status should be determined and vaccine administered if indicated (see Chapter 1). A broad-spectrum antibiotic may be administered for 10 days (Augmentin 875/125 mg by mouth twice a day).

Domestic Animals

If the rabies status of the domestic animal (e.g., cat, dog, ferret) is unknown, the animal should be quarantined and observed for 10 days; prophylaxis can be postponed if suspicion is relatively low. If the animal is rabid or if the presence of rabies is highly suspected, human rabies immunoglobulin (RIG) and human diploid cell rabies vaccine (HDCV) should be administered.

- RIG: 20 IU/kg, 50%into the wound and 50%given IM.
- HDCV: Given on days 0, 3, 7, 14, and 28.

Wild Animals

Regard all wild animals (e.g., bats, foxes, coyotes, raccoons, skunks) as rabid. Test the animal if captured and administer RIG and HDCV to all patients as indicated above.

Snake

The majority of snakes are nonvenomous; therefore, snakebite wounds will likely heal without extensive intervention. Venomous snakebites, however, can pose a severe threat to the local soft tissues or cause life-threatening systemic reactions. The family Viperidae is the largest family of venomous snakes worldwide. The subfamily Crotalinae (pit vipers) includes rattlesnakes, cottonmouths, and copperheads; pit vipers are the most common type of venomous snake in the United States. The family Elapidae is the next largest family of venomous snakes. Coral snakes are commonly found in the southern and southwestern regions of the United States, while cobras, mambas, and kraits are not indigenous to the United States, but are exotic snakes that can be found in zoos or are kept by private collectors.

The identification of the snake's species is important in determining if envenomation is expected. Commonly patients will present with knowledge of the type of snake that was involved (see **Table 4.1** for some types of venomous snakes). Alternatively, the differentiation between a venomous and a nonvenomous snakebite can be made using the pattern of the bite or physical features of the snake if brought for presentation (**Fig. 4.1**).

Evaluation

Obtain thorough history that includes
- Time of the bite.
- Description of the snake.

Assess the timing of events and onset of symptoms. (Early and intense pain implies significant envenomation.)

Determine history of prior exposure to antivenin or snakebite.

Table 4.1 Venomous snake species

Family	Geographic range	Common names	
Viperidae	Africa, Europe, Asia, North and South America	Subfamily Crotalinae (pit vipers) includes rattlesnakes (diamondback, timber), cottonmouths, copperheads	Pit vipers are the most common type of venomous snake found in the U.S.
Elapidae	North America, Europe, Africa, Asia, Australia	Coral snakes, cobras, mambas, kraits	Coral snakes are commonly found in the U.S. in the southern and southwestern states

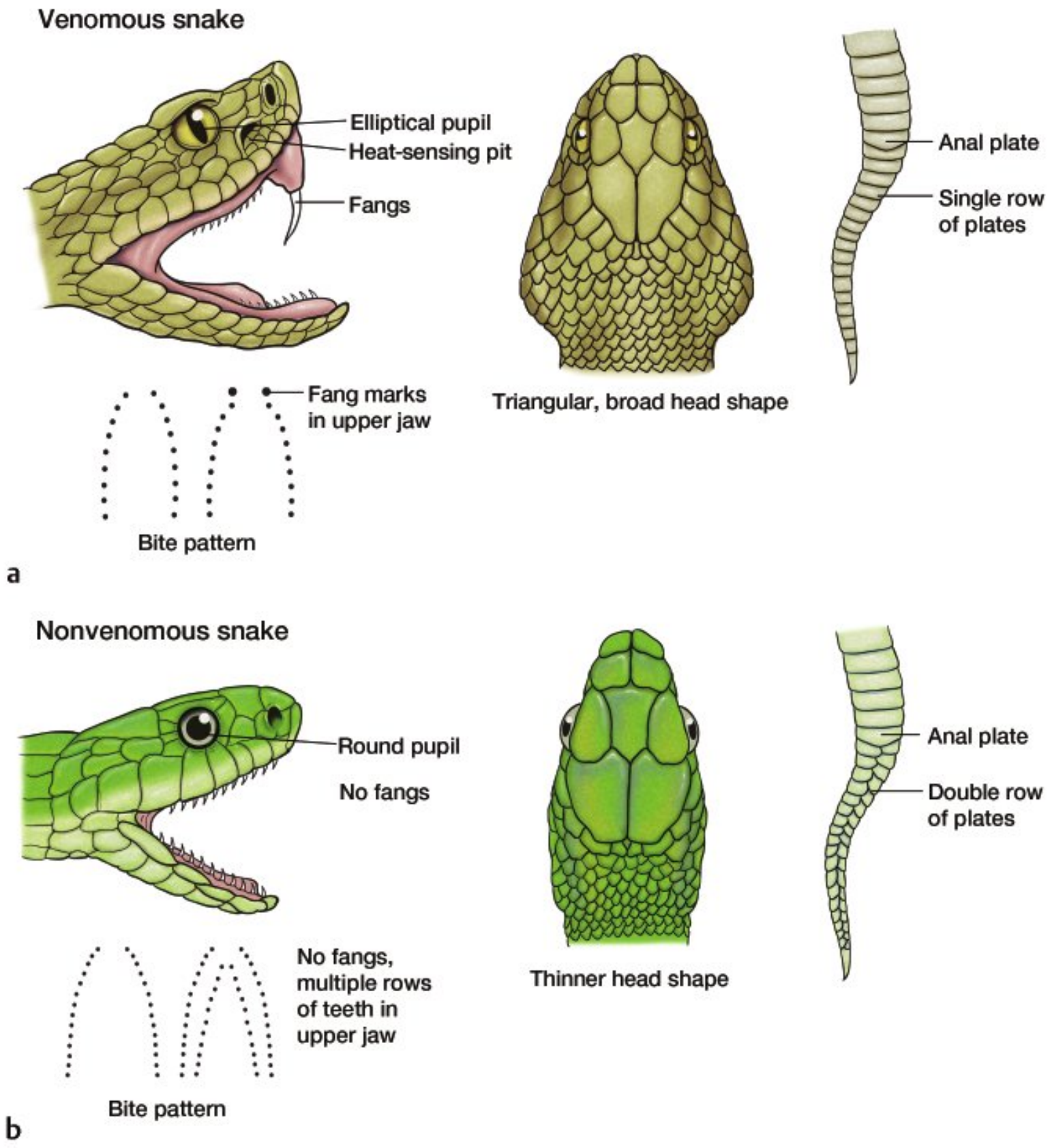


Fig. 4.1 Characteristics of **(a)** venomous versus **(b)** nonvenomous snakes and their bite patterns.

Assessment and physical examination should detail the following:

1. Fang marks.
2. Edema.
3. Bullae.
4. Erythema.
5. Necrosis.
6. Crepitus.
7. Petechiae.
8. Paresthesia.
9. Hemoptysis.
10. Presence of compartment syndrome if the bite occurs on an extremity (**Fig. 4.2**).

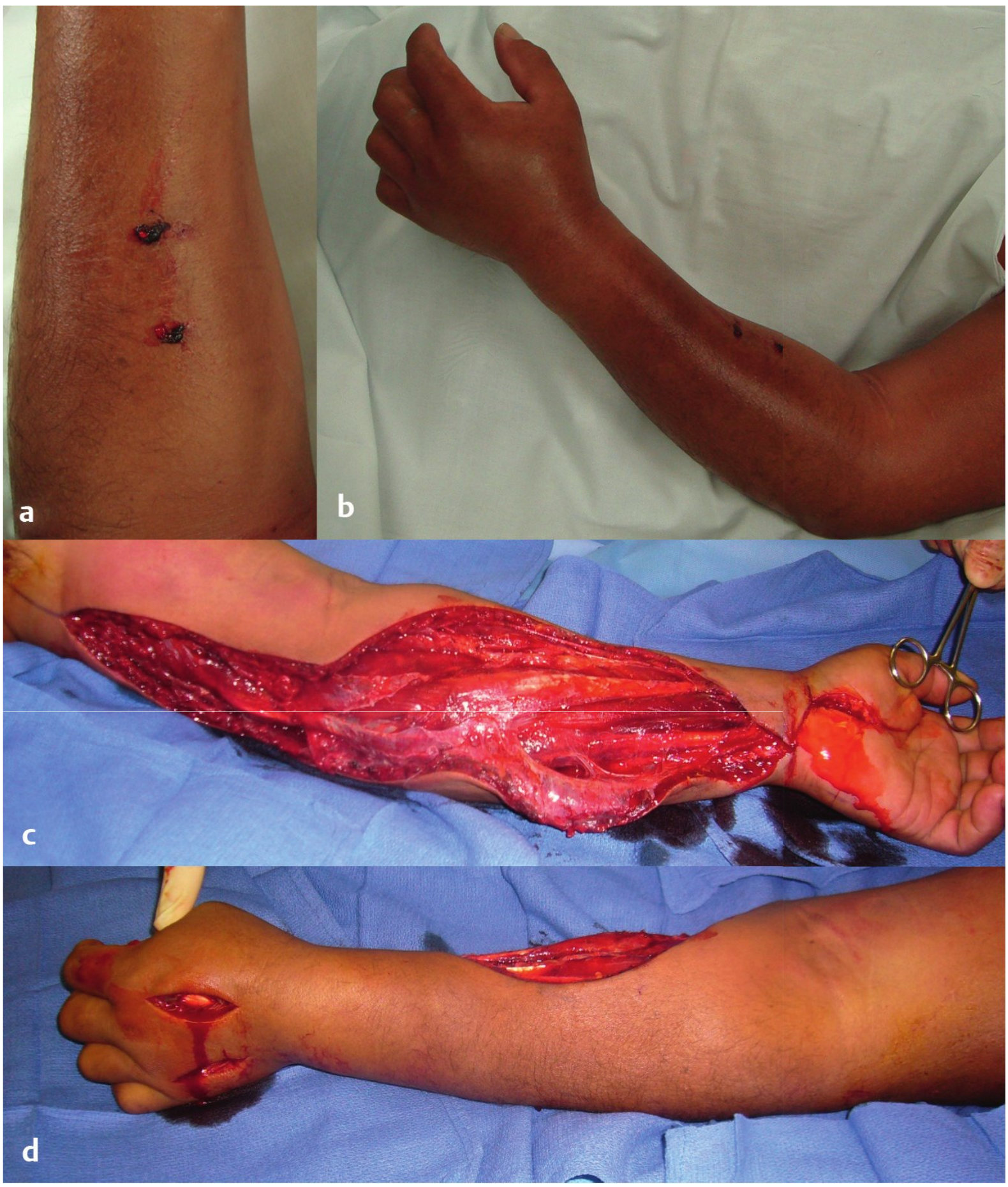


Fig. 4.2 **(a)** Fang marks characteristic of a venomous snakebite. **(b)** Signs of severe local reaction and compartment syndrome. **(c,d)** Forearm and hand fasciotomy required for the treatment of compartment syndrome secondary to the reaction from a venomous snakebite.

Treatment

1. Review the ABCs and evaluate the patient for signs of shock (e.g., tachypnea, tachycardia, dry pale skin, mental status changes, hypotension).
2. Obtain baseline laboratories (including PT, PTT, and INR) and CXR; type and crossmatch patient for FFP and PRBCs.
3. Rule out compartment syndrome and assess every 4 hours for signs of compartment syndrome (see Chapter 19).
4. Tetanus prophylaxis.
5. Prophylactic antibiotic use is controversial; however, some recommendations include the following: Rocephin (Roche Pharmaceuticals) 1 g IV every 12 hours or Timentin (GlaxoSmithKline) 3.1 g IV every 6 hours.
6. Immobilization, neutral positioning (splint) of extremity, and supportive care. Suction devices on the bite can be effective in the first 15 to 30 minutes. Do not attempt incision over the bite, mouth suctioning, tourniquets, or ice packs.
7. **Elevate the involved extremity.** This may require the aid of an IV pole, with which the extremity is hung using a stockinette.

Grading of Envenomation

1. Mild envenomation.
   a. Local pain and edema.
   b. Signs of systemic toxicity are absent.
   c. Laboratory values are normal.
2. Moderate envenomation.
   a. Local reaction—severe pain, edema greater than 12 inches surrounding the wound.
   b. Mild systemic toxicity present, including nausea, vomiting.
   c. Abnormal laboratory values—decreased hematocrit or platelet values.
3. Severe envenomation.
   a. Severe local reaction and generalized petechiae, ecchymosis.
   b. Severe systemic reaction—respiratory distress, airway edema, blood-tinged sputum, hypotension, renal dysfunction.
   c. Changes in coagulation profile—PT, APTT, and DIC.

Antivenin is given for moderate and severe cases of snake envenomation. Serum sickness is possible with antivenins, which are made with horse or sheep serum venom. A test dose is recommended; watch for an anaphylactic reaction. CroFab (BTG International Inc.) is a purified pit viper antivenin

that has fewer hypersensitivity reactions, so that serum sickness is less of an issue. CroFab is the preferred antivenin for pit viper envenomation.

Antivenin is given in ampules. One should start with 5 to 10 vials and continue therapy for up to 24 hours from the initial bite. If the patient responds (a decrease in both local and systemic reaction), then a dosing regimen of antivenin can be weaned. If the patient responds partially, plan to redose the antivenin. Patients should be monitored in an ICU setting during administration of antivenin for signs of allergic reaction. **Bites from coral snakes** ("red on yellow kills a fellow") are not treated with antivenin in the United States due to lack of production of specific coral snake antivenin and a halt by the FDA on the production of Wyeth's North American coral snake antivenin. Because a specific antivenin for coral snakes is not available, patients are currently treated with monitored supportive care.

Spider

There are over 20,000 species of spiders on Earth. Dangerous species often encountered in North America include the brown recluse, the black widow, the hobo or aggressive house spider, and the yellow sac spider. Of these, only the brown recluse and the black widow have ever been associated with significant disease (**Fig. 4.3**).

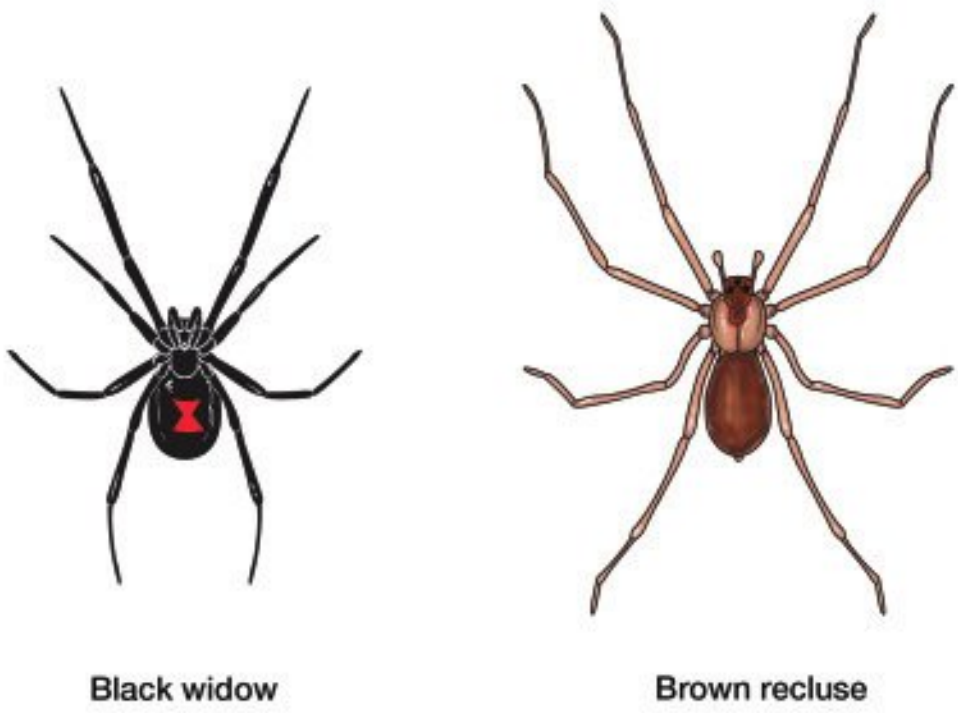


Fig. 4.3 The black widow and brown recluse spiders.

The Brown Recluse Spider

The brown recluse spider has six eyes and a violin-shaped pattern on its thorax and is found almost exclusively in the Midwestern and southeastern states. Although the venom is more toxic than that of the rattlesnake, morbidity is usually not as severe because of the small amount of venom that can actually be injected by the creature. One of the specific enzymes in the venom causes destruction of skin, fat, and blood vessels. This process eventually leads to soft tissue necrosis at the site of the bite.

The venom also has a profound effect on the immune response, triggering the release of various inflammatory cytokines, histamines, and interleukins that can themselves cause further injuries and systemic responses. Although rare, these include hemolysis, thrombocytopenia, coagulopathy, acute renal failure, coma, or death.

One should carefully assess the patient for any of the above symptoms, and admission is warranted for anyone exhibiting systemic toxicity. Apply ice to decrease pain and swelling, and elevate the site of injury above the heart. Wash the area thoroughly with soap and water, and instruct the patient to avoid any strenuous activity, which can facilitate the spread of the venom. Do not place heat on the area; this can accelerate tissue destruction. Do not attempt to suction the venom out, and the use of steroid creams is not advised.

Brown recluse spider bites are usually painless at first, and symptoms are slow to develop. Pain will usually present around 4 hours after the initial bite, with the bite wound presenting with a bull's-eye appearance. Blistering is then commonly seen 12 to 24 hours later, with soft tissue necrosis to follow. Early débridement is not indicated, and necrotic lesions should be kept clean and carefully dressed until spreading stops and the area of necrosis is well defined. A wide area of tissue around the necrotic skin can then be removed, with subsequent skin grafting as needed.

1. Baseline laboratories should include CBC, Chem-7, PT, PTT, and INR.
2. There is no antivenin available; however, dapsone 100 mg by mouth daily can be reserved for people with severe systemic disease (anemia, DIC, acute renal failure).
3. Acetaminophen or opiates for pain. Avoid aspirin, ibuprofen (Motrin [Pfizer Pharmaceuticals], Advil [Wyeth Pharmaceuticals]), and naproxen (Aleve [Bayer Consumer Care]).
4. Diphenhydramine 25 to 50 mg by mouth every 6 hours as needed.
5. Antibiotics should be administered if significant soft tissue necrosis ensues. Patients should be watched very closely, with follow-up the next day if possible.

The Black Widow Spider

Black widow spiders are nocturnal and are found in the southern states. This spider has a distinctive red-colored hourglass figure on its underbelly. Its initial bite is usually associated with local pain followed by systemic reactions that can carry mortality as high as 5%(usually in children or the elderly). Generalized symptoms usually include

- Nausea, vomiting.
- Faintness, dizziness.
- Chest pain.
- Hypotension.
- Tachycardia.
- Respiratory difficulties.
- Abdominal pain mimicking gallbladder disease or appendicitis.

There is minimal tissue toxicity, and the wound should be irrigated and cared for in the usual manner. Treatment for systemic symptoms is supportive, and an antivenin is available for severe cases. It should only be used if the patient is unstable.

Cold compresses have been used to ease the pain at the site, as well as over-the-counter pain medications. Over-the-counter pain medications (e.g., acetaminophen, naproxen, ibuprofen, Advil) can be used, as well as Benadryl 25 to 50 mg by mouth every 6 hours for itching. In general, antibiotic prophylaxis and extensive medical follow-up is not needed.

5 Burns and Frostbite

Evaluation and management of the acutely burned patient is a common requirement of the plastic surgeon on call. Rapid assessment, stabilization, and triage are essential for decreasing morbidity and mortality associated with burn injury. Commonly, the initial encounter will be as a consultant subsequent to the evaluation performed by the emergency room personnel. It is imperative, however, to remember to initiate measures to stop the burning process and practice universal safety precautions to confer increased safety for both the patient and the caregiver. Burn injury is often associated with trauma; therefore, a complete assessment of other injuries should be performed. If a child is burned and the mechanism of injury does not fit the burn pattern or if the patient was burned under unlikely circumstances or conditions, consider abuse.

Thermal Burns

Initial Assessment—Starting with the ABCs

Airway

- Establish a patent airway and begin oxygenation.
  - Employ manual techniques—chin lift, jaw thrust.
    - Utilize nasal trumpets and oral airways.
    - Consider creating a surgical airway when there is upper airway obstruction (cricoidectomy, tracheostomy).
- Assess for inhalation injury (**Fig. 5.1**).
  - Determine whether the burns occurred while the patient was in an enclosed space.
  - Signs and symptoms of inhalation injury.
    - Soot deposits in the oropharynx.
    - Carbonaceous sputum.
    - Singed nasal hair.
    - Facial edema, tongue edema, hoarseness.
  - Measure carboxyhemoglobin level.
    - > 10% requires oxygen therapy and is highly suggestive of an inhalation injury that requires intubation.

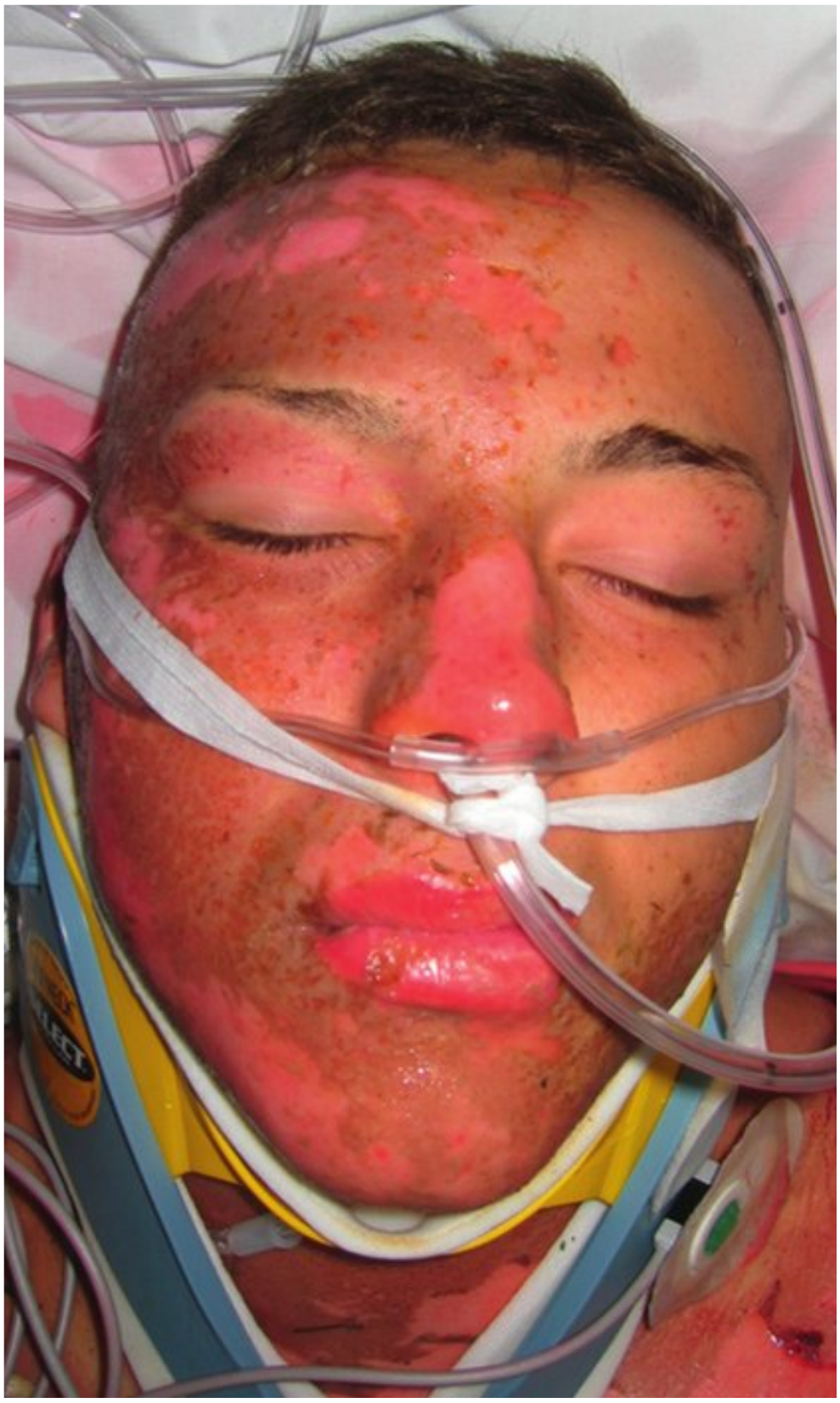


Fig. 5.1 Signs of potential airway burns and inhalation injury.

- General criteria for intubation.
  - Glasgow Coma Scale score < 8 (**Table 5.1**).
  - Inhalation injury.
  - Deep facial and neck burns.
  - Facial burns with associated TBSA burns > 40%
  - Large TBSA burns—to allow adequate resuscitation.
  - Oxygenation or ventilation compromise.
    - $PaO_2 < 60$.
    - $PCO_2 > 50$.
    - RR > 40.

Patients who present with burns of the head and neck and inhalation injury may require early intubation to protect the airway from late edema and edema that occurs during resuscitation.

Table 5.1 The Glasgow Coma Scale (score = E+ M+ V)

Eye opening (E)	
Spontaneous	4
To speech	3
To pain	2
No response	1
Best motor response (M)	
Obeys verbal command	6
Localizes painful stimulus	5
Flexion: withdrawal	4
Flexion: abnormal	3
Extension	2
No response	1
Best verbal response (V)	
Converses and oriented	5
Converses but disoriented	4
Inappropriate words	3
Incomprehensible sounds	2
No response	1

Breathing

- Provide humidified oxygen by face mask.
- Expose the chest to assess ventilation, chest excursion, degree of chest wall injury, and presence of circumferential burns to the thorax.
- Consider thoracic escharotomy for deep injury to the chest with associated ventilatory compromise.

Circulation

- Establish vascular access with large-bore, high-flow venous cannulation. Avoid the injured area if possible.
- Initiate monitoring: BP, pulse, temperature.
- Consider invasive arterial lines for monitoring and frequent laboratory blood draws.

Disability

- Gross assessment of neurologic status (mnemonic tool = AVPU).
  - Alert.
  - Responds to Vocal stimuli.
  - Responds only to Painful stimuli.
  - Unresponsive to all stimuli.
- Glasgow Coma Scale (**Table 5.1**).

Exposure

- Remove all clothing and debris to assess for gross injuries and for burn severity.
- Prevent hypothermia by increasing the room temperature, covering the patient with clean warm linens, and infusing warm IV fluids.

Burn Severity Assessment

For initial acute resuscitation, the following information is necessary:

- Height, weight, and age of the patient.
- Depth of the burn injury.
  - Universal burn wound classification (**Fig. 5.2**).
- Percentage of the total body surface area burned that is second or third degree.
  - The percentage of total body surface area (TBSA) can be estimated by the "rule of nines" (**Fig. 5.3**).
  - More accurate calculation can be done with burn charts (**Table 5.2**), which is important in the pediatric population.
  - Generally, the patient's hand (palm and fingers) is estimated as 1% of their total body surface area.

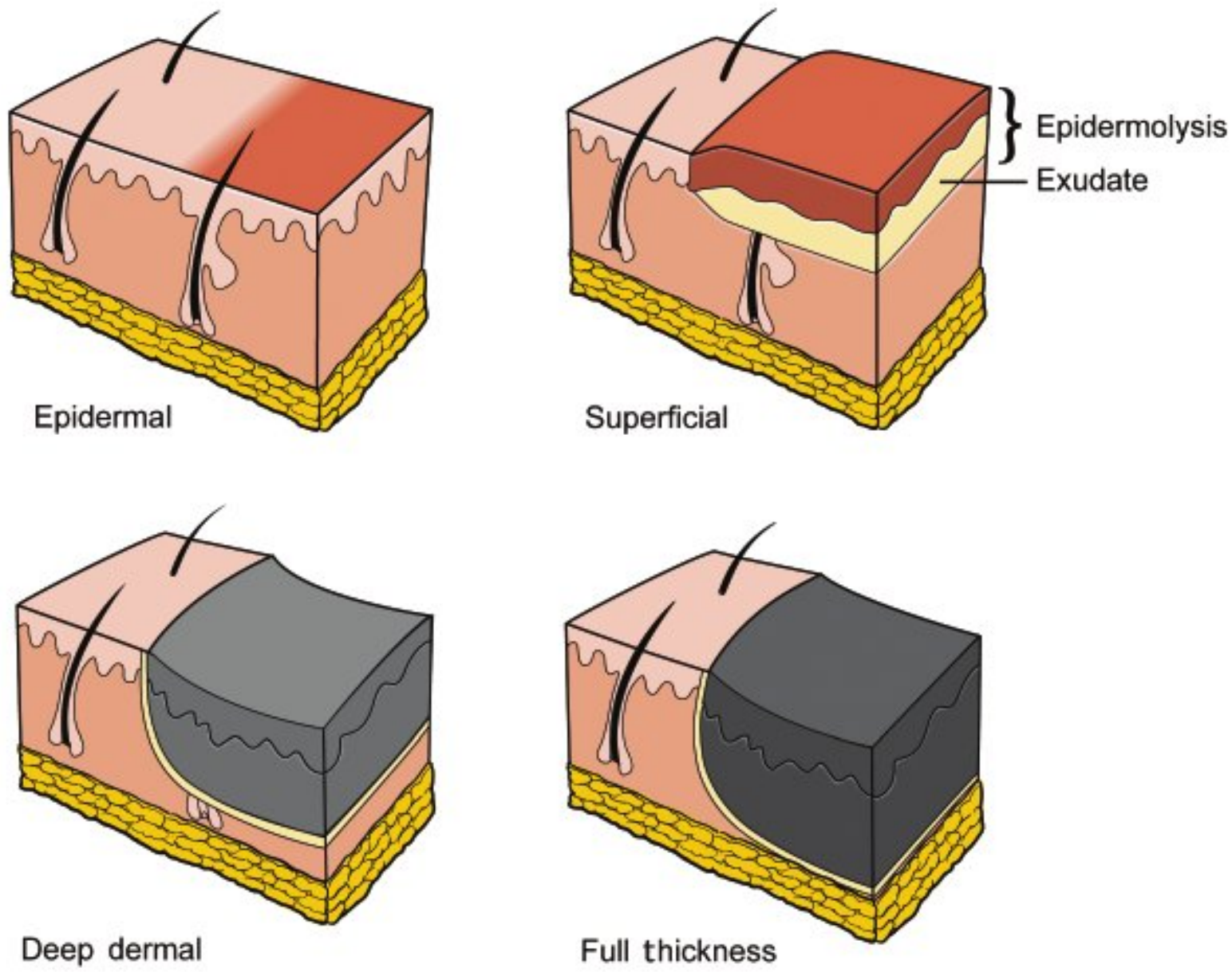


Fig. 5.2 Burn wound classification.

Epidermal Burns, First Degree (Fig. 5.4)

- Zones of injury are confined to the epidermis.
- Similar to sunburn.
- Nonblanching erythema.
- Very painful.
- Heals in 1 week.
- No significant scarring.

Partial-Thickness Burns, Superficial Second Degree (Fig. 5.5)

- Confined to the upper third of the dermis.
- The edema layer between the injured layer and normal dermis causes blistering.
- Commonly, these are the result of brief hot-liquid exposure.
- Wounds are wet, pink, and blistering.
- Wounds heal in 10 to 14 days with minimal scarring.

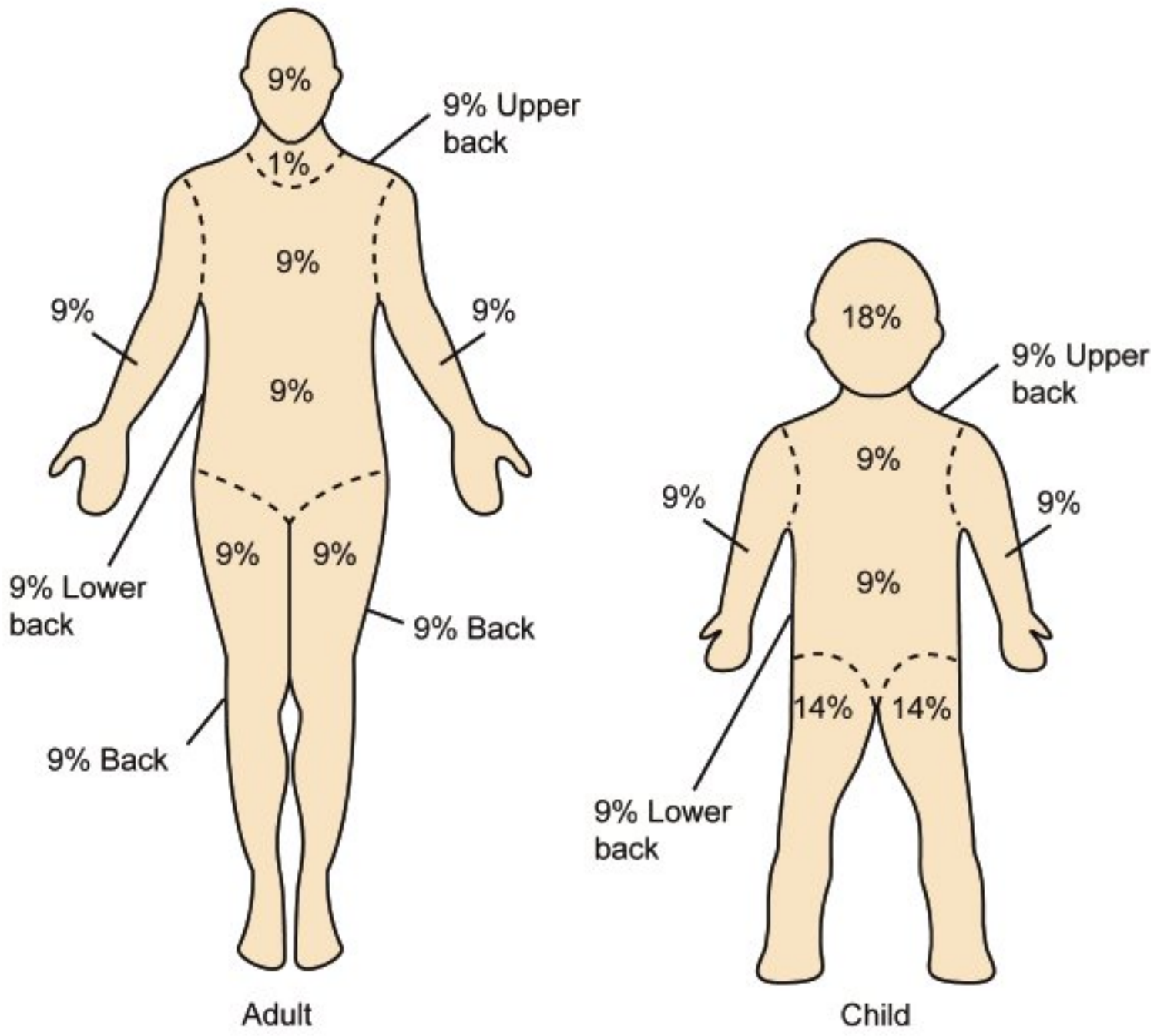


Fig. 5.3 The rule of nines for adults and children.

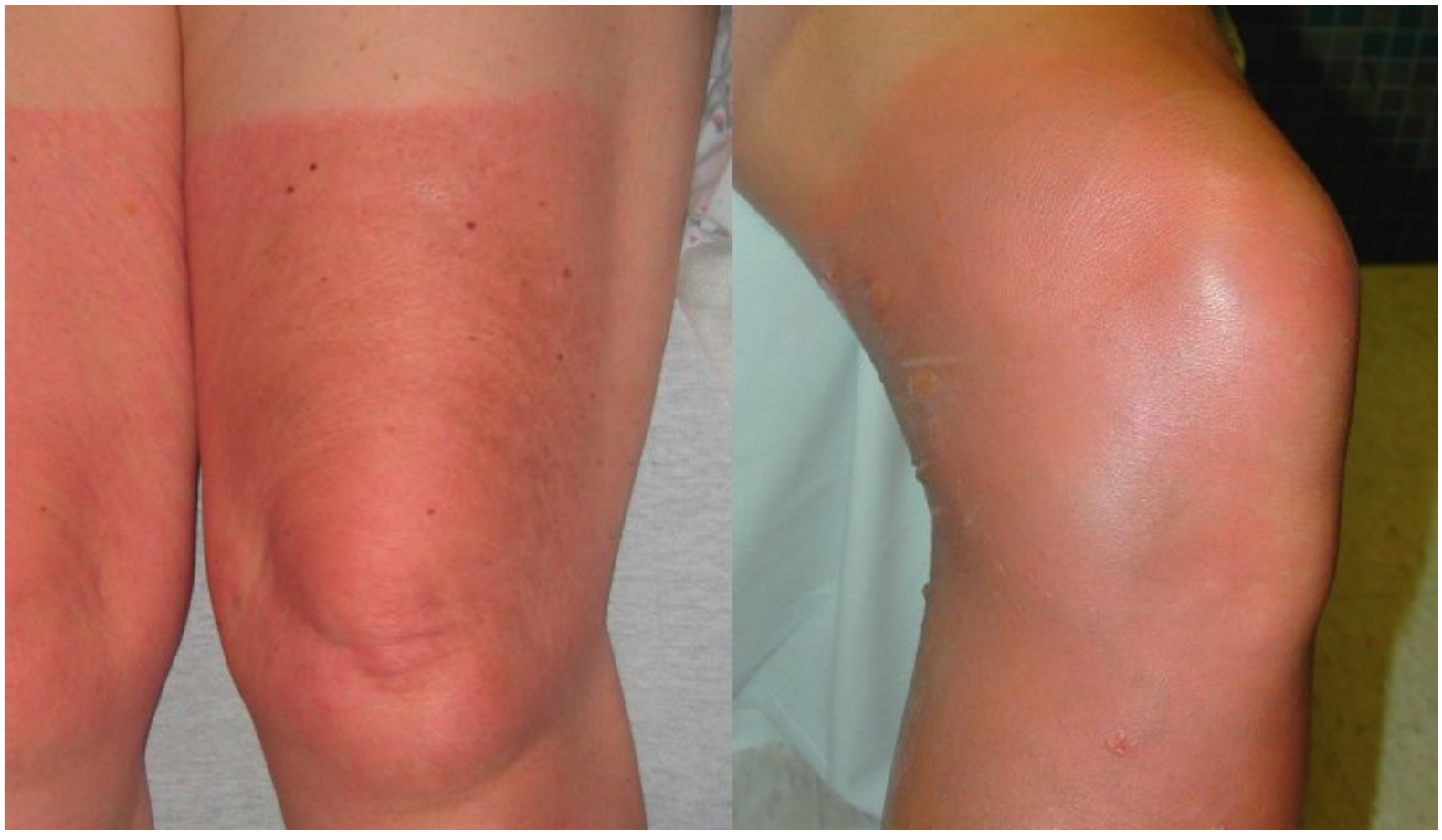


Fig. 5.4 First-degree epidermal burn.

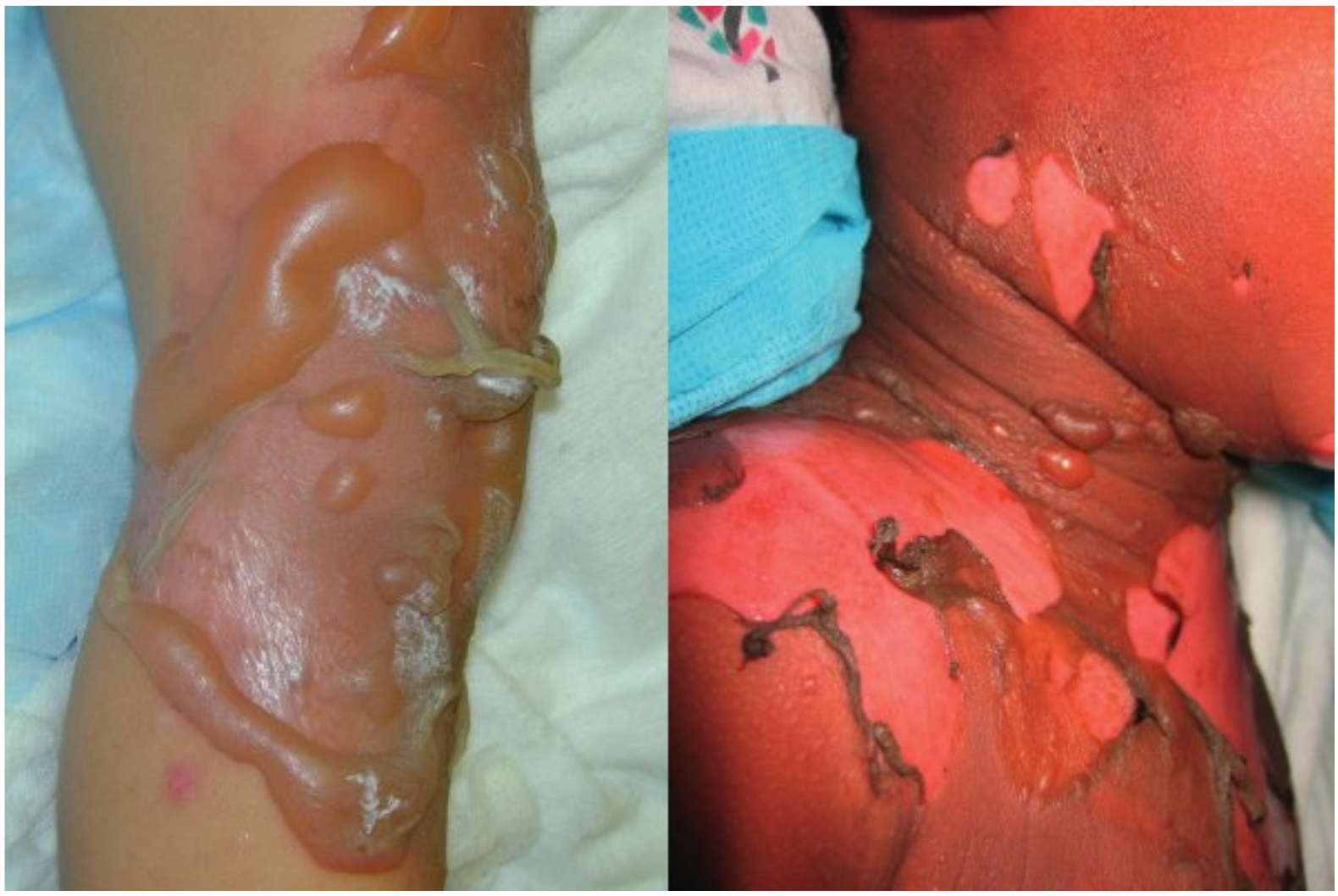


Fig. 5.5 Superficial second-degree burn with blistering and epidermolysis.

Partial-Thickness Burns, Mid-dermal Second Degree (Fig. 5.6)

- Result from longer hot-liquid exposure, grease, and flash flames.
- Wounds are red with minimal exudates and moderately painful.
- Wounds heal in 2 to 4 weeks with moderate scarring.

Partial-Thickness Burns, Deep Dermal Third Degree (Fig. 5.7)

- Result from exposure to flames, grease, chemicals, and electricity.
- Wounds are usually dry, white, and minimally painful (due to damage to nerve endings).
- Generally, wounds heal in 3 to 8 weeks with severe hypertrophic scarring.
- Excision and grafting will accelerate closure.

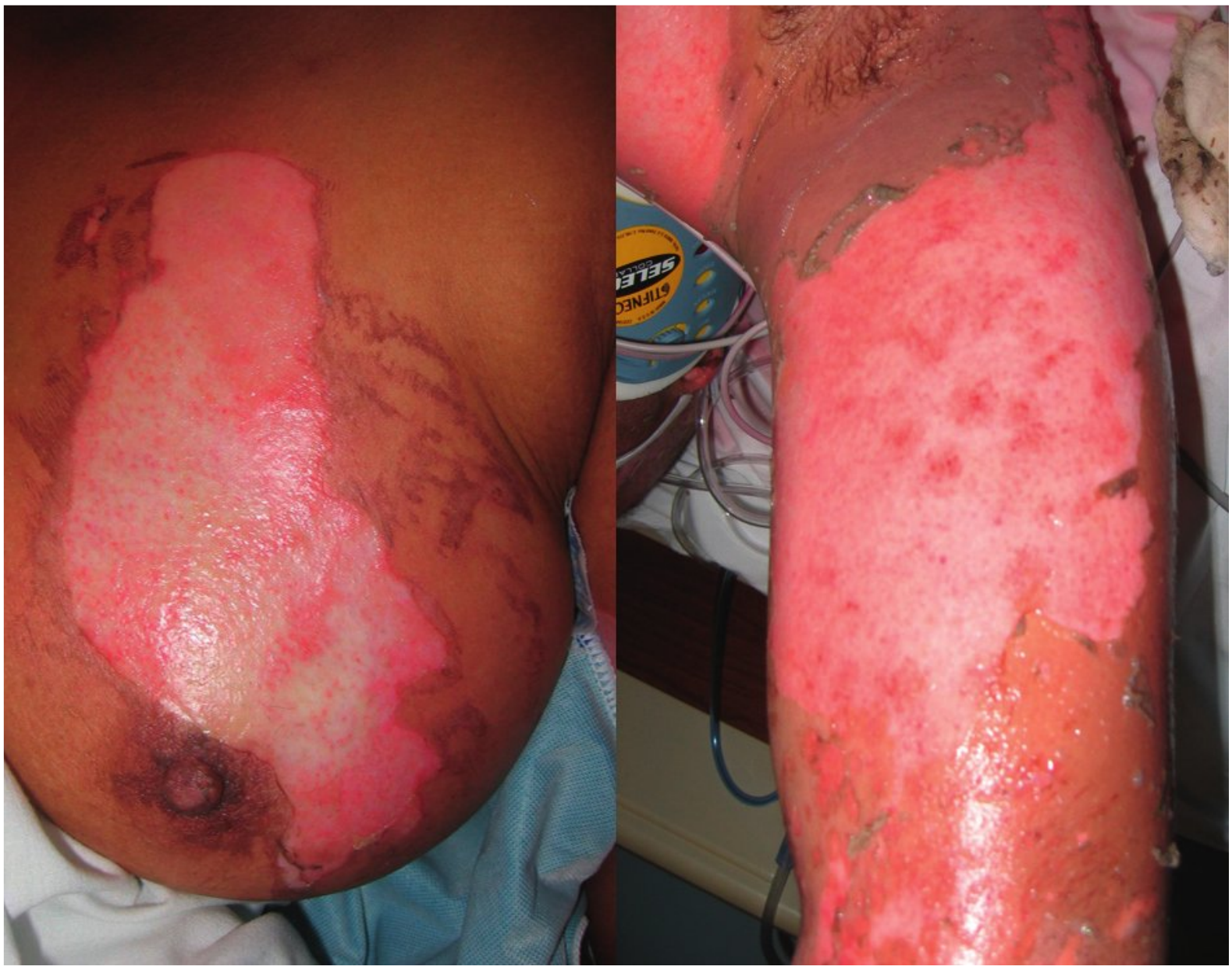


Fig. 5.6 Mid-dermal second-degree burn.

Full-Thickness Burns, Third Degree (Fig. 5.8)

- Result from high energy and prolonged thermal exposure (chemicals, flames, electricity, explosions).
- Wounds are dry and white, or exhibit immediate eschar formation.
- Wounds are painless and insensate.
- These wounds need débridement and grafting to promote healing.

Burn Patient Resuscitation

Patients who require intravenous crystalloid resuscitation and possibly fluid balance monitoring with a Foley catheter placement are

- Adults with second- and third-degree burns > 20%TBSA.
- Children (< 14 years of age) with burns > 15%TBSA.
- Infants (< 2 years of age) with burns > 10%TBSA.

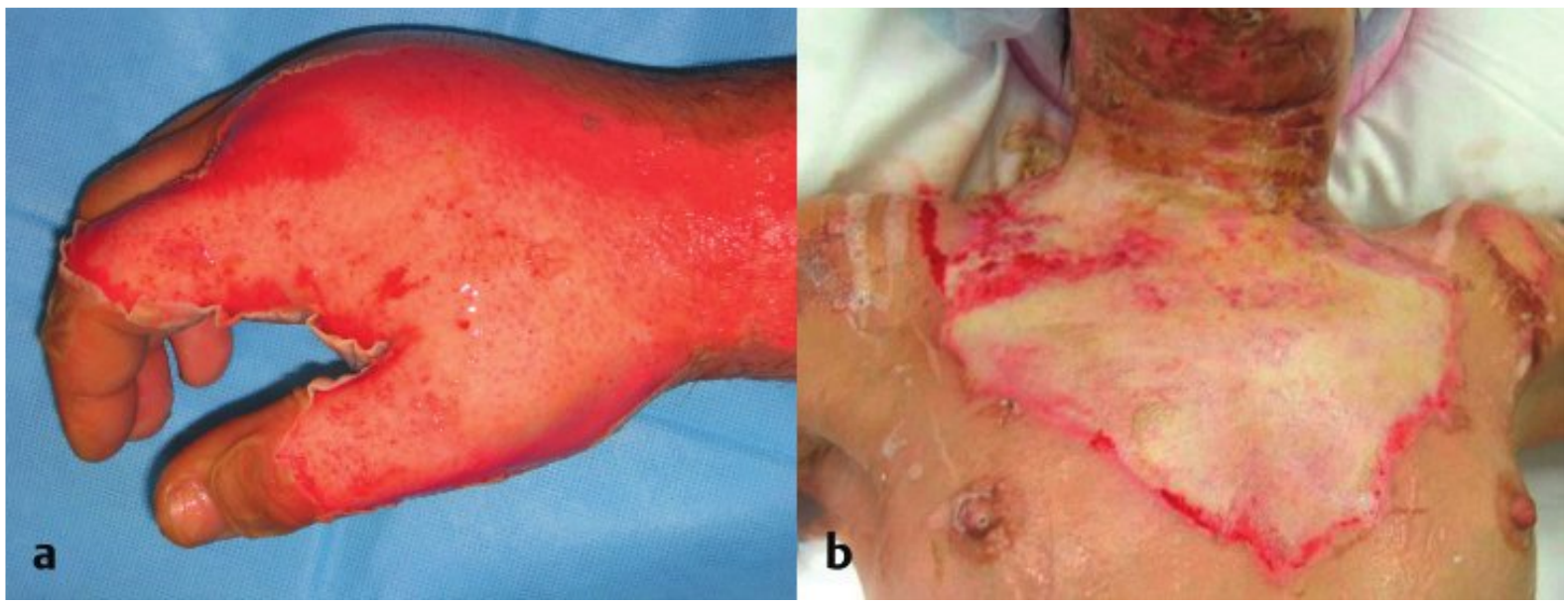


Fig. 5.7 **(a)** Deep dermal third-degree burn. **(b)** Deep dermal burn with areas of full-thickness involvement.

All other patients can be managed with oral hydration.

Urine output is used to gauge the success of fluid resuscitation. If there is any question as to the patient's ability to pass urine, place a Foley catheter. Lactated Ringer's solution should be started as soon as possible after the time of the burn. The volume of fluid given in the first 24 hours for adult victims is determined by the Parkland formula:

$$4 \times \text{weight (kg)} \times \%\text{BSAB} = \text{volume of fluid for 24 hours}$$

These estimates are based on second- and third-degree burn injuries only.

Pediatric patients have increased fluid requirements secondary to differences in BSA-to-weight ratio and require larger volumes of urine for excretion of waste products. The volume required in the first 24 hours for the burned pediatric patient is estimated using the Galveston formula (established at the Shriners Institute for Burned Children, Galveston, TX):

$$\text{fluid volume for first 24 hours of resuscitation (mL)} = [2{,}000\ \text{mL} \times \text{TBSA}] + [5{,}000\ \text{mL} \times \text{BSA}\ (\text{m}^2)]$$

Total Body Surface Area (TBSA)

$$\text{TBSA}\ (\text{m}^2) = 0.007184 \times (\text{height in cm})^{0.725} \times (\text{weight in kg})^{0.425}$$

Burn Surface Area (BSA)

$$\text{BSAB}\ (\text{m}^2) = \text{TBSA} \times \%\text{surface area burned (using rule of nines or calculated from burn \%chart)}$$

The rate of infusion when using the Parkland and Galveston formulas is as follows:

- Half of the determined volume is given within the first 8 hours of the **time of the burn.**
- The remaining volume is given during the succeeding 16 hours.

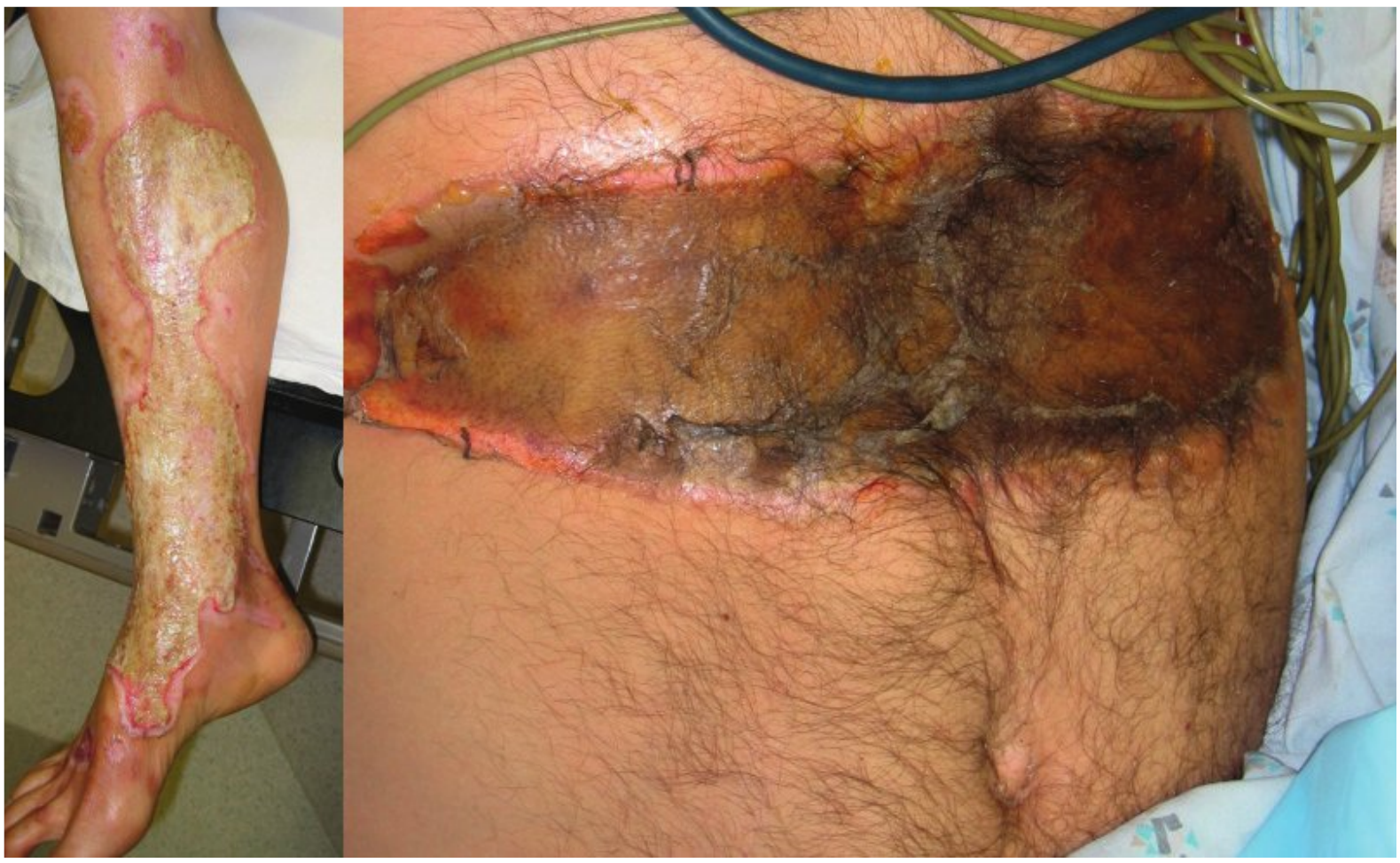


Fig. 5.8 Full-thickness burn injury.

Fluid requirements beyond the first 24 hours are determined based on the patient's weight and evaporative losses, and adjusted according to the patient's response (i.e., urine output). **Maintenance volume of fluid** is calculated in L/d as

- 100 mL/kg for first 10 kg.
- 50 mL/kg for second 10 kg.
- 20 mL/kg for each additional kg of body weight.

In addition:

$$\text{evaporated losses related to the burn wounds per day} = 3{,}750\ \text{mL} \times \text{BSAB}\ (\text{m}^2)$$

This volume is then added to the maintenance volume and divided over 24 hours.

Alternatively, the maintenance volume per day in the postacute resuscitation period is calculated as

$$[1{,}500\ \text{mL} \times \text{TBSA}\ (\text{m}^2)] + [3{,}750\ \text{mL} \times \text{BSAB}\ (\text{m}^2)]$$

Ultimately, this calculation should be adjusted to ensure adequate end-organ perfusion as monitored by the patient's urine output, which should be > 0.5 mL/kg/h for adults or 1 mL/kg/h for children. Bolus additional IV fluid to maintain adequate urine output.

Escharotomy

Late tissue edema may lead to vascular compromise secondary to decreased elasticity of a burn scar. This is particularly hazardous in deep burns of the extremities and circumferential burns of the chest wall. An escharotomy is performed early for circumferential deep dermal and full-thickness burns to the extremities and chest. Generally, escharotomies should be performed by a surgeon or a physician experienced in the procedure to decrease morbidity.

Procedure (Fig. 5.9, Fig. 5.10)

- Use electrocautery or a scalpel to incise the burned skin.
- Extend down through eschar into the subcutaneous fat.
- Cut midmedially or midlaterally.

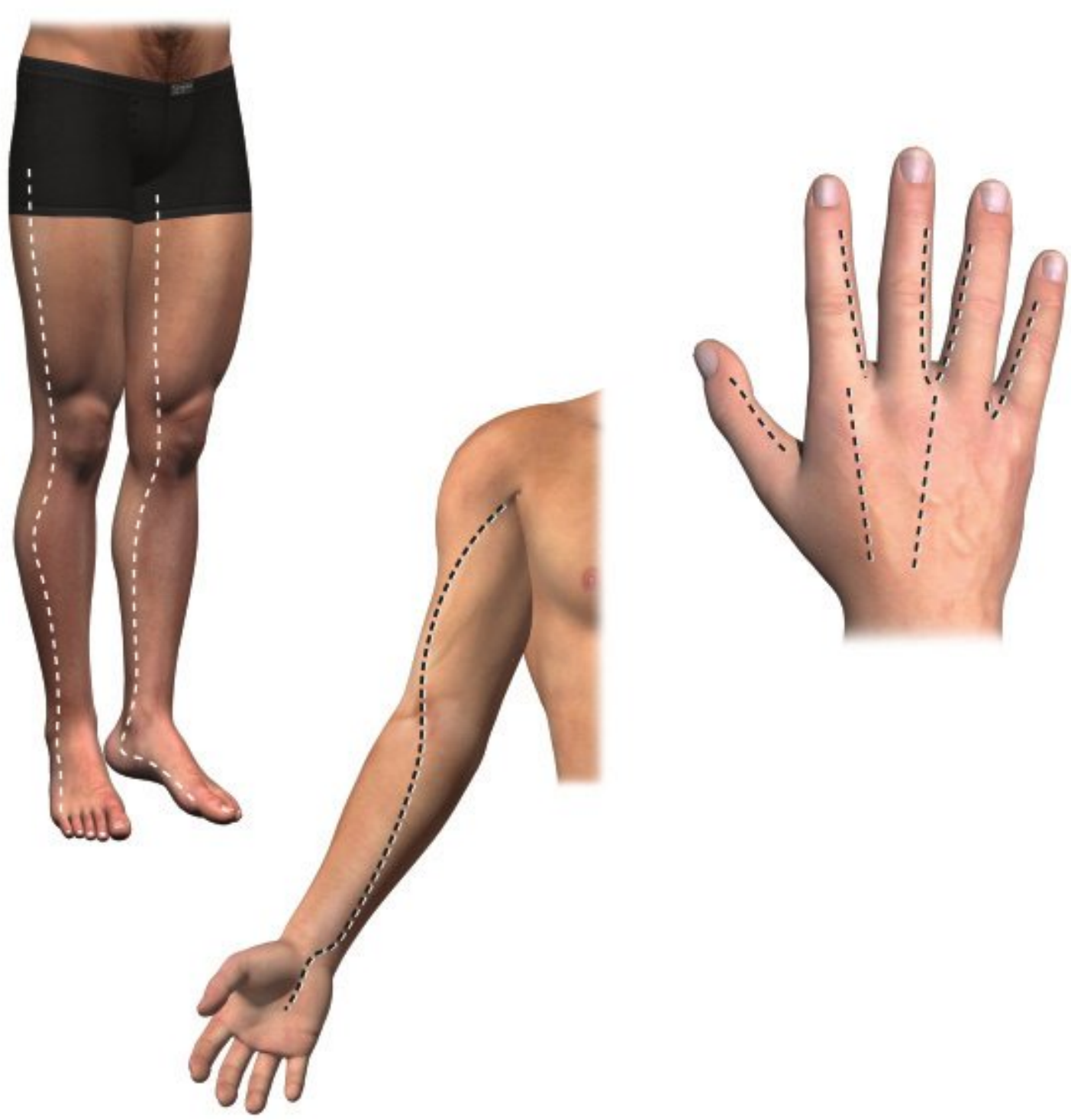


Fig. 5.9 Incision locations for escharotomy.

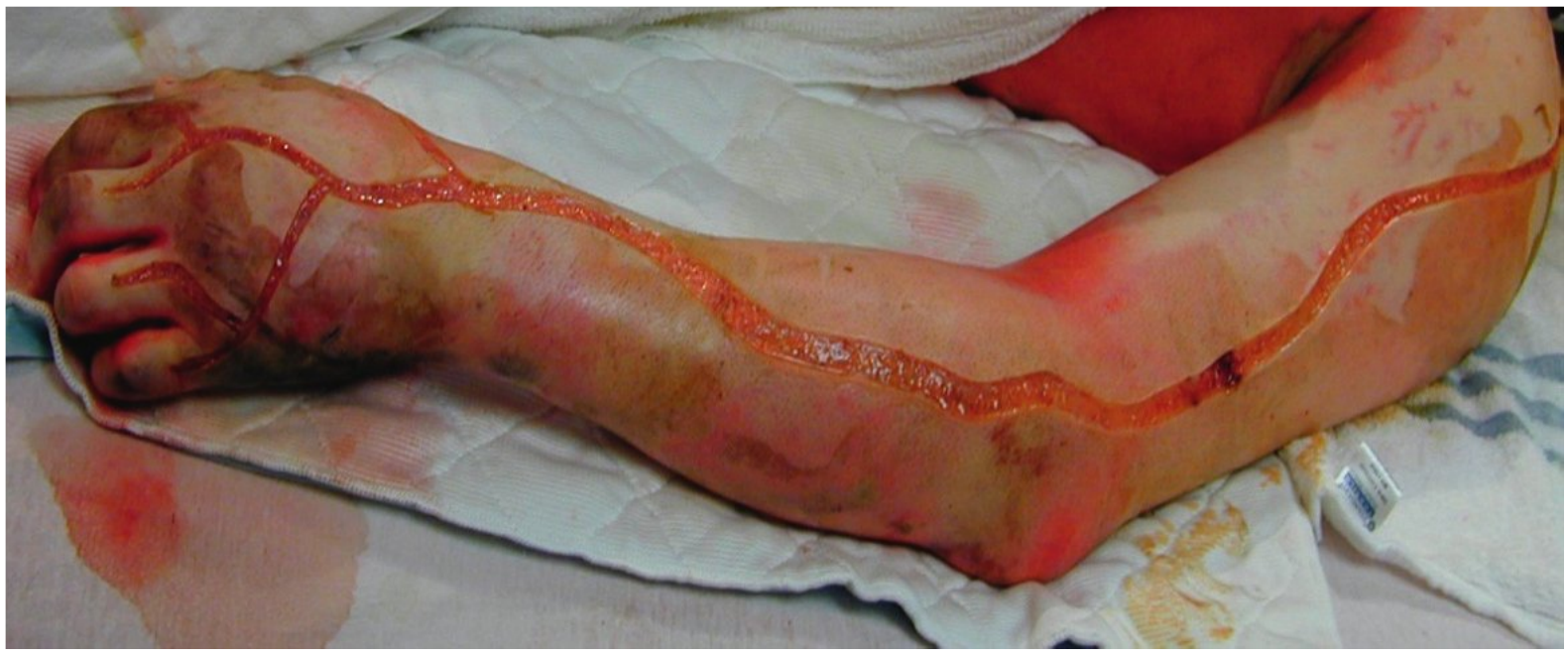


Fig. 5.10 Lateral and dorsal escharotomy of the upper extremity.

- Extend the incision the length of the constricting burn eschar and across involved joints.
- Avoid major vessels, nerves, tendons, and pressure surfaces.

Associated Conditions

Inhalation Injury

The leading cause of death in fires is smoke inhalation, not burns. Inhalation injury is present in one-third of burn patients and doubles the mortality rate from burns.

Signs and Symptoms of Inhalation Injury

- Anatomical distortion of the face and neck edema.
- Inability of the patient to clear secretions.
- Altered mental status.
- Decreased oxygenation.
- Increased carboxyhemoglobin.
- Lactic acidosis.

Management of Inhalation Injury

- Evaluate patient for intubation.
- Perform a fiberoptic laryngoscopy and bronchoscopy for diagnosis and soot/secretion removal.
- 100% oxygen supplementation.
- Assess for carbon monoxide poisoning.
- Elevate chest/head to 20 to 30 degrees at all times.

- Liberal use of bronchodilators such as albuterol.
- Transfer patient to a burn center or critical care setting.

For advanced management of severely burned airway:

- Intubate; apply positive pressure ventilation.
- Positive end-expiratory pressure (PEEP); maintain patency of smaller airways.
- Give the patient *N*-acetylcysteine.
- Administer nebulized heparin.
- Transfer patient to a burn center or critical care setting.

Carbon Monoxide Toxicity

Carbon monoxide toxicity is one of the leading causes of death associated with fires and is produced in the process of O_2 combustion. Carbon monoxide preferentially binds to hemoglobin in place of oxygen and forms carboxyhemoglobin (COHb), which shifts the oxyhemoglobin dissociation curve to the left, reducing oxygen delivery. Signs and symptoms of carbon monoxide poisoning are outlined in **Table 5.3.**

Management of Carbon Monoxide Toxicity

- Administer high-flow oxygen by mask (FiO_2 100%) until carboxyhemoglobin is < 10%
- For obtunded patients.
  - Intubate.
  - 90 to 100%oxygen via positive pressure ventilation.

If the patient is not responding to 100%oxygen:

- Consider advanced modes of ventilating.
  - Volume Diffusive Respirator (VDR; Percussionaire), high frequency percussive ventilation
  - Bi-level inverse ratio ventilation (IRV).
    - Hyperbaric therapy.

Table 5.3 Symptoms of carbon monoxide poisoning

COHb (%)	Symptoms
0–10	Normal value
10–20	Headache, confusion
20–40	Disorientation, fatigue, nausea, visual changes
40–60	Hallucination, combativeness, coma, shock state
> 60	Mortality > 50%

Burn Patient Triage

Once the burned patient is stabilized, the appropriate facility to care for the patient is determined. Triage of the burn patient includes outpatient management, inpatient management, management by a trauma service, or referral to a specialized burn center. The criteria for referral to a burn center are outlined in **Table 5.4**.

The criteria for the management of burn victims as **outpatients** include the following:

- Burns are < 10%partial-thickness burns without inhalation injury.
- Patients are responsive to oral analgesics.
- Victims are compliant patients who will care for their wounds and present for follow-up evaluation within 3 to 5 days.
- There is no immediate or delayed risk to specialized areas (i.e., circumferential burns).

All other burn victims require hospital admission for more extensive treatment or monitoring. At times, patients with minor burns must be admitted for pain control or personal safety/caution, as in the case of abuse or patients with multiple preexisting comorbidities or trauma.

Table 5.4 Burn Center Referral Criteria

A burn center may treat adults, children, or both. Burn injuries that should be referred to a burn center include the following:

- Partial-thickness burns of greater than 10 percent of the total body surface area.
- Burns that involve the face, hands, feet, genitalia, perineum, or major joints.
- Third-degree burns in any age group.
- Electrical burns, including lightning injury.
- Chemical burns.
- Inhalation injury.
- Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality.
- Burns and concomitant trauma (such as fractures) when the burn injury poses the greatest risk of morbidity or mortality. If the trauma poses the greater immediate risk, the patient's condition may be stabilized initially in a trauma center before transfer to a burn center. Physician judgment will be necessary in such situations and should be in concert with the regional medical control plan and triage protocols.
- Burns in children; children with burns should be transferred to a burn center verified to treat children. In the absence of a regional pediatric burn center, an adult burn center may serve as a second option for the management of pediatric burns.
- Burn injury in patients who will require special social, emotional, or long-term rehabilitative intervention.

Source: From Committee on Trauma: American College of Surgeons. Reproduced with permission.

Burn Wound Management

The patient should be premedicated with analgesics prior to wound treatment to decrease discomfort and increase patient cooperation.

General Principles

- Cleanse the wounds of particles and débride devitalized tissue.
- Initiate tetanus prophylaxis.
- Daily or twice-daily wound cleansing and dressing.
- Antibiotics only for gross soft tissue infection.
- Aggressive pain control.

Use chlorhexidine, 0.5%silver nitrate combined with chlorhexidine gluconate, normal saline, or soap and water to cleanse the burn wound. To prevent wound infection and deeper wound conversion, topical antimi-

crobials are used until epithelialization of the wound is complete. The topical antimicrobials are provided via gauze applications, ointments, creams, or solutions; dressings are changed at least twice a day. Commonly used topical antimicrobials are outlined in **Table 5.5** and antimicrobial dressings in **Table 5.6**.

Outpatient Wound Dressings

Epidermal First-Degree Burns

- Heal spontaneously with little intervention required.
- Moisturize the wound to alleviate pain.

Partial-Thickness Burns, Superficial Second-Degree Burns

- Treat blisters.
  - Minor blisters over a small surface area require no intervention.
  - Large, tense, turbid, painful blisters.
    - Using aseptic technique, aspirate with a large-bore needle, leaving epidermis as a biologic dressing.
    - Débride epidermis if wounds are contaminated.
      - Clean wound thoroughly and dress.
        - Antibiotic impregnated petroleum gauze.
        - Biobrane (UDL Laboratories, Inc.) for clean scald burns.
        - Aquacel Ag (ConvaTec) for wounds with excessive exudate.
- Apply soft bulky gauze dressing.
- Administer analgesics as needed.
- Follow up in 2 to 3 days. If the patient is free of pain and wounds are healing, then instruct the patient or caregiver on how to continue dressing changes at home.

Mid- to Deep Dermal Burns, Second- and Third-Degree Burns

- Clean wounds thoroughly with chlorhexidine.
- Débride superficial devitalized tissue.
- Dress wounds with antimicrobial material.
  - Silvadene.
  - Sulfamylon for burns with eschar formation.
  - Acticoat (Smith & Nephew) and Mepilex Ag (Mölnlycke Health Care) are great alternatives to creams that offer a more organized and easier application with a greater antimicrobial spectrum.
- Apply soft bulky gauze dressing.

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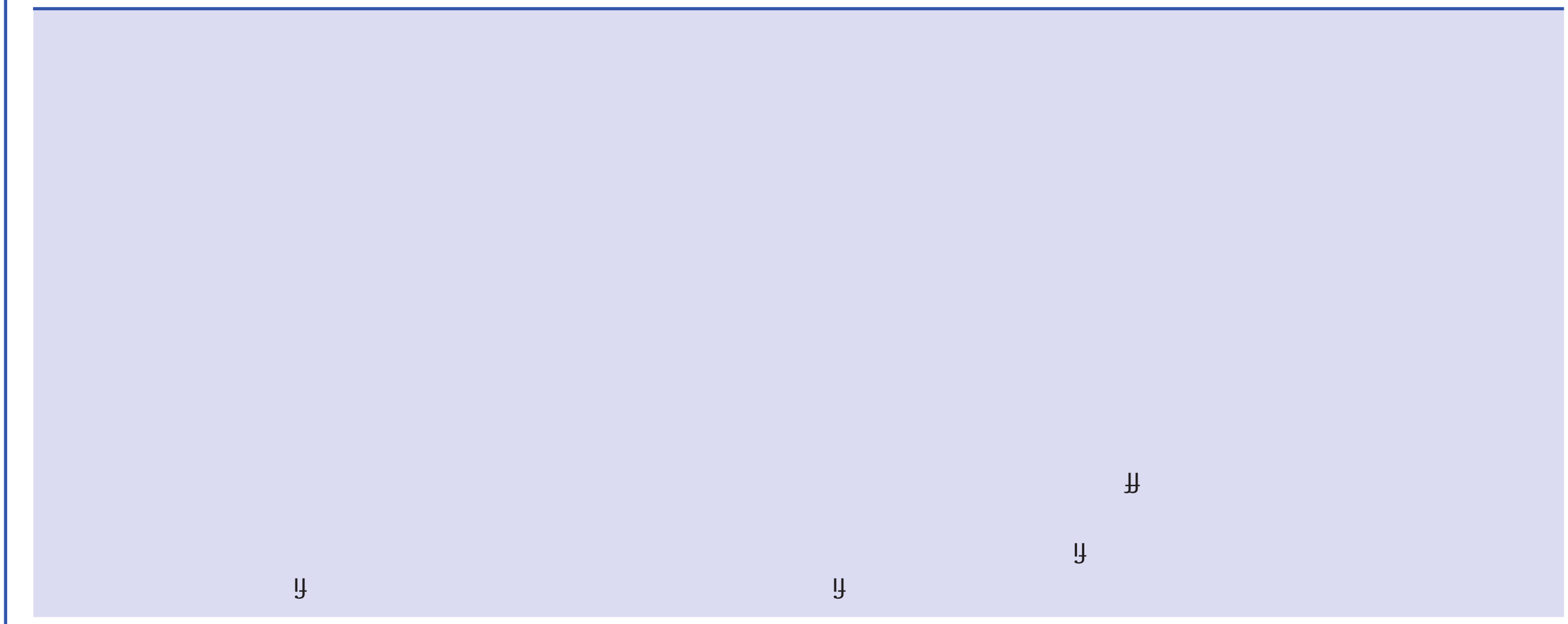


- Administer analgesic.
- Administer antibiotic (Bactrim DS by mouth twice a day) for signs of infection (i.e., cellulitis).
- Follow up in 3 to 5 days.
- Refer for possible excision and grafting if healing is delayed beyond 3 weeks.

Management of Burns to Specific Anatomical Regions

Hand Burns

- Assess for neurovascular compromise.
- Perform escharotomies for deep circumferential injury.
- Stabilize open joint deformities with K-wires.
- Elevate.
- Splint in a position of safety.
- Refer patient for occupational therapy.
- Wound care.
  - Apply Xeroform gauze.
  - Use Biobrane glove for superficial burns.
  - Silver-impregnated dressings for deeper injury.
- Full-thickness burns are referred for early excision and grafting to prevent scarring and contracture leading to dysfunction.

Facial Burns

- Evaluate for inhalation injury.
- Assess for injury to eyes and ears.
- Keep head elevated.
- For superficial and deep burns.
  - Daily cleansing.
  - Apply bacitracin ointment.
- For full-thickness burns.
  - Allow 5 to 7 days of healing before committing to grafting.
  - Cover temporarily with amnion or bacitracin.

Ear Burns

- Assess external canal and drum for otitis media or externa and tympanic membrane perforation.
- Apply topical Sulfamylon or gentamicin ointment to exposed cartilage.
  - Beware of chondritis.
  - Avoid placing pillows under the head.

Eyelid Burns

- Irrigate the affected eye with buffered saline solution.
- Perform fluorescein examination to identify corneal injury; consult an ophthalmologist.
- Superficial burns.
  - Thin layer of bacitracin ointment—do not contaminate eye.
- Full-thickness burns.
  - Excise and graft with full-thickness skin early to prevent ectropion and corneal exposure.

Burns to the Genitalia

- Insert Foley catheter to maintain patency of urethra.
- Penile escharotomy for circumferential injury.
- Partial-thickness burns heal spontaneously with conservative management—amnion, Polysporin (Pfizer Pharmaceuticals), bacitracin.
- Refer full-thickness burns for grafting; dress with Silvadene.

Electrical Burns

Electrical burn injury results from a spectrum of low- to high-voltage electrical exposure from lightning, direct electrical contact (electric shock), and electrical arching. The passage of the electric current through the body causes thermoelectric burns. **Flash burns** are thermal burns caused by the heat generated by an arc of electricity. **Flame burns** may result from ignition of clothing. The systemic manifestations of electrical injury are generally greater than the local tissue injury and are potentially fatal. The systemic complications of electrical injury are outlined in **Table 5.7**.

Table 5.7 Systemic Effects of Electrical Exposure

- Cardiovascular
  - Changes in the permeability of myocyte membranes
  - Cardiac arrest and ventricular fibrillation
  - Conduction defects
  - Creation of arrhythmogenic foci due to myocardial necrosis
- Neurologic
  - Loss of consciousness
  - Confusion
  - Amnesia
  - Seizures
  - Visual disturbances
  - Delayed onset paralysis
- Respiratory
  - Apnea from damage to cerebral respiratory center
- Renal
  - Acute tubular necrosis
  - Myoglobinuria
- Musculoskeletal
  - Myonecrosis
  - Rhabdomyolysis
  - Compartment syndrome
  - Fractures and dislocations from tetany

Management of Electrical Burns

- Acute airway management and resuscitation.
- Admit for observation or refer to burn unit.
- 24-Hour continuous cardiac monitoring and serial assessment of myocardial damage (creatine kinase [CK], troponin, L-lactate dehydrogenase [LDH]).
- Evaluation for rhabdomyolysis and myoglobinuria.
  - Diagnosis:
    - Increased urine pigment—red.
    - Urine dipstick is heme positive, but no RBCs are seen on microscopic evaluation.
    - Increased urine myoglobin.
  - Treatment:
    - Increase renal perfusion.
    - Aggressive resuscitation with intravenous fluids.

        - To maintain adequate urinary output, at least 0.5 mL/kg/h of urine (35 mL/h for a 70-kg patient), but preferably 50 to 100 mL/h.
        - Mannitol 0.25 to 1 g/kg over 20 minutes every 4 to 6 hours.
    - Alkalinize urine.
        - Add sodium bicarbonate—1 to 2 mEq/kg/d to IV fluids; adjust dose according to serum and urinary pH.
- Evaluation of the limbs for compartment syndrome and need for escharotomy.
- **MRI or CT evaluation for deeper injuries.**
- Ophthalmologic and otoscopic evaluation.
- CT scan of the head is indicated in all high-voltage injuries.
- Evaluation for hidden injuries—spinal cord injury, blunt thoracic or abdominal trauma.
- Supportive care.

Chemical Burns

Approximately 3% of all burns are secondary to chemical exposure, and 30% of burn deaths are due to chemical injuries. More than 25,000 home or industrial products are available that can cause chemical injury. The resultant injury from chemical solutions causes tissue protein coagulation and necrosis. The offending agent continues to destroy the tissues until the agent is neutralized or completely removed. Deeper penetration of the chemical compound can result in severe systemic toxicity. Common household agents and neutralizing substances are outlined in **Table 5.8.**

Characteristics of Chemical Burns

- Acid burns.
  - Tissue damage leads to **coagulation necrosis.**
  - Cause exothermic reactions with exposure.
  - Associated with hypocalcemia and hypomagnesemia.
  - Exposure may lead to inhalation injury.
  - Systemic toxicity may lead to hepatic or renal failure.
- Alkali burns.
  - Constituent of lye exposure.
  - Tissue damage leads to **liquefaction necrosis** and saponification of fats.
  - Tissue injury appears less severe than the actual depth of injury.
  - Alkali burns are associated with a higher incidence of systemic toxicity.

Table 5.8 Common household agents associated with chemical injury

Agent	Common use	Treatment
Phenol	Deodorant Sanitizer Plastics Dyes Fertilizers Explosives Disinfectants	Polyethylene glycol Vegetable oil Bacitracin ointment
Phosphorus	Explosives (fireworks) Poisons Insecticides Fertilizers	Lavage with 1%copper sulfate Castor oil
Sodium hypochlorite	Bleach	Milk
Potassium permanganate	Deodorizer	Egg white
	Disinfectant	Paste Starch
Lye	Drain cleaner	Water lavage Mafenide acetate
Chromic acid	Metal cleansing	Water lavage

Management of Chemical Burns

- Obtain a thorough history to identify offending agent.
- Carefully inspect hands, face, and eyes.
- Remove all clothing and sources of chemical contact.
- Immediately irrigate with water (except phenol). If the patient presents with a severe chemical exposure, plan on irrigation for hours in a shower (especially lye exposures). Small exposures can be treated with smaller volumes of fluid. Always err on the side of more fluid irrigation than needed.
- Resuscitate based on amount of surface exposed and monitor urine output.
- Consider antidote—refer to toxicologist, poison control center, or local burn center for assistance with management.
- Monitor electrolytes and obtain blood gas to assess for systemic toxicity.

- Provide supportive therapy in a monitored environment for large burns.
- Once irrigated, dress wounds with Silvadene or silver-impregnated dressing.
- Refer patients to burn centers and specialized facilities for excision and graft of mid-dermal to full-thickness chemical burns.

Treatments for Specific Chemical Burns

- Sodium or lithium metal, mustard gas.
  - Cover with oil, sand, or Class D fire extinguisher; excise immediately.
  - Do not irrigate with water.
- Phenol.
  - Wipe with polyethylene glycol.
  - Do not irrigate with water.
- Phosphorus.
  - Copper sulfate irrigation.
- Hydrofluoric acid.
  - Irrigate with 5%calcium gluconate or massage with 2.5%calcium gluconate gel. If pain persists, inject 5%calcium gluconate subcutaneously until pain is relieved.
  - Magnesium sulfate subcutaneous injection may also be used.

Chemical Burn Triage

Due to the unique mechanism of chemical burn injury, specialty assistance should be sought from the American Association of Poison Control Centers' help line (800–222–1222) or a local burn unit. Patients with the following characteristics should be admitted and possibly referred to a burn unit:

- Chemical injury > 15%TBSA.
- Full-thickness burns.
- Burns to the perineum, eye, foot, hand.
- Multiple comorbidities.
- Patients at extremes of age.

Intravenous Injection Injuries (IV Infiltrates)

Injection injuries are a form of chemical burn that results from extravisation of an irritating chemical during intravenous injection. The result is an interstitial inflammatory process that will lead to local tissue necrosis and possibly systemic toxicity. The degree of injury is dependent on the tox-

icity, amount, and concentration of the material injected. The mechanism of injury is by either induced ischemia, osmotic derangement within the tissue, or direct cytotoxicity (**Table 5.9**). The reaction is progressive and worsens in the acute phase that can last days (**Fig. 5.11**). The local reaction is painful and begins as erythema that can progress to full-thickness necrosis of the skin and even deeper penetration through the fascia to the muscle.

Table 5.9 Common agents associated with injection injuries

- Ischemia-inducing agents
  - Dobutamine
  - Dopamine
  - Epinephrine
  - Norepinephrine
  - Vasopressin
- Hypertonic solutions
  - Calcium chloride
  - Calcium gluconate
  - Intravenous contrast agents
  - Potassium
  - TPN
  - 30%urea
  - 10%dextrose
- Cytotoxic agents
  - Chemotherapy drugs
  - Digoxin
  - Doxorubicin
  - Nafcillin
  - Tetracycline
  - Sodium bicarbonate

Treatment of Injection Injuries

The majority of injection injuries involving dangerous agents occur in a monitored environment. Therefore, the amount of exposure is limited and the management is conservative due to the relatively small burden of injury.

The basic principles of conservative management are as follows:

- Remove the IV line.
- Obtain a history of the agents being infused.
- Elevate the affected area.

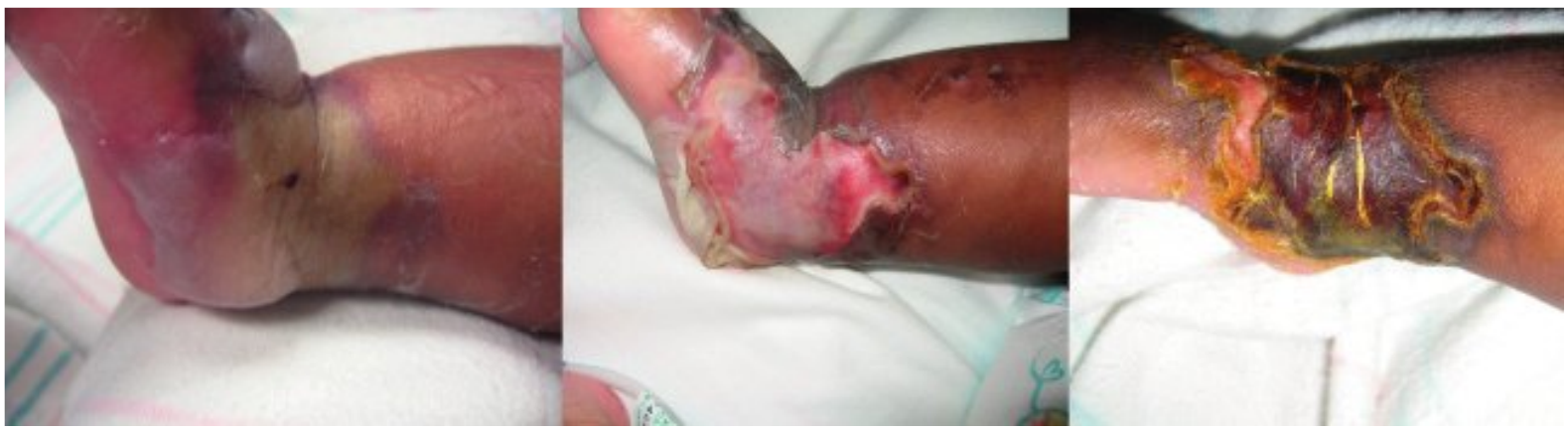


Fig. 5.11 IV infiltrate injury to the lower extremity demonstrating the progression from superficial to full-thickness injury.

- Apply a cold compress.
- If the hand is involved, utilize a splint in the safe position (wrist in dorsiflexion, MCPs flexed).
- Diligent observation.
  - The soft tissue reaction may progress for 4 to 5 days.
- Antibiotics for signs of cellulitis.

In rare situations where there is more severe progressive tissue damage, consider a more aggressive approach. This would include the instillation of an antidote into the affected soft tissue. Possible antidotes include:

- **Hyaluronidase: Hyperosmolar and cytotoxic agents.**
- **Phentolamine: Vasoconstricting agents.**
- **Corticosteroids: Chemotherapy drugs.**

Early surgery is rarely indicated and is reserved for cases of massive soft tissue injury associated with injection, compartment syndrome, or vascular compromise. The patient is referred for excision and reconstruction of areas that have failed after 2 to 3 weeks to heal with conservative management.

Frostbite

Cold injury results from both tissue freezing (frostbite) and nonfreezing injury (trench foot). Frostbite is the result of tissue freezing after exposure to temperatures < 28°F (–2°C). At such temperatures, ice crystals form intracellularly that cause tissue destruction, and intravascular crystals contribute to microvascular occlusion. The pathogenesis of trench foot is secondary to exposure, usually of an extremity to a moist environment at temperatures of 32 to 50°F (1 to 10°C) for long periods. This creates a scenario of excessive heat loss in the involved region. There is also ischemic perfusion secondary to vasoconstriction. Patients with cold thermal injury will commonly experience severe pain, pruritus, numbness, paresthesias, and hyperemia, which may last up to 6 weeks.

Management of Cold Thermal Injury

- Rapid rewarming of the involved area.
  - Water immersion—heated to 104°F (40°C).
- Administer.
  - Parenteral analgesics.
  - Tetanus prophylaxis.
  - Systemic prostaglandin inhibitors—ibuprofen.
  - Topical thromboxane inhibitors, e.g., aloe vera.
- Débride necrotic tissue.
  - Whirlpool (hydrotherapy débridement).
  - Allow complete wound demarcation before committing to radical surgical débridement.
- Elevate affected areas.
- Begin early passive range of motion to all involved extremities.
- Dress wound twice a day and protect from further injury.

6 General Assessment and Management of Facial Trauma

Facial trauma varies in severity from a simple laceration to life-threatening injury (**Fig. 6.1**). All patients require an appropriate trauma evaluation beginning with the ABCs (airway, breathing, circulation). The patient must also be evaluated for other serious injuries before focusing on treating the facial injury. Usually, treatment of any intra-abdominal, thoracic, or neurologic injury takes precedence. Evaluation and treatment should be coordinated between the trauma, thoracic, vascular, ENT, orthopaedic, ophthalmic, and neurosurgical services.

The exam should start with a detailed medical, surgical, social, and previous craniofacial injury history. The mechanism of injury should be ascertained to gauge the force of contact and to determine the location of potential fractures or soft tissue injuries. Additional considerations include loss of consciousness, breathing difficulties, vision and hearing deficits.

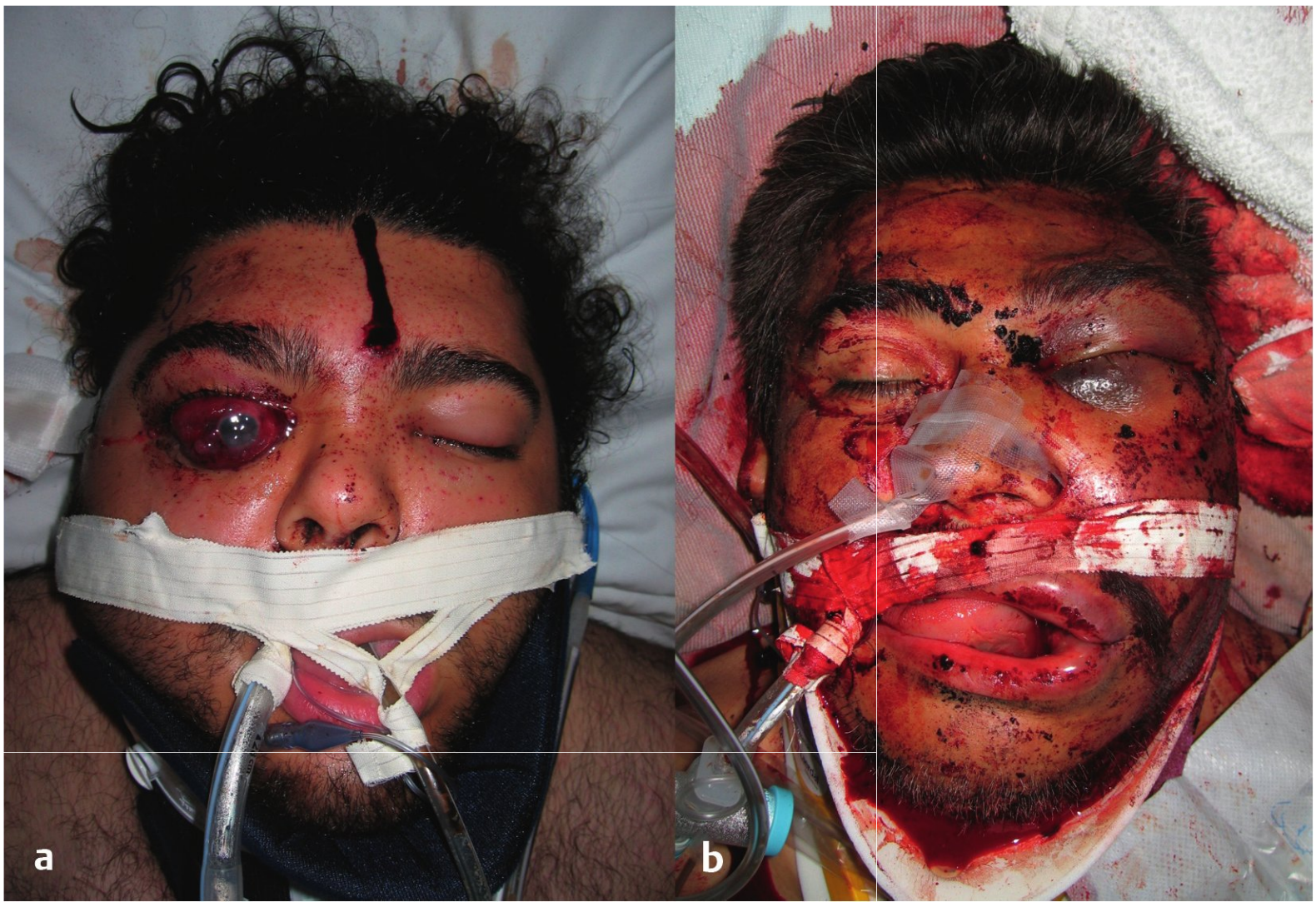


Fig. 6.1 Severe **(a)** penetrating and **(b)** blunt trauma to the face.

Airway Establishment

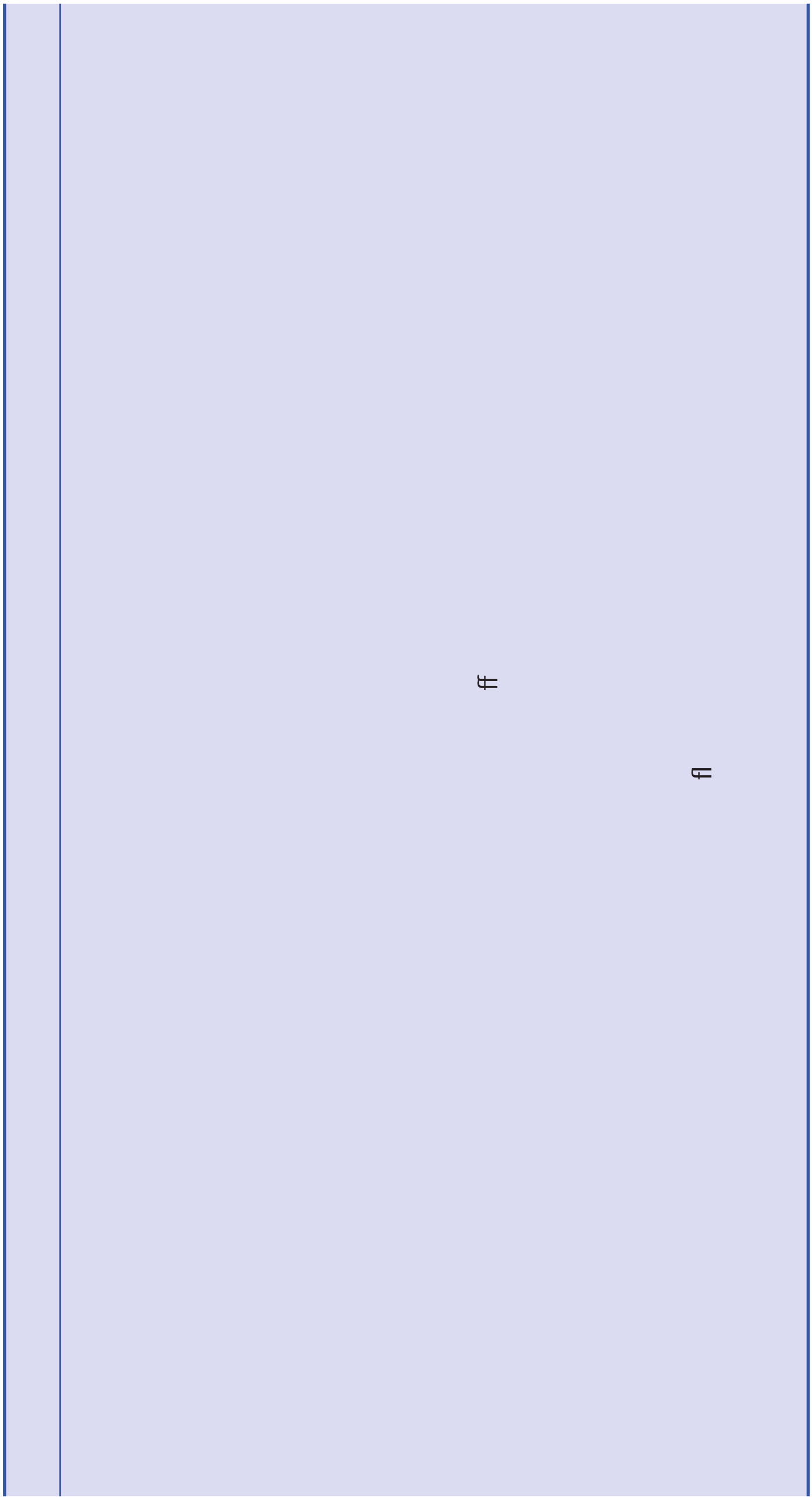
Avoid nasal intubation in patients suspected of having a skull base fracture or excessive midface trauma to prevent the possibility of intracranial disruption. Elective oral endotracheal intubation should be considered in patients with severe panfacial trauma, especially in the midface and mandible. Patients with large posterior base of tongue injuries (including lacerations) should be electively intubated. Any intubation should be done with cervical spine (C-spine) precautions: 10% of facial traumas harbor a C-spine injury. Tracheostomy should be considered in complex cases, particularly when nasal or oral trauma precludes upper airway cannulations.

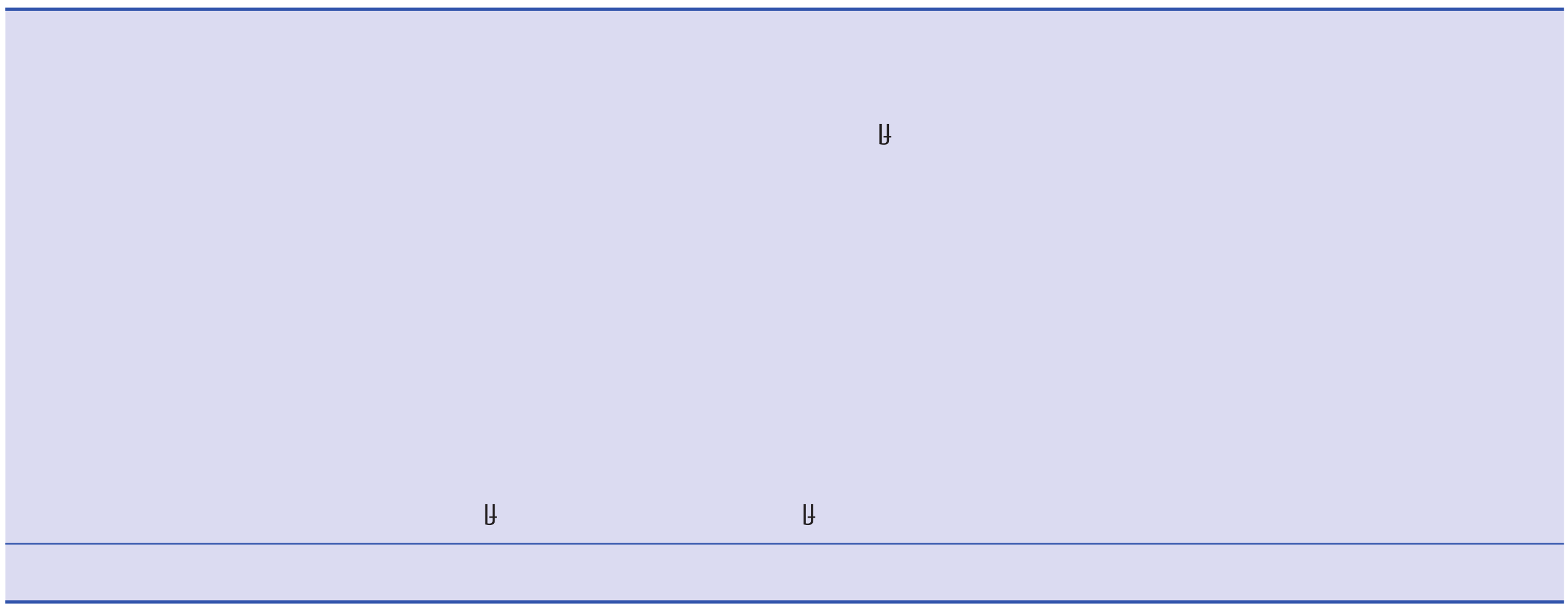
Patient Evaluation

Examination

Remove all articles of clothing and jewelry. Irrigate dirt, debris, foreign bodies, and dry crusted blood to avoid obscuration of the injury. Note all lacerations, asymmetries, bleeding, bruising, or foreign bodies. An organized, systematic approach is recommended to avoid any missed injuries. A careful examination should include assessment for

- Raccoon eyes (periorbital ecchymosis)—skull base fracture.
- Battle's sign (postauricular ecchymosis)—skull base fracture.
- Otorrhea—skull base fracture, condylar fracture.
- Hemotympanum—skull base fracture.
- Perforated tympanic membrane.
- Epistaxis—nasal fracture.
- CSF rhinorrhea—cribriform plate fracture, NOE fracture.
- Intraoral injury.
  - Edema.
  - Bleeding.
  - Gingival bleeding.
  - Fractured/loose/displaced teeth.
  - Dental caries.
- Nasal septal hematoma.
- Assess the patient's dental occlusion and have the patient compare this with the preinjury occlusion. Abnormal occlusion is highly suggestive of mandibular, maxillary, and Le Fort fractures.
- Test for motor and sensory deficits (**Table 6.1**). Test all muscles of facial expression and follow with a detailed sensory exam.





Facial Palpation

- Tenderness.
- Crepitus/subcutaneous emphysema.
- Bony step-offs.
- Scalp—gently palpate to uncover depressions/crepitus.
- Forehead—frontal sinus fracture.
- Orbital rim.
- NOE (naso-orbital-ethmoid)—palpate intranasally and inward from medial canthus; bony movement to diagnose NOE fracture.
- Nasal bridge.
- Zygoma.
- Maxilla—gently depress the maxilla with both thumbs to rule out Le Fort fractures. If mobile, grab the central incisors between thumb and index finger with one hand and hold the nasal spine with other hand. Movement of the entire dental alveolus indicates a Le Fort I fracture; movement of the nasal bridge indicates Le Fort II or III.
- Mandible—preauricular pain on palpation can be indicative of a condylar fracture.
- Neck exam—performed with caution in relation to the C-spine.

Ophthalmic Assessment

- Inspection.
  - Corrective lens (contacts or eyeglasses).
  - Enophthalmos/exophthalmos.
  - Retrobulbar hematoma.
  - Interpupillary distance—normally 30 to 32 mm; greater than normal may indicate an NOE fracture.
  - Hyphema—blood layering in the inferior aspect of the anterior chamber. An ophthalmologist should be consulted immediately based upon the potential increase in intraocular pressure.
  - Corneal abrasion.
  - Subconjunctival hemorrhage.
  - Chemosis—scleral edema.
  - Upper eyelid ptosis may indicate superior fissure syndrome.
  - Fat protrusion.
- Visual acuity.
  - Test each eye separately by measuring patient's ability to read legible fine print (ID card).
  - Diplopia.

  - Red color saturation—*first color affected* in impending optic nerve injury.
  - Compare and contrast red color perception in each eye individually.
  - Variation in exam may indicate optic nerve injury.
- Extraocular muscle function—muscle entrapment; perform forced duction test (Chapter 9).
- Pupillary response—reactivity, dilated, constricted.
  - Consensual light response.
    - If one eye is exposed to light, there should be ipsilateral and contralateral constriction of the pupils.
    - An injured eye may be fixed and dilated secondary to intrinsic damage to that eye, but maintain normal afferent optic nerve function. In this scenario, there will be loss of ipsilateral pupillary constriction; however, contralateral constriction is maintained.
  - When contralateral pupillary constriction is lost, this indicates an afferent pupillary defect (APD) in the affected eye.
    - In a patient suspected of an afferent pupillary defect, exposing the unaffected eye to light will cause both pupils to constrict.
    - When the light is brought back to the affected pupil, this pupil will dilate (due to consensual relaxation), instead of constricting, confirming an afferent pupillary defect in this eye.
  - Medial/lateral canthal tendon stability—traction test: pull laterally on medial aspect of lower eyelid; laxity is indicative of medial canthal tendon disruption.

Immediate attention should be paid to any signs of acute optic compressive neuropathy, penetrating globe injuries, or vision loss. Any questionable injury or condition warrants an ophthalmologic consult/evaluation.

Radiographic Evaluation

If facial fractures are suspected, a CT scan of the face is warranted (**Fig. 6.2**). High-resolution (axial, sagittal, and coronal) views should be obtained, and 3D reconstructions obtained if possible (**Fig. 6.3**).

In the case of a mandible fracture, a Panorex radiograph is the only plain film that should still be routinely obtained even if a CT scan of the face is also obtained (**Fig. 6.2**). Panorex radiograph films are excellent for evaluating fractures and condyles, and they provide a single plain film view of the entire mandible. They are also useful in evaluating dentition such as impacted molars. Panorex films are not good for evaluating fractures of the symphyseal region.

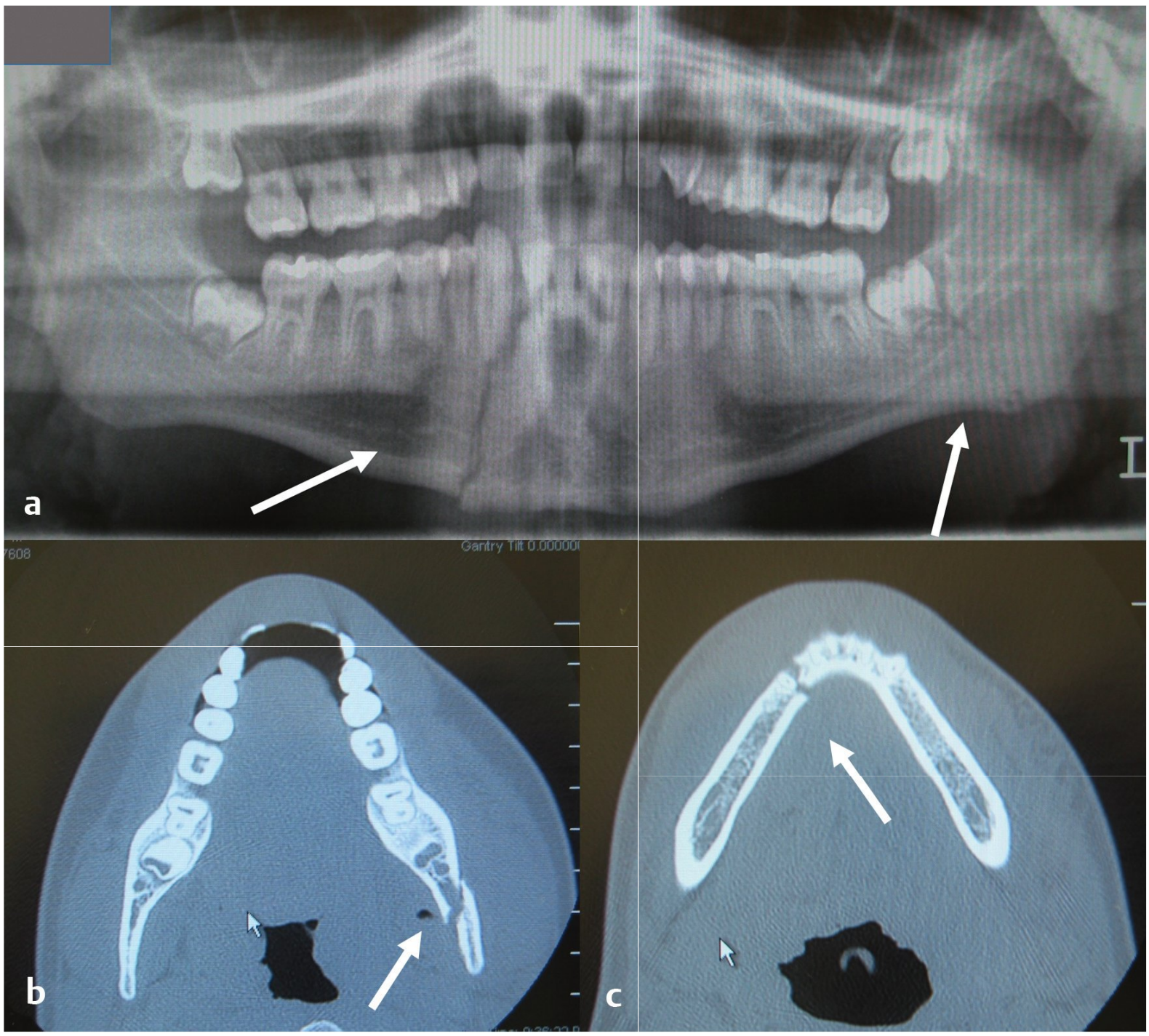


Fig. 6.2 **(a)** Panorex radiograph of a mandible with fractures of the parasymphysis and angle. CT scan of the mandible demonstrating the same fractures of **(b)** angle and **(c)** parasymphysis.

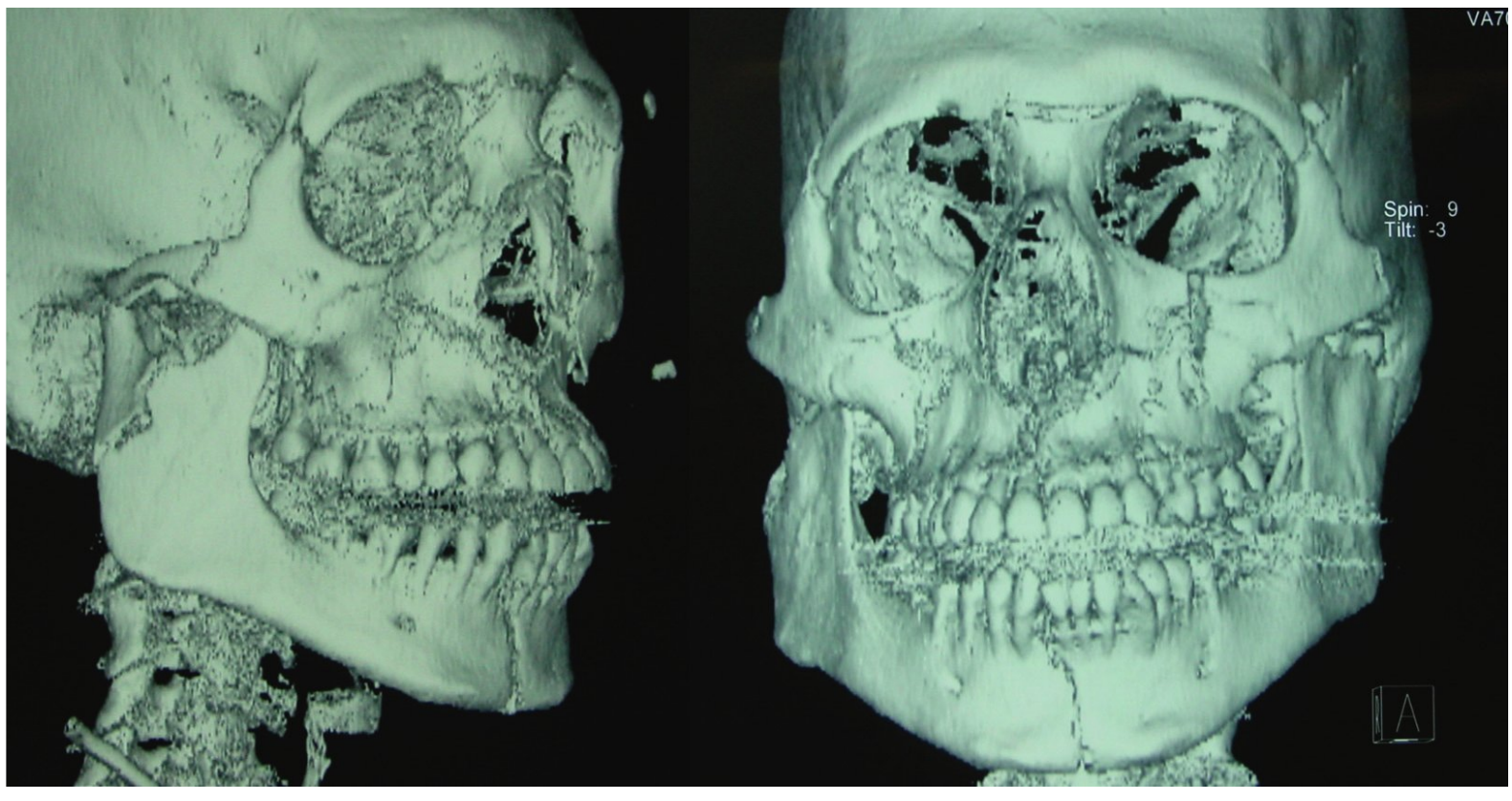


Fig. 6.3 3D reconstruction of the facial bones.

Additional plain films (rarely used) include:

- *Waters view:* PA view that requires neck extension. Occipital-mental projection that optimizes superior and inferior orbital rims, nasal bones, zygoma, and maxillary sinuses.
- *Caldwell view:* PA view that requires neck flexion. Occipital-orbital projection that optimizes frontal bones and sinus, lateral walls of the maxillary sinus, orbital rims, and zygomaticofrontal sutures.
- *Lateral view:* Optimizes anterior frontal sinus wall, anterior and posterior maxillary sinus walls.
- *Submental view:* Optimizes view of zygomatic arches.

Urgent Interventions

Hemorrhage Control

Large, deep lacerations can first be treated with irrigation, followed by application of pressure to control bleeding. Active arterial bleeders can be tied off or suture ligated with 4–0 Vicryl suture. Wounds can be extended with a scalpel to gain exposure. Because of the extensive collateral vascular supply of the face and scalp, even lacerations to the facial artery can be ligated if necessary to control bleeding. To avoid injury to nerves and other vital structures, do not blindly clamp any vessels. If direct visualization is not possible due to excessive bleeding, place 4x4 gauze on the wound, place pressure on the wound, and take the patient to the operating room.

Epistaxis can be controlled with anteroposterior nasal packing. Obtain a nasal speculum and bayonet forceps, along with a Cottonoid (Codman & Shurtleff) soaked in epinephrine (1:200,000) (**Fig. 6.4a**). Four percent cocaine may also be used, but with great caution (Afrin in an alternative). If a Cottonoid is not available, cut Xeroform gauze into strips and layer those into the nasal cavity. Under direct visualization using a nasal speculum, layer (do not stuff or pack) the gauze or Cottonoid into the posterior nasal pharynx (**Fig. 6.4**). When nasal packing is used, start the patient on prophylactic antibiotics to prevent streptococcal toxic shock syndrome.

Occasionally, midface and mandibular fractures can result in severe bleeding. Common vascular structures include the maxillary artery, alveolar artery, retromandibular vein, facial artery/vein, or buccal branches of the facial artery. Access to these structures is difficult, and attempts should be made to obtain some crude reduction to tamponade the bleeding. A Barton bandage can be applied using Kerlix reinforced with an Ace bandage to apply compression to the face. The Kerlix is wrapped coronally multiple times to hold the mandible in occlusion, then wrapped around the forehead. Reinforce with an Ace wrap (**Fig. 6.4c**).

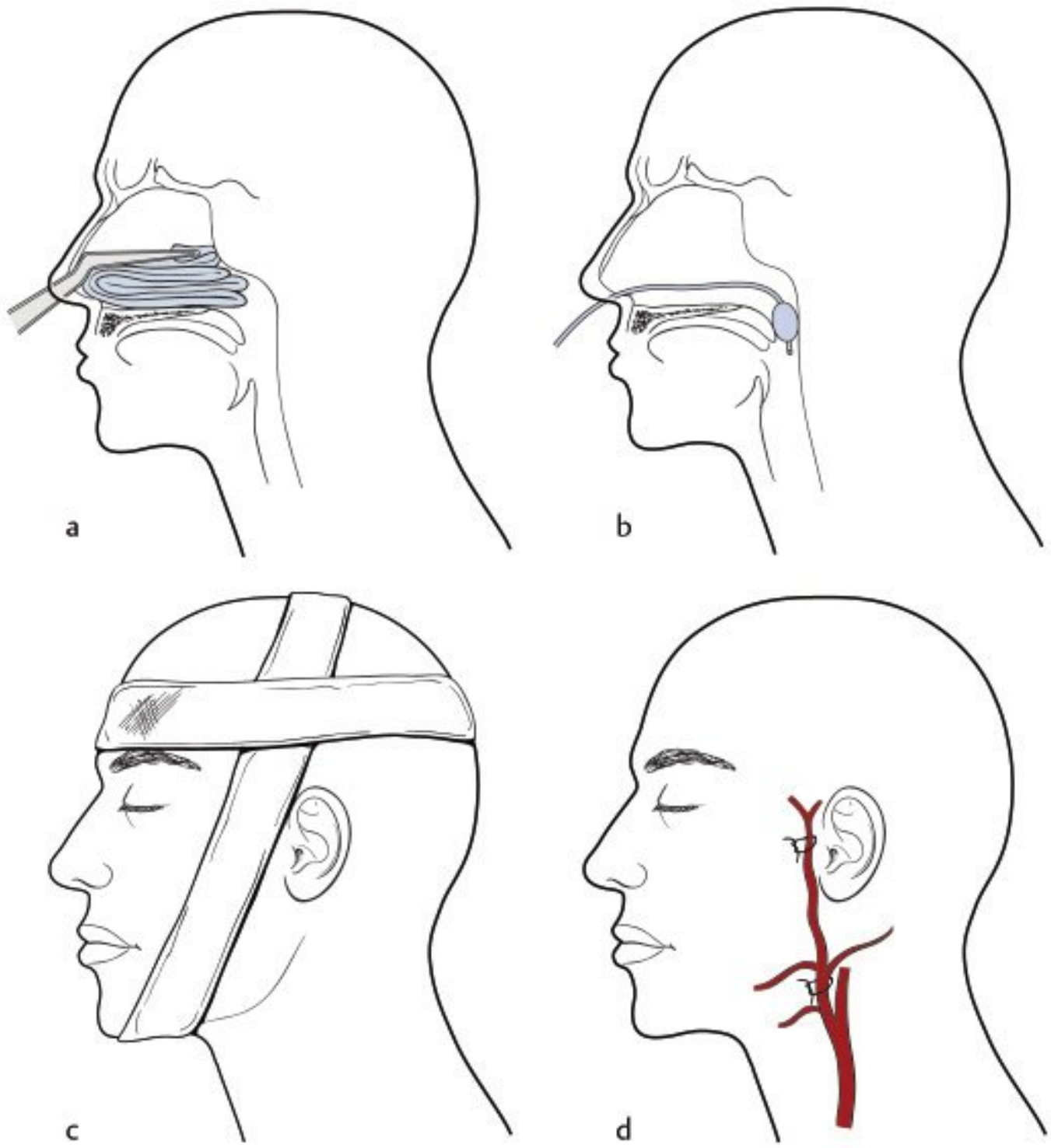


Fig. 6.4 Techniques for achieving hemostasis. **(a)** Anterior nasal packing. **(b)** Posterior nasal packing. **(c)** Barton bandage head wrap. **(d)** Selective arterial ligation or embolization.

Mandibular Stabilization

Comminuted or complex fractures of the mandible can be acutely stabilized to reduce oral airway edema and reduce pain. A bridle wire (25-gauge wire passed around two teeth flanking a fracture) can be used to help stabilize a fracture. Stabilization will also help to offset the patient's pain. A more effective technique may be Essig wiring. This involves passing a 25-gauge wire around two teeth on either side of the fracture and then placing interdental wires above and below the first wire.

Nasal Septal Hematomas

Nasal septal hematomas should be drained in the emergency room to prevent septal necrosis. Using an 18-gauge needle or 11-blade scalpel, make a small perforation in the mucosa with a nasal speculum under direct visualization. Evacuate the hematoma and apply a compressive dressing (Xeroform layered packing with bacitracin ointment) to prevent reaccumulation (**Fig. 6.4a**). Note that if there was no epistaxis at the time of the injury, the presence of a nasal bone fracture is less likely.

Auricular Hematomas

Auricular hematomas should be treated like septal hematomas. Drain with a scalpel or an 18-gauge needle (aspirate) and apply pressure dressing. Bolster the ear with rolled-up Xeroform gauze sutured in place with through-and-through 2–0/3–0 nylon or Prolene (**Fig. 6.5**).

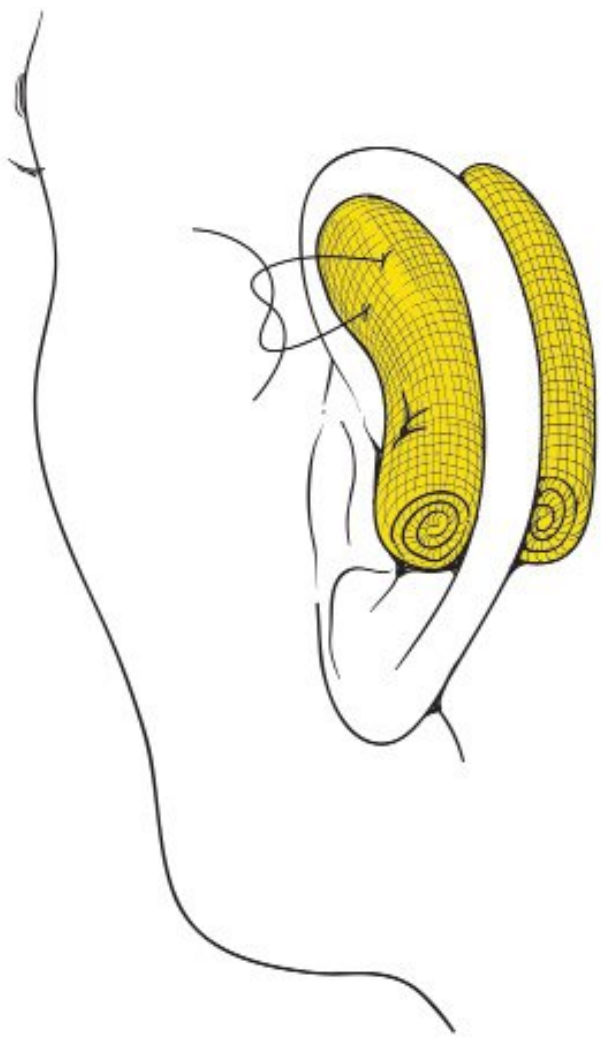


Fig. 6.5 Ear bolster dressing technique.

Acute Optic Compressive Neuropathy

Acute optic compressive neuropathy requires emergent lateral canthotomy (**Fig. 6.6**) along with mannitol, acetazolamide, and methylprednisolone to decrease intraocular pressure and to control orbital nerve edema (see Chapter 8).

- Mannitol: 50 to 100 g (1.5 g/kg) of 5% solution IV over 2 hours; repeat dose to maintain urine output (> 30 to 50 mL/hour), with a maximum of 200 g/d. Test dose with 200 mg/kg.
- Acetazolamide: 250 mg by mouth every morning or 5 mg/kg IV every 24 hours.
- Methylprednisolone: Loading dose of 30 mg/kg; then, after 2 hours, 15 mg/kg every 6 hours.

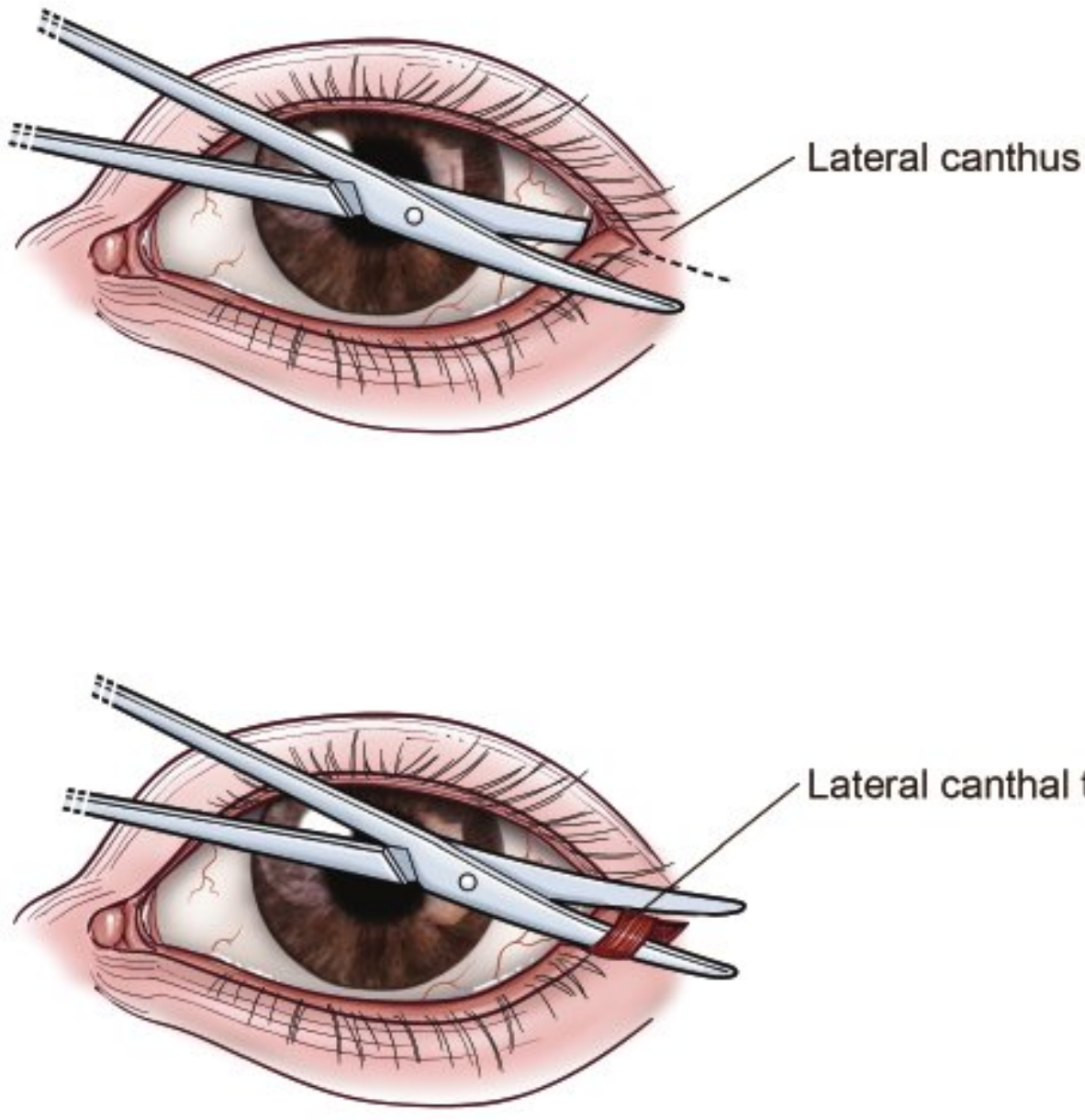


Fig. 6.6 **(a)** Lateral canthotomy. **(b)** Release of the lateral canthal tendon.

Treatment Sequence and Timing

Polytrauma patients will be admitted to the trauma service. Bony repair is delayed until the patient is stable. If possible, repair should be performed immediately to avoid excessive edema or delayed from 10 days to 2 weeks after injury to allow the edema to subside. Soft tissue injuries should be irrigated and repaired within 8 hours. Do not leave open wounds on the face to granulate; attempt closure by any means. Remember that closure does not need to be definitive because revisions can be made later during bony repair.

7 Facial Lacerations

Facial soft tissue injuries range from simple lacerations to complex avulsions of the facial skin and appendages (**Fig. 7.1**). These injuries contribute to a large number of visits to emergency rooms in the United States each year. The mechanism of injury varies from penetrating to blunt trauma and also includes shearing modalities. Motor vehicle accidents (MVAs) comprised the majority of these injuries. Advanced automobile safety equipment has reduced the prevalence of these injuries. However, the overall incidence of facial injuries due to sports- and job-related injuries, animal bites, and factors related to domestic and interpersonal violence has remained constant. Treatment ranges from simple laceration repair to repair of specialized anatomical structures and microvascular replantation.

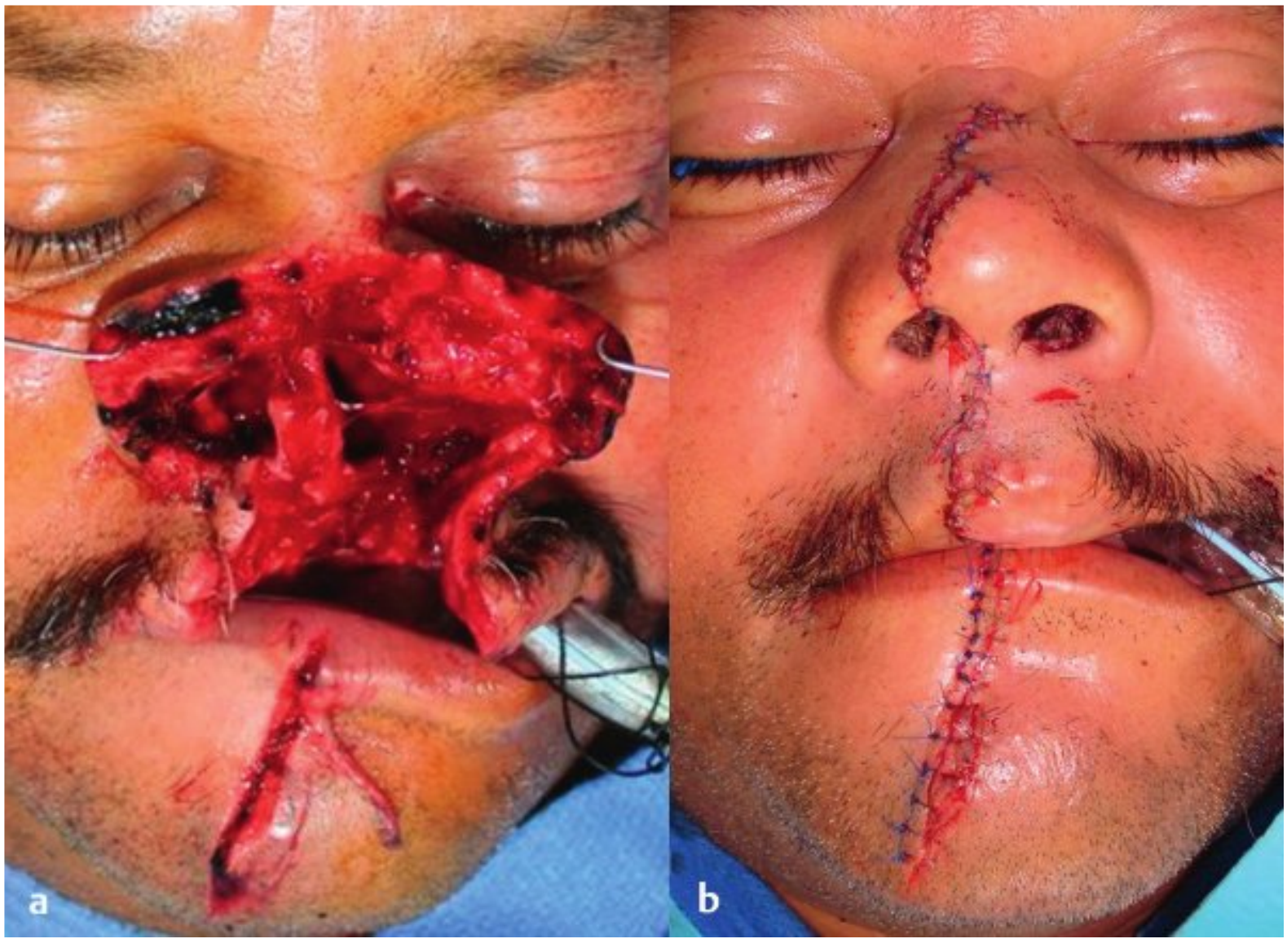


Fig. 7.1 Complex facial laceration **(a)** before and **(b)** after repair.

Assessment

The immediate priority for patients with facial injuries is to rule out a life-threatening airway or intracranial injury and to control the airway and stop any bleeding. Detailed evaluation of the facial trauma patient is outlined in Chapter 6.

The physical examination proceeds with inspection and palpation of the patient. Using a systematic approach from the scalp to the base of the clavicles, inspect for lacerations, localized areas of edema, and ecchymosis that may indicate underlying injury. Care must be taken to adequately remove any debris and dried blood in this region, which can easily camouflage lacerations and lead to missed injuries.

Diligently assess cranial nerve function by specific provocative maneuvers. Facial injuries associated with lacerations are grossly identified by inspection of facial asymmetry at rest and during animation and by assessing sensory function (see **Table 6.1** in Chapter 6). However, be cognizant that soft tissue edema may limit normal facial movement, leading to false readings of facial nerve function.

Utilizing palpation, appreciate focal areas of tenderness, depressions, crepitus, and edema that may indicate hematoma or a bony fracture. Patients who have injuries suspicious for facial fractures should have their wounds thoroughly irrigated, débrided, and closed, and should be referred for radiographic evaluation (see Chapter 6, Radiographic Evaluation).

Treatment

General Procedures

- Follow basic laceration closure procedures (see Chapter 3).
- Measure the laceration and assess for anatomical distortion of specialized structures.
- Irrigate and perform conservative débridement. Be mindful that overly aggressive débridement around the nose, eyelids, and brow may lead to severe disfigurement.
- For patients with severe “road rash” or a blast injury, clean the wound meticulously under loupe magnification. This procedure is time-consuming, but you will have better results.
- Tetanus prophylaxis.

- Local anesthesia prior to débridement and laceration repair will help facilitate the procedure.
  - Field blocks with 1%lidocaine/1:100,000 epinephrine through a 25- or 27-gauge needle.
  - Consider regional blocks for large lacerations isolated to a single nerve distribution.
  - Regional blocks of the trigeminal nerve are performed by instillation of 2 to 4 mL of local anesthesia (1%lidocaine or 0.25% Marcaine [Abbott Laboratories]) above the periosteum in the region of the nerve (**Fig. 7.2**).
- Manage superficial lacerations with minimal disfigurement conservatively.
  - Cleanse abrasions daily and apply antibiotic ointment (bacitracin twice a day).
  - Close small wounds with Steri-Strips (3M) or Dermabond or Dermabond Prineo (Ethicon).
- Close larger lacerations as soon as possible; waiting 2 or 3 days will compromise the results. Lacerations that present 6 to 8 hours after injury need to be thoroughly irrigated and the wound edges freshened prior to closure.

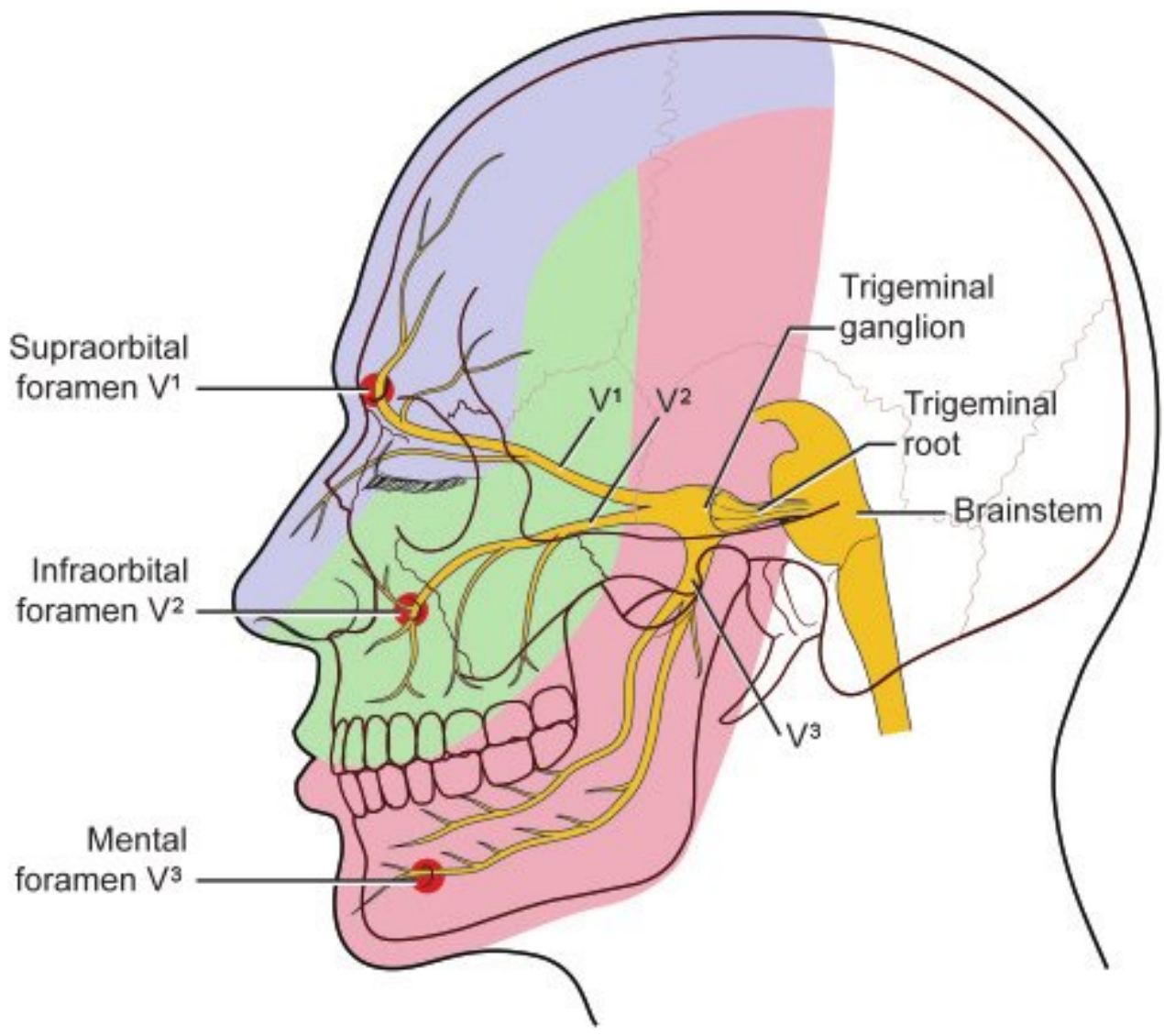


Fig. 7.2 Placement procedures for regional blocks of the trigeminal nerve for the repair of facial soft tissue trauma.

- To avoid depressed scarring, close deep tissue with the appropriate sutures.
  - Muscle—Monocryl 4–0, Vicryl 4–0.
  - Skin.
    - Deep layer—Monocryl 5–0, 6–0.
    - Superficial layer—nylon/Prolene 6–0, 7–0.
  - Mucosa—chromic 3–0, 4–0.

Pediatric patient considerations:

- Pursue radiographic evaluation to rule out any associated fractures.
- Use conscious sedation (Chapter 2) to reduce emotional trauma for the patient and to repair difficult lacerations in specialized areas safely (e.g., periorbital region).
- Use absorbable sutures.
  - Skin.
    - Deep layer—Monocryl 5–0, 6–0.
    - Superficial layer—fast-absorbing plain gut 5–0, 6–0.
  - Mucosa—chromic 5–0.
  - Muscle—Vicryl 4–0.

Lip Lacerations

- Approximate each layer of a full-thickness laceration (**Fig. 7.3**).
  - Muscle—Monocryl 3–0, 4–0; Vicryl 3–0, 4–0.
  - Skin—nylon/Prolene 6–0, 7–0.
  - Mucosa (all surfaces of lip)—chromic 3–0, 4–0.
- Instruct the patient to minimize oral movement as much as possible for 5 days after repair.
- Recommend a liquid diet for large intraoral lacerations, and cleansing wounds 5 times a day with Peridex mouthwash—swish and discard.

Lacerations through the white roll (skin–vermilion border):

- Align the white roll (skin–vermilion junction), philtral columns, and cupid's bow *before* injection with local anesthesia. These anatomical landmarks will be distorted by the edema that occurs after injection.
- Approximate the white roll exactly to obtain the best cosmetic outcome.
- Approximate the orbicularis oris at the appropriate height to avoid a depressed scar.
- The skin is approximated with a nonabsorbable suture (nylon, Prolene 6–0) at the white roll.
- Suture the vermilion with 5–0 chromic gut sutures.

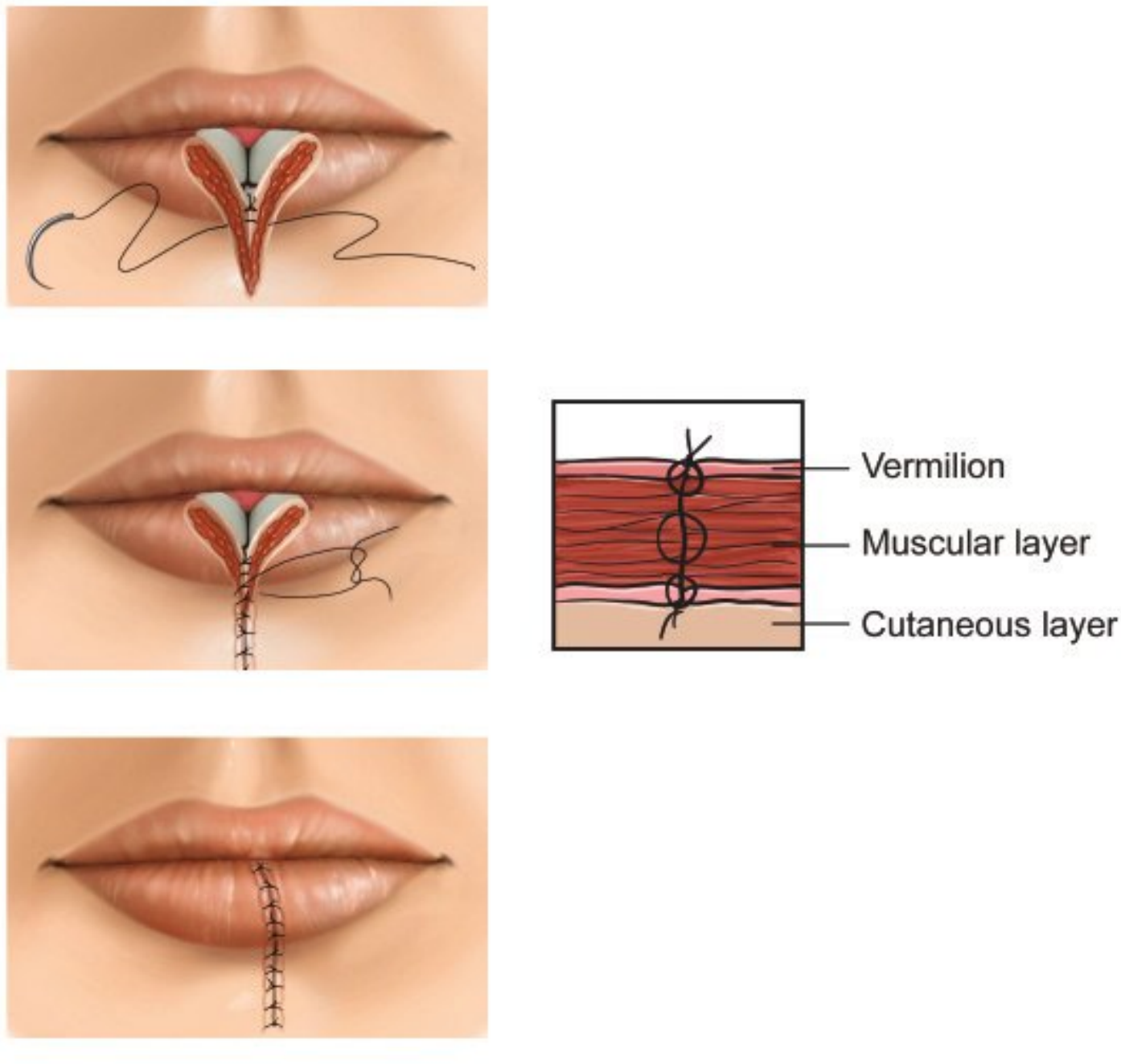


Fig. 7.3 Layered closure of lip lacerations.

Ear Lacerations

- Irrigate ear lacerations thoroughly, but débride conservatively to prevent cartilage exposure.
- Ensure skin closure over cartilage to avoid chondritis.
- Approximate skin and perichondrium in a single bite using nonabsorbable sutures (Prolene, 5–0 or 6–0).
- Prescribe oral prophylactic antibiotics (Bactrim DS by mouth twice a day) for 5 to 7 days.
- Clean and cover incisions twice a day with antibiotic ointment (Sulfamylon or gentamicin ointment).
- Prepare dressings to avoid hematoma formation (**Fig. 6.5**).
- Apply Xeroform with fluffed gauze in a pressure dressing with circumferential head wrap.
- Assess for perichondrial hematomas (Chapter 6).

Large Ear Defects

Large defects with either composite or excessive skin loss may require secondary reconstructive procedures for closure (i.e., skin graft, partial composite resection).

- Cover ear with Xeroform and employ frequent dressing changes until reconstruction to avoid desiccation of the cartilage.

Avulsion Injuries (Fig. 7.4)

- Treat immediately to avoid vascular compromise.
- Assess for perfusion of the avulsed fragment.
  - Laser photography with indocyanine green perfusion test (Spy, Novadaq) is useful to determine if there is perfusion to the partially amputated part.
- Débride, trim, and attach small avulsion fragments as a composite graft (< 1.5 cm).

Amputation and large avulsions may require microvascular attachment depending on the site of avulsion and residual vasculature. Alternatively, the cartilage architecture may be preserved by dermabrasion of the avulsed part with storage under a postauricular flap or in the abdominal subcutaneous tissue ("pocket principle"). This will allow use of this fragment for delayed reconstruction. However, this procedure is not as optimal as reattachment of the ear, if reattachment is possible. Microsurgical

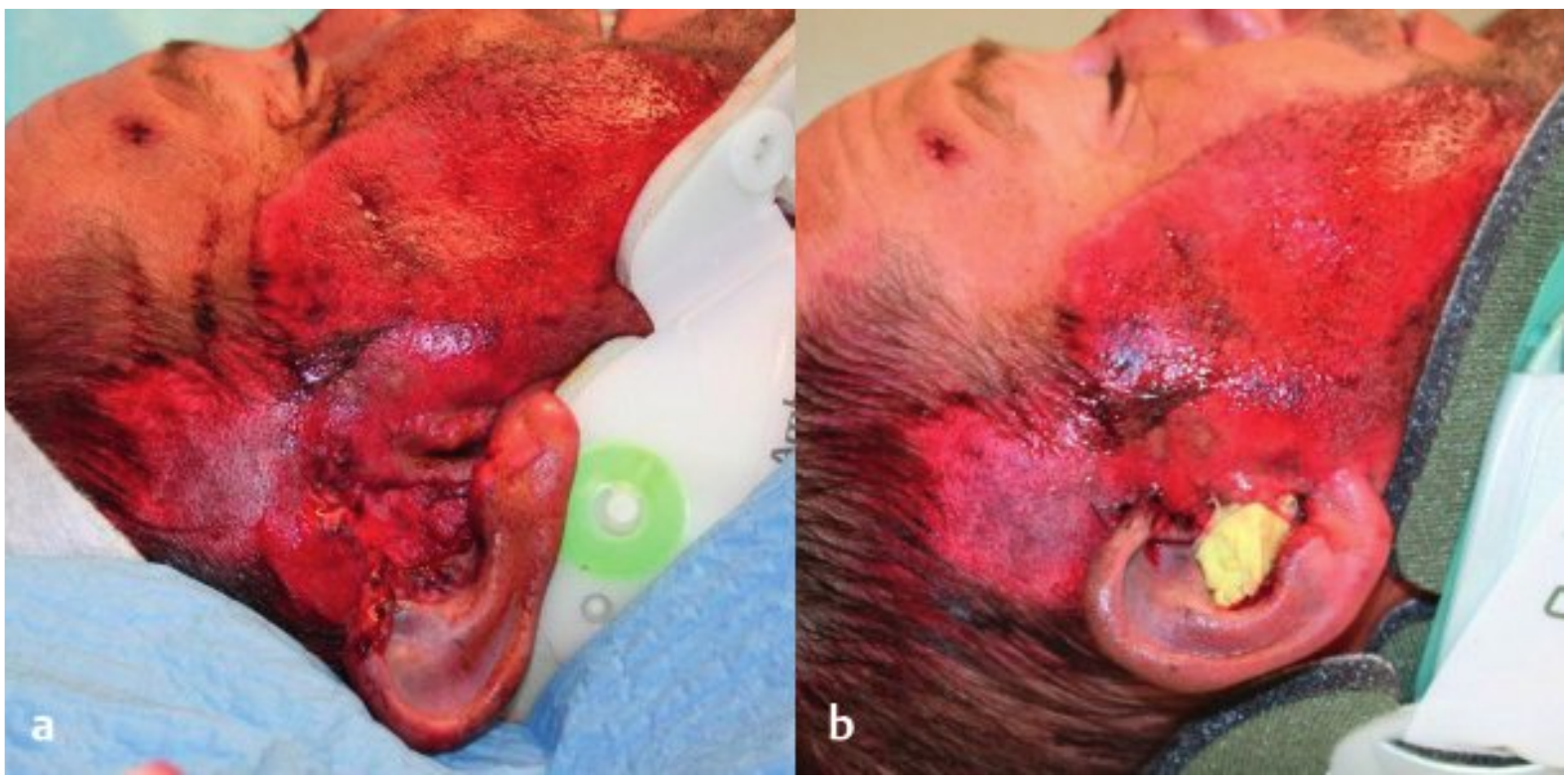


Fig. 7.4 **(a)** Ear avulsion injury with preservation of the posterior circulation **(b)** allowing attachment with complex repair.

replantation can be performed to the superficial temporal artery or the posterior auricular artery. Venous outflow is provided by drainage to the external jugular vein. Utilize vein grafts when necessary.

Venous congestion is a common problem after repair of avulsions. Large avulsed fragments and amputated ears will require leech therapy for survival.

Scalp Lacerations

- Rule out intracranial injury.
- Promote hemostasis with a pressure dressing until the environment is appropriate for exploration.
- Identify all lacerations by carefully removing debris and blood with hydrogen peroxide and water. Shaving is rarely necessary.
- Irrigate wounds with ample amounts of normal saline and remove any missed foreign bodies.
- Layered closure.
  - Galea—Vicryl or Monocryl 2–0.
  - Skin—full-thickness bites, continuous suture for hemostasis; Prolene (blue) 3–0, 4– 0, or staples.
- Use smooth pickups to pull hair out from the wound and from underneath the sutures.
- Use a Penrose drain cut longitudinally under scalp flap for wound drainage for 1 to 2 days, closed suction drains for large scalp avulsions.
- Large scalp avulsions (**Fig. 7.5**).
  - Determine adequacy of perfusion.
    - Look for bleeding at the skin edges.
    - Utilize indocyanine green angiography.
  - Thorough irrigation and layered closure over drains.
- Scalp amputations.
  - A sharp cutting amputation may provide vital targets for microsurgical attachment. Leeches can be applied for venous congestion if veins are weak or unavailable.
  - Shearing and shredding injuries may not have reliable vessels in the amputated portion for successful microsurgery.
    - Consider defatting the amputated part and grafting to the galea if intact.

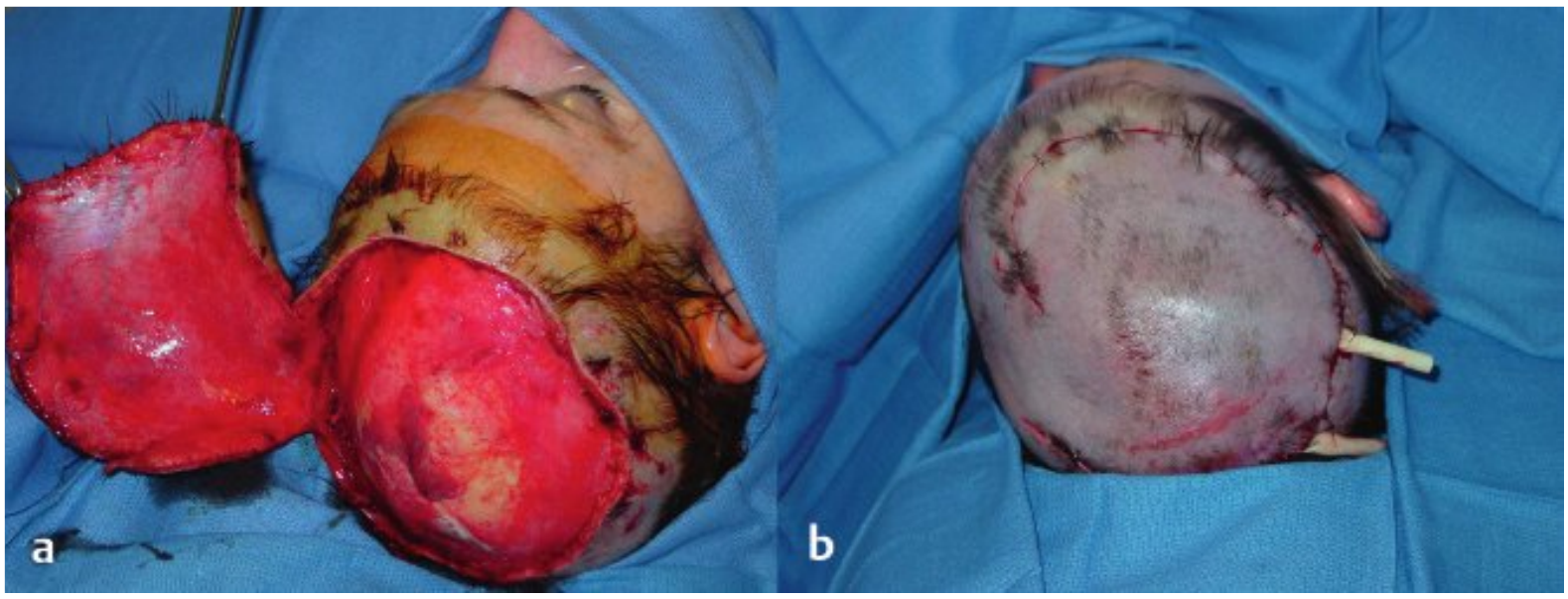


Fig. 7.5 **(a)** Large scalp avulsion perfused by lateral circulation. **(b)** Closure after irrigation and débridement over Penrose drains.

Eyelid and Eyebrow Lacerations

- Rule out ocular injuries (see Chapters 9 and 10).
- Beware of lacrimal duct injury.
- Copious irrigation to remove ocular foreign bodies.
- Layer-by-layer closure of conjunctiva, tarsus, and skin using 6–0 fast-absorbing gut with inverted knot away from the cornea. Conjunctival closure is not always needed when there is good integrity of the other layers.
  - Orbicularis—6–0 Vicryl suture.
  - Skin—6–0 fast-absorbing gut or 6–0 nylon.

For eyebrow lacerations:

- Do not shave.
- Layered closure.
  - Deep layer and muscle—Monocryl, Vicryl 5–0.
  - Skin—exactly align brow elements using Prolene 5–0, 6–0.

Eyelid Margin Lacerations (Fig. 7.6)

- Approximate lid margin.
- Evert the lid margin to prevent lid notching with vertical mattress suture.
- Antemarginal tarsus.
  - Two or three 6–0 Vicryl sutures.
  - One-half to three-quarters of the thickness of the tarsus.
  - Knot ends directed away from the cornea.
  - SkinProlene 7–0.

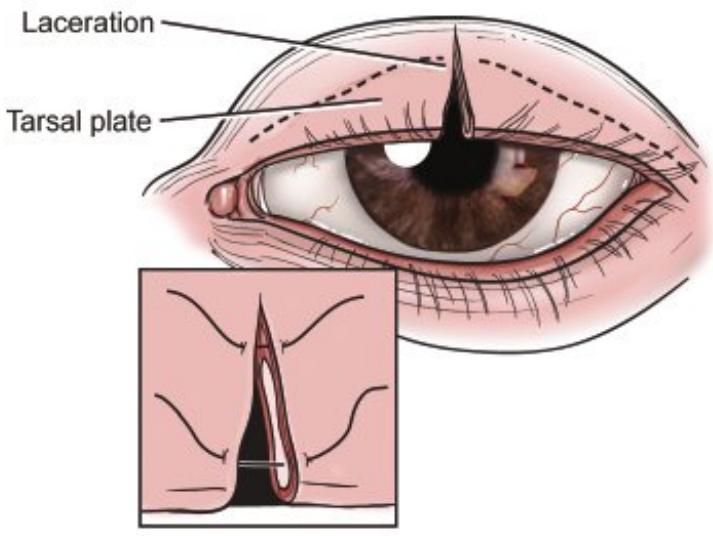


Fig. 7.6 Repair of full-thickness eyelid laceration with repair of tarsus.

Nasal Lacerations

- Achieve nasal hemostasis (see Chapter 6).
- Inspect nasal cavity to rule out nasal septal hematoma.
  - Drain hematoma under direct vision with an 11-blade scalpel.
- Layered closure of full-thickness lacerations.
  - Mucosal layer—plain gut 4–0.
  - Align skin and cartilage together with Prolene 6–0.
- Splint nose with Steri-Strips.

Facial Hematomas

- Evaluate for intracranial injuries and C-spine injury.
- Administer pain medication.
- Apply cold compresses for 48 hours, and then apply warm compresses until resolution.
- Drain hematomas that compromise the airway or visual axis.
- Explore hematomas that are expanding despite adequate pressure therapy.
- Evacuate hematomas that predispose the overlying skin to pressure necrosis.
- When a hematoma is coupled with a laceration, use the laceration as an access point for evacuation.
- Aspirate hematomas that occur in the malar region using an intraoral incision to avoid inflicting additional facial scars.

Facial Nerve Injuries

- Lacerations through the superficial musculoaponeurotic system (SMAS) and facial musculature put the nerve at risk at any point along its route (**Fig. 7.7**).
- Injury to the temporal or zygomatic branch causes an inability to elevate the brow or close the eye, respectively.
- Damage to the buccal branch causes loss of the nasolabial crease and an inability to elevate the lip.
- Marginal mandibular nerve injury causes weakness of the lower lip depressors (frowning).

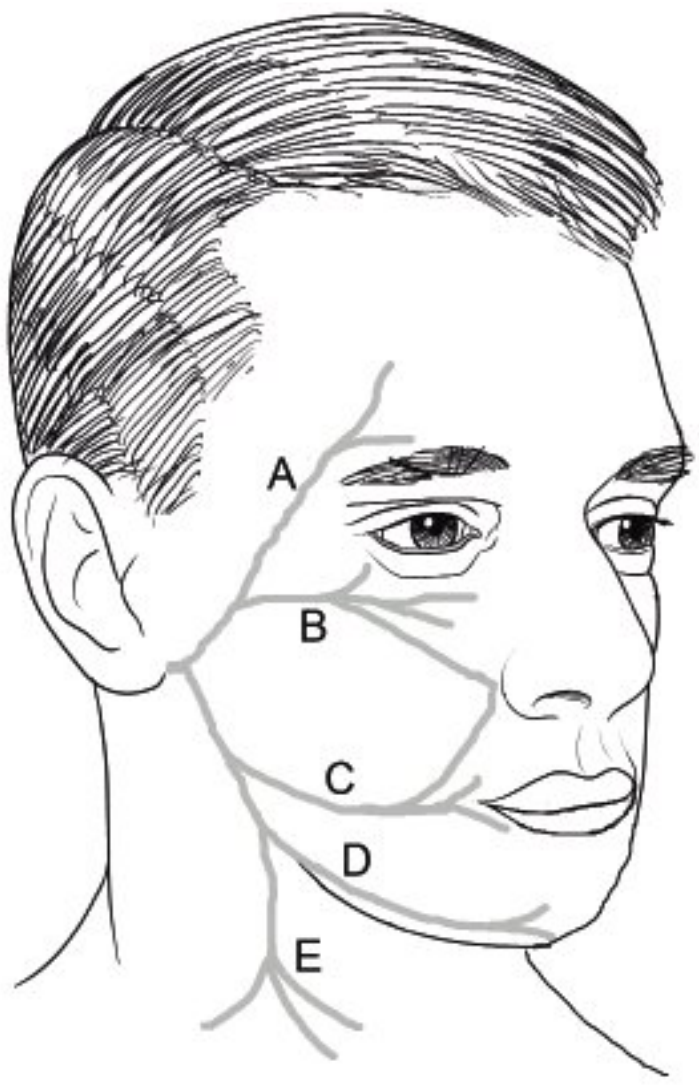


Fig. 7.7 The course of the facial nerve: **(a)** frontotemporal branch, **(b)** zygomatic branch, **(c)** buccal branch, **(d)** marginal mandibular branch, and **(e)** cervical branch.

Facial Nerve Lacerations

- Once the laceration is identified, nerve repair should be performed within 72 hours. Repair within this time frame allows for the identification of severed nerve ends using a nerve stimulator before the motor end plates are depleted of neurotransmitters.
- Attempted repair after 72 hours is extraordinarily difficult secondary to contraction of the cut segment and the inability to stimulate the distal end for exact matching to the proximal end.
- Repair the nerve in the operating room under loupe magnification or with the microscope.
- Identify and trim the proximal and distal nerve ends prior to anastomosis.
- Anastomose the fresh nerve ends using tension-free 9–0 or 10–0 nonabsorbable (nylon) sutures in an epineural fashion. Fibrin glue is also useful.
- Significant nerve loss or tension may require nerve grafting or the use of artificial nerve conduits.

For blunt injuries to the face that cause neuropraxia to the facial nerve:

- These do not require immediate operative measures.
- Monitor for signs of improvement over the course of 3 weeks.
- If there is no evidence of healing, refer the patient for electrodiagnostic testing (ENoG, EMG).
  - Identification of advanced architectural injury to the nerve at this point warrants exploration and repair.

Parotid Duct Injuries

The parotid duct traverses in a plane from the tragus to the middle of the upper lip. The duct orifice is in the buccal mucosa opposite the second maxillary molar. Extraoral or intraoral lacerations in this location place these structures at risk for injury. Injury to the buccal branch of the facial nerve should also raise suspicion for parotid duct injury. A patient with a suspected parotid duct laceration can be tested easily by placing tooth-paste in the patient's oral cavity. Excessive saliva will be expressed from the laceration.

- Evaluate a parotid duct injury by cannulating the intraoral segment with a 22-gauge Angiocath (BD Medical).
- Inject 1 mL of milk or methylene blue to assess patency.
- Repair lacerated duct over a stent in the operating room using 7–0 monofilament nylon sutures (**Fig. 7.8**).

- Keep the stent in place for 5 days to allow patency and prevent fistula formation.
- Give the patient prophylactic antibiotics during this period—clindamycin.
- Reconstruct ostia for the proximal segment or duct ligation if there is severe irreparable damage of the parotid duct.
- Oversew parotid gland injuries without duct injuries with absorbable suture—Monocryl 3.0, 4.0, or Vicryl.

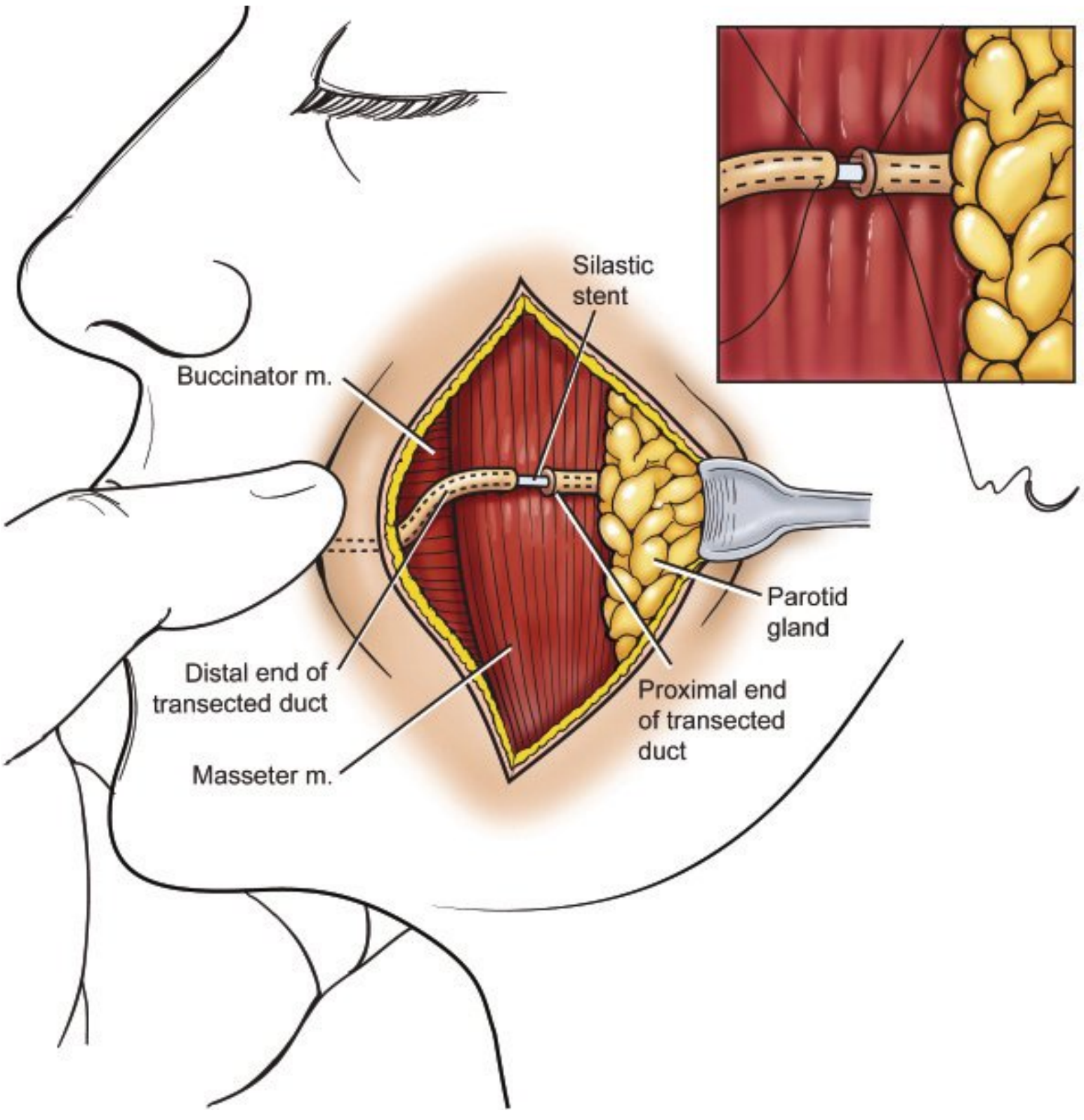


Fig. 7.8 Repair of parotid duct injury over Silastic (Dow Corning) stent.

8 Orbit and Zygoma Fractures

The Orbit

Anatomy

The orbit is composed of seven bones:

- Zygoma.
- Greater and lesser wing of the sphenoid.
- Ethmoid.
- Frontal.
- Palatine.
- Maxilla.
- Lacrimal.

These seven bones create a bony pyramid with the optic canal at the apex. The orbit is composed of the following structures:

- Floor: Roof of the maxillary sinus.
- Medial wall: Lamina papyracea of the ethmoid bone and the lacrimal bone.
- Lateral wall: Zygoma and greater wing of the sphenoid bone.
- Roof: Frontal bone, floor of the frontal sinus.

The medial wall is the weakest structure, followed by the floor. The roof and the lateral wall are generally the strongest. The optic nerve exits the optic canal situated superomedially and approximately 40 to 45 mm from the inferior orbital rim. The superior orbital fissure separates the greater and lesser wings of the sphenoid.

- Structures that pass through the superior orbital fissure.
  - Oculomotor nerve (CN III).
  - Trochlear nerve (CN IV).
  - Abducens nerve (CN VI).
  - Ophthalmic division of the trigeminal nerve (CN V_1).
- Structures that pass through the inferior orbital fissure.
  - Maxillary division of trigeminal nerve (CN V_2).
  - Branches of sphenopalatine ganglion.
  - Branches of the inferior ophthalmic vein.

Physical Examination

Orbital fractures are usually associated with blunt trauma. Nearly 30% of orbital fractures will have injuries to the globe. It is important to perform a detailed ophthalmic exam that includes visual acuity, pupillary reaction, retinal exam, and red color saturation, as described in Chapter 6. Any deviation from normal warrants an emergent ophthalmic consultation.

- Pathologic physical findings.
  - Orbital ecchymosis.
  - Periorbital edema.
  - Subconjunctival hemorrhage.
  - Epistaxis.
  - Orbital rim/zygoma bony step-offs.
  - Diplopia.
  - Extraocular muscle entrapment.
    - Examine the active range of motion of the extraocular muscles to rule out mechanical entrapment.
    - In unconscious patients, perform the **forced duction test:** using Adson forceps, grasp the inferior capsulopalpebral fascia of the inferior rectus muscle and gently rotate the globe, while feeling for any restrictions.
  - Intraorbital edema.
  - Optic nerve neuropraxia.
  - Pupillary shape—oblong pupil is suggestive of ocular perforation.
  - Pupillary response—afferent pupillary defect (see Chapter 6)
  - Supraorbital, infraorbital, alveolar nerve paresthesias.
  - Crepitus/subcutaneous emphysema—disruption of maxillary or ethmoid sinus mucosa.
  - Enophthalmos—noticeable with > 2-mm shift; however, rarely evident immediately after injury because of edema.
  - Proptosis/exophthalmos.
  - Hyphema—fluid in the anterior chamber of the eye.
  - **Superior orbital fissure (SOF) syndrome**, resulting from fractures of the SOF.
    - Fixed dilated pupil (CN III).
    - Upper lid ptosis (CN III).
    - Loss of corneal reflex (CN V1).
    - Ophthalmoplegia (CN IV, CN VI).
  - **Orbital apex syndrome**—SOF syndrome plus impairment of optic nerve as it exits the optic canal; blindness.
  - Nausea, vomiting, bradycardia—oculocardiac response to extraocular muscle entrapment (**Fig. 8.1**).

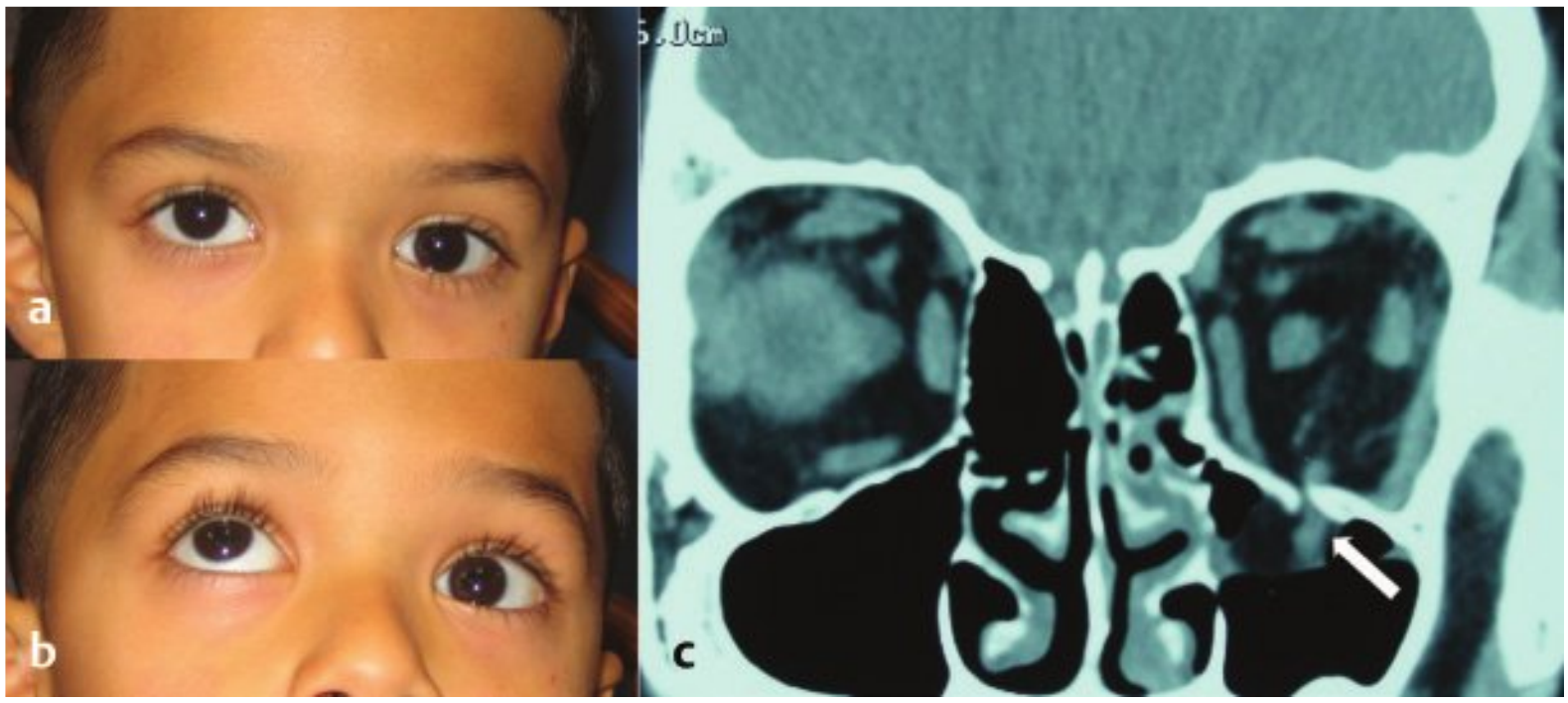


Fig. 8.1 **(a)** Hypoglobus at rest. **(b)** Diplopia with upper gaze associated with entrapment of the inferior rectus muscle limiting ocular mobility. **(c)** Coronal CT scan; arrow indicates inferior rectus within the trapdoor of the fracture.

Acute Compressive Optic Neuropathy (ACON)

Orbital injuries sustained from high-velocity trauma can result in a rapid increase in orbital pressure due to the accumulation of edema or blood (retrobulbar hematoma) in the orbital pyramid. This can cause vision-threatening compression of the optic nerve.

- Signs and symptoms of acute compressive optic neuropathy.
  - Proptosis.
  - Severe eye pain.
  - Diplopia.
  - Vision loss.
  - Reduced ocular motility.
  - Chemosis.

Treatment of Acute Compressive Optic Neuropathy

Patients suspected of having acute compressive optic neuropathy should undergo **emergent decompression**.

- Decompression is performed with a lateral canthotomy (**Fig. 6.6**) or by fracturing the medial orbital floor.
- Start methylprednisolone (load 30 mg/kg followed in 2 hours by 15 mg/kg every 6 hours), acetazolamide (250 mg by mouth twice a day), and mannitol (1 g/kg IV, repeat every 6 hours as needed).

To perform a lateral canthotomy, retract the upper and lower lid superiorly and inferiorly, respectively, with your index and third finger. Incise the lateral canthal skin 4 to 5 mm, then palpate the lateral canthal tendon with fine scissors and release overlying soft tissue lateral to the conjunctiva all the way down to the lateral bony orbit. Disinsertion of the canthal tendon will result in a more freely mobile eye, along with complete mobility of the lower lid.

To fracture the medial orbital floor, first manually retract the lower lid. With a pair of fine hemostats, push through the floor medially to allow drainage into the maxillary sinus.

Traumatic Optic Neuropathy (TON)

A subset of patients with ocular injuries will present with vision loss secondary to optic nerve trauma (compression or edema) without increased extraocular orbital pressure. These patients are suspected of having traumatic optic neuropathy. The etiology may be direct secondary to bony fragments within the optic canal. Indirect injury is secondary to ischemia and edema of the optic nerve. Emergent high-resolution CT of the orbit is performed to identify specific anatomical optic nerve pathology. Patients with decreased light perception should be started on a megadose of steroids for 48 hours (methylprednisolone load 30 mg/kg followed in 2 hours by 15 mg/kg every 6 hours). Patients who exhibit worsening light perception or who present with no light perception should be considered for operative optic nerve decompression.

Types of Orbital Fractures

Orbital fractures can occur anywhere along the medial or lateral walls, floor, roof, and apex. Most commonly, they will be localized to the medial wall and floor, the weakest structures. Medial wall fractures are part of a complex of fractures associated with the nasal and ethmoid bones; they are discussed in Chapter 10.

Orbital Floor

Orbital floor fractures (blow-out) most commonly occur at the medial wall and floor of the orbit along the infraorbital groove (paresthesia). A fracture defect may entrap periorbital fat and possibly the inferior rectus muscle (**Fig. 8.2**). The pathomechanics of the injury include two theories:

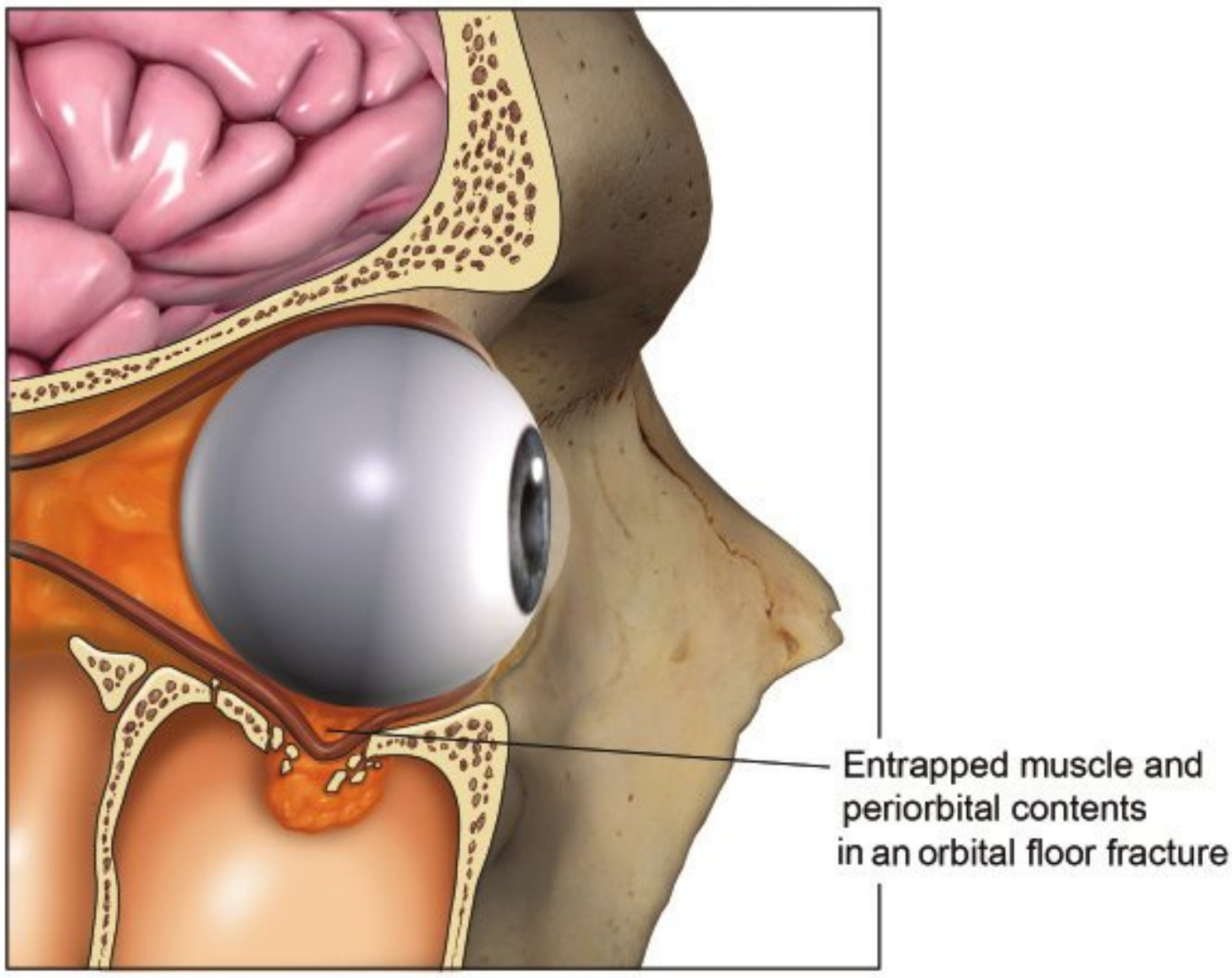


Fig. 8.2 Schematic of an orbital floor fracture with entrapment of inferior rectus.

1. The hydraulic theory—direct trauma to the globe leads to increased intraorbital pressure resulting in a decompressing fracture at the weakest point.
2. The bone conduction theory—an indirect transmission of forces around the orbital rim leading to fracture of the floor.

Orbital Roof

Fractures of the orbital roof are rare due to protection by the supraorbital rim and strong frontal bone. These fractures are more common in children secondary to the differences in the architecture of the cranium. When fractures in the roof occur, displacement can be either into the anterior cranial fossa or, more commonly, into the orbit, causing a "blow-in" fracture. Evaluation of these patients by CT should rule out both intracranial and intraocular involvement. Blow-in fractures are characterized by a decreased orbital volume (i.e., exophthalmos) and commonly warrant urgent surgical intervention to decrease the increased intraocular pressure (**Fig. 8.3**). Additionally, injury to the supraorbital artery can result in a retrobulbar hematoma.

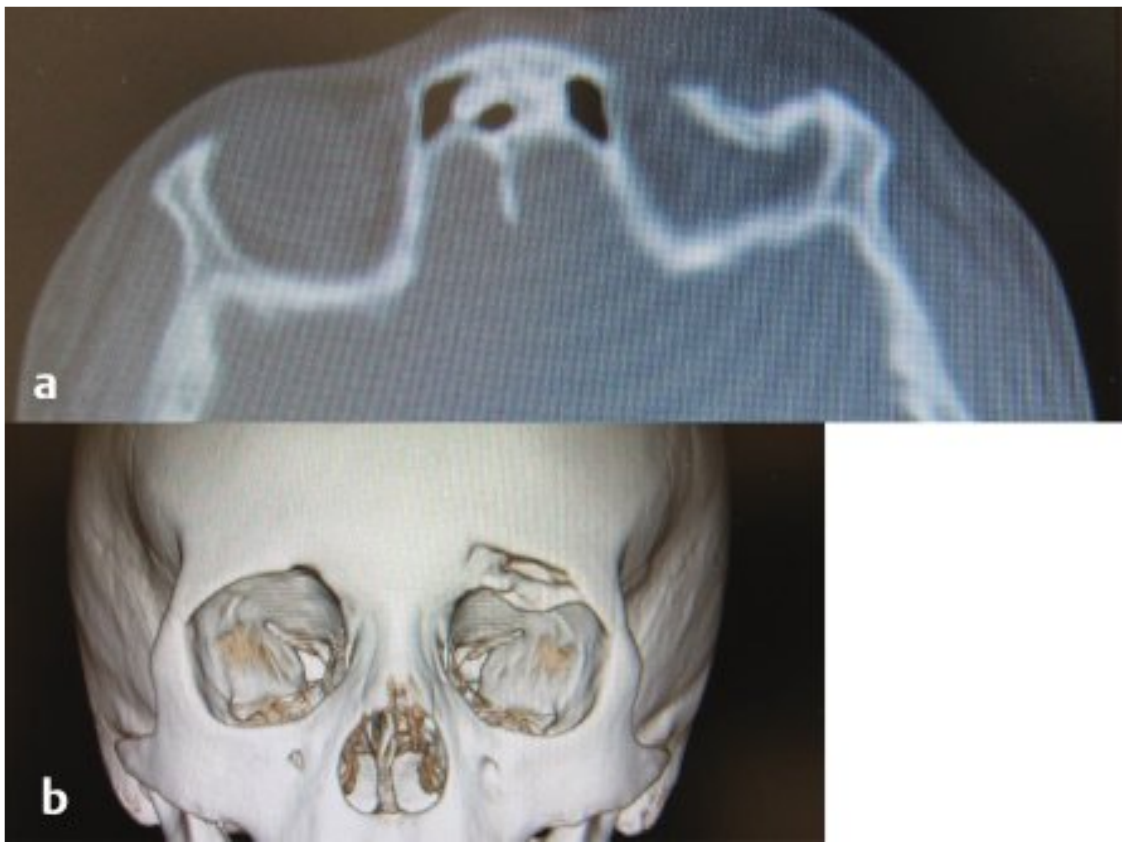


Fig. 8.3 **(a)** Axial and **(b)** 3D reconstruction CT scans of a patient with an orbital roof fracture.

Radiographic Evaluation

CT scans should be obtained with 1.5-mm thin cuts through the orbit with sagittal and coronal reconstructions. Evaluate radiographs for the following:

- Displaced fracture fragments.
  - Trapdoor fracture.
  - Bony fragment impingement on the optic canal.
- Area of floor defect.
- Soft tissue entrapment.
- Enophthalmos.
- Lens dislocation.
- Retrobulbar hematoma.
- Other associated fractures (medial wall fracture).

Management

Patients without evidence of entrapment, TON, retrobulbar hematoma, ACON, or any signs of globe injury can be discharged home. Nondisplaced fractures may be associated with diplopia secondary to edema and blunt trauma to the globe. Diplopia alone is not an indication for surgery. Patients should be followed closely for 2 weeks to ensure resolution of symptoms. Pain medication should be prescribed as indicated; antibiotics are not indicated. Patients should be instructed to use artificial tears to keep the eye lubricated, and to minimize nose blowing to avoid orbital emphysema and displacement of a fracture.

Patients who do require delayed surgical intervention should be seen by an ophthalmologist prior to surgery to rule out open globe injuries. Fractures are optimally operated on after 2 to 4 weeks once edema resolves.

Emergent surgery is indicated for those patients who have clear bony displacement into the optic canal or globe as confirmed by CT, entrapment, or signs and symptoms suggestive of oculocardiac response.

Surgical Indications—Orbital Floor Fractures

Urgent surgical indications are

- Orbital entrapment.
- Oculocardiac response—bradycardia, nausea, syncope.

Indications for delayed surgical intervention are

- Early enophthalmos > 2 mm within 6 weeks.
- Enophthalmos with symptomatic diplopia lasting longer than 2 weeks (primary field).
- Displaced fracture with floor defect > 1 cm^2.
- Hypoglobus—low-lying globe.

Zygoma/Zygomaticomaxillary Complex Fractures

The zygoma articulates with the frontal, sphenoid, maxillary, and temporal bones comprising the characteristic tetrapod (**Fig. 8.4**). It is composed of two faces, the malar face, which comprises the lateral orbit, and the body, which gives projection to the cheek. The zygomatic process of the temporal bone articulates with the body of the zygoma to create the zygomatic arch. The zygoma has multiple muscular attachments; most important is the masseter, which produces a major inferior deforming force on the body and arch when fractured. Fractures and disarticulations of the zygoma usually result in an inferior displacement, leading to increased intraorbital volume producing enophthalmos. These fractures are most commonly referred to as tripod, tetrapod, or a zygomaticomaxillary complex (ZMC) fracture, so called because it involves separation of all of the major attachments of the zygoma to the rest of the face (**Fig. 8.5**). Occasionally, there can be an isolated fracture of the zygomatic arch or lateral wall without concomitant ZMC fracture (**Fig. 8.6**).

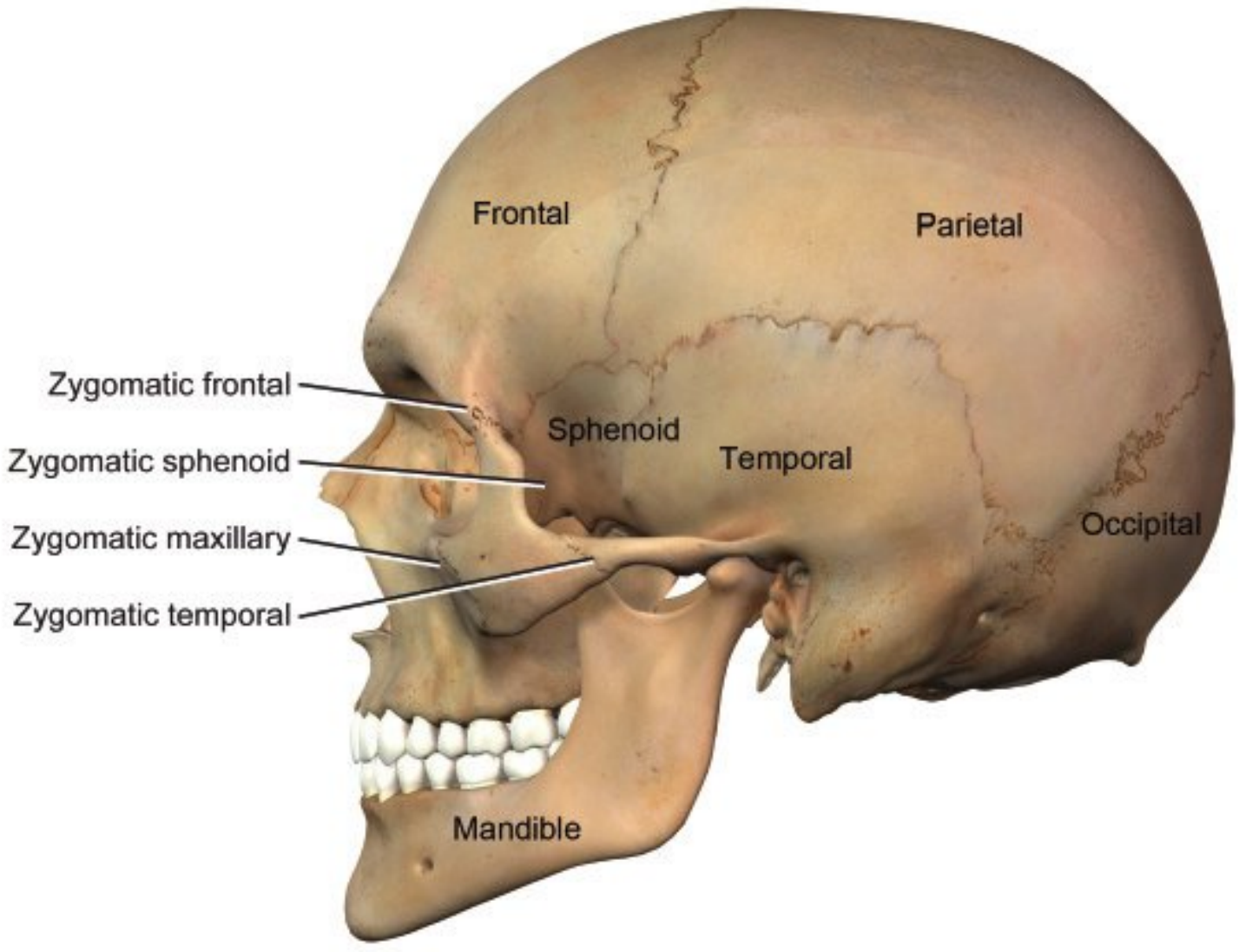


Fig. 8.4 Zygomatic tetrapod bone articulations.

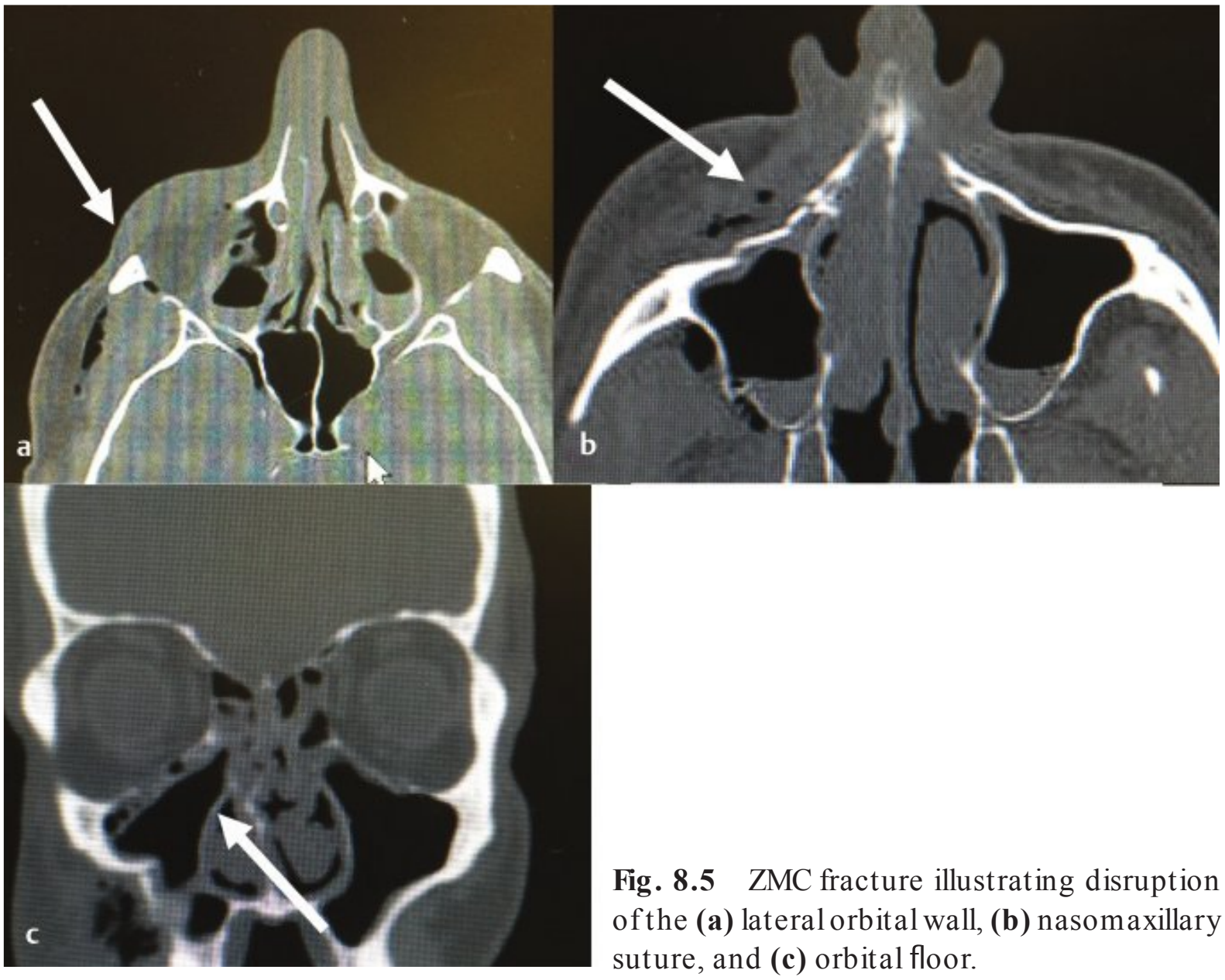


Fig. 8.5 ZMC fracture illustrating disruption of the **(a)** lateral orbital wall, **(b)** nasomaxillary suture, and **(c)** orbital floor.

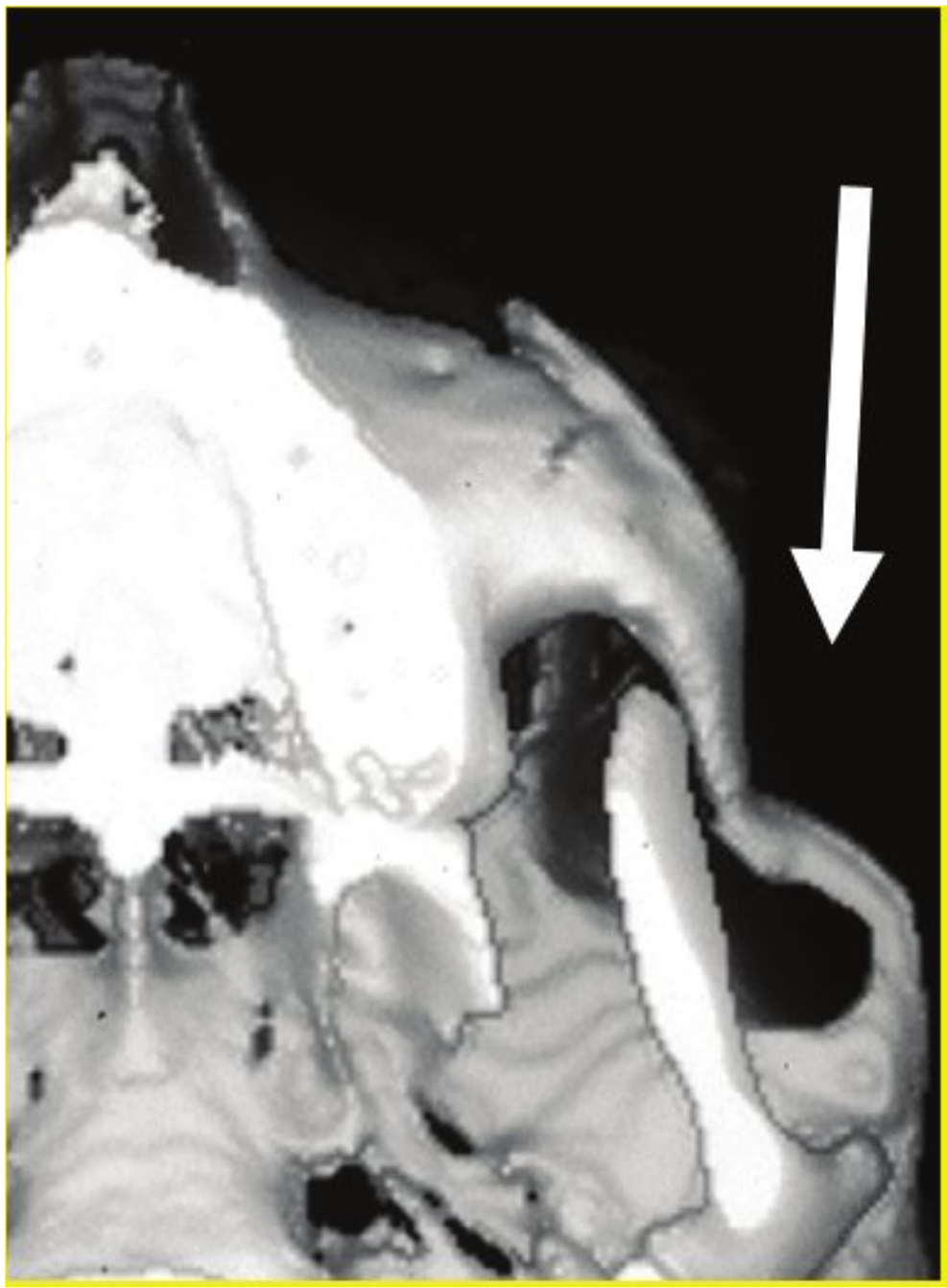


Fig. 8.6 Displaced zygomatic arch fracture with impingement of the coronoid process of the mandible.

Symptoms and Physical Findings

- Enophthalmos.
- Flattening of the midface/malar asymmetry.
- Diplopia.
- Trismus.
- Impingement on the coronoid process.
- Periorbital and subconjunctival hematoma (**Fig. 8.7**).
  - "Flame sign."
- Epistaxis.
- Inferior displacement of globe.
- Inferior displacement of lateral canthus.
- Infraorbital nerve injury—paresthesia of the cheek, upper lip, anterior incisors, and ala of the nose; V2 distribution.
- Mandibular occlusion and range-of-motion disturbances—trismus.
- Intraoral hematoma.

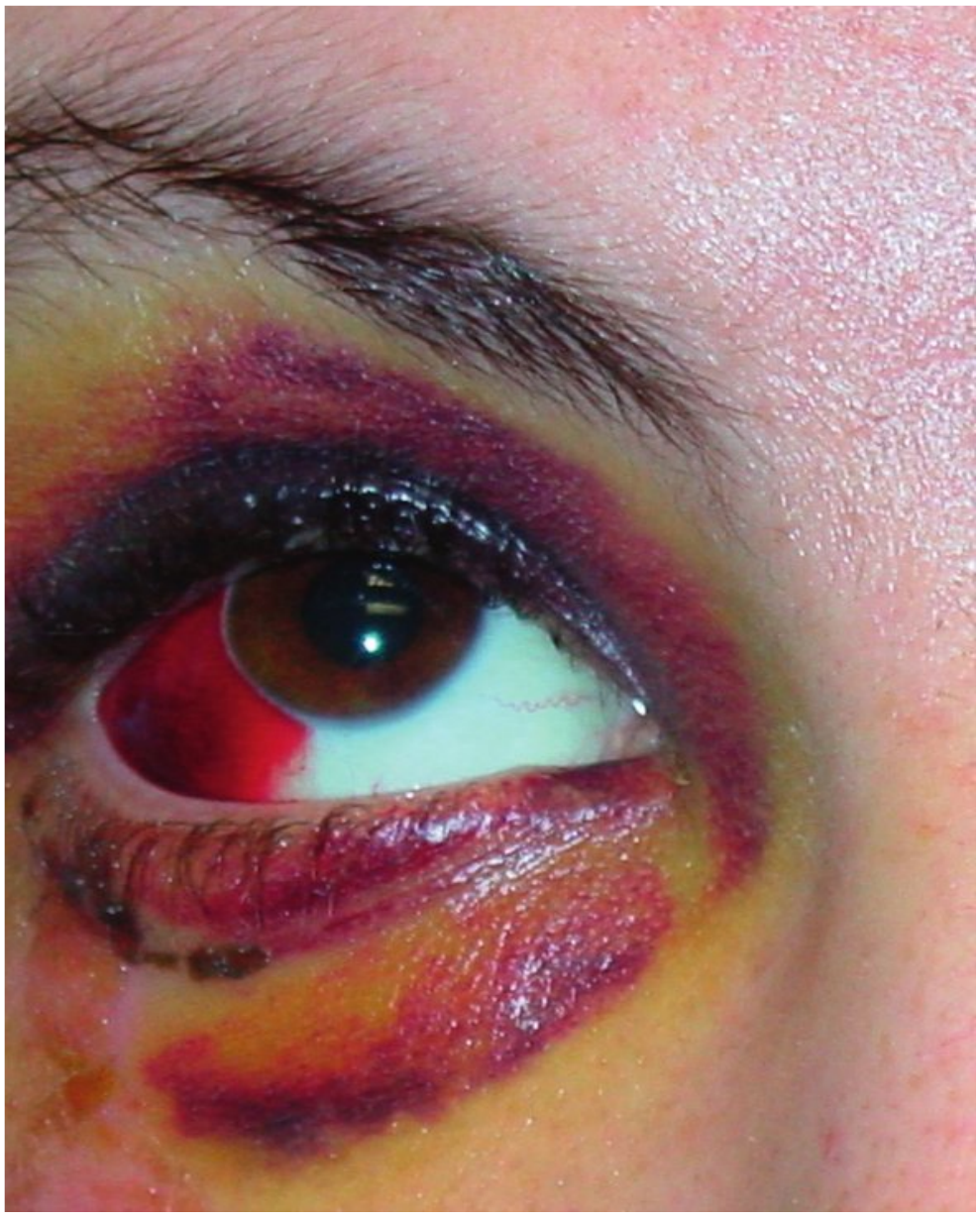


Fig. 8.7 Flame sign—suggestive of an orbitozygomatic fracture.

Radiographic Evaluation

- CT scan.
  - Axial.
  - 1.5 mm cuts.
  - Coronal.
  - Orbital evaluation.
  - Reconstructions.
    - 3D reconstructions.
- Plain radiographs (less commonly used).
  - Caldwell view.
  - Submental vertex.
  - Waters view.
    - Most helpful plain film.
    - 30 degrees of occipitomental projection, extension.
    - Visualization of zygomatic buttresses.

Management

Patients with **nondisplaced ZMC fractures** can be discharged home, observed, and treated conservatively. Antibiotics are not indicated. Keep patients on a soft diet (nonchew) for 6 weeks with protection of the malar eminence. Follow-up should be in 2 weeks to assess for displacement and enophthalmos. This can occur over the long term with the masseter pull on the fractured zygoma.

Patients with **displaced ZMC fractures** should be prepared for surgery to realign the lateral orbital wall and floor and to correct contour irregularities of the malar eminence. An ophthalmologist's evaluation is warranted with orbital involvement. Orbital floor and rim fractures are commonly associated with ZMC fractures. Impacted ZMC fractures may hide orbital defects on radiographs. If there is an indication for urgent intervention due to orbital involvement (i.e., entrapment), then the patient should be admitted and prepared for reduction of the ZMC and orbital floor reconstruction.

Patients with **nondisplaced isolated zygomatic arch fractures** require no surgical intervention. They can be discharged home with malar eminence protection. Patients with **displaced isolated zygomatic fractures** do not need admission and can be discharged home to have their fracture repaired electively. Repair can be done within 24 hours or delayed until 2 weeks after edema resolves.

Those patients with trismus secondary to impingement on the coronoid or masseter and cosmetic temporal deformities also warrant consideration for operative reduction.

9 Nasal and Naso-Orbital-Ethmoid (NOE) Fractures

Injuries to the midface involve the complex union of the nose, the orbit, and the base of the skull. Fractures of these areas can occur in isolation or in conjunction at the union of these structures (NOE).

Anatomy

Nasal anatomy (**Fig. 9.1**):

- Nasal bone.
- Frontal processes of the maxilla.
- Nasal cartilage.
- Nasal septum.
- Quadrilateral cartilage.
- Perpendicular plate of the ethmoid.
- Vomer.

Blood supply:

- Ophthalmic artery is the first branch of the internal carotid.
- Anterior and posterior ethmoidal branches of internal carotid.
- Facial artery branches.
- Superior labial branch.
- Internal maxillary branches of external carotid (sphenopalatine, greater palatine, and infraorbital).

External innervation:

- Nasociliary nerve V_1.
- Supratrochlear nerve V_1.
- Infraorbital nerve V_2.

Internal innervation:

- Anterior ethmoid nerve V_1.
- Greater palatine nerve—lateral wall.
- Nasopalatine nerve V_2.

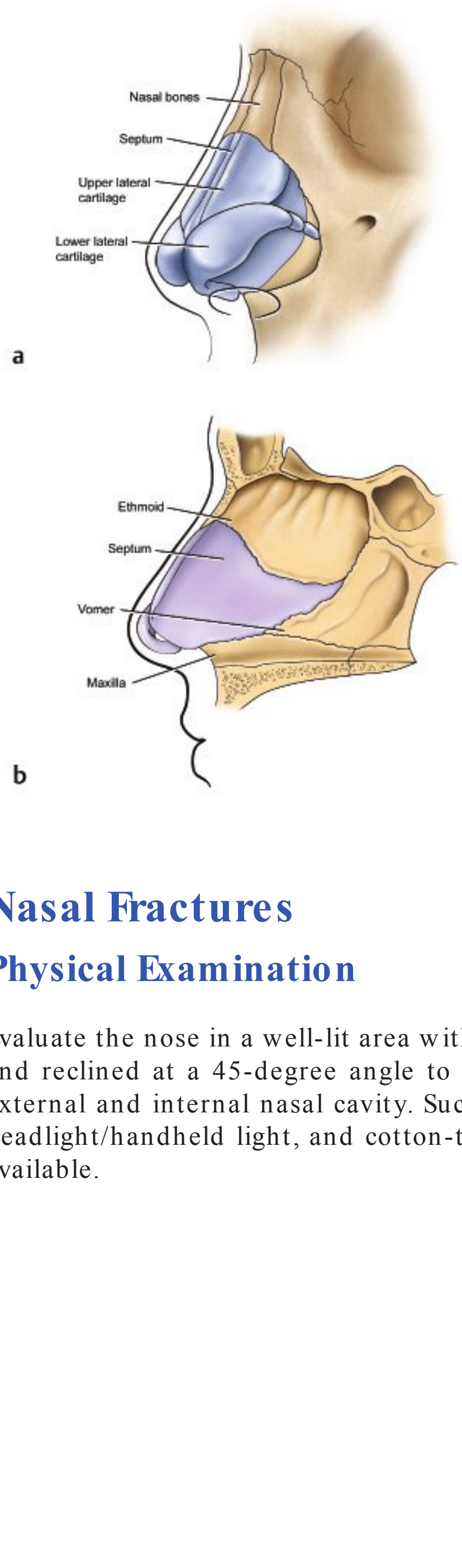


Fig. 9.1 **(a)** Bony and cartilaginous vault anatomy. **(b)** Nasal septal anatomy.

Nasal Fractures

Physical Examination

Evaluate the nose in a well-lit area with the patient comfortably seated and reclined at a 45-degree angle to facilitate inspection of both the external and internal nasal cavity. Suction, irrigation, nasal speculum, headlight/handheld light, and cotton-tip applicators should be readily available.

Common physical findings include

- Tenderness.
- Crepitus.
- Nasal deviation.
- Mobility.
- Epistaxis.
- Airway obstruction.
- Septal deviation.
- Septal hematoma.
- Saddle deformity.
- Mucosal laceration.

Septal Hematomas

Septal hematomas are caused by bleeding between the septum and mucosa. Diagnosis is made by direct visualization of a hematoma beneath the mucosa (**Fig. 9.2**). Septal hematomas require immediate drainage in the acute setting. If left undrained, the accumulation of blood in the mucoperichondrium can lead to septal ischemia with potential septal necrosis. Complications include perforation, loss of dorsal support, and saddle deformity. Nasal septal hematomas should be drained appropriately, with the proper pressure dressing applied (see Chapter 6, **Fig. 6.4a**). Packing should be removed on day 3 to prevent sinusitis or toxic shock. Place the patient on clindamycin or Augmentin while packing is in place.

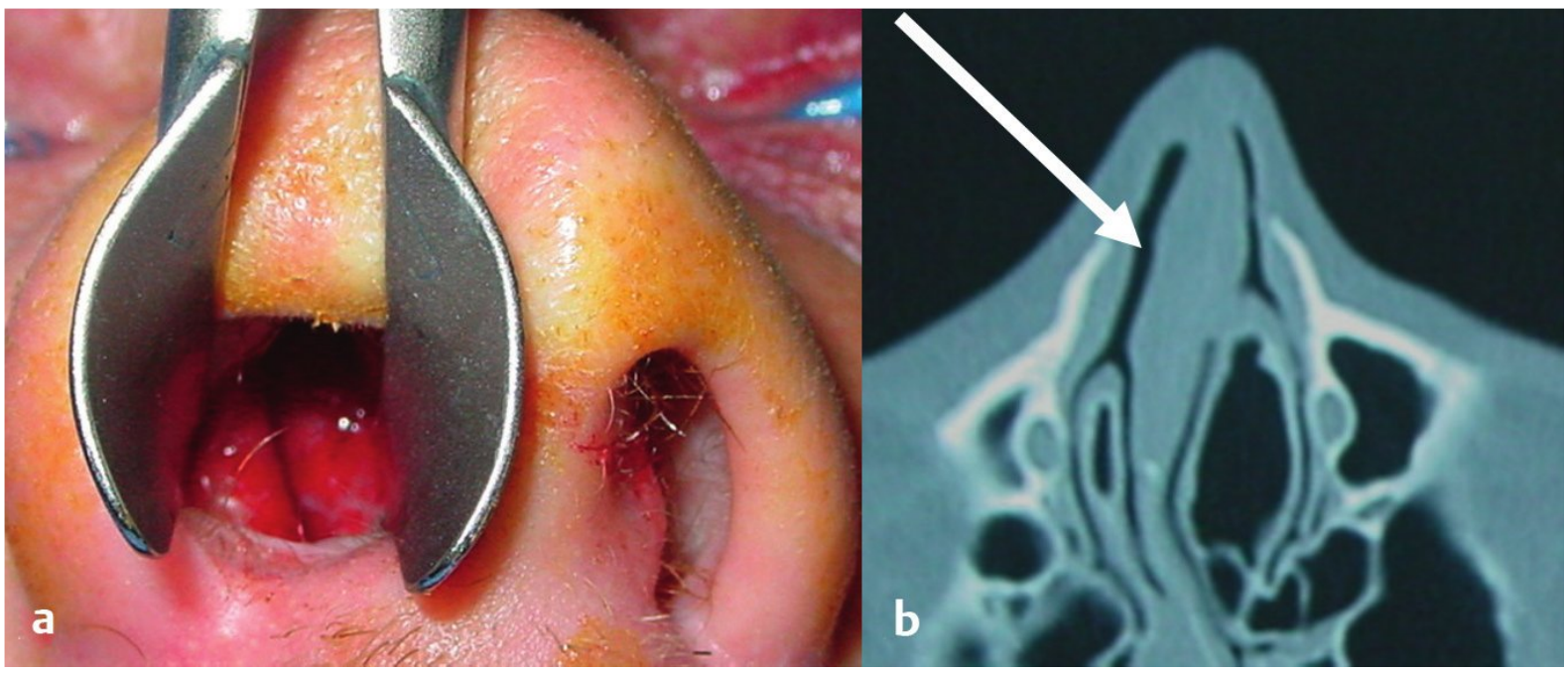


Fig. 9.2 **(a)** Nasal septal hematoma that requires immediate drainage. **(b)** CT scan confirms diagnosis.

Radiographic Evaluation

Plain films and CT scans (**Fig. 9.3**) are not absolutely necessary. They become more relevant if other injuries are suspected (e.g., naso-orbital-ethmoid fractures [NOE], orbital floor fracture, intracranial bleeding). If one has a low clinical suspicion of any other injury, nasal fractures in general do not require any radiography. In selected clinical scenarios, a nasal series (anterior and lateral view) can be ordered to aid in diagnosis and for documentation.

Stranc-Robertson Nasal Fracture Classification (Fig. 9.4)

- Type I.
  - Anterior portion of the nasal pyramid.
  - Septum.
- Type II.
  - Comminution of the nasal pyramid.
  - Dislocation of the septum.
- Type III.
  - Frontal processes of the maxilla.
  - NOE fractures.

Nasal Fracture Treatment

The timing of the repair is usually bimodal and correlated with the amount of edema. Any repair should be performed within the first 2 hours, before the onset of significant edema. A patient rarely presents within this time, and typically, repairs are performed after 1 to 2 weeks, when the edema subsides.

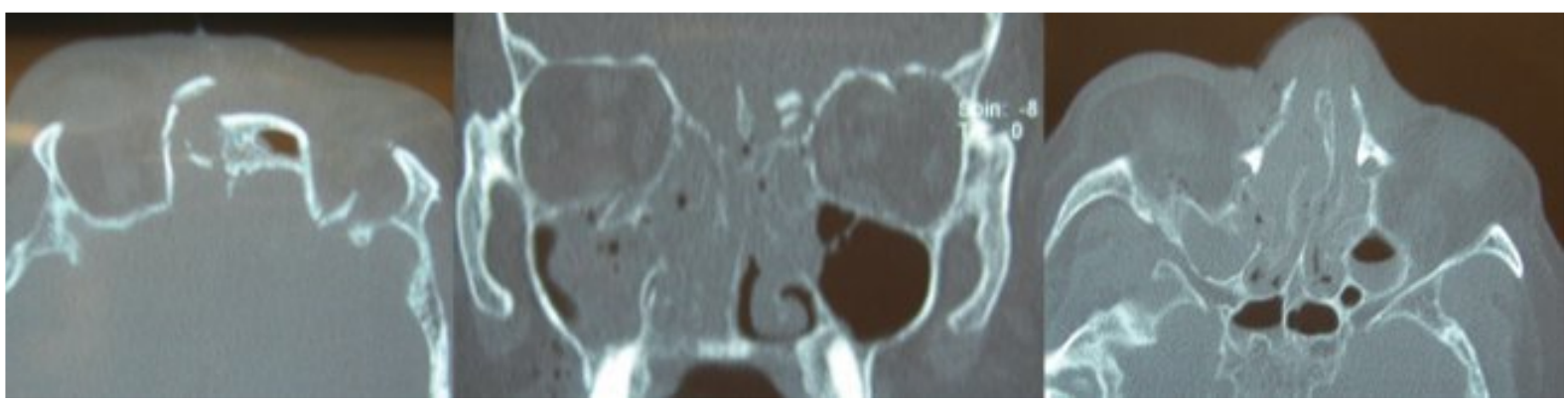


Fig. 9.3 CT scan of nasal bone and septal fracture with orbital component.

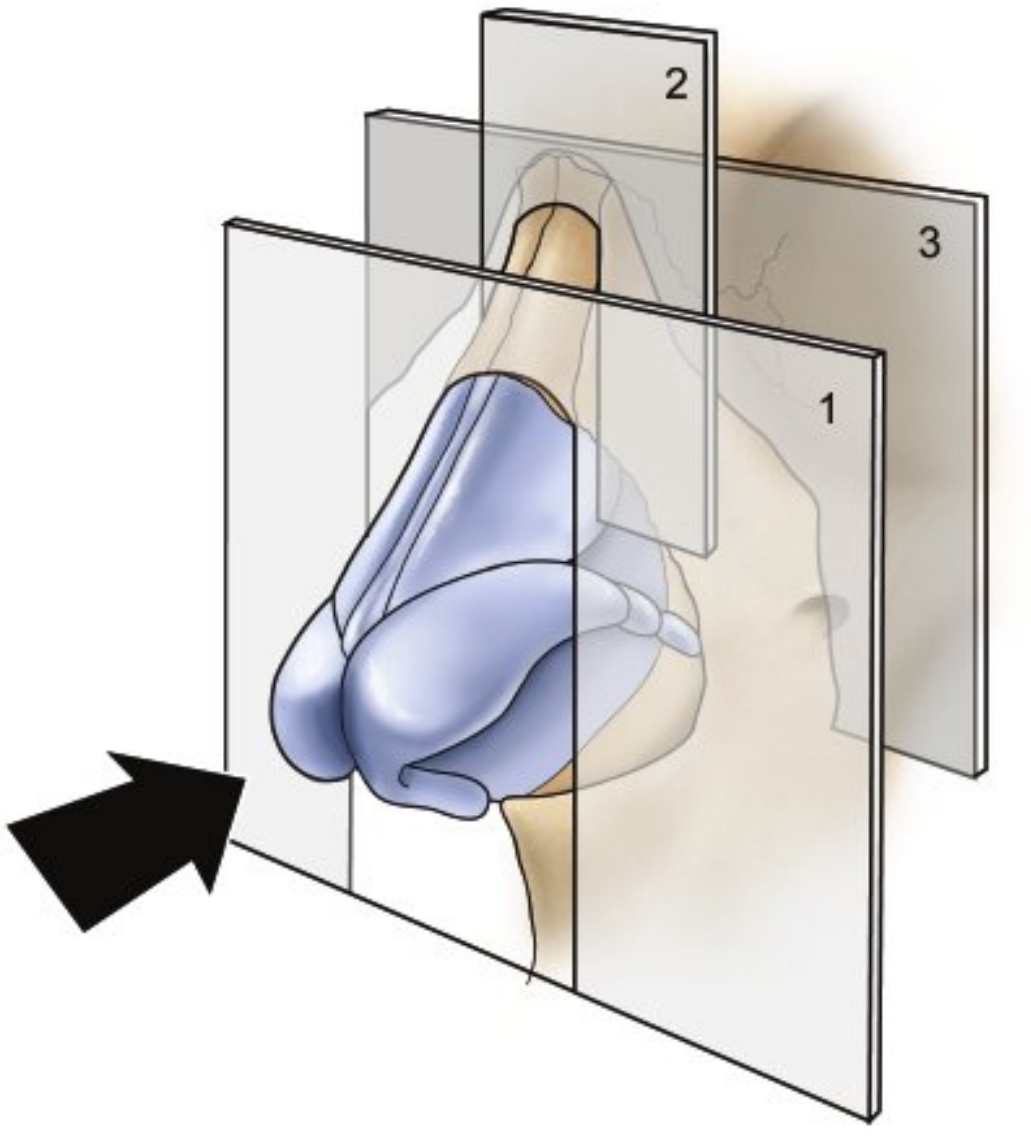


Fig. 9.4 Stranc-Robertson nasal fracture classification.

Closed Reduction

Adequate anesthesia can be achieved locally if a reduction is attempted in the emergency care setting. Epinephrine 1:100,000 or 4% cocaine-soaked Cottonoid/pledgets or Afrin (Schering-Plough Corp.) spray on pledgets can be applied intranasally for 5 minutes.

A regional block—1% lidocaine with 0.25% Marcaine mixed 1:1—provides long-lasting pain relief with fast onset. Epinephrine can also be added at 1:100,000.

- Regional block (see Chapter 7, **Fig. 7.1**).
  - Nasociliary nerve.
  - Infratrochlear nerve.
  - Infraorbital nerves.
  - Tip—columella.

Asch or Walsham forceps can be used to realign and reduce the fracture (**Fig. 9.5**). The blunt end of a scalpel handle can also be used. Reduction should be aimed at repositioning the nose to the midline. Reshaping the nasal pyramid often involves "outfracturing" of the nasal sidewalls. Assess reduction by visualization and palpation.

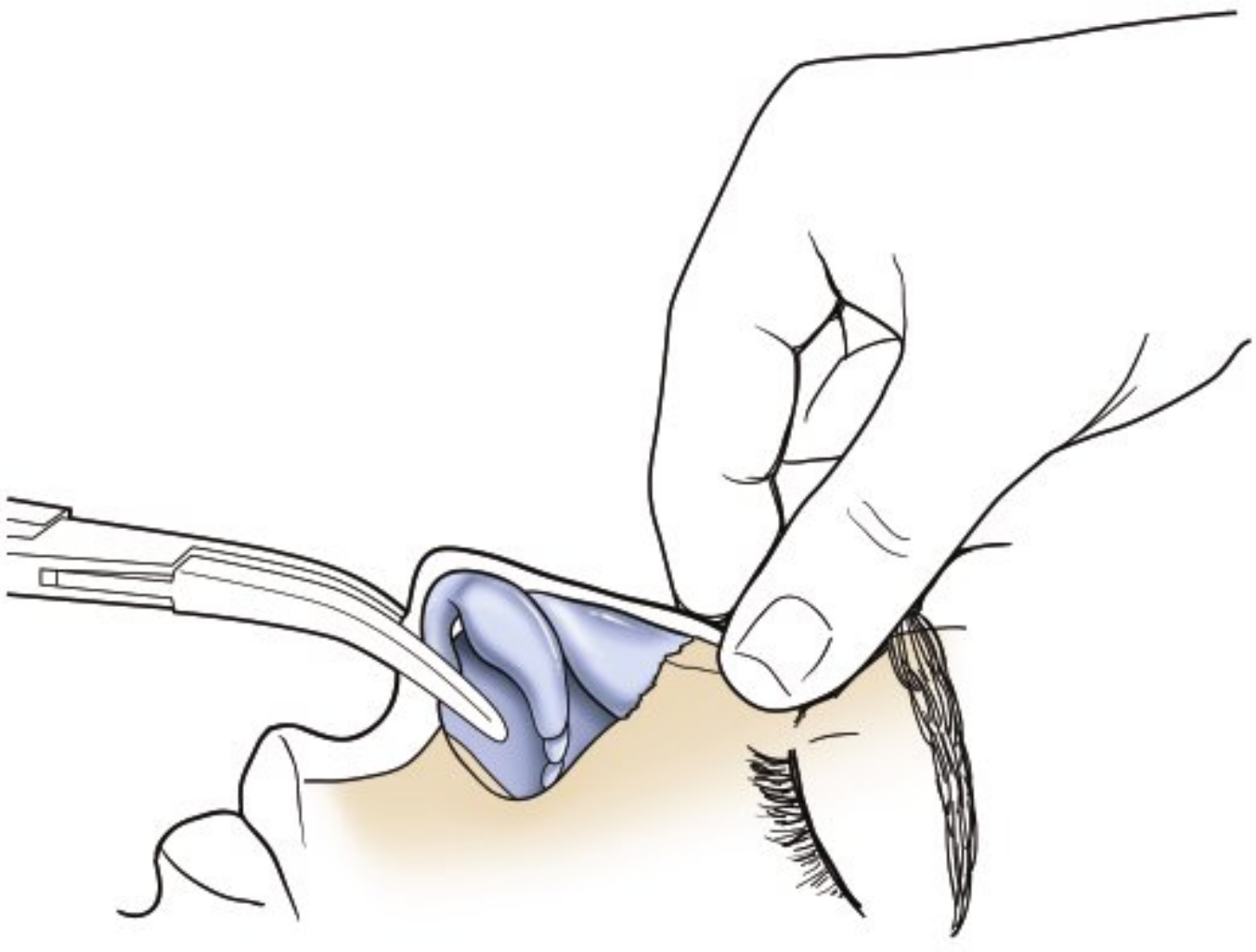


Fig. 9.5 Closed reduction of nasal fracture using Walsham forceps.

Postreduction Care

- Packing—place packing in distinct layers, if necessary, to achieve hemostasis (see Chapter 6, **Fig. 6.4**).
  - Merocel (Merocel Surgical Products), Xeroform gauze, Vaseline (Unilever PLC)/bacitracin-impregnated gauze, Cottonoid soaked with epinephrine 1:100,000.
  - Remove packing within 3 days to avoid sinusitis or toxic shock (see Chapter 6, **Fig. 6.4a**).
  - Prescribe antibiotics for patients with intranasal packing.
    - Augmentin 875 mg by mouth twice a day × 3 days or clindamycin 450 mg by mouth four times a day × 3 days.
    - A silicone internal nasal splint can also be used if bleeding is controlled (**Fig. 9.6**).
      - Internal splints are sutures through the septum (2–0 Prolene) to prevent migration and are removed in 2 weeks.

- Splint—apply an external nasal splint to the dorsum (**Fig. 9.6**); keep splints in place for 7 to 10 days.
  - Fashion a splint out of a small piece of plaster over Steri-Strips if prefabricated thermoplastic splints are not available.
- Have the patient avoid nose blowing for several weeks.
- Have the patient avoid contact to nose.
- Follow up within 1 week.

Patients with nasal fractures that are significantly displaced or with significant edema hindering reduction in the acute setting can be discharged home with contact precautions. Antibiotics are not needed, and patients should follow up in 2 weeks for attempted closed or open operative reduction.

Naso-orbital-ethmoid Fractures

NOE fractures result from force directly over the nasal pyramid. The nose is depressed between the orbits, resulting in fractures of the nasal bone and medial orbital wall. Fractures are commonly bilateral, but one-third of the time they are unilateral. The high-velocity impact will often be accompanied by orbital blow-out fractures or can extend into the cranial base (**Fig. 9.7**).

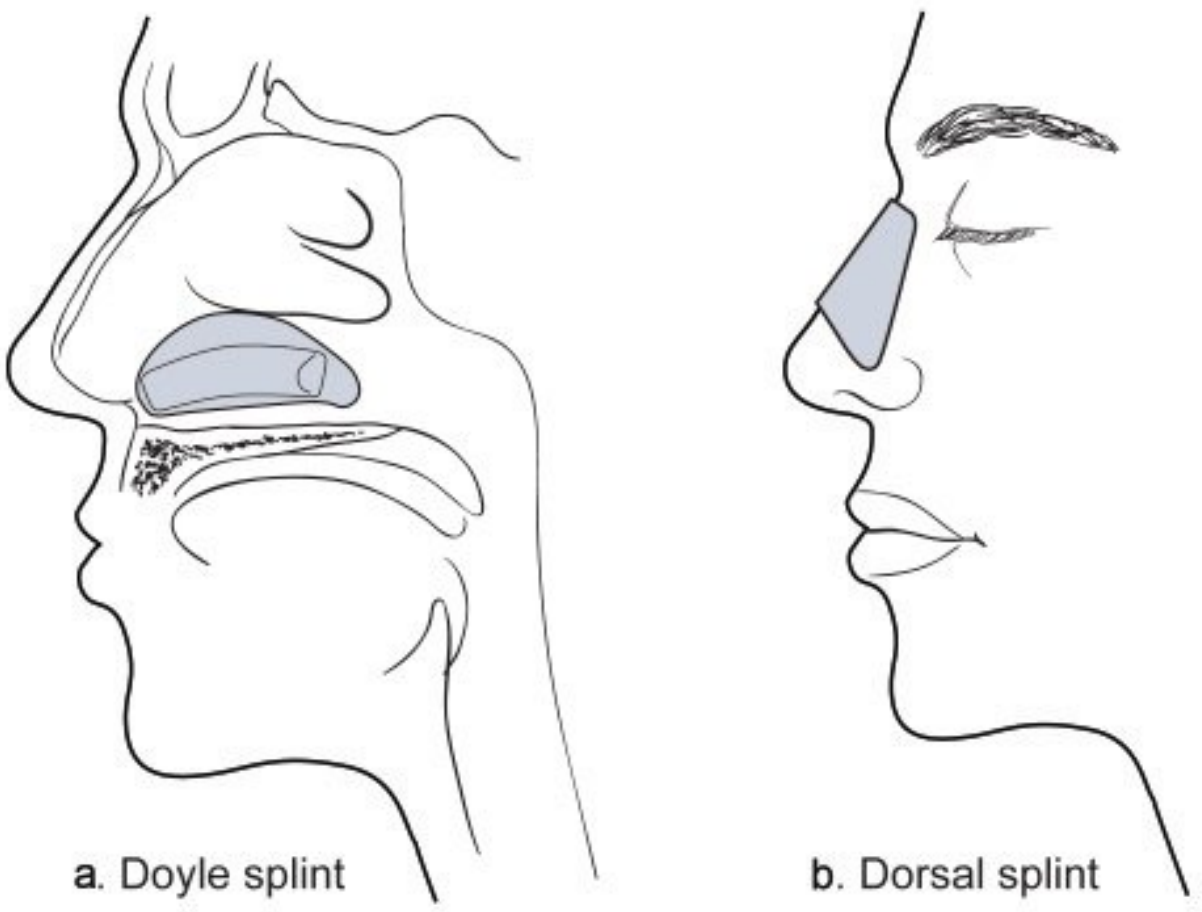


Fig. 9.6 Postreduction intranasal and dorsal splints.

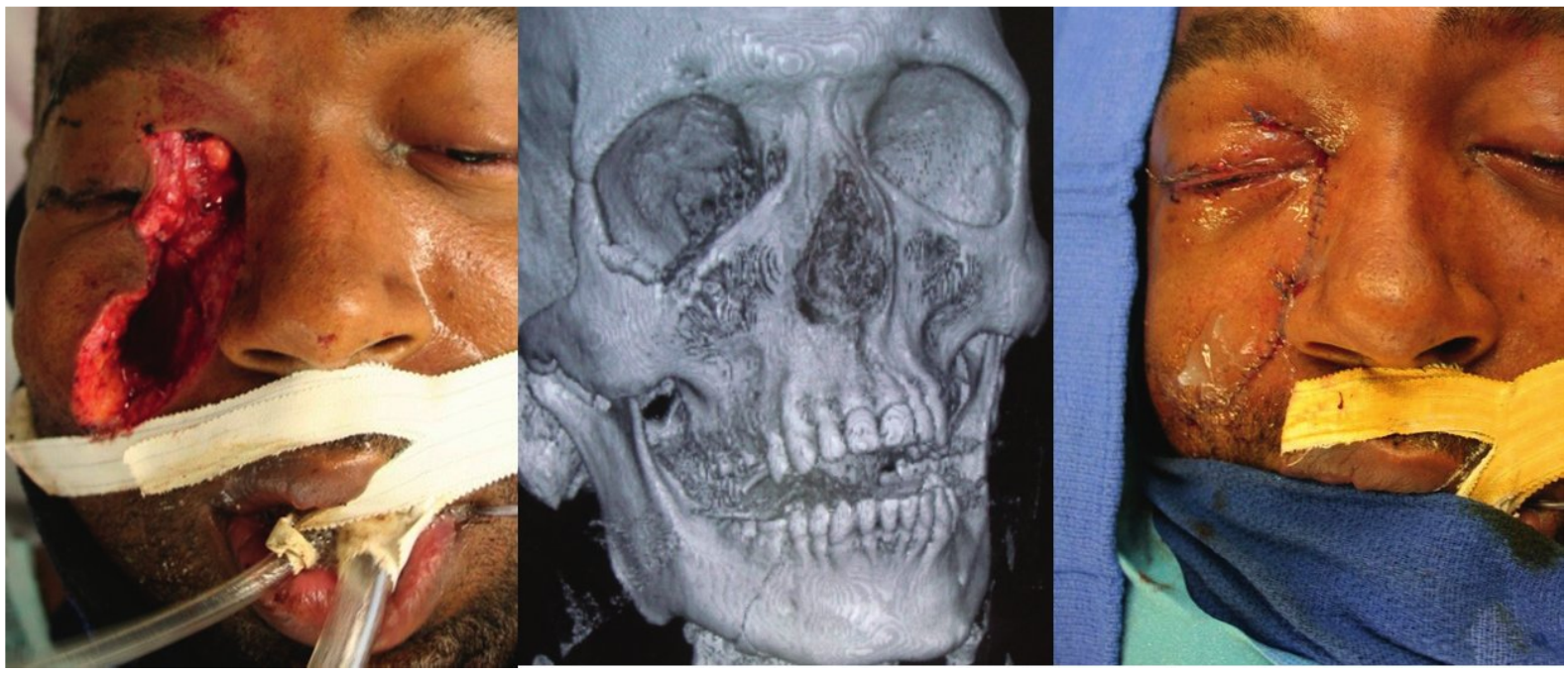


Fig. 9.7 Open complicated NOE fracture with disinsertion of the medial canthal tendon extending into the orbital floor and maxilla.

Anatomy

- Posterior.
  - Sphenoid bone.
- Roof.
  - Anterior cranial fossa.
- Lateral extension of interorbital space.
  - Medial orbital walls.
- Anterior structures.
  - Maxilla, frontal and nasal bones.

The medial canthal ligament is the direct extension of the orbicularis oculi muscle with insertion onto the medial orbital wall. The ligament is composed of three limbs, which help provide medial support to the globe, along with keeping the eyelids tangential to the globe. The superior, anterior, and posterior limbs together form a tent that houses the lacrimal sac. This ligament is important in the classification of NOE fractures.

Physical Findings That May Indicate an NOE Fracture

- Loss of dorsal-nasal prominence (saddle deformity).
- Glabellar, periorbital, nasal ecchymosis.
- Bony crepitus over canthal region.
- Telecanthus ≥ 35 mm (normal 30–32 mm).
- Bowstring test—lateral traction of lower eyelid will result in telecanthus if ligament is disrupted.
- Rhinorrhea—indication of a cribriform plate fracture.
- Olfactory disturbance.

Radiographic Evaluation

- CT scan—1.5-mm cuts axial and coronal.

Markowitz Classification (Fig. 9.8)

- Type I: Single-segment central fracture with medial canthal tendon attached.
- Type II: Comminuted fracture with medial canthal tendon attached.
- Type III: Comminuted fracture with avulsed medial canthal tendon.

Treatment and Management

Patients with NOE fractures need to be admitted and monitored, and intracranial injury should be ruled out. Urgent ophthalmologic evaluation is warranted to rule out injury to the globe. The patient should be assessed for leakage of cerebrospinal fluid (CSF), which may indicate damage to the cribriform plate, frontal sinus, or anterior cranial fossa. CSF rhinorrhea is evaluated by performing the halo test (formation of a halo when CSF is placed on tissue paper) or by laboratory analysis of glucose or β-transferrin in the nasal drainage. If potential dural contamination is suspected, a neurosurgical consult is appropriate.

- Place patient on IV antibiotics (clindamycin 600 IV every 6 hours, Rocephin 1 g IV every 24 hours).
- Fractures will likely be explored and repaired.
- Elevate head of bed.
- No nose blowing.
- Follow appropriate preoperative procedures (nothing by mouth, IV fluids, etc.) if surgery is planned.

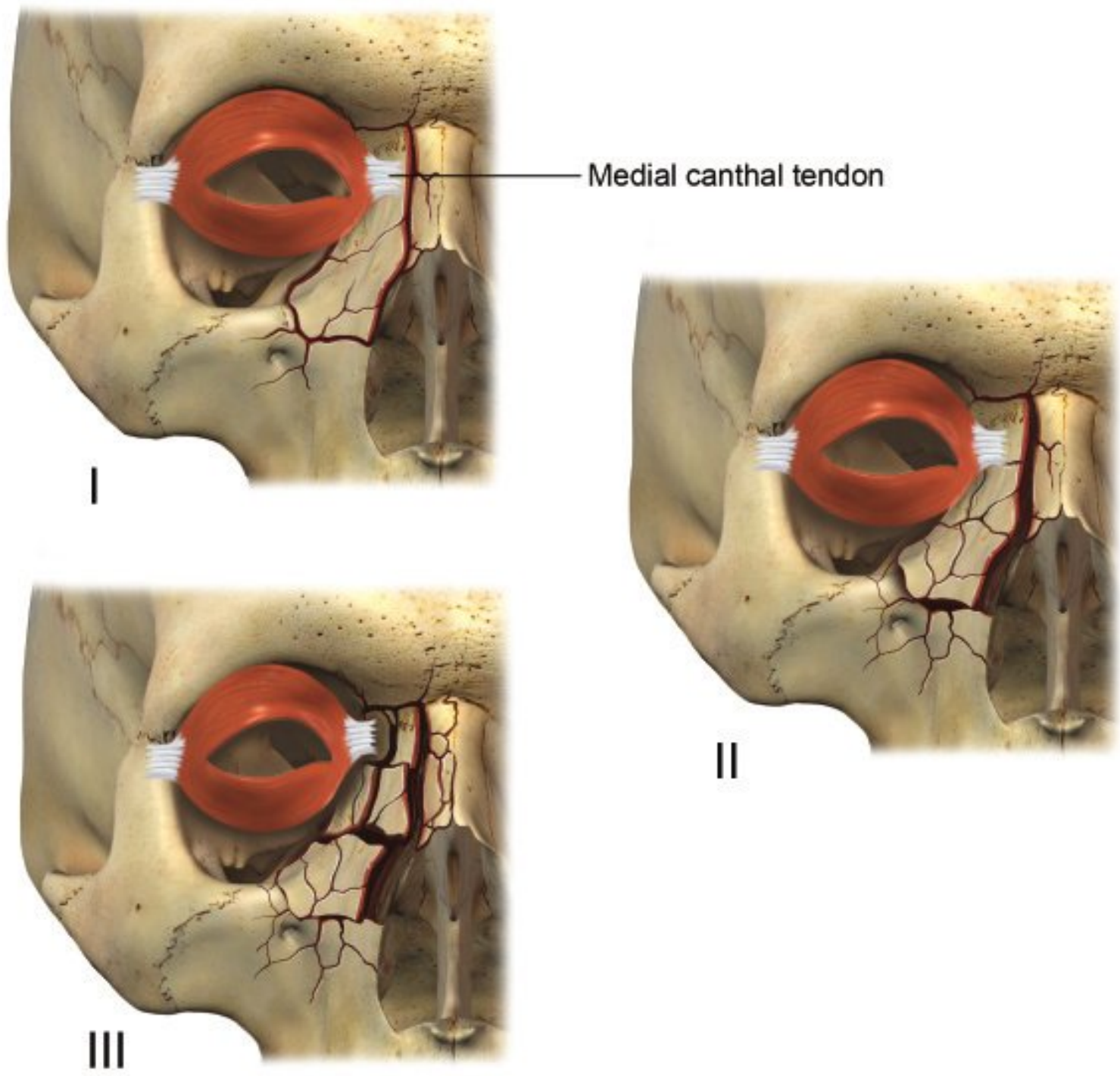


Fig. 9.8 Markowitz classification of naso-orbital-ethmoid fractures.

10 Frontal Sinus Fractures

The frontal bone is the strongest bone of the face. The anterior table can withstand 800 to 2,200 lb of force; therefore, a direct isolated high-energy impact is usually needed to fracture this bone. The frontal sinuses are absent in 4% of individuals, rudimentarily developed in 5%, and unilateral in 10% of individuals. The development of the frontal sinus begins at age 2; it becomes radiographically present at age 8 and continues to develop until the age of 12. Frontal sinus fractures are rare in the pediatric population. Instead, the forces applied to this area are transmitted to the orbital rim (upper or the weaker lower) and the nasal ethmoid area in younger patients.

Anatomy

The anatomy of the frontal sinuses comprises (**Fig. 10.1**)

- Two paired irregular cavities.
  - Anterior wall = anterior table.
  - Posterior wall = posterior table.

Physical Examination

Physical findings that suggest a frontal sinus fracture:

- Forehead contusion.
- Forehead laceration.
- Forehead or orbital hematomas.
- Epistaxis.
- Otorrhea or rhinorrhea from dural tears—test for halo sign on paper towel; send fluid for glucose and β-transferrin analysis.
- Palpable step-off deformity secondary to underlying fracture; may be obscured by overlying swelling in the acute setting.
- Paresthesias in the supraorbital nerve distribution.
- Extension into the supraorbital rim and superior orbital fissure can lead to superior orbital fissure syndrome (see Chapter 8).

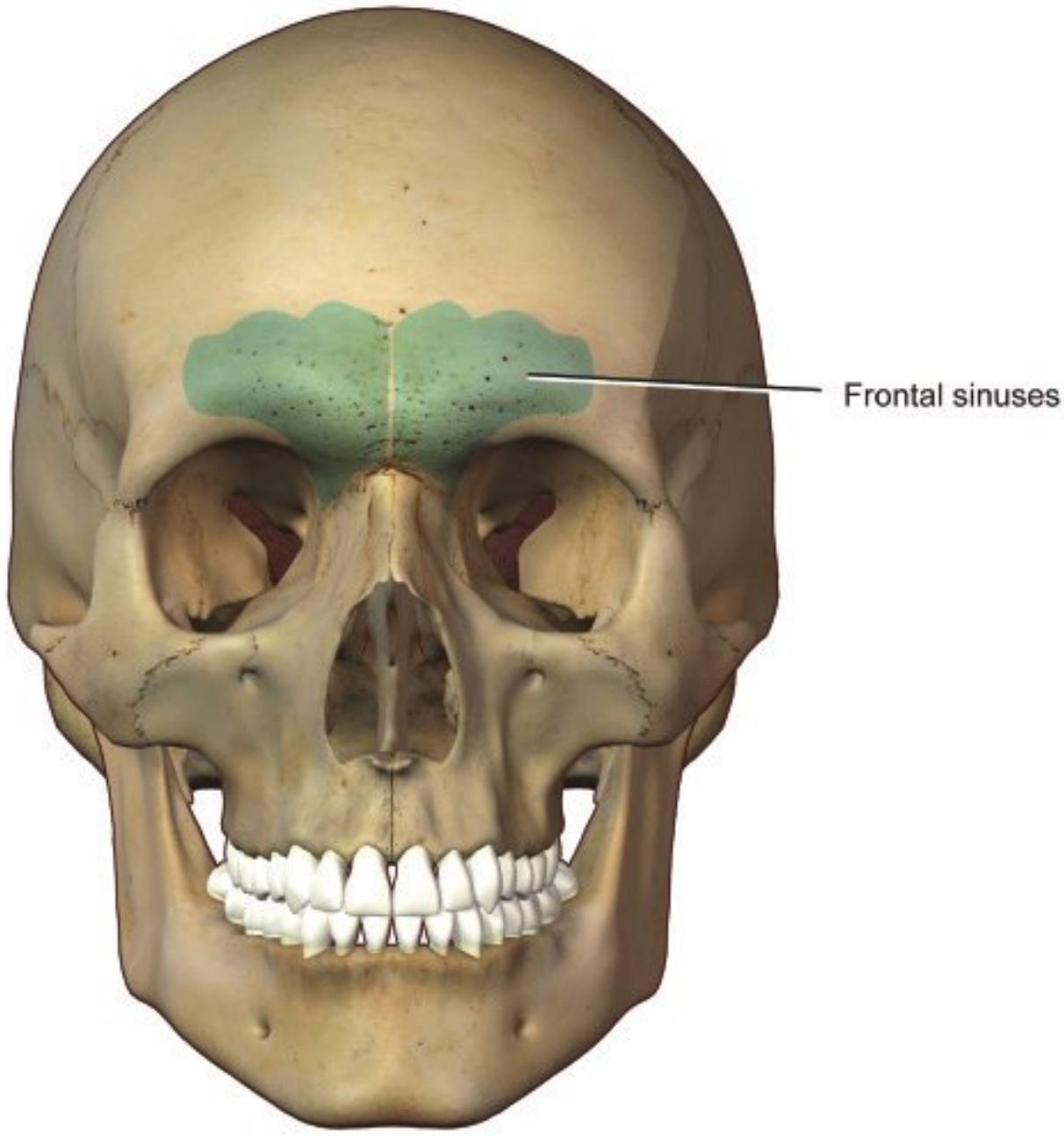


Fig. 10.1 The frontal sinuses.

Radiographic Evaluation

CT of the face with 3-mm axial cuts and coronal reconstructions is the most sensitive modality for diagnosing frontal sinus fractures. Management will often be dependent on whether or not there is a nasofrontal duct injury. Fractures that are located inferiorly and medially should raise a high level of suspicion for nasofrontal duct injury (**Fig. 10.2, Fig. 10.3**).

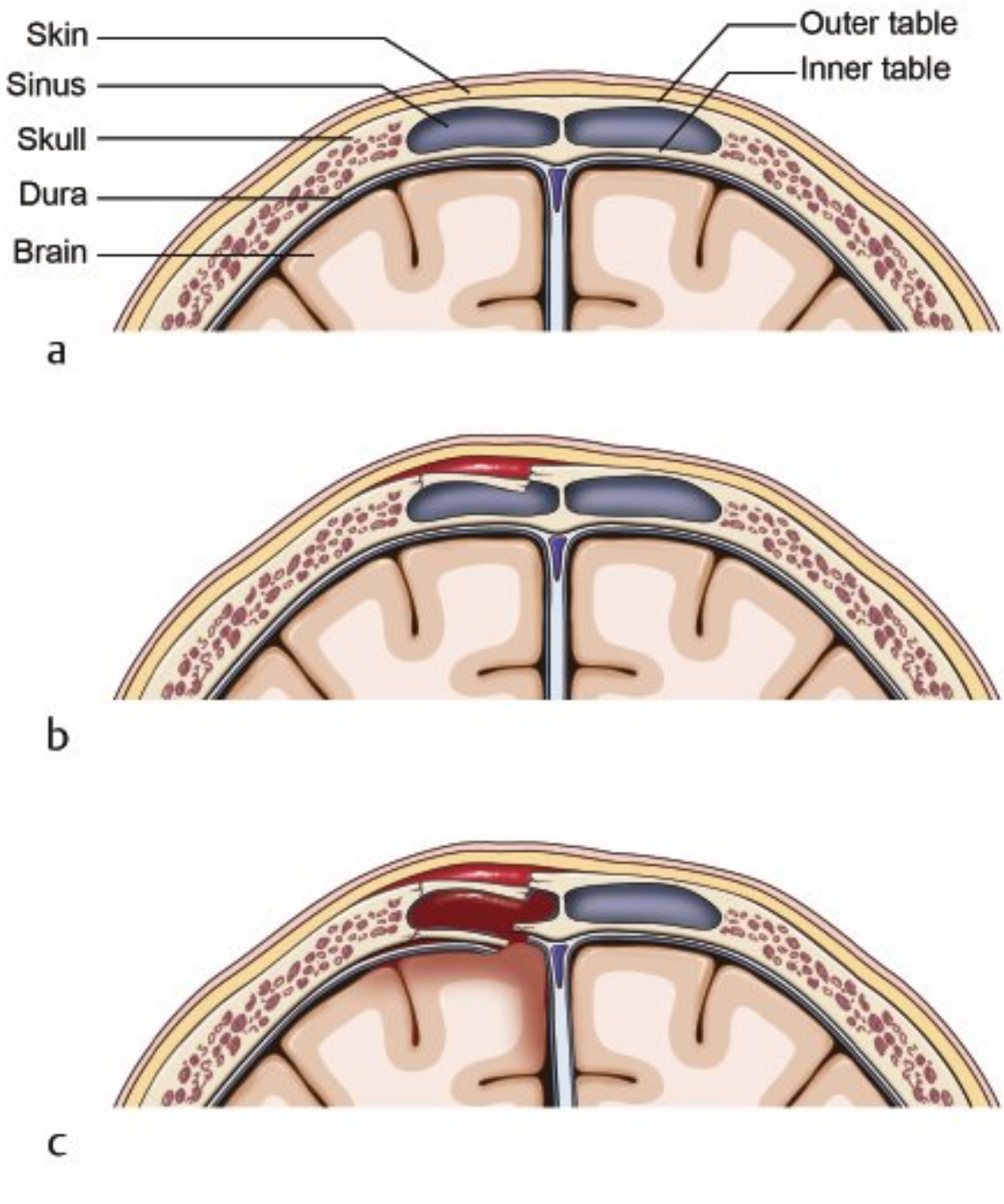


Fig. 10.2 Frontal sinus fracture patterns. **(a)** Normal relationship. **(b)** Anterior table. **(c)** Comminuted anterior and posterior table.

Management

All patients with frontal sinus fractures should be admitted and observed.

- Initial evaluation.
  - Subarachnoid hemorrhages.
  - Subdural hematomas.
  - Epidural hematomas.
  - Cerebral contusions.
  - Pneumocephalus.
- Elevate head of bed to minimize edema.
- Begin intravenous antibiotics.
  - Ceftriaxone 1 to 2 g IV every 24 hours.
- Have the patient avoid activities that could increase intracranial contamination from the sinuses.
  - Avoid nose blowing.
  - Cough and sneeze with mouth open and not through nose.

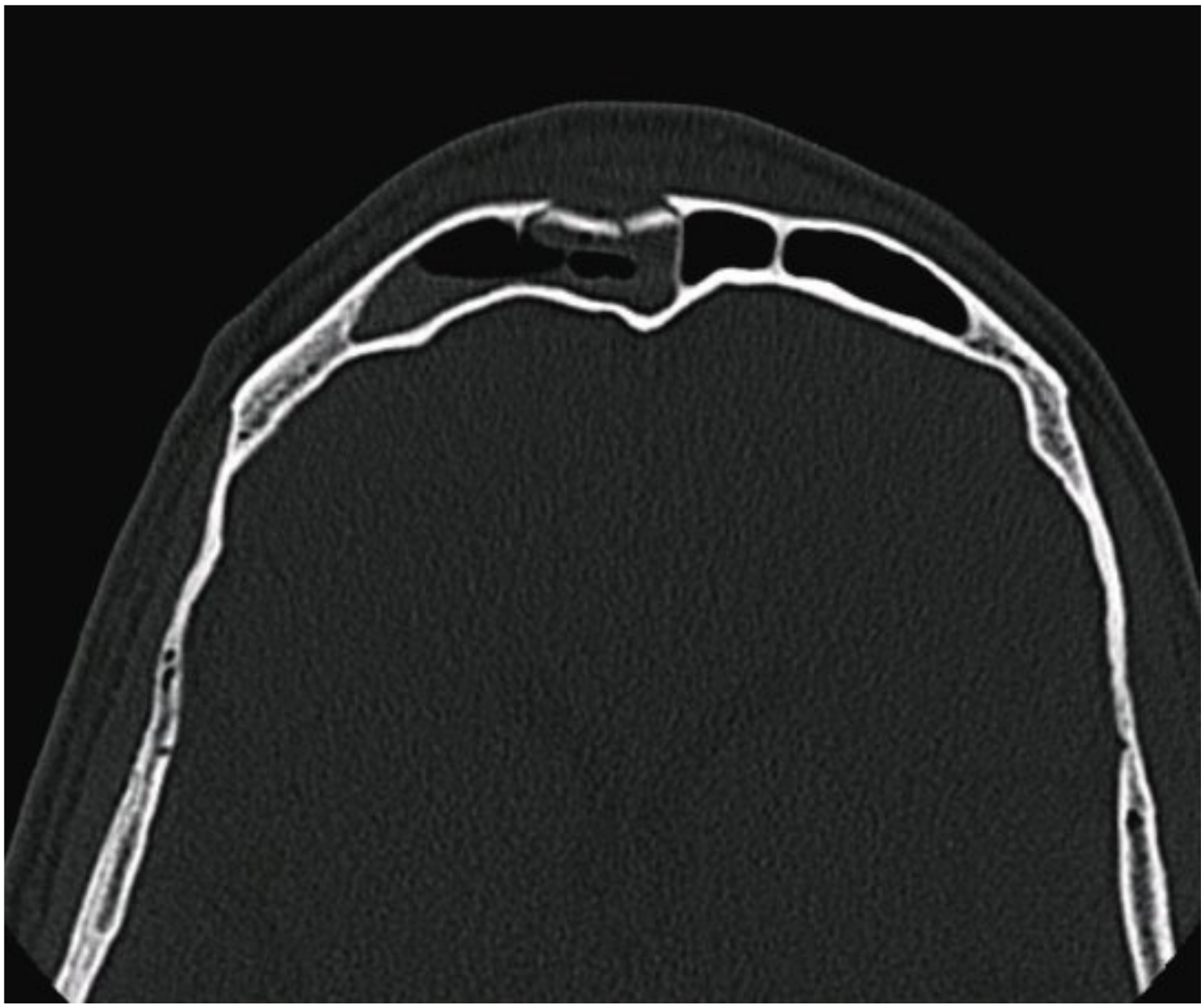


Fig. 10.3 CT of anterior table fracture.

Operative management is dependent on degree of fracture displacement, nasofrontal duct involvement, and dural integrity. Anterior table fractures induce a cosmetic deformity. Nondisplaced fractures do not require operative intervention. Obliteration of the nasofrontal duct is indicated when the duct is involved in the fracture. Otherwise, displaced fractures may by reduced and fixed in a delayed fashion.

Posterior table fractures occur in combination with anterior table fractures and can produce the same sequelae, with the addition of the potential for anterior cranial fossa involvement and dural penetration. CSF leak is evident when the patient presents with significant rhinorrhea that is positive for β2-transferrin or creates a yellow ring on tissue paper (halo test). If the posterior table is not displaced, the patient is observed for 4 to 7 days. Patients with persistent leakage of CSF or displacement and comminution of the posterior table require cranialization. Specific fracture management strategies are outlined in **Fig. 10.4**.

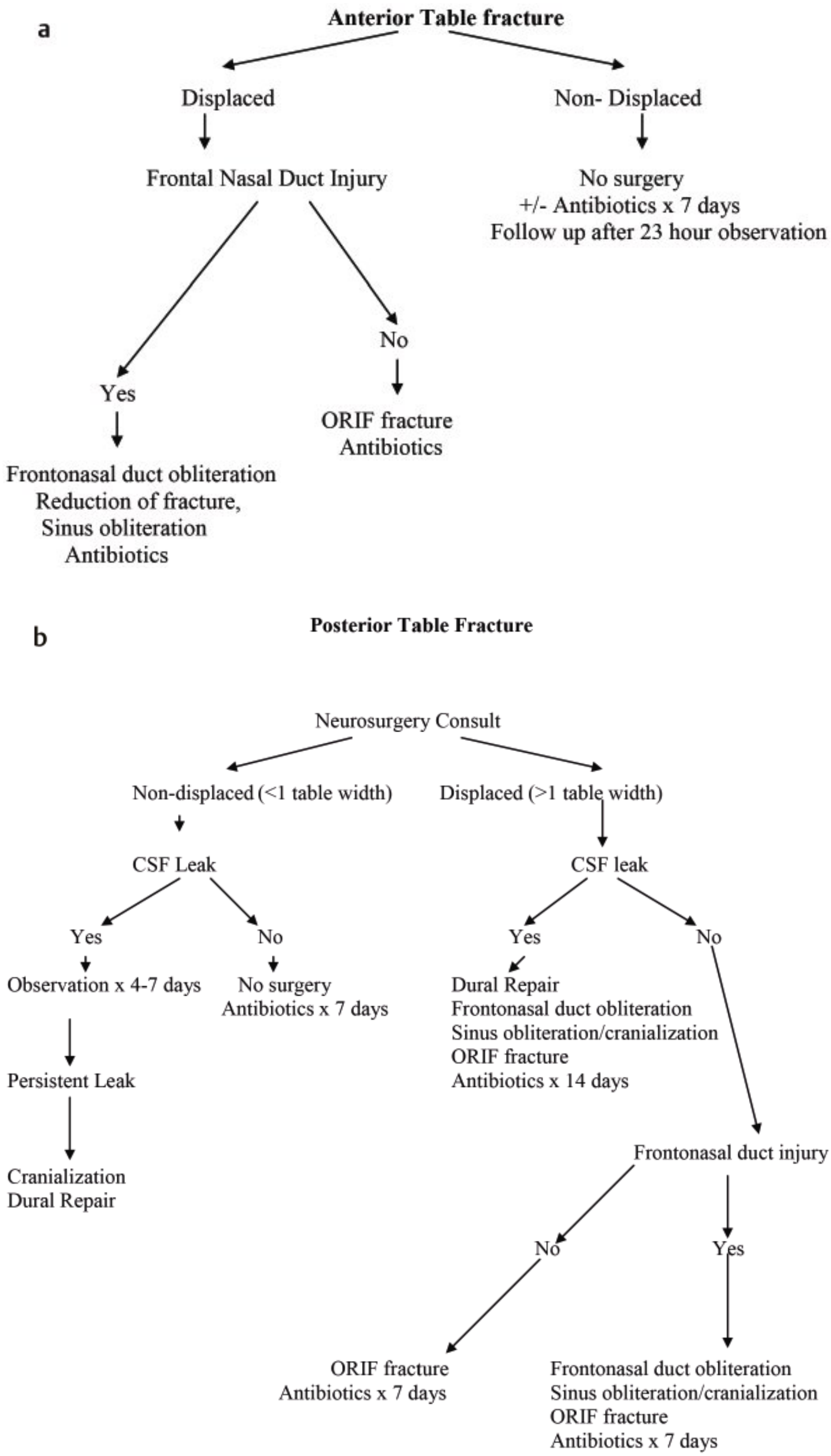


Fig. 10.4 **(a)** Algorithm for anterior table fracture. **(b)** Algorithm for posterior table fracture.

11 Mandibular Fractures

Anatomy

- A U-shaped bone that contains two equal segment hemimandibles.
- Structures unite at midline called symphysis.
- Each hemimandible consists of several structures (**Fig. 11.1**).
  - Body.
  - Angle.
  - Ramus.
  - Coronoid process.
  - Condyle.
- Muscles of mastication.
  - Jaw protrusion.
    - Lateral pterygoid (lateral pterygoid plate to condylar neck).
  - Jaw elevators.
    - Temporalis (temporal fossa to coronoid).
    - Masseter (zygomatic arch to the body).
    - Medial pterygoid (medial pterygoid plate to angle).
  - Jaw depressor-retractors.
    - Lateral pterygoid.
    - Digastric.
    - Geniohyoid.
    - Mylohyoid.
    - Genioglossus.
- Condyle articulates with cranium at the glenoid fossa of the temporomandibular joint (TMJ).
- Blood supply of mandible.
  - Inferior alveolar artery from the internal maxillary artery enters at mandibular foramen and exits at mental foramen.
  - Branches from the muscles of mastication.
- Nerve supply.
  - Inferior alveolar nerve from CN V_3 enters at mandibular foramen and exits at mental foramen.
- Mental foramen.
  - Located between first and second premolars.

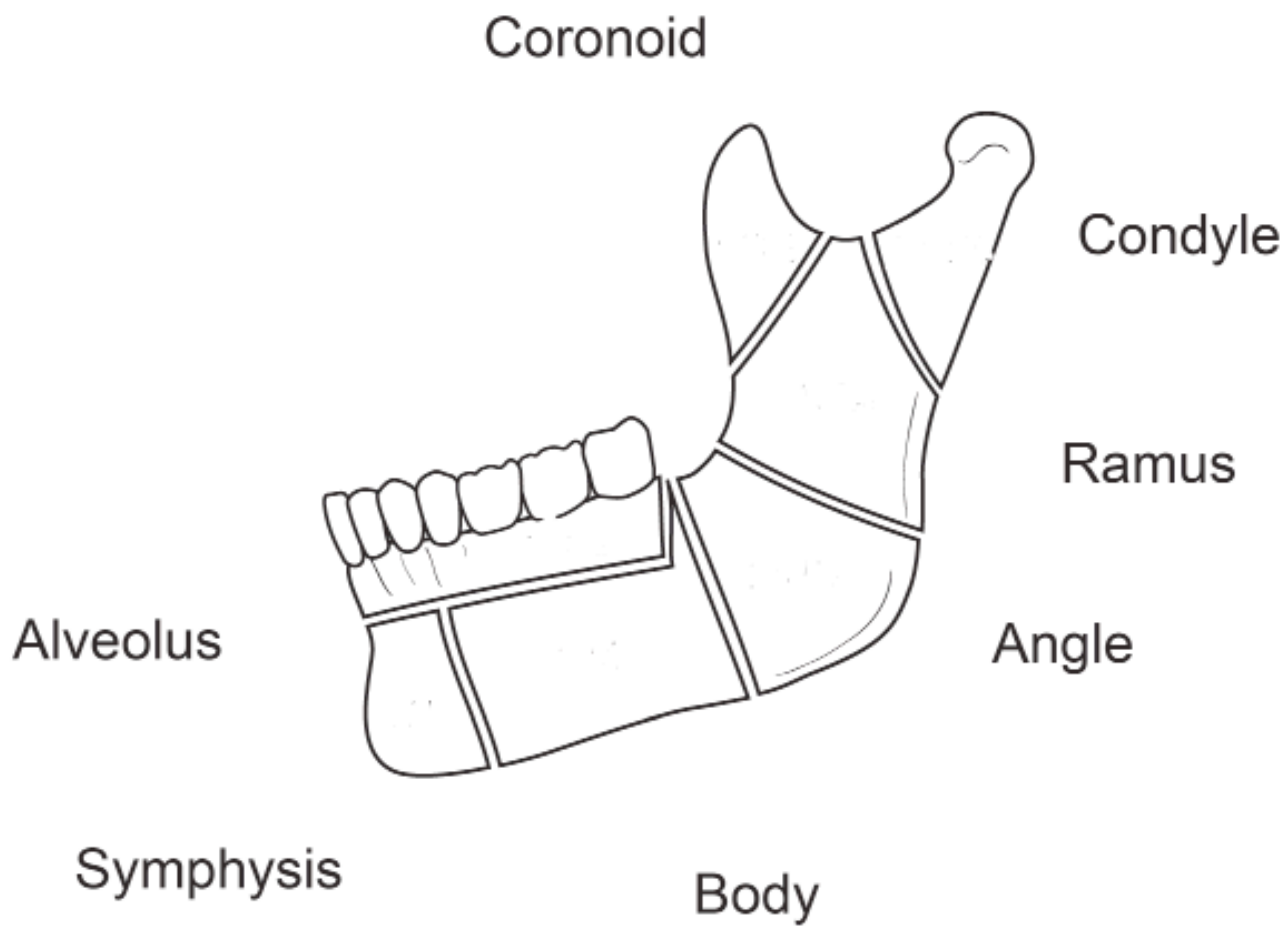


Fig. 11.1 Anatomy of the mandible.

Dental Relationships

Child:

- 20 deciduous or primary teeth labeled A through T.
  - Right—A B C D E F G H I J.
  - Left—T S R Q P O N M L K.

Adult:

- 32 permanent teeth labeled 1 through 32.
  - Numbering begins with the third right maxillary molar as tooth no. 1 and the last maxillary molar as no. 16.
    - Numbering continues with the mandibular left third molar as no. 17 and ends with the mandibular right third molar as no. 32.

Each hemimandible or hemimaxilla consists of

- One central and one lateral incisor.
- One canine (cuspid).
- First and second premolar (bicuspid).
- First, second, and third molar.

Angle Classification of Occlusion

Based on the first maxillary molar and its position relative to the first mandibular molar (**Fig. 11.2**):

- Class I—normal occlusion.
  - Mesiobuccal cusp of the maxillary first molar occludes with buccal groove of the mandibular first molar.
- Class II—overbite.
  - Lower first molar is distal (posterior) to the upper first molar.
- Class III—underbite.
  - Lower first molar is mesial (anterior) to the upper first molar.

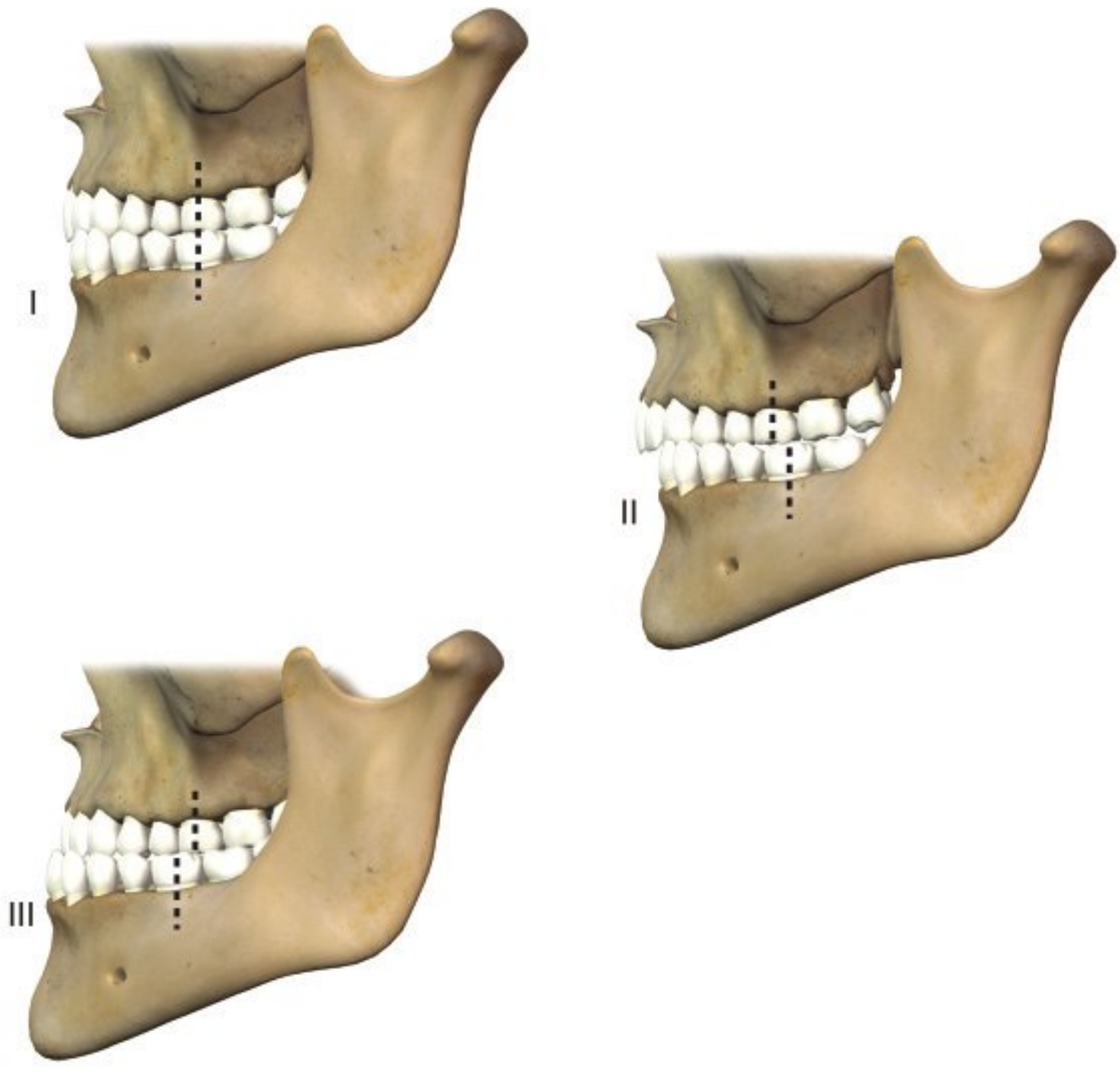


Fig. 11.2 Angle classification of occlusion.

Mandibular Fractures

Fractures of the mandible are often the result of physical altercations and have the highest frequency in men between the ages of 25 and 35. Fractures commonly arise in the thinnest portions of the bone in the angle and condylar region. The thick ramus is fractured the least (**Fig. 11.3**). Mandibular fractures often occur at two sites on the mandible due to the coup-contrecoup phenomenon.

Symptoms and Physical Findings

- Pain.
- Malocclusion—document the angle class of occlusion.
- Trismus—inability to completely open mouth due to pain.
- Crepitus/bony step-offs.
- Mandibular instability.
- Edema and ecchymoses over fracture site.
- Contusions, lacerations, and excoriation.
- Intraoral.
  - Dental infection/abscess.
  - Buccal or lingual ecchymosis.
  - Avulsed teeth/loose teeth.
    - Use the numbering system to account for avulsed, loose, fractured, or missing teeth.
- Open bite.
- Deviation of jaw on opening—suggestive of condylar fracture.
- Paresthesia/anesthesia—document function of inferior alveolar, lingual, and mental nerves.
  - Transection of the inferior alveolar nerve can result in paresthesia/anesthesia at the lips, teeth, and gums.
- TMJ dislocation or derangement.
  - Assess TMJ with a finger in external auditory canal—condylar head should translate anteriorly without significant pain if joint is not injured.

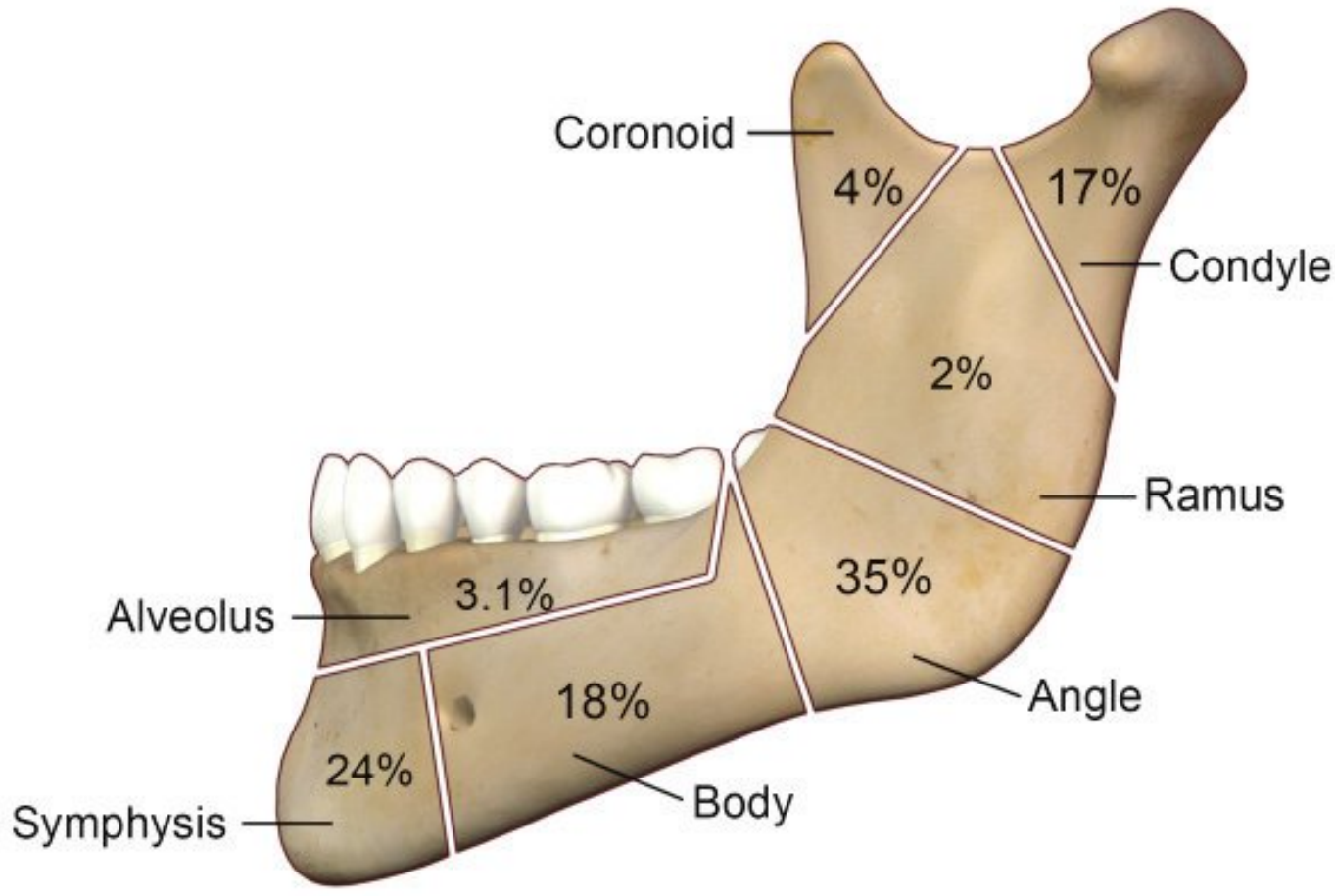


Fig. 11.3 Incidence of mandibular fractures by region.

Radiographic Evaluation

A Panorex radiograph (**Fig. 11.4**) offers the best diagnostic tool in suspected mandible fractures. It is a quick and inexpensive radiograph that offers a complete view of the mandible. It provides an easy means of identification of symphyseal and angle fractures, as well as showing the relation of the fracture line to teeth. Some minimally displaced fractures at the symphysis may be difficult to visualize on a Panorex. Patients are required, however, to have their C-spines cleared because the Panorex radiograph is taken in the sitting position. Otherwise, an intubated or obtunded patient can undergo a panoramic zonography or Zonarc (a panoramic evaluation in the supine position).

CT evaluation is cost-effective and offers nearly 100%sensitivity for diagnosing mandibular fractures (**Figs. 11.5**). A CT scan of the face with coronal reconstructions should be ordered for patients who demonstrate a high index of suspicion for a mandible fracture. Each CT scan should be complemented with a Panorex radiograph to show the relation of the fracture line to the teeth. This detailed information on dental occlusion in relation to the fracture is not easily seen on CT images and is important when assessing which teeth may need to be extracted to allow for optimal mandibular union. Coronal CT evaluation is also helpful in diagnosing mandibular coronoid and condyle fractures (**Fig. 11.5c**).

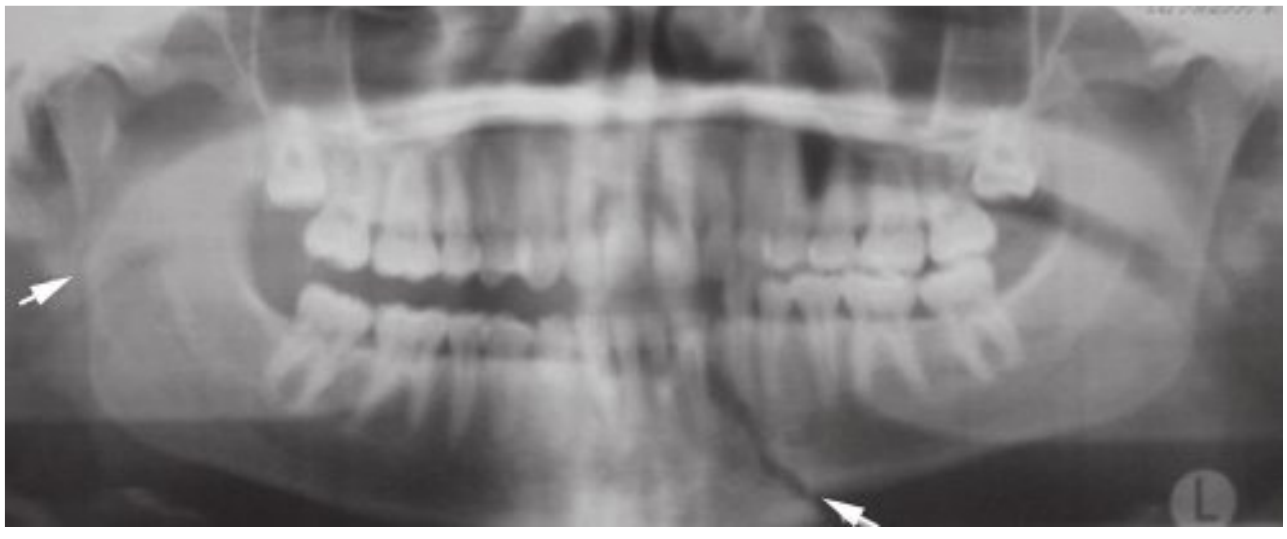


Fig. 11.4 Mandibular fractures of the symphysis commonly occur in combination with fractures of the contralateral condylar region. Panorex radiograph clearly illustrating the fracture of the parasymphysis.

Classification of Fracture

Types of Fracture

- Closed versus open.
- Displaced versus nondisplaced.
- Complete versus incomplete.
- Linear versus comminuted.
- Favorable—when the muscles draw the bony fragments together.
- Unfavorable—when the fragments are displaced by the forces of the muscles.

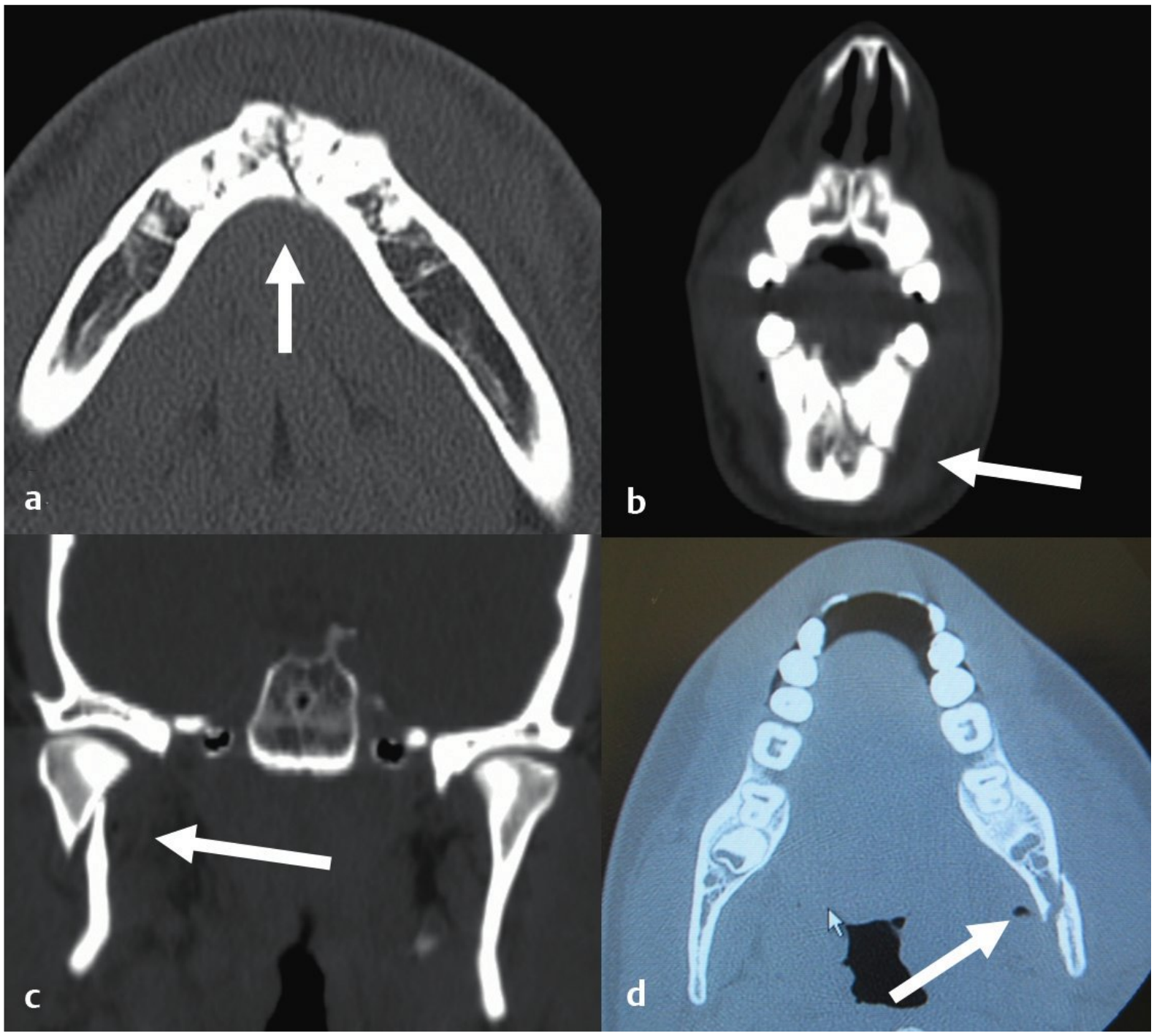


Fig. 11.5 (a) CT evaluation of a fracture of the symphysis. **(b)** Evaluation of the parasymphyseal fracture by coronal CT. **(c)** CT coronal scan of the same patient as in Fig. 11.4 demonstrating a subcondylar fracture not easily seen on the Panorex radiograph. **(d)** CT evaluation of a fracture of the angle.

Location of Fracture (Fig. 11.1)

- Symphyseal—between central incisors.
- Parasymphyseal—between distal border of canine and central incisor.
- Body—between distal edge of canine and distal border of third molar.
- Angle.
- Ramus.
- Coronoid.
- Condyle.
  - Condylar head.
  - Condylar neck.

Nonoperative Management

The absolute goal in the treatment of mandibular fractures is reestablishment of preinjury occlusion. Additionally, attention should be placed on reestablishment of facial contour, height, symmetry, and projection. These goals are accomplished by achieving anatomical reduction of the fracture fragments without infection and with normal mandibular motion.

Immobilization techniques depend on the degree of displacement and the fracture location. Nonoperative management is instituted when the fractures are single and nondisplaced and when patients exhibit preinjury occlusion (**Table 11.1**).

These patients are counseled to comply with a nonchew diet and to perform aggressive oral hygiene for 6 weeks. Nonoperative candidates treated conservatively should be monitored closely at 1- or 2-week intervals until fracture healing. During this observation period, patients should be evaluated for maintenance of occlusion and signs of infection. Deviation from a normal prognosis may portend operative management.

- Nonoperative home discharge regimen.
  - Nonchew diet for 6 weeks.
  - Good oral hygiene—toothbrushing and Peridex mouthwash, swish and spit every 2 to 4 hours.
  - Follow-up in clinic within 2 weeks—obtain additional Panorex radiographs and assess occlusion.

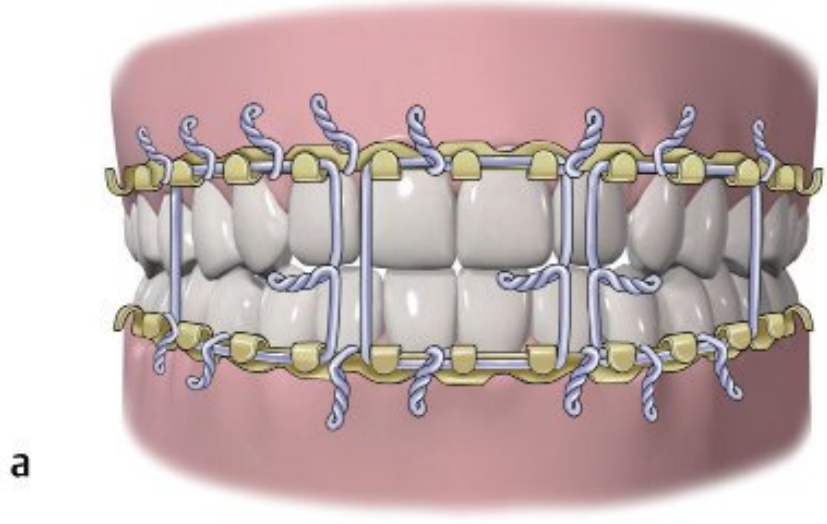
Table 11.1 Criteria for nonoperative and operative treatment of mandibular fractures

Indications for nonoperative treatment of mandibular fractures	Indications for operative treatment of mandibular fractures
Isolated to one region	Fractures of multiple regions
Nondisplaced	Displaced
Simple	Comminuted
Patient exhibits preinjury occlusion	Poor occlusion
	Failed nonoperative management
	Associated infection

Surgical Treatment

Generally, surgical treatment for mandibular fractures is recommended for patients with comminuted, displaced, infected, or multiple injuries. Treatment strategies in the acute setting include bridle wiring and closed reduction in maxillomandibular fixation (MMF) with arch bars and wires or elastics (**Fig. 11.6**).

The specific fracture management depends on the region. Once operative treatment has been decided, reduction of the fragments should be undertaken to reduce the possibility of infection, pain, and malunion.



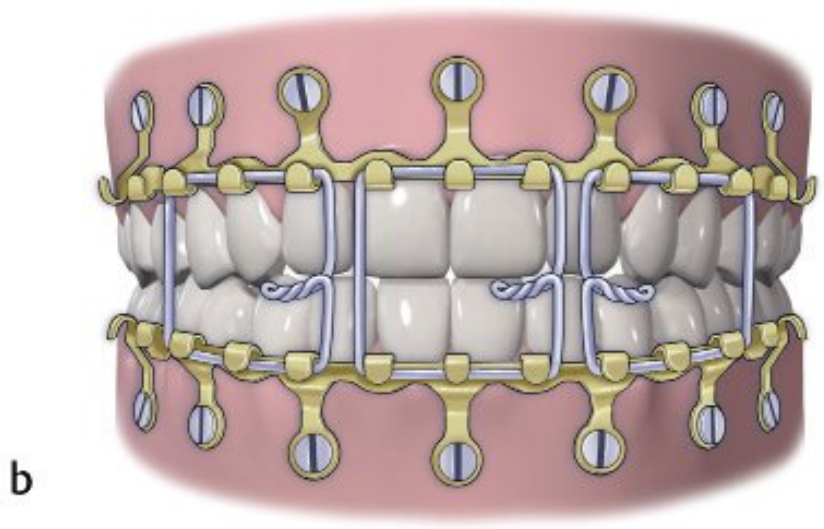


Fig. 11.6 **(a)** Closed reduction of mandible fractures utilizing arch bars with elastics or wire fixation. **(b)** Rapid placement of MMF can be accomplished using the Hybrid MMF system (Stryker).

If fracture reduction is to be delayed more than 5 days, the fracture fragments should be stabilized with a Barton bandage (see Chapter 6, **Fig. 6.4**), a cervical collar externally, or alternatively with MMF until surgery. Fixation of open fractures should be attempted within 72 hours.

Instructions for patients admitted for operative treatment:

- Prophylactic antibiotics—clindamycin 600 mg IV every 8 hours.
- Ensure patent airway. Patients with mandibular fractures may have tongue-based airway obstruction (lacerations, etc.) that may require tracheostomy.
- Rule out C-spine injuries.
- Clear liquid or nonchew diet.
- Oral hygiene—toothbrushing and Peridex mouthwash, swish and spit every 2 to 4 hours.
- IVFs to prevent dehydration secondary to poor oral intake.
- Preoperative work-up.
  - Nutrition consult

Condylar Fractures

Condylar fractures are treated conservatively with closed reduction or open reduction depending on the degree of displacement and laterality. Treatment strategies in this region are employed to decrease the incidence of ankylosis of the TMJ. Closed reduction is advocated in children or when the fracture pattern is high and contained within the capsule. Open reduction and internal fixation (ORIF) is advocated when there is significant displacement outside the capsule of the TMJ or into the middle cranial fossa (**Fig. 11.7**). Foreign bodies within the capsule and failed closed reduction are additional indications for open reduction and internal fixation.

Unilateral nondisplaced fractures in patients with normal occlusion can be treated conservatively. Patients are placed on a nonchew diet and encouraged to perform rehabilitation protocols to prevent ankylosis.

Displaced unilateral fractures in patients with malocclusion are treated with closed reduction for 7 to 10 days, after which rehabilitation is begun.

Bilateral nondisplaced fractures in a patient with a stable midface are treated with closed reduction. However, bilateral displaced fractures or bilateral fractures in a patient with an unstable midface should be considered for ORIF of at least one side to preserve mandibular height and ensure occlusion.

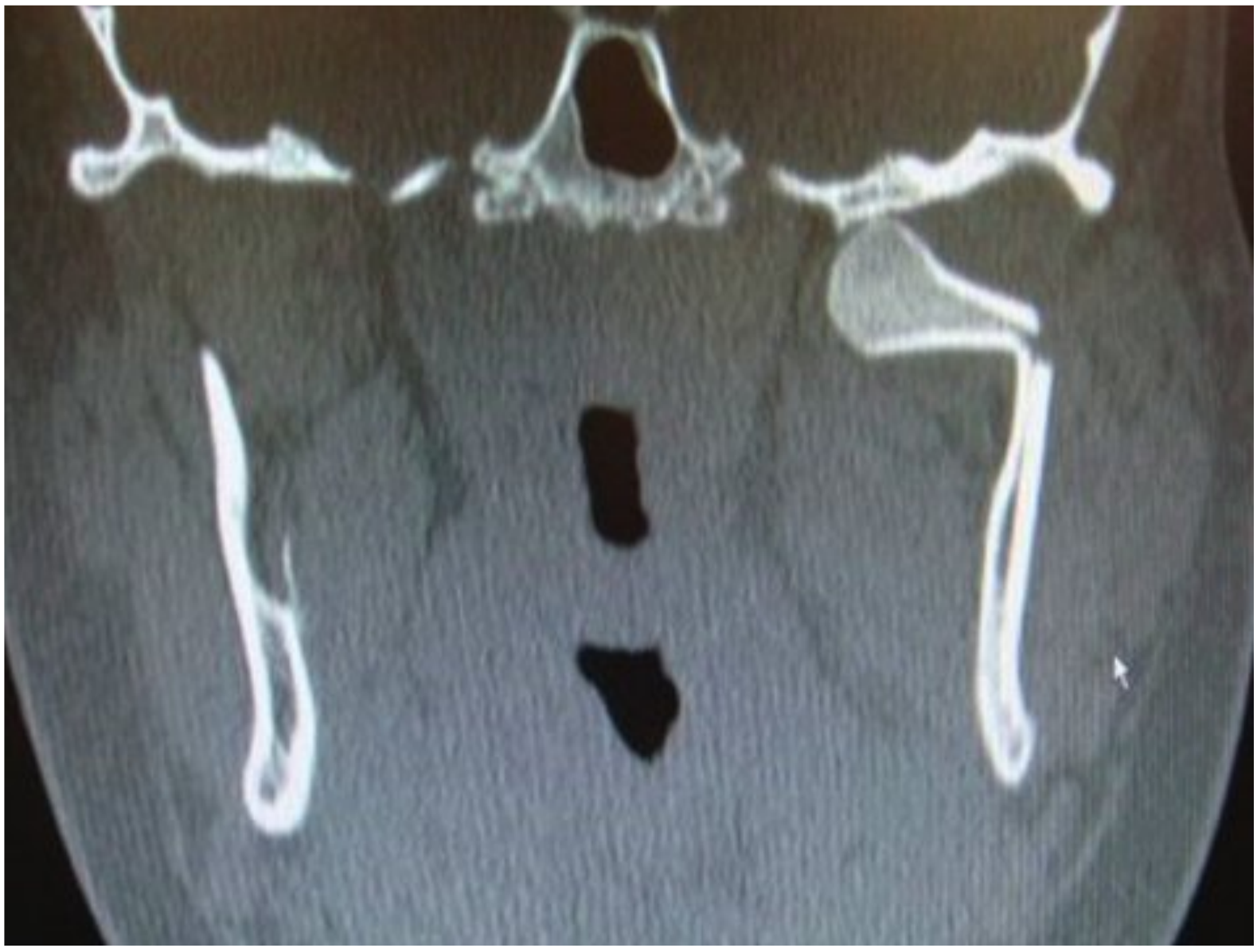


Fig. 11.7 Subcondylar fracture with extracapsular displacement of the condylar head should be considered for ORIF.

Coronoid and Ramus Fractures

Coronoid fractures, if isolated, are managed conservatively provided the patient can open and close the mouth normally. Those patients who are unable to range normally or who have significant pain should undergo ORIF. When coronoid fractures occur in combination with other mandible fractures, ORIF of the concomitant fracture is recommended over closed reduction to prevent ankylosis.

Due to the splinting mechanism of the insertion of the muscles of mastication on the ramus, ramus fractures are stable unless severely displaced. Ramus fractures in isolation are treated with closed reduction.

Angle Fractures

The angle of the mandible is the thinnest portion of the bone and is additionally weakened by the presence of the third molar. Fractures in this region commonly occur secondary to direct trauma and in isolation. The angle lacks dentition and incurs significant distracting forces from

the masseter and the temporalis, which negates the ability of closed reduction to establish occlusion. Therefore, angle fractures are treated with ORIF.

Body and Symphyseal Fractures

Due to the presence of dentition in the body and symphysis, fractures in this region are treated with closed reduction if they are single and easily reducible. If significant dentition is missing or the fracture pattern is comminuted or irreducible, ORIF is considered. Fractures in this region of the mandible commonly occur with contralateral fracture to the subcondylar area. Therefore, careful examination of the condyles is warranted in these patients.

Severely Comminuted Fractures

Severely comminuted fractures are associated with severe bony displacement, as well as possible soft tissue loss. These fractures may require external fixation and débridement in the operating room. Establishment of an airway and ruling out C-spine injury are of paramount importance.

12 Examination of Hand Injuries

When evaluating a patient with a hand injury, begin with a detailed physical examination.

History

- Age.
- Sex.
- Hand dominance.
- Occupation.
- Other medical problems.
- Location of the injury.
- Cause of the injury.
- Time of the injury.
- Duration of the injury process.

Physical Examination

- Verify any physical hand deformities.
- Establish if there is any bleeding, pain, swelling, or ecchymosis.
- Confirm open wounds.
- Note old scars.
- Assess posture of the hand.
  - Angulation of digits signals possible dislocations and fractures.
- Palpate fingers, palm, and wrist for tenderness.
- Determine the temperature of the hands and if they are dry or moist.

After a history is taken and a physical examination is performed, obtain appropriate radiographs (standard X-rays in three views).

All patients with hand injuries should have radiographs made to evaluate for fractures and foreign bodies.

Range of Motion

Check the resting hand position; this may indicate tendon injuries if the natural arcade is disrupted. Examine the motor function, strength, and mobility of all joints of the hand. Observe movement globally and each individual joint's movement. Start with the fingertips and move proximally. **Table 12.1** lists the normal ROM for each joint in the hand.

Distal Interphalangeal Joint

Evaluate for tuft fractures distal to the DIP joint. Normal ROM is 0 degrees of extension and 65 degrees of flexion. Stabilize the middle phalanx with the PIP joint extended to test flexion of the flexor digitorum profundus (FDP).

Lack of extension of this joint may indicate mallet finger, which is a result of the avulsion of the terminal extensor tendon, leaving the DIP joint in a flexed position.

Table 12.1 Normal range of motion for joints of the hand

Joint	Degrees of flexion
Finger DIP	65
Finger PIP	110
Finger MCP	85
Thumb IP	90
Thumb MCP	45–60

Proximal Interphalangeal Joint

Full ROM is from 110 degrees of flexion to 0 degrees of extension in the PIP joint. Inability to flex the PIP joint can result from disruption of the flexor digitorum superficialis (FDS) tendon/muscle, volar plate disruption, or contracture of the intrinsic muscle of the hand. Inability to extend the joint may be a result of extensor mechanism injury (boutonnière deformity) or contracture of the flexor mechanism.

Metacarpophalangeal Joint

The digit MCP joints progress through 85 degrees of flexion and 0 degrees of extension. Often, tendons or the joint capsule may be exposed in cases of laceration. In cases of assault, look for an open laceration over the joint along with decreased prominence of the fifth metacarpal head. This signals the possibility of fracture of the fifth metacarpal neck (boxer's fracture). Joint dislocations may also be present. These may be difficult to reduce if tendon or volar plate entrapment occurs.

The Thumb

Normal MCP joint ROM for the thumb is 45 to 60 degrees of flexion and 0 degrees of extension. Examine for radial and ulnar deviation and pain in the MCP and CML joints. Radial deviation at the MCP joint is a sign of weakness of the ulnar collateral ligament (gamekeeper's thumb).

Common Hand Deformities

Boutonnière Deformity

- PIP flexion with DIP extension caused by disruption of the extensor insertion of middle phalanx and volar migration of the lateral bands.

Swan Neck Deformity

- PIP hyperextension with DIP flexion caused by lateral band tightness and volar plate laxity.

Extrinsic Muscles of the Hand (Table 12.2)

Flexors

Each of the extrinsic flexors is responsible for flexion across one or more joints. Care must be taken to isolate and test each of these tendons individually. The flexors can be injured at their muscle bellies in the forearm or their tendinous portions in the hand. The extrinsic flexors of the digits include the FDP, the FDS, and the flexor pollicis longus (FPL). The wrist is flexed by the combination of the flexor carpi ulnaris (FCU) and flexor carpi radialis (FCR) muscles and secondarily with flexion of the finger flexors.

Table 12.2 Intrinsic and extrinsic flexors and extensors of the hand by joint

Joint	Flexion	Extension
Finger DIP	FDP	Lumbricales, interossei
Finger PIP	FDP, FDS, FDM	EDC, lumbricales, interossei
MCP	Lumbricales, interossei	EDC, EIP, EDM
Thumb IP	FPL	EPL
Thumb MCP	FBP	EPB
Wrist	FCR, FCU, PL	ECU, ECRL, ECRB

Abbreviations: FDP, flexor digitorum profundus; FDS, flexor digitorum superficialis; FDM, flexor digiti minimi; FPB, flexor pollicis brevis; EDC, extensor digitorum communis; FPL, flexor pollicis longus; EIP, extensor indicis proprius; EDM, extensor digiti minimi; EPB, extensor pollicis brevis; EPL, extensor pollicis longus; FCR, flexor carpi radialis; FCU, flexor carpi ulnaris; PL, palmaris longus; ECU, extensor carpi ulnaris; ECRL, extensor carpi radialis longus; ECRB, extensor carpi radialis brevis.

Testing

FDP. Hold the patient's PIP joint in extension and ask the patient to flex the DIP joint. The FDP can flex both joints if one is not immobilized (**Fig. 12.1**).

FDS. Hold all fingers in full (PIP and DIP) extension except the one digit whose FDS tendon you are testing. Ask the patient to flex his or her finger. If the FDS is uninjured, then the PIP will flex. The muscle bellies of the FDS tendon can work independently of each other when the FDP tendons are pulled together. Therefore, immobilize all of the digits in extension so the FDS tendon can be tested for each digit (**Fig. 12.2**). The only exception to this rule is the FDS of the index finger. To determine if the FDS is intact in the index finger, have the patient hold a sheet of paper between the thumb and index finger. If the PIP joint in the index finger is flexed (due to the presence of the FDS), then the FDS is intact. If the PIP joint is extended, then the FDS is not intact. Fifteen percent of the general population do not have a small finger FDS; it is not functional in another 15%

FPL. Elicit flexion of the thumb IP joint.

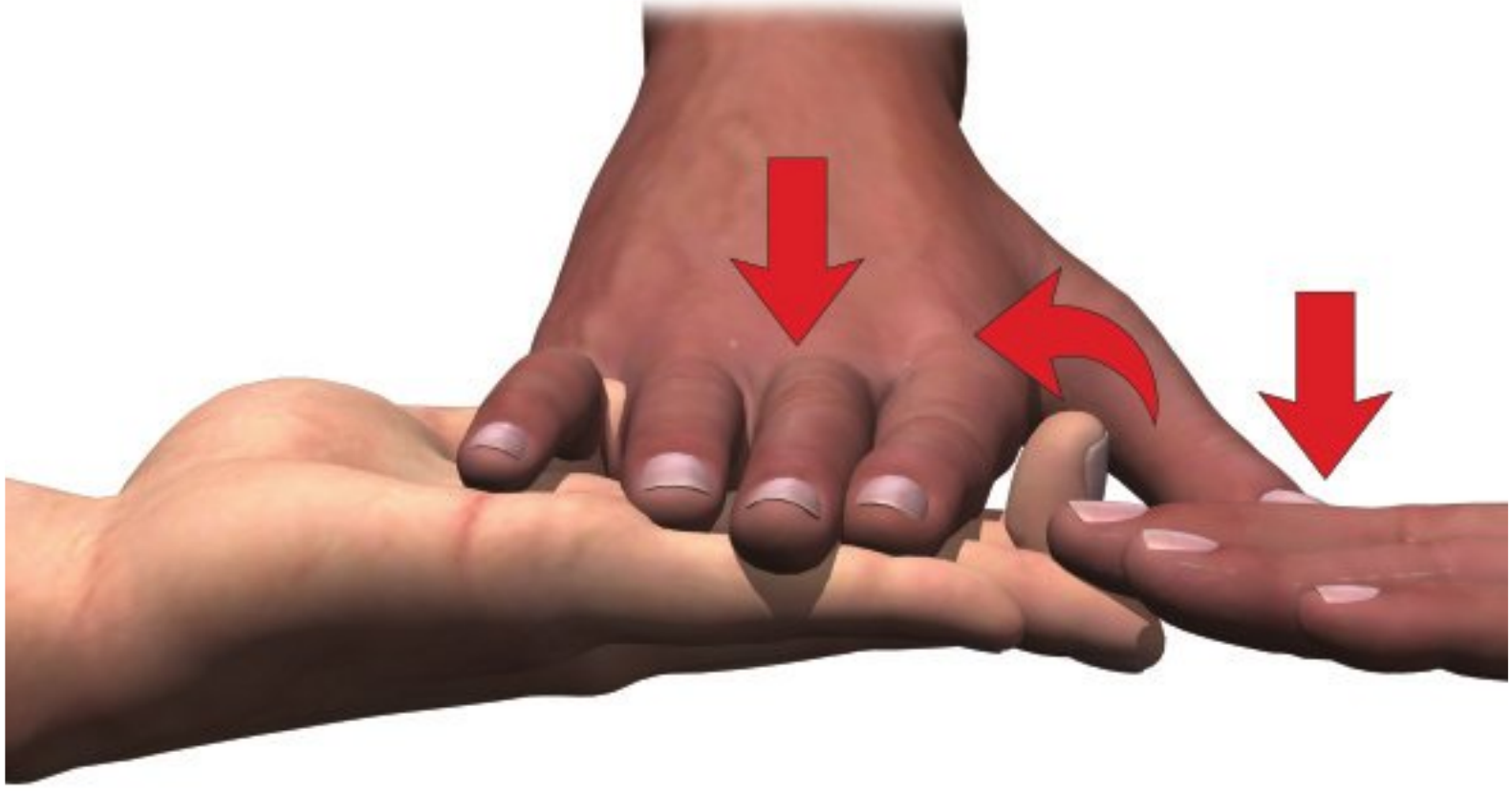


Fig. 12.1 Physical examination of flexor digitorum profundus (FDP). Isolate the FDP by immobilizing the PIP joint, thereby minimizing the contribution of the FDS tendon.

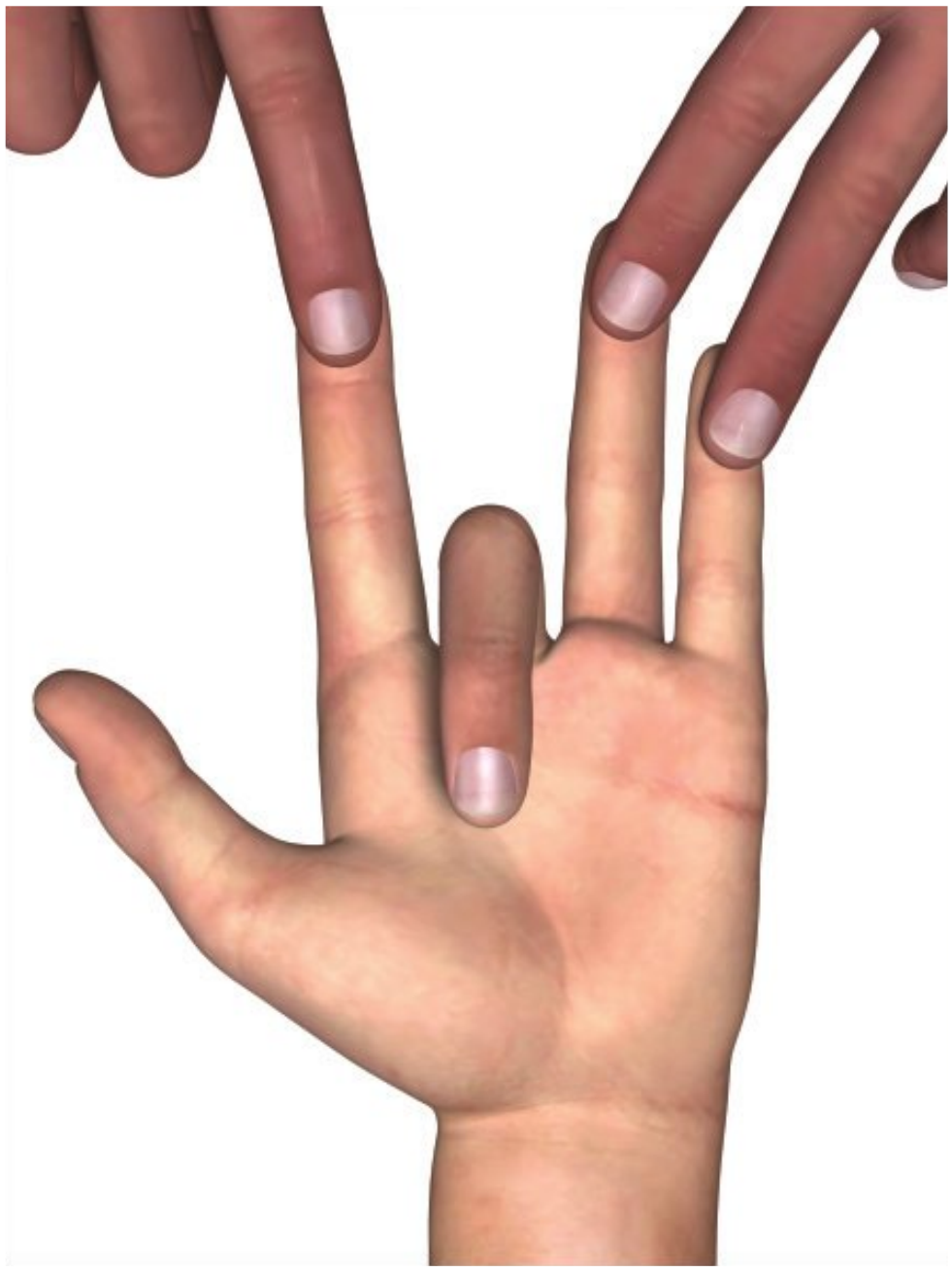


Fig. 12.2 Physical examination of the flexor digitorum superficialis (FDS). Immobilize other digits in extension to minimize contribution of FDP to finger flexion.

Extensors

The extrinsic extensors can be damaged from their muscle bellies in the dorsal forearm all the way to the distal phalanx. They are grouped into six compartments. The MCP joint is extended by the extrinsic extensors only while the PIP and DIP joints are extended by the combination of the intrinsic and extrinsic extensors. The compartments and tests of the extrinsic extensors are as follows:

- Compartment 1: Abductor pollicis longus (APL; abducts thumb) and extensor pollicis brevis (EPB; extends MCP joint).
  - APL: Abduction of thumb on flat surface (**Fig. 12.3**).
  - EPB: Extension of thumb MCP joint.
  - Finkelstein's test: Tests for de Quervain tenosynovitis. Have the patient make a fist over thumb and deviate hand ulnarly. This reproduces patient's pain over the first compartment (**Fig. 12.4**).
- Compartment 2: Extensor carpi radialis longus and brevis, extend wrist.
  - Make a fist and extend wrist against resistance.

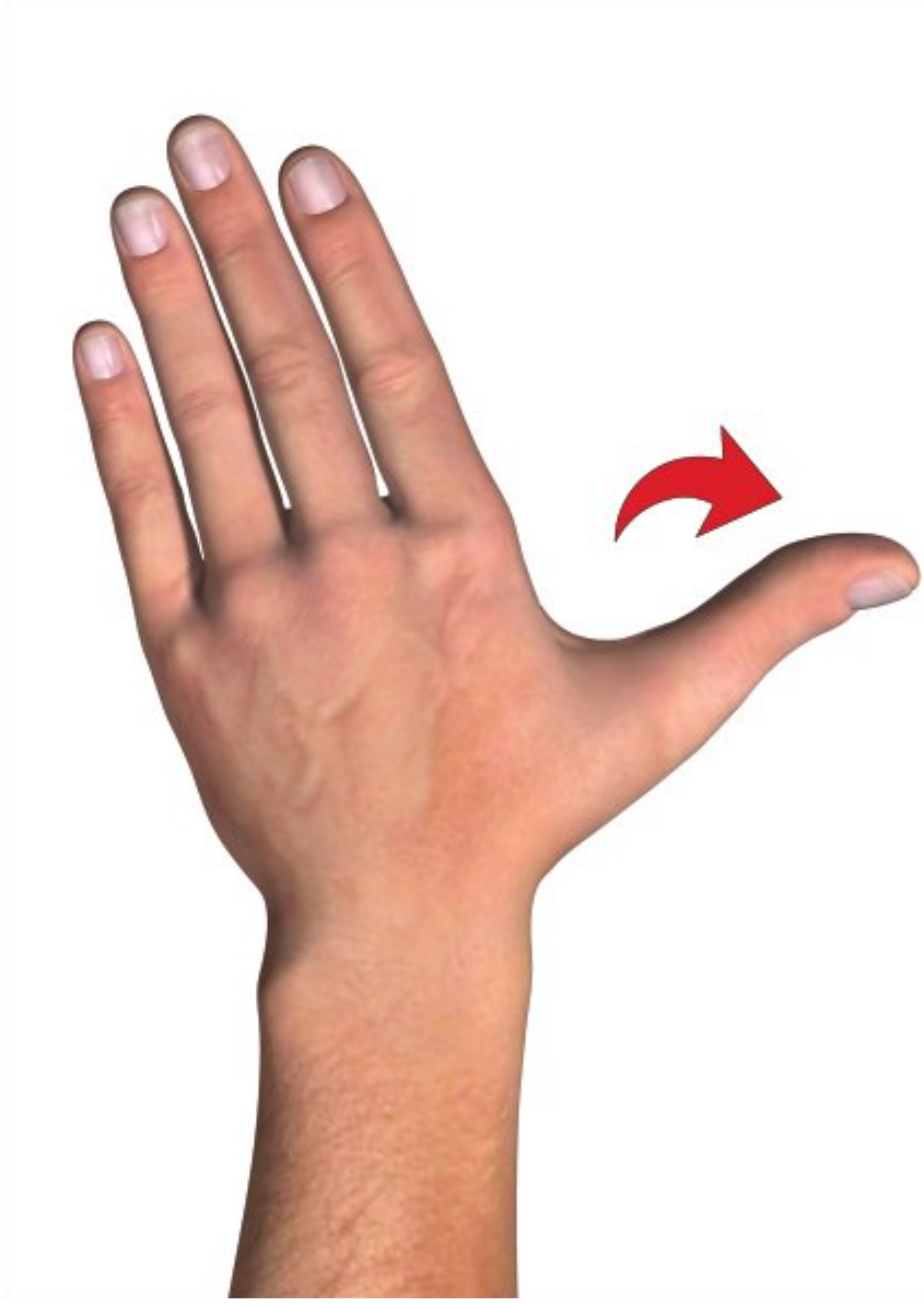


Fig. 12.3 Physical examination of abductor pollicis longus (APL) and extensor pollicis brevis.

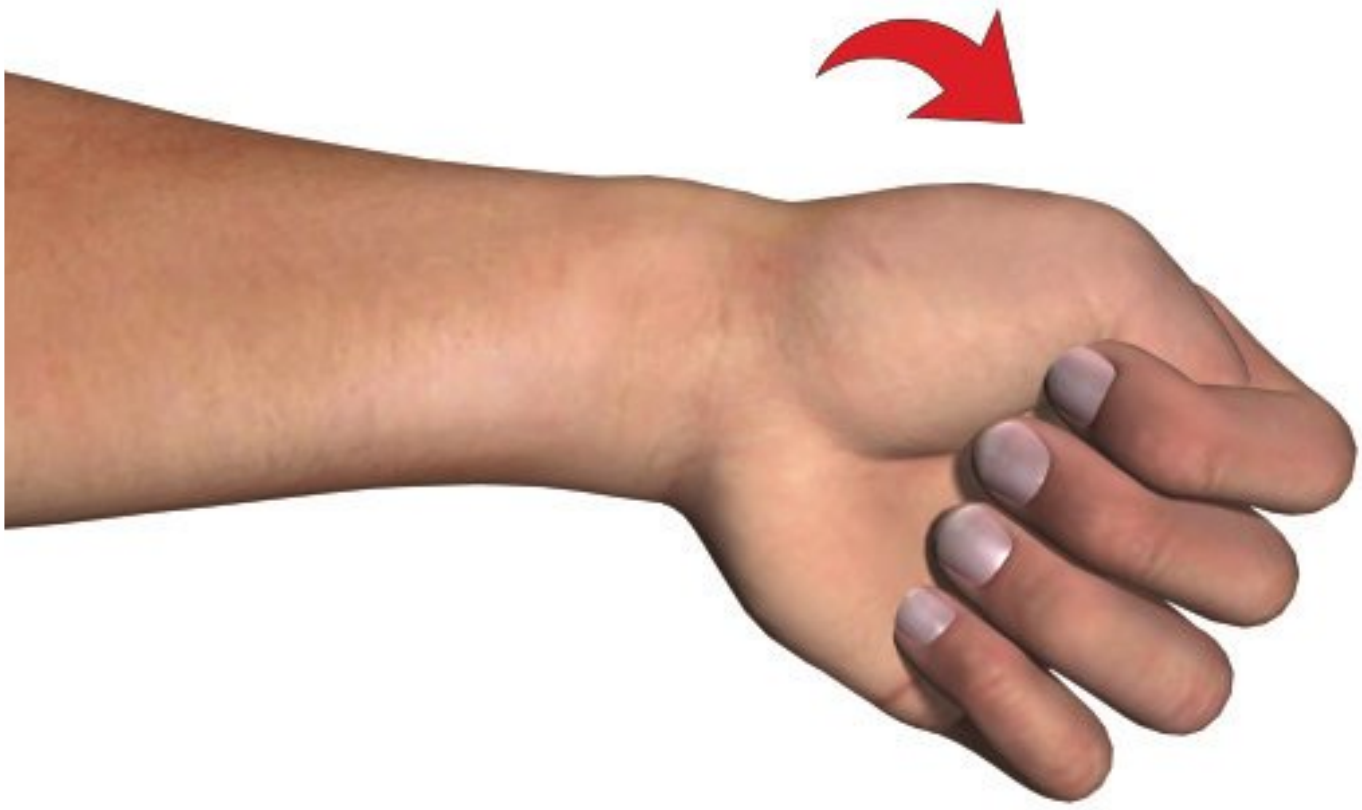


Fig. 12.4 Finkelstein's test. See text for details.

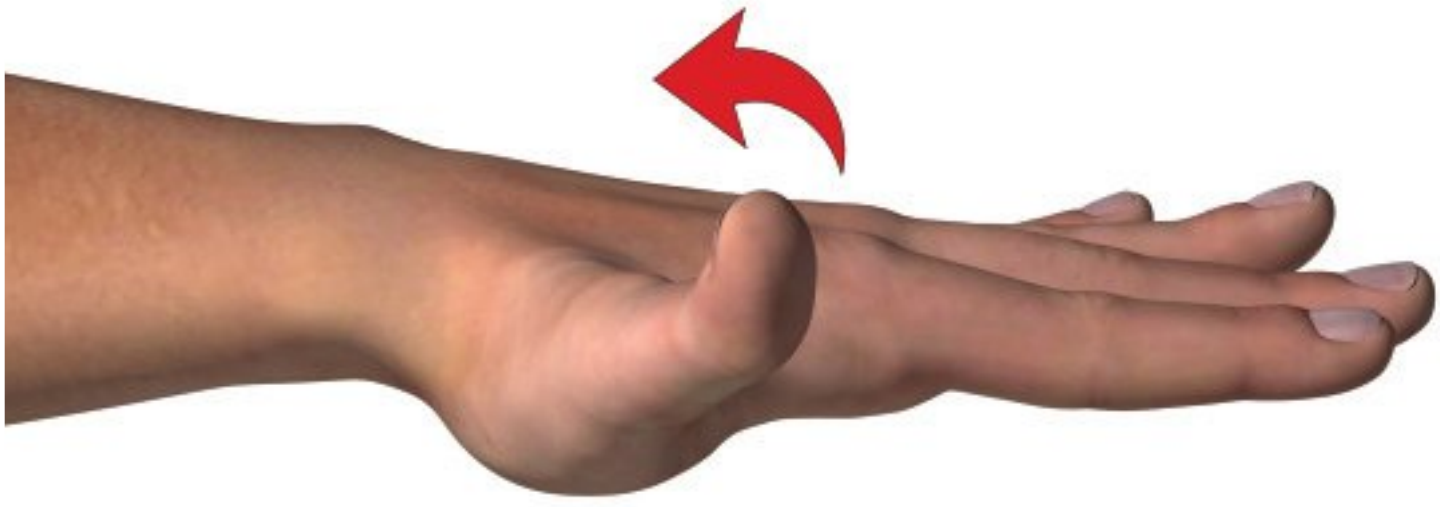


Fig. 12.5 Physical examination of extensor pollicis longus.

- Compartment 3: Extensor pollicis longus.
  - Put hand on a table and raise thumb off the table (**Fig. 12.5**).
- Compartment 4: Extensor digitorum communis (EDC) and extensor indicis proprius (EIP).
  - EDC: Extend all fingers.
  - EIP: Ask patient to hold the index finger in extension while flexing other fingers. EDC tendons are grouped and therefore cannot act independently of each other.
- Compartment 5: Extensor digiti minimi.
  - Hold small finger in extension while making a fist with other fingers.
- Compartment 6: Extensor carpi ulnaris.
  - Extend the wrist ulnarly and palpate tendon over fifth metacarpal.

Intrinsic Muscles of the Hand (Table 12.2)

The muscle bellies and tendons of the intrinsic muscles of the hand are contained within the hand. Together these muscles act to flex the MCP joint while extending the IP joints. The intrinsic muscles of the hand are listed below, together with the appropriate tests.

- Thenar muscles: Abductor pollicis brevis, opponens pollicis, flexor pollicis brevis.
  - Palpate thenar eminences. If hypotrophic, consider median nerve damage. To test these muscles, ask the patient to perform thumb-pulp-to-small-finger-pulp opposition.
- Adductor pollicis: Involved in pinching.

- Hypothenar muscles: Abductor digiti minimi, flexor digiti minimi, and opponens digiti minimi.
  - Palpate hypothenar eminence. Ask the patient to abduct small finger.
- Interosseous muscles: MCP flexion and IP extension.
  - Dorsal: Digital abduction.
  - Palmar: Digital adduction.
    - Hold IP joints in extension and ask the patient to flex the MCP joint.
- Lumbricales muscles: MCP flexion and IP extension.
  - Hold MCP in flexion and ask patient to extend IP joints.

Vascular Examination

- Test to see if the hand is cold, congested, or edematous.
- Check capillary refill by pinching fingertips and counting the time it takes to refill; 2 to 3 seconds is normal.
- Check for blue or necrotic spots on the fingertips.
- Palpate radial and ulnar arteries. If not palpable, then use a handheld Doppler probe.
- Perform Allen test to determine the integrity of the palmar arch.
  - First, ask the patient to make a fist while you occlude the radial and ulnar arteries.
  - Then have the patient open the exsanguinated hand.
  - Let go of the radial vessel and determine if the hand returns to its normal pink hue. Repeat this procedure and let go of the ulnar artery. Take special note of return of vascularity to the thumb.
  - If the patient cannot make a fist, then use Doppler ultrasound to find the palmar arch. Occlude the radial or ulnar artery and check to see if there is a Doppler signal in the palmar arch. Perform test on both radial and ulnar artery.
  - Make sure to examine the arch throughout its course in the palm.
- Examine each of the digital arteries by assessing temperature, color, and capillary refill.

Neurologic Examination

The radial, ulnar, and median nerves supply the hand; **Table 12.3** gives distribution and innervation. First determine if the patient has sensation over the radial distribution (the back of the hand).

Next, turn the hand volar and determine if the patient has general sensation over the radial three digits and palm (median nerve). Be sure to examine the proximal portion of the palm for sensation. Finally, examine the volar and dorsal ulnar portion (ulnar nerve distribution).

To determine if digital nerves are intact, perform the Weber test. Using a caliper or bent paper clip, measure the minimum distance of two-point discrimination. Normal is 2 to 3 mm in the finger pulp. Patients involved in occupations where heavy labor is required may have a two-point discrimination of 5 to 6 mm. Patients who are blind may have a discrimination of 1 to 2 mm. The patient needs to be correct in 7 tests out of 10 for good two-point discrimination.

Testing of motor nerve function of the upper extremity is performed by eliciting contraction of specific motor units.

- *Musculocutaneous nerve:* Flexes the elbow.
- *Radial nerve:* Elbow extension.
- *Median nerve:* Wrist, finger (index, long) flexion, and thumb flexion.
- *Ulnar nerve:* Wrist, finger (ring, small), and intrinsic hand motility, including abduction and adduction of the fingers.
- *Radial nerve:* Wrist, finger (MCP joint), and thumb extension.

Specific nerve–muscle associations are listed in **Table 12.3**. In assessing motor function, the Medical Research Council muscle scale is useful for quantifying strength (**Table 12.4**).

Table 12.3 Nerve and motor innervations of the hand

Nerves	Motor	Sensory
Radial	Triceps	Dorsal wrist capsule
	Anconeus	Dorsal radial hand
	Brachioradialis	Dorsal thumb, index finger, middle finger, and radial half of ring finger to PIP joint
	Supinator	
	Extensor carpi radialis brevis	
	Extensor carpi radialis longus	
	Extensor carpi ulnaris	
	Extensor digitorum communis	
	Extensor indicis proprius	
	Extensor digiti minimi	
	Abductor pollicis longus	
	Extensor pollicis longus	

(Continued)

	Extensor pollicis brevis	
Ulnar	Flexor carpi ulnaris	Ulnar half of the dorsum of hand
	Flexor digitorum profundus (small and ring fingers)	Volar and dorsal aspect of small finger, ulnar side of ring finger
	Palmaris brevis	
	Dorsal interosseous muscles	
	Palmar interosseous muscles	
	Ring and small finger lumbricales	
	Adductor pollicis	
	Flexor pollicis brevis deep belly	
	Hypothenar muscles:	
	Abductor digiti minimi	
	Flexor digiti minimi	
	Opponens digiti minimi	
Median	Pronator teres	Volar wrist, thumb, index finger, middle finger, and radial half of ring finger extending to the DIP joint
	Pronator quadratus	
	Palmaris longus	
	Flexor carpi radialis	
	Flexor digitorum superficialis	
	Flexor digitorum profundus (index and middle fingers)	
	Flexor pollicis longus	
	Index and middle finger lumbricales	
	Thenar muscles	
	Abductor pollicis brevis	
	Opponens pollicis	
	Superficial belly of flexor pollicis brevis	

Table 12.4 The Medical Research Council muscle grading system

Observation	Muscle grade
No contraction	0
Flicker or trace of contraction	1
Active movement, with gravity eliminated	2
Active movement against gravity	3
Active movement against gravity and resistance	4
Normal power	5

13 Anesthesia and Splinting of the Hand and Wrist

Anesthesia

The application of nerve blocks not only provides comfort to patients but also assists the physician in exposing and repairing injuries to the upper extremity.

Common local anesthetics available in the emergency setting are:

- Lidocaine 1 to 2%
  - Toxic dose > 4 mg/kg.
- Lidocaine 1%with epinephrine 1:100,000.
  - Toxic dose > 7 mg/kg.
- Marcaine 0.25%
  - Toxic dose > 2.5 mg/kg.
- 1:1 mixture of lidocaine/Marcaine.
  - Toxicity is the same for both agents.
  - Toxicity is not additive.

Injection of Local Anesthetics

There are several techniques to consider when administering local anesthesia to increase patient comfort and to reduce complications:

- Dilute the concentration.
  - Dilute with sterile injectable saline.
    - Provides additional volume for injection over a larger area without increasing the total dose administered.
    - Aids in decreasing the total dose required.
- Administer the local anesthetic agent slowly.
  - Toxicity develops due to peak serum concentration.
  - Inject each site sequentially rather that all at once.
  - Spread the total dose of local anesthetic over a longer period; this leads to lower peak serum levels.
- Add epinephrine.
  - Effective concentrations 1:1,000,000.
  - Improves safety and allows administration of lower doses.
  - Improves hemostasis, thus decreasing duration of procedures.

  - Helps prevent the need for subsequent injection.
  - Beware of epinephrine use in patients with cardiac history.
  - Avoid administering epinephrine in the digits and to pediatric patients.
- Add bicarbonate.
  - Decreases burning on administration.
  - Add 1 mL of a 1-mEq/mL bicarbonate for every 9 mL of local anesthesia.
- Consider mixing agents.
  - Use more than one local anesthetic to take advantage of the unique properties of each local anesthetic.
  - Use a short-acting local anesthetic (lidocaine) with a long-acting agent (Marcaine).
  - Provides prolonged anesthesia without causing toxicity from either agent.
  - The toxicity of the mixture does not exceed the individual toxicity of each agent.
  - Toxicity of multiple agents in a solution is not additive.
- **Draw back prior to injection to ensure no anesthetic is given intravascularly**.

Digital Nerve Block

Two volar and two dorsal nerves innervate the digit. The common digital nerve and dorsal sensory nerves are blocked via a dorsal approach with one needlestick. Using a 25-gauge needle, a 1-mL wheal is made over the extensor mechanism at the level of the MCP joint to block the dorsal sensory nerve. The needle is then advanced volarly on either side of the joint in the web space until it is palpated in the palm. An additional 1 mL of local anesthetic is placed on each side to block the digital nerve.

Alternatively, a digital nerve block may be performed with direct instillation of the 1 to 3 mL of anesthetic agent in the adjacent web spaces dorsally and dorsal over the MCP joint (**Fig. 13.1**). Care must be taken not to perform circumferential instillation around the digit that may subsequently impair perfusion.

Wrist Block

Wrist blockade includes anesthesia of the median, ulnar, and radial nerves. The efficacy of a wrist block is increased by application of a tourniquet at the midforearm. Wrist blocks are applied as follows.

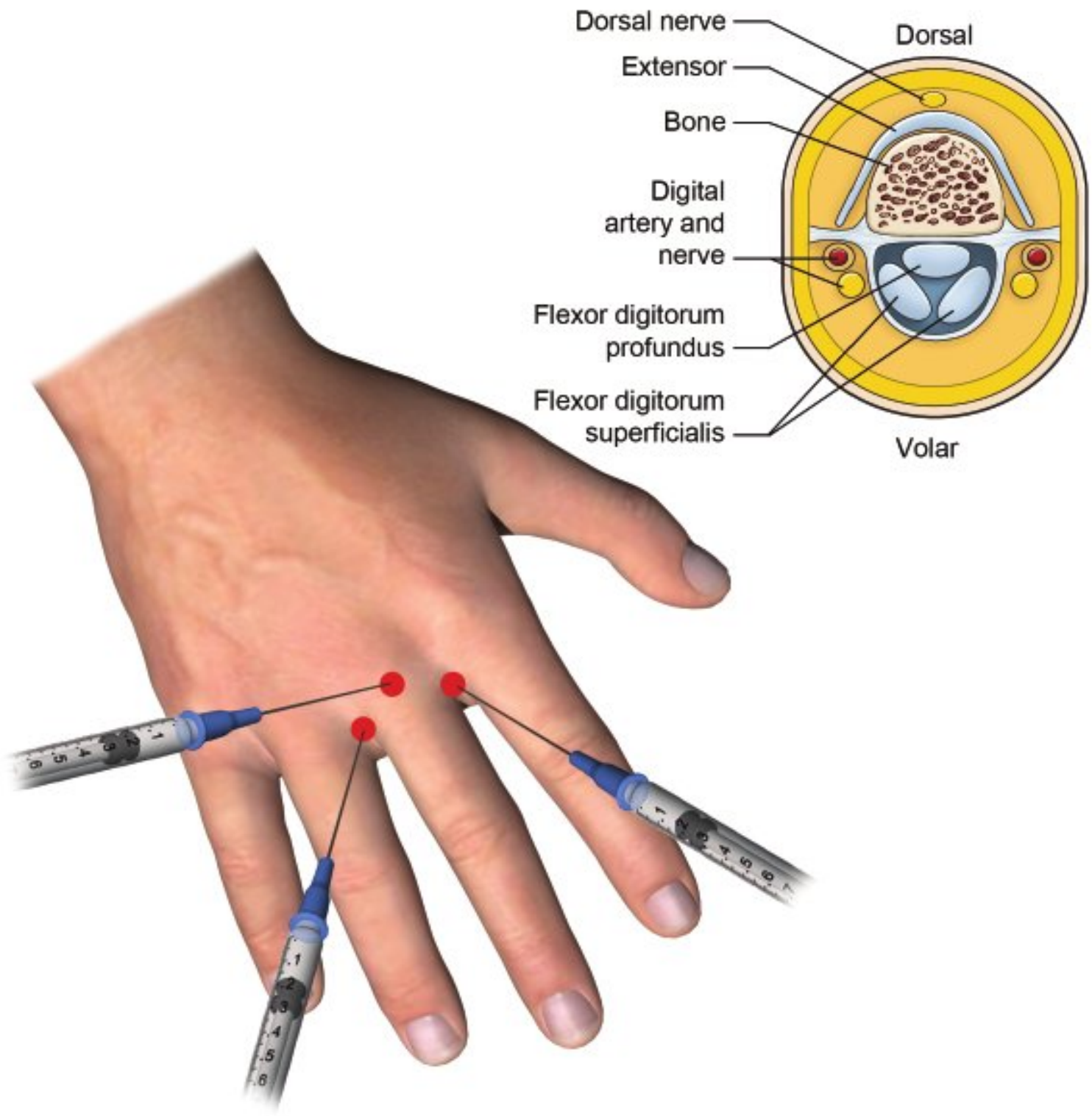


Fig. 13.1 Digital nerve block.

Median nerve. Inject 5 mL of agent between the palmaris longus and flexor carpi radialis tendons at the proximal wrist crease using a 25-gauge needle. **Avoid injection directly into the median nerve**. If the patient feels tingling during injection, withdraw the needle 1 to 2 mm and reinject.

Ulnar nerve. Inject 5 mL of agent radial to the flexor carpi ulnaris tendon at the wrist crease with the wrist in flexion. Take care not to inject into the ulnar artery. Remember to draw back on the syringe first.

Radial nerve—superficial branch. Inject 5 mL of agent from the midpoint of the dorsum of the wrist to the radial border of the anatomical snuffbox (**Fig. 13.2**). Draw back on the syringe so as not to inject into the radial artery.

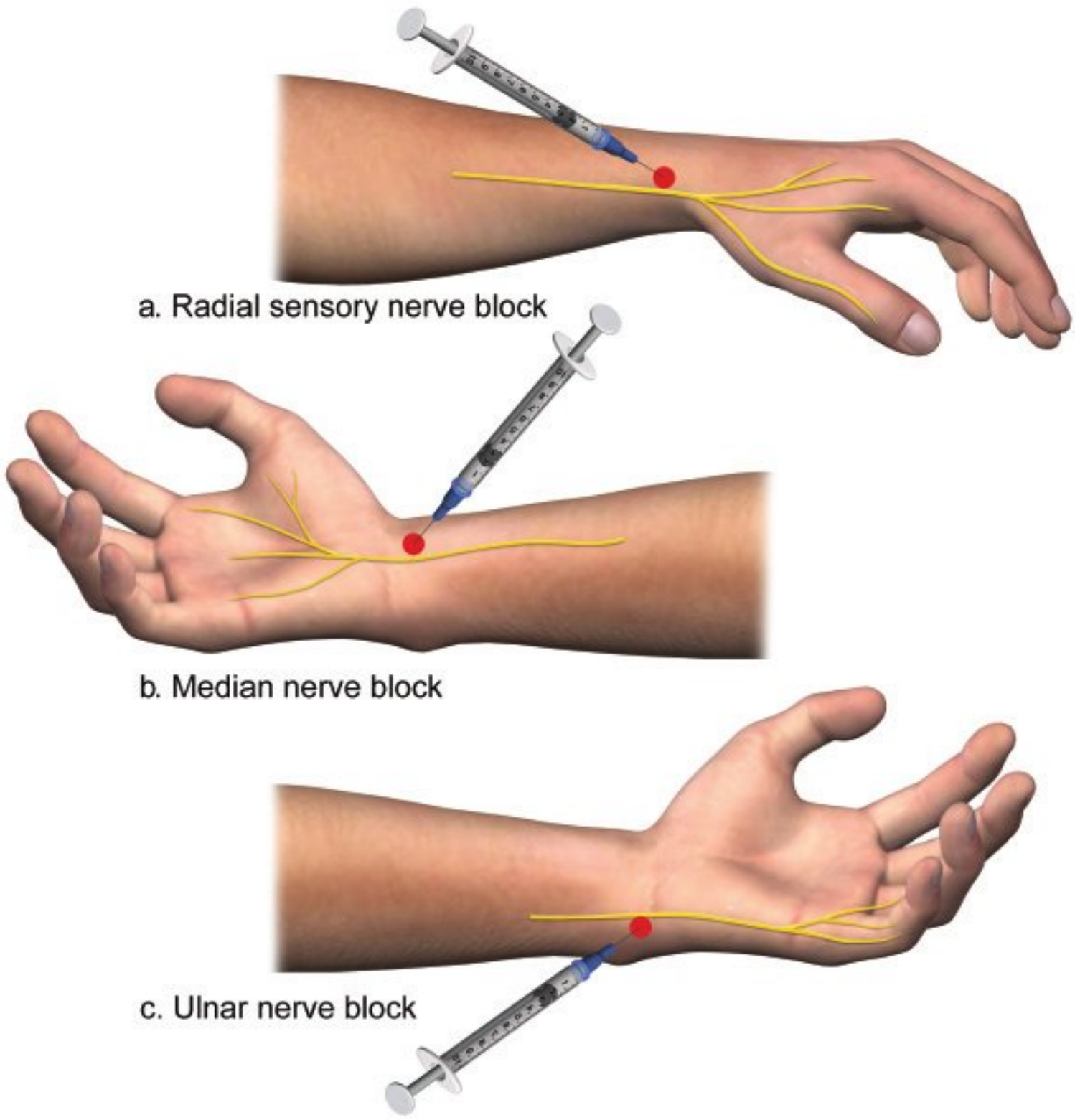


Fig. 13.2 Wrist blocks. **(a)** Radial sensory nerve block. **(b)** Median nerve block. **(c)** Ulnar nerve block.

Splinting

The proper splinting of fractures and dislocations is of paramount importance. The use of a splint in tendon, nerve, and artery repair protects against traction and disruption of the repair. Splints of the hand for infections and soft tissue trauma prevent dysfunctional bone and soft tissue contractures. The use of a splint also helps to decrease a patient's pain and discomfort.

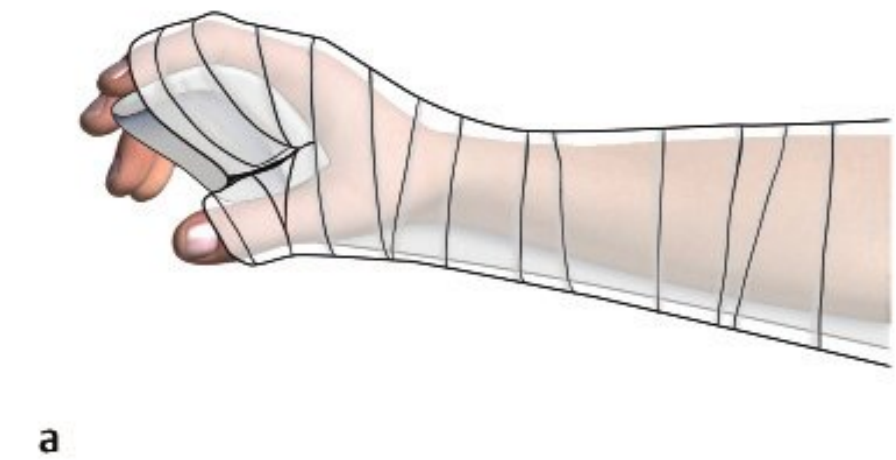
General Procedures (Fig. 13.3a)

- Use local and regional blocks to allow painless manipulation of the extremity while splinting.
- Splinting materials used.
  - Kerlix gauze.
  - 4-inch (10-cm) Webril roll.
  - 4-inch plaster material.
  - 4-inch Ace bandage.
  - 4-inch Coban (3M).
- After injury repair, clean and dry the extremity.
- Place a single layer of Webril (Kendall Company) around the hand and forearm loosely (**Fig. 13.3b**).
- Measure the length of the area to be splinted; cut a 10-ply piece of plaster material at that length. Alternatively, premade plaster/gauze of fiberglass composites (e.g., OCL [Orthopedic Casting Lab manufacturers] splint roll) can be cut to length and used (**Fig. 13.3c**).
- Place plaster in Webril wrap (**Fig. 13.3d**).
- Wet the plaster material and place it on the hand/forearm in the desired position (**Fig. 13.3e**).
- Hold the plaster material in position by wrapping with a single layer of Kerlix (**Fig. 13.3f**).
- Hold the hand in the desired position (**Fig. 13.3 g**).
- Apply either Ace or Coban in a single loose layer over the splint (**Fig. 13.3h**).
- Apply circumferential wraps *loosely* to avoid constriction.
- Elevate the extremity to prevent dependent edema while in the splint.
- Prescribe a short-term follow-up protocol, especially in outpatient treatment, to allow assessment for edema, digital perfusion, and splint displacement.

Splint Types

Volar Splint (**Fig. 13.3**)

- Most versatile splint for radial-sided injuries.
- Index and long finger fractures.
- Index and long finger infections.
- Wrist neurovascular injuries.
- Forearm infections.
- Metacarpal fractures.



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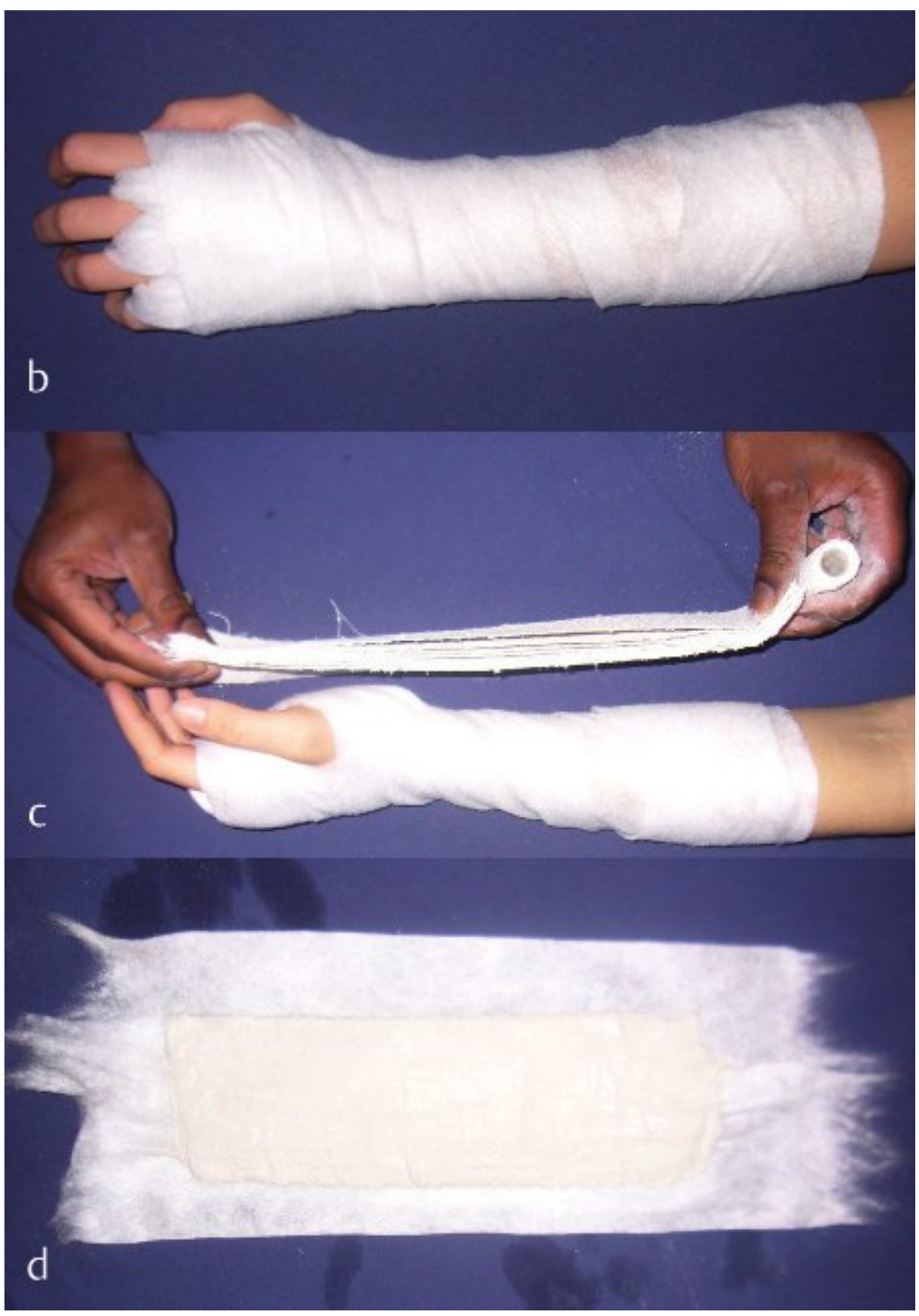


Fig. 13.3 **(a)** Volar splint. The gray shading denotes plaster position. **(b–g)** Placement of a plaster, step-by-step.

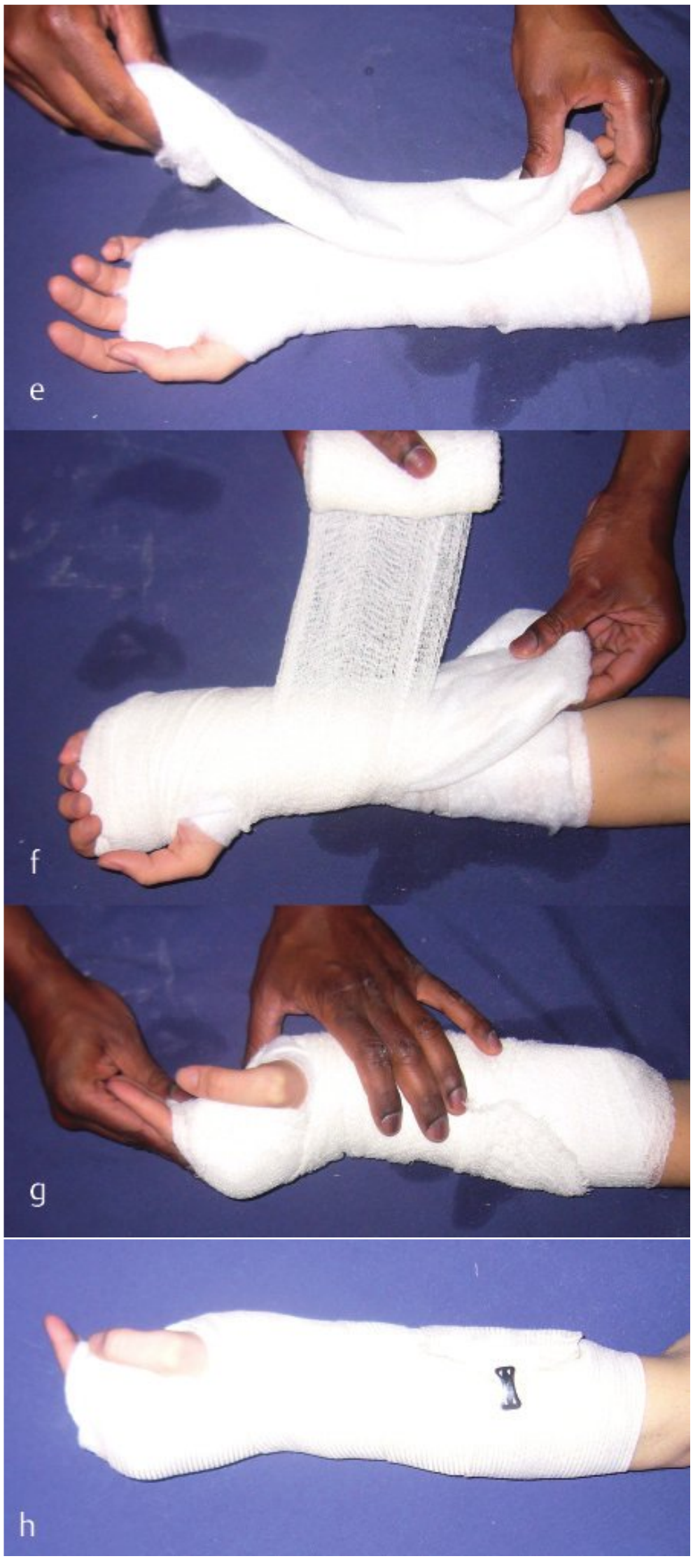


Fig. 13.3 (Continued) **(b–g)** Placement of a plaster, step-by-step. **(h)** Plaster in place.

Position Intrinsic Plus Position - “Safe Position”

- Proximal forearm to DIP joint.
- Apply on volar surface of the forearm/wrist and hand.
- Include index through small finger.
- Keep thumb free.
- Extend wrist 35 degrees.
- Flex MCP joint 90 degrees.
- Flex PIP/DIP joints 0 to 10 degrees.
- For extensor tendon injuries, place MCP joint in extension.

Ulnar Gutter Splint (**Fig. 13.4**)

- Ulnar-sided injuries.
- Ring and small finger fractures.
- Ring and small finger infections.
- Extensor tendon injuries.
- Ulnar-sided metacarpal fractures.

Position

- Proximal forearm to DIP joint.
- Apply on ulnar volar surface of the forearm/wrist and on the hand to the mid-dorsum.
- Include ring and small fingers.
- Keep thumb and index and long fingers free.
- Wrist extended 35 degrees.
- Flex MCP joint 90 degrees.
- Flex PIP/DIP joints 0 to 10 degrees.

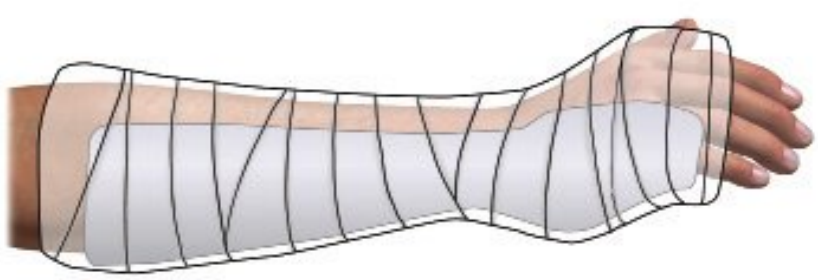


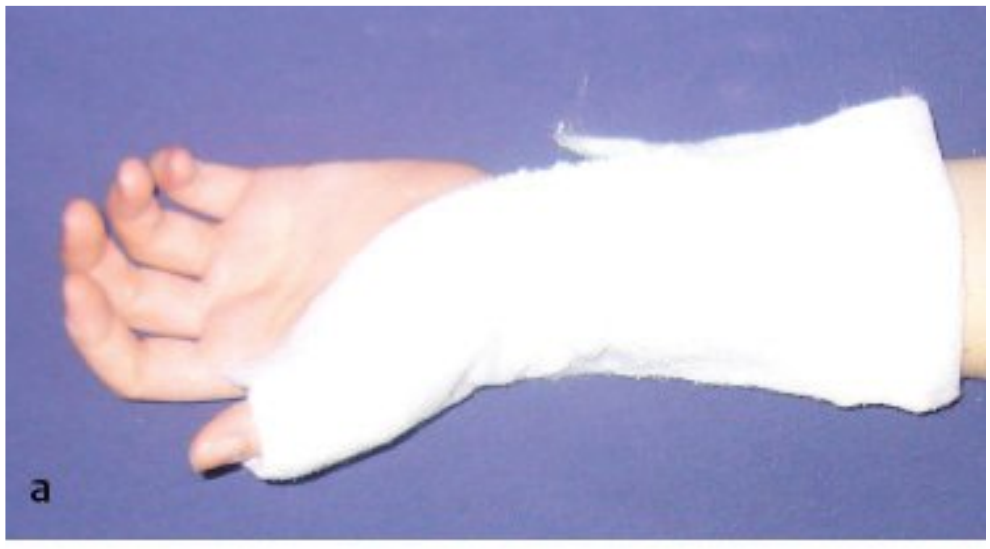
Fig. 13.4 Ulnar gutter splint. The gray shading denotes plaster position.

Thumb Spica Splint (**Fig. 13.5**)

- Most versatile splint for thumb injuries.
- Thumb fractures and dislocations.
- Thumb and thenar infections.
- Thumb tendon injuries.
- Scaphoid injuries.
- First metacarpal fractures.

Position

- Proximal forearm to IP joint.
- Apply two plaster splints.
  - One on volar surface of the forearm/wrist and thumb.
  - One radially to the mid-dorsum.
- Keep index through small finger free.
- Extend wrist 35 degrees.
- Flex MCP joint 10 to 15 degrees.
- Flex IP joint 0 to 10 degrees.



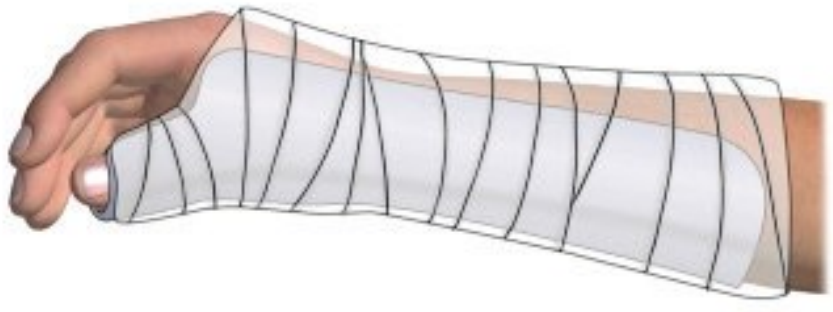


Fig. 13.5 (a,b) Thumb spica splint. The gray shading denotes plaster position.

Extension Block Splint (**Fig. 13.6**)

- Flexor tendon injuries.
- Proximal and middle phalangeal fractures.

Position

- Splint from proximal forearm to DIP joint.
- Apply on dorsal surface of the forearm/wrist and hand.
- Include index through small finger.
- Flex wrist 45 degrees.
- Flex MCP joint 90 degrees.
- Flex PIP joint 45 degrees.
- Flex DIP joint 20 degrees.
  - Splint from proximal forearm to DIP joint.
  - Apply on dorsal surface of forearm.

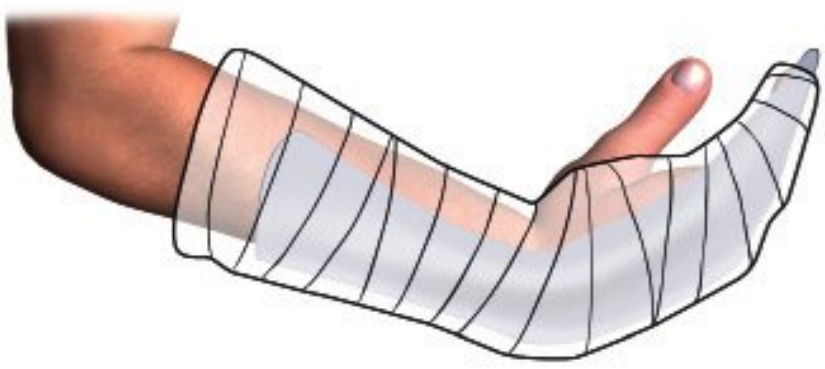


Fig. 13.6 Extension block splint. The gray shading denotes plaster position.

Proximal and middle phalangeal fractures

- Consider intrinsic plus position with plaster dorsal to block extension.
  - Include adjacent digits.
    - Wrist in neutral position or slight extension.
    - MCP joint in 70 to 90 degrees of flexion.
    - IP joint extended.

14 Hand and Wrist Fractures and Dislocations

Hand Fractures

Physical Examination

Patients with suspected hand fractures require a thorough physical examination (Chapter 12).

- Perform a complete physical examination to determine the integrity of the nerves, the arteries (perfusion), the tendons, joint mobility, and the soft tissues.
- Radiographs are paramount for establishing and confirming the correct diagnosis.
  - Plain radiographs—a hand series should include AP, true lateral, and oblique views.
  - In selected cases of carpal fractures and more complex wrist injuries, a CT scan may be indicated.
  - Consider radiographs of the joint proximal to the injury so as not to miss associated injuries.

Fracture Classification

- Open versus closed.
- Displaced versus nondisplaced.
- Transverse versus oblique versus spiral versus comminuted or avulsion.
- Traumatic versus pathologic.
- Adult versus pediatric
  - In pediatric patients—greenstick versus epiphyseal plate.
  - Epiphyseal plate fractures—Salter-Harris classification (**Fig. 14.1**).

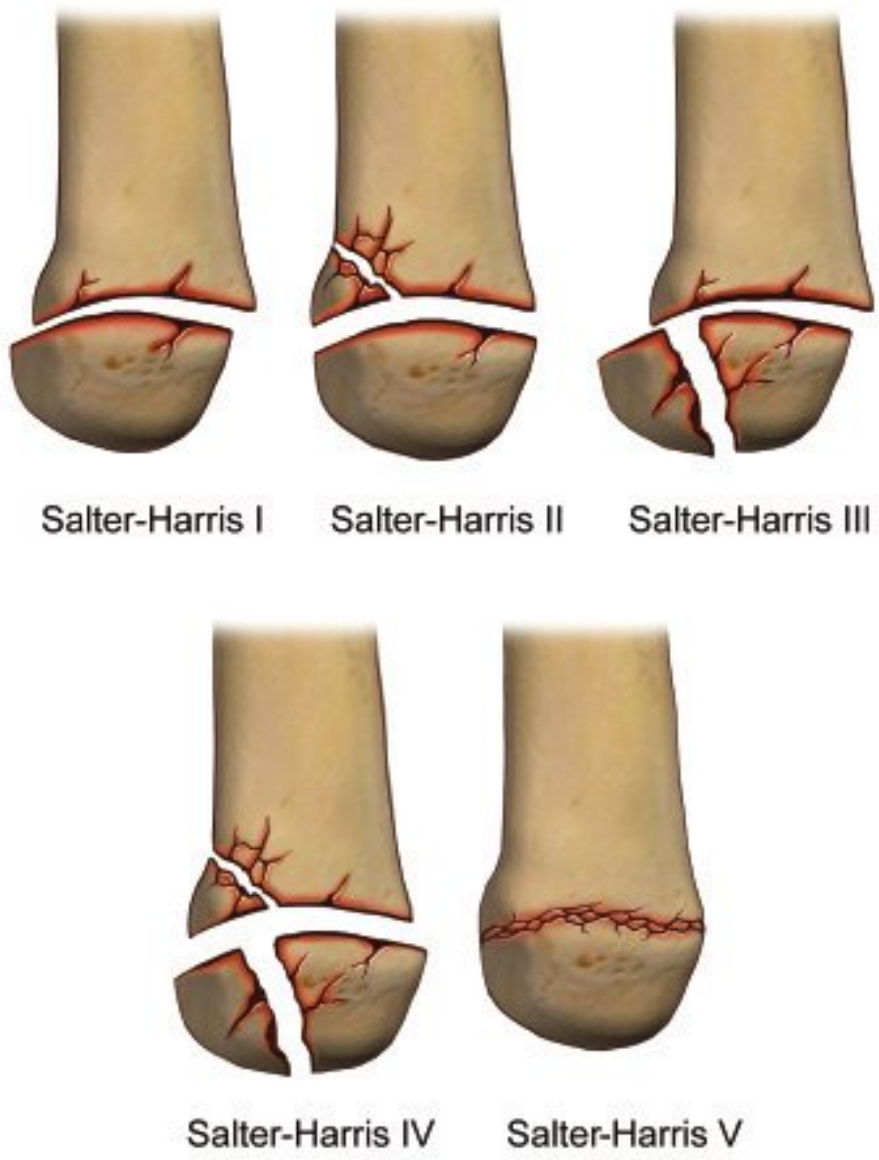


Fig. 14.1 Salter-Harris classification of epiphyseal fractures.

Fracture Treatment

In general, hand fractures can be treated in the emergency room with closed reduction and splinting. However, if the fracture is open, displaced, or unstable, or if the angulation is not acceptable, then operative treatment may become necessary.

Open Fractures

- Perform a digit or wrist block.
- Culture and irrigate open fractures profusely.
- Administer IV antibiotics (ER treatment or inpatient).

  - Ampicillin 500 g IV every 8 hours + gentamicin 3 to 5 mg/kg daily divided every 8 hours (check peak and trough serologic levels).
  - Vancomycin 1 g IV every 12 hours + ceftriaxone 1 to 2 g IV every 24 hours.
  - Outpatient prophylactic antibiotics for patients with a plan for later surgery includes Bactrim DS by mouth twice a day.
- Irrigate the wound and splint the patient in preparation for operative reduction.

Phalangeal and Metacarpal Fractures

Indications for Operative Treatment

- Intra-articular fractures.
- Irreducible fractures.
- Malrotation.
- Subcapital phalangeal fractures.
- Open fractures if displaced or angled.
- Bone loss.
- Multiple fractures.
- Fractures with soft tissue injury.

Phalangeal Fractures

Distal Phalanx Fractures

Distal phalanx fractures are the most common fractures in the hand. The thumb and middle finger are most likely involved. Patients present with tuft fractures, shaft fractures, and intra-articular injuries due to crush.

Tuft Fractures

Open Fractures

- Perform finger or wrist block.
- Remove nail.
- Irrigate.
- Repair nail bed with 6–0 to 7–0 chromic suture and stent the nail matrix (see Chapter 17, **Fig. 17.2**).
- Immobilize DIP joint in extension with tongue blade or aluminum splint for 3 to 4 weeks with PIP free.

- In cases of severe comminution, soft tissue repair is adequate for splinting fractures.
- Treat with Bactrim DS by mouth twice a day ×5 days.

Closed Fractures

- Perform finger or wrist block.
- If a hematoma is present under the nail, drain it using a puncture technique.
  - Sterile 18-gauge needle tip, heated paper clip, or electrocautery.
  - If the hematoma > 50% of nail bed, likely nail bed injury
    - Remove and repair nail bed and splint with piece of foil from chromic package or use the nail itself (see Chapter 17, **Fig. 17.2**).
      - Splint finger for 2 weeks.
      - Treat with outpatient antibiotics ×5 days.

Shaft Fractures - Distal Phalanx

- Nondisplaced.
  - Repair soft tissue.
  - Splint for 3 weeks.
  - Bactrim DS by mouth twice a day.
- Displaced.
  - Likely nail bed laceration.
  - Repair nail matrix (see Chapter 17, **Fig. 17.2**).
  - Stabilize fracture with K-wire or 18-gauge needle.
  - Splint finger with PIP free for 3 weeks.
  - Outpatient antibiotics ×5 days.

Intra-articular Fractures - DIP

- Open fracture.
  - Repair nail bed.
  - Splint DIP joint in extension for 6 to 8 weeks.
  - Outpatient antibiotics.
- Closed fracture.
  - Splint DIP joint in extension.

Dorsal Base

An intra-articular fracture of the dorsal base (mallet fracture) is a hyperflexion injury in which a portion of the dorsal bone breaks off with extensor mechanism. It causes extensor lag with a mallet finger deformity. Treatment requires strict patient compliance. In the pediatric population this may require a K-wire through the DIP joint.

- Treat with splint in extension for 6 to 8 weeks.

Volar Base (FDP Avulsion)

An intra-articular fracture of the volar base is a hyperextension injury in which the flexor digitorum profundus (FDP) pulls off the distal phalanx.

- Treat with ORIF because FDP may retract into palm.
- Splint hand in emergency room with tongue blade or aluminum splint.
- If open, wash out, repair nail bed, start antibiotics, and splint.

Middle and Proximal Phalanx Fractures

Middle and proximal phalanx fractures are caused by crushing forces rather than direct blow, twisting, or angular forces. If these fractures are nondisplaced or stable, simply buddy tape or splint with IP extended for 3 to 4 weeks. A comminuted, displaced fracture of the middle or proximal phalanx to the articular surface is called a pilon fracture.

Articular Fractures - PIP

- In ER setting.
  - Fracture of single digit—ensure involved joint is in extension.
    - Aluminum or tongue blade splint.
  - Multiple fractures—splint hand in intrinsic plus.
  - Follow up in clinic for operative management.
- Nondisplaced—inherently unstable.
  - Operative management using either closed or open reduction or fixation by multiple K-wires or screws or a combination.
  - If nonoperative management chosen, then close follow-up required.
- Displaced.
  - Dorsal base fractures of middle phalanx.
    - ORIF to avoid boutonnière deformity.

  - Dorsal base fractures of proximal phalanx.
    - Require ORIF.
- Unicondylar (displaced).
  - Inherently unstable—either closed or open reduction and fixation with multiple K-wires or screws.
  - Extension splint 2 to 3 weeks.
- Bicondylar.
  - Requires ORIF.
  - Noncomminuted.
    - Fix condyle to condyle first, then to the shaft with K-wires or screws.
  - Comminuted.
    - Difficult to treat.
    - DIP joint.
      - Minimal displacement—closed reduction.
        * Splint 2 weeks in extension.
        * Physical therapy in 2 weeks.
      - Displaced.
        * ORIF with K-wire/screw fixation.
        * Early motion at 2 weeks.
    - PIP joint.
      - Skeletal traction of the middle phalanx for 3 to 4 weeks with forearm splint.
      - Active flexion of PIP joint immediately.

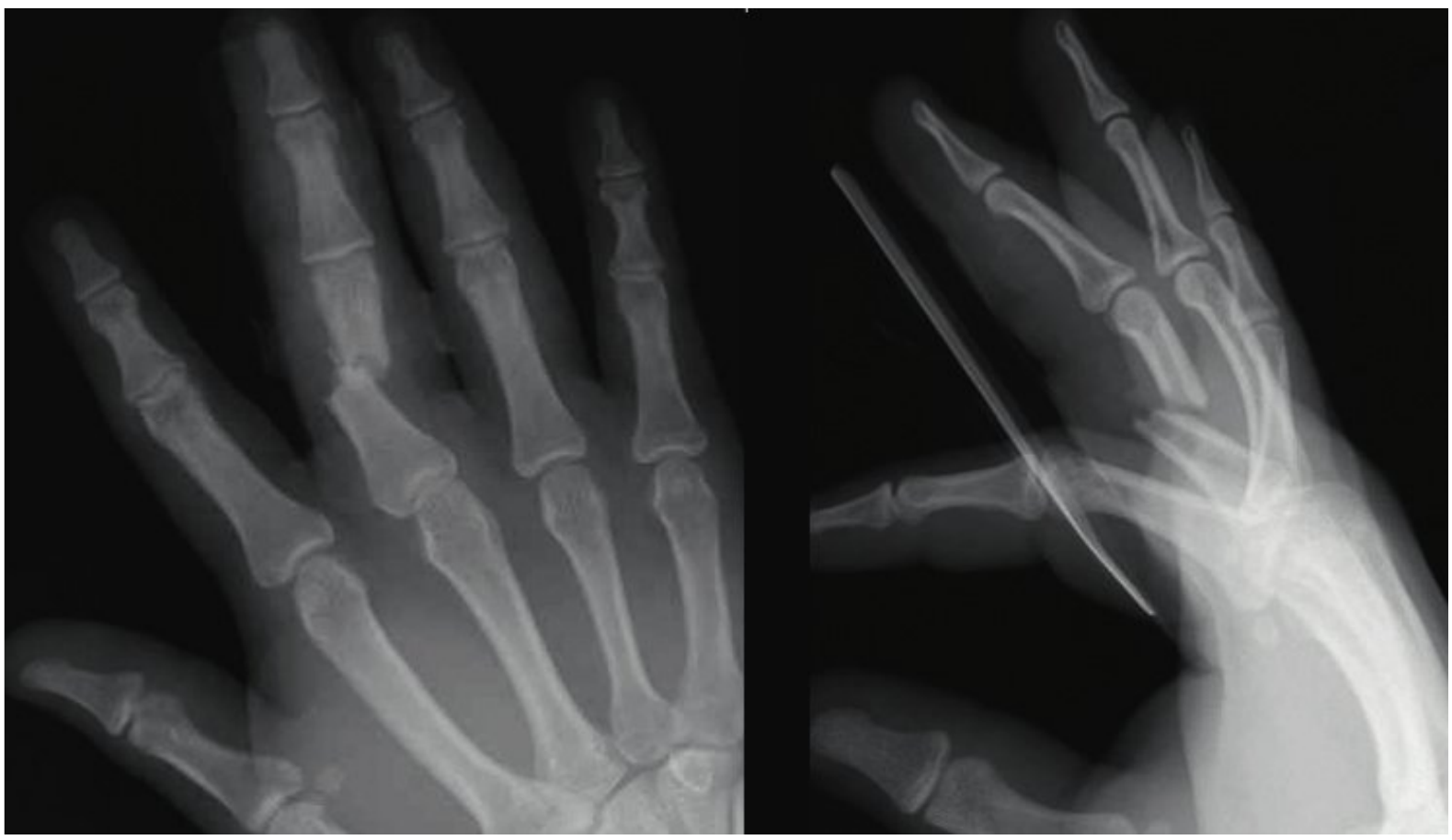


Fig. 14.2 Transverse fracture of the proximal phalanx.

Nonarticular Fractures

- Shaft.
  - **Nondisplaced and stable—not** rotated, angulated, or comminuted.
    - Splint the finger in extension with an aluminum splint.
      - Must cover proximal and distal joint.
      - Duration of 1 week.
      - Once pain and swelling resolve, buddy tape to adjacent finger and begin range of motion.
  - Displaced but amenable to stable closed reduction.
    - Usually **transverse** fractures (**Fig. 14.2**), not oblique or spiral.
    - Attempt reduction and stabilization.
      - Perform digit block (See Chapter 13, **Fig. 13.1**).
      - Flex MCP joint maximally.
      - Flex distal fragment to correct volar angulation.
      - Dorsal splint in intrinsic plus position.
        - Plaster should be placed dorsally for extension blocking—MCP 90 degrees, IP extended, include adjacent digits in splint for stabilization.
      - Splint for 3 weeks, then buddy tape for additional 2 weeks.
  - Unstable—if potential for rotation or angulation exists.
    - Open, oblique, spiral, comminuted fractures.
    - Radiographically angulated.
    - Assess by having patient flex finger.
      - Fingers overlap.

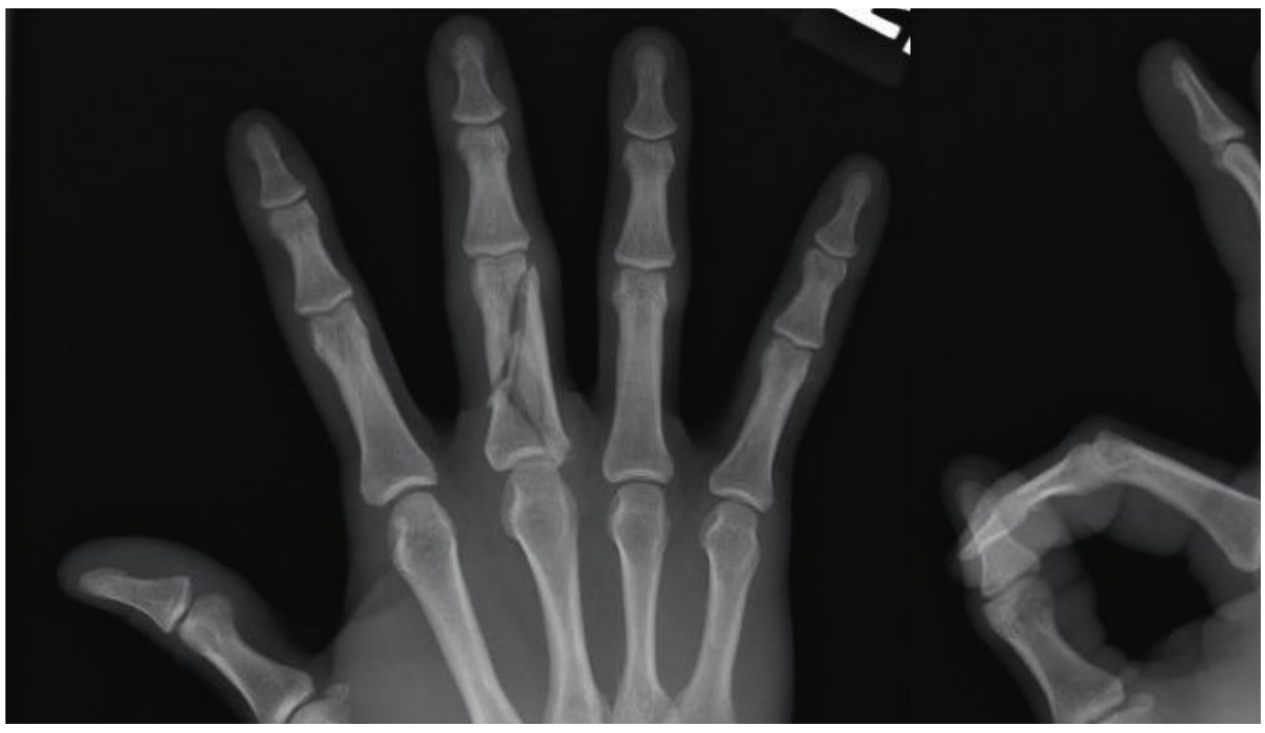


Fig. 14.3 Comminuted fracture of the proximal phalanx.

    - Plan closed reduction with percutaneous pinning within 3 to 4 days.
    - Use 0.035- to 0.045-inch K-wire.
  - Unstable transverse fractures.
    - Intramedullary longitudinal fixation through metacarpal head with K-wire.
    - Extension block splint in intrinsic plus position with IP joints free for 3 to 4 weeks.
  - Comminuted fractures (**Fig. 14.3**).
    - Require operative management.
    - Attempt closed K-wire fixation.
    - External fixation device often indicated for complex comminution and shortening.
      * Preserves length.
      * Assists with management of soft tissue injuries.
  - For unsuccessful percutaneous pinning, perform ORIF with plates or interosseous wiring.

Base Fractures of Proximal Phalanx

- Extra-articular.
  - Angulation of 25 degrees in adults and 30 degrees in children requires treatment.
  - To reduce.
    - Flex MCP maximally.
    - Flex distal fragment to correct volar angulation.
  - Splint in intrinsic plus (dorsal plaster) for 3 weeks.
  - Failed closed reduction.
    - K-wire fixation.

Metacarpal Fractures

Head Fractures

- Open fractures secondary to closed-fist injury, or fight bite (also see Chapter 15, **Fig. 15.9**).
  - Wrist or local block.
  - High-pressure irrigation and débridement.
  - Leave wound open.

  - Delay fixation until signs that inflammation or infection has subsided.
  - Splint in intrinsic plus volar splint (see Chapter 13, **Fig. 13.3**).
  - Augmentin 875 mg by mouth twice a day × 10 days or Bactrim DS by mouth twice a day.
  - Short-term follow-up.
- Index finger most commonly involved due to axial loading, and often intra-articular.
- AP, lateral, and oblique X-rays; if not clear, then Brewerton view.
- Nondisplaced—splint in volar splint for 4 weeks (see Chapter 13, **Fig. 13.3**).
  - If > 25% of articular surface or > 1 mm step-off, splint in safe position, plan ORIF.
  - Miniplate fixation preferred, to allow early mobilization (**Fig. 13.3**).
- If comminuted, perform wrist block and wash out wounds.
  - Splint acutely in safe position.
  - Plan for immobilization for 2 weeks with skeletal traction, external fixation, or arthroplasty.

Neck Fractures

- Indications for reduction.
  - Pseudoclawing (clawing of fingers with ulnar nerve intact).
  - MCP hyperextension/PIP flexion.
  - Rotational deformity.
  - Scissoring of fingers.
  - Unacceptable angulation.

Apex dorsal angulation occurs from intrinsic muscle contraction.

Treatment is based on angulation:

- Small digit (boxer's fracture)—50 degrees angulation acceptable.
- Ring finger—30 to 40 degrees angulation acceptable.
- Middle and index finger—10 to 15 degrees acceptable.

If angulation is unacceptable, and pseudoclawing or rotation deformity is present:

- In a fresh fracture, attempt closed reduction.
- Fracture > 7 days old may require operative reduction.
- Closed reduction by Jahss maneuver (**Fig. 14.4**)—first perform a wrist block (wrist block for boxer's fracture).
- Next, flex MCP joint to 90 degrees and PIP joint to 90 degrees.

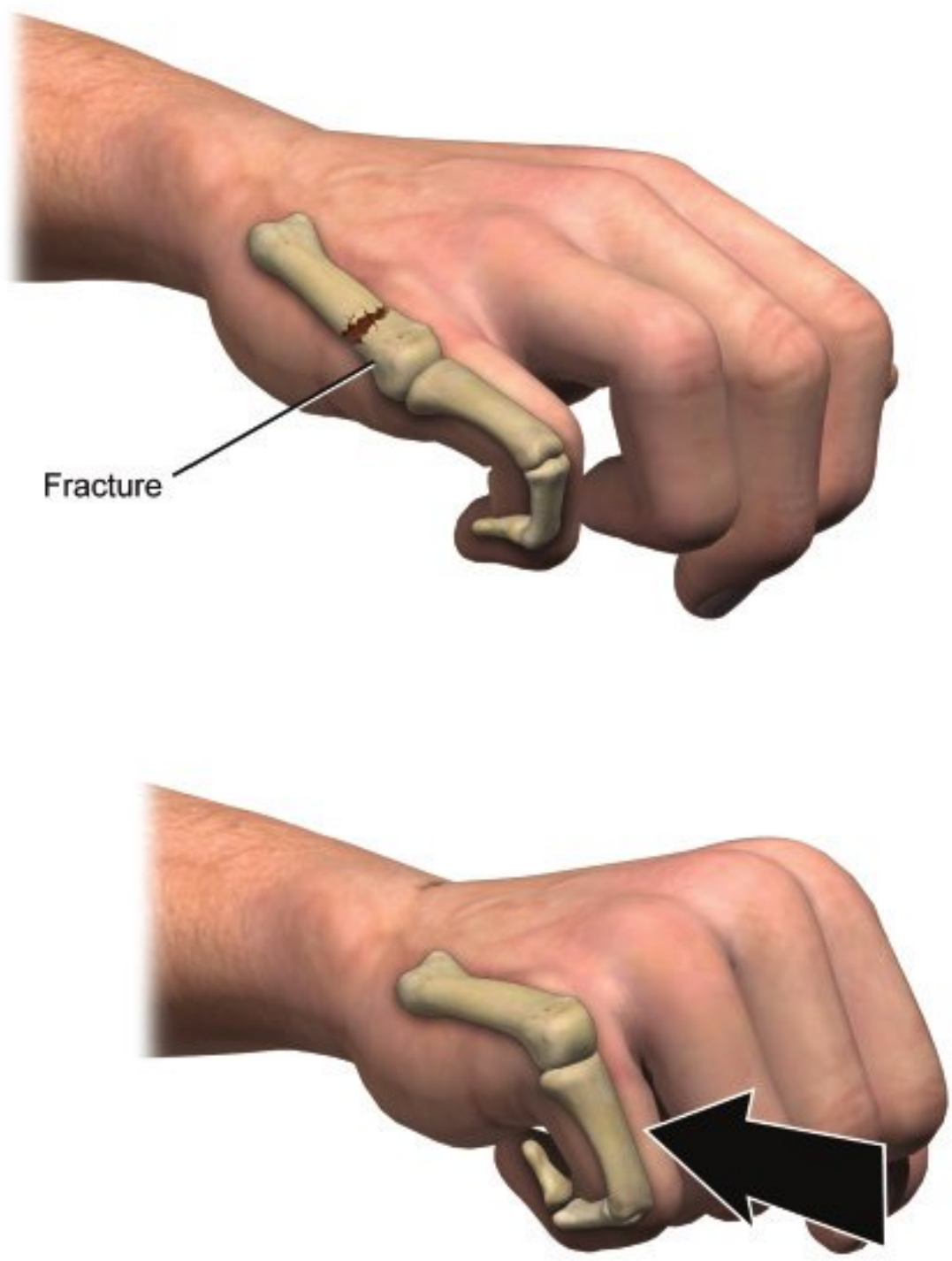


Fig. 14.4 Jahss maneuver for reduction of metacarpal fractures.

- Apply upward pressure on the proximal phalanx while pressing down on the metacarpal shaft.
  - If reduced, splint in safe position for 3 to 4 weeks and monitor reduction regularly (**Fig. 13.3**) for second and third metacarpal.
  - For fourth and fifth metacarpal fracture, use ulnar gutter splint (see Chapter 13, **Fig. 13.4**).
  - If not reducible, use internal fixation with K-wires, plates, or dorsal tension band wires.
    - Splint in a safe position acutely.
  - After reduction, take radiograph to confirm reduction.
  - Also consider splinting with MCP joints extended
  - PIP joints are left free of immobilization

Shaft

The types are:

- *Spiral:* Torsional forces with 5 degrees of malrotation causing a 1.5-cm digital overlap.
- *Oblique:* Lateral bending forces with axial load.
- *Transverse:* Lateral bending force versus axial load.
- *Comminuted:* Direct impact on the metacarpal, which may cause shortening.

If angulation is acceptable (see Neck Fractures above), then close reduce the fracture using traction and a wrist block.

- Flex MCP joint.
- Press on the fracture apex dorsally with a palmar-directed force.
- Place in volar intrinsic plus splint.

If there are multiple fractures (**Fig. 14.5**), the fracture is unstable/open, or there is scissoring and severe angulation, then perform ORIF with K-wires, interosseous wires, plates, lag screws, or external fixation. In the emergency room, place the patient in a safe-position splint.

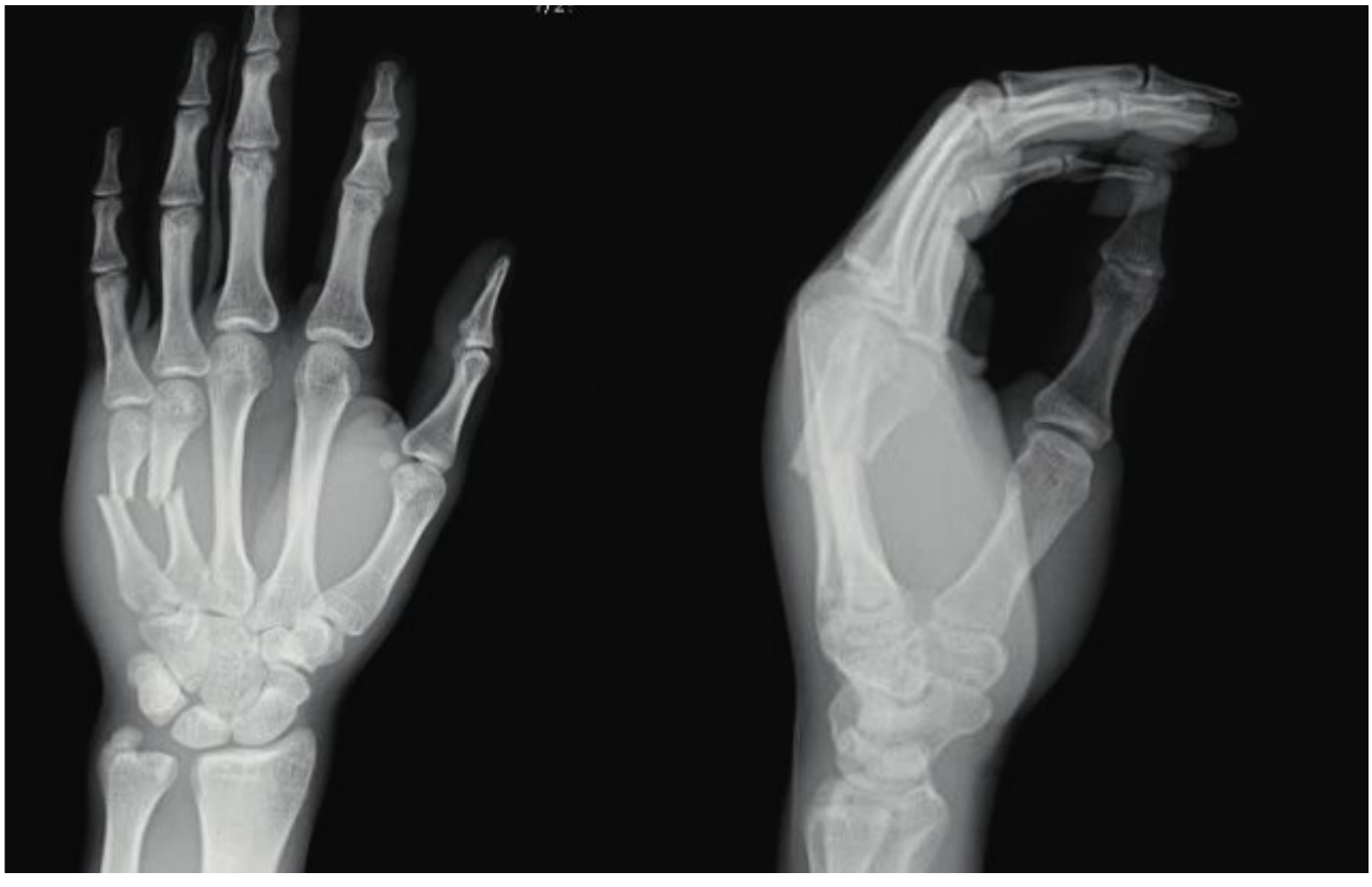


Fig. 14.5 Fractures of multiple metacarpals.

Base Fractures/CMC Joint Fracture-Dislocation

Base fractures and CMC joint fracture-dislocation are inherently unstable fractures caused by axial load versus direct blow. Index fingers and middle fingers are less likely to undergo this type of fracture because these joints are less mobile. A hamate–fifth metacarpal intra-articular fracture is a **baby (reverse) Bennett fracture**.

- Because the extensor carpi ulnaris pulls on the fifth metacarpal, in the X-rays you will see that the ulnar portion subluxates proximally and dorsally.
- Closed reduction with K-wires versus ORIF. In the emergency room, place the patient in a volar splint in safe position (see Chapter 13, **Fig. 13.3**).

Thumb Fractures

Injuries occur from direct trauma and angular or rotary forces.

Thumb Phalangeal Fractures

Extra-articular:

- Proximal phalanx fracture with > 20 to 30 degrees apex volar angulation is unacceptable and requires reduction.
- Comminuted fractures require reduction and either open or closed fixation (K-wire) (**Fig. 14.6**).
  - Severe comminution may require external fixation to preserve length.
- In the emergency room, splint in a thumb spica splint and plan operative treatment (**Fig. 13.5**).
- Distal tuft—associated with subungual hematoma, nail bed injuries, and comminuted fractures.
  - Finger block.
  - Remove nail.
  - Irrigate thoroughly.
  - Repair nail bed.
  - Splint 3 to 4 weeks in extension with tongue blade or aluminum splint.
- Transverse shaft.
  - Finger or wrist block.
  - Close reduce.
  - Splint in extension; if unstable, then ORIF.

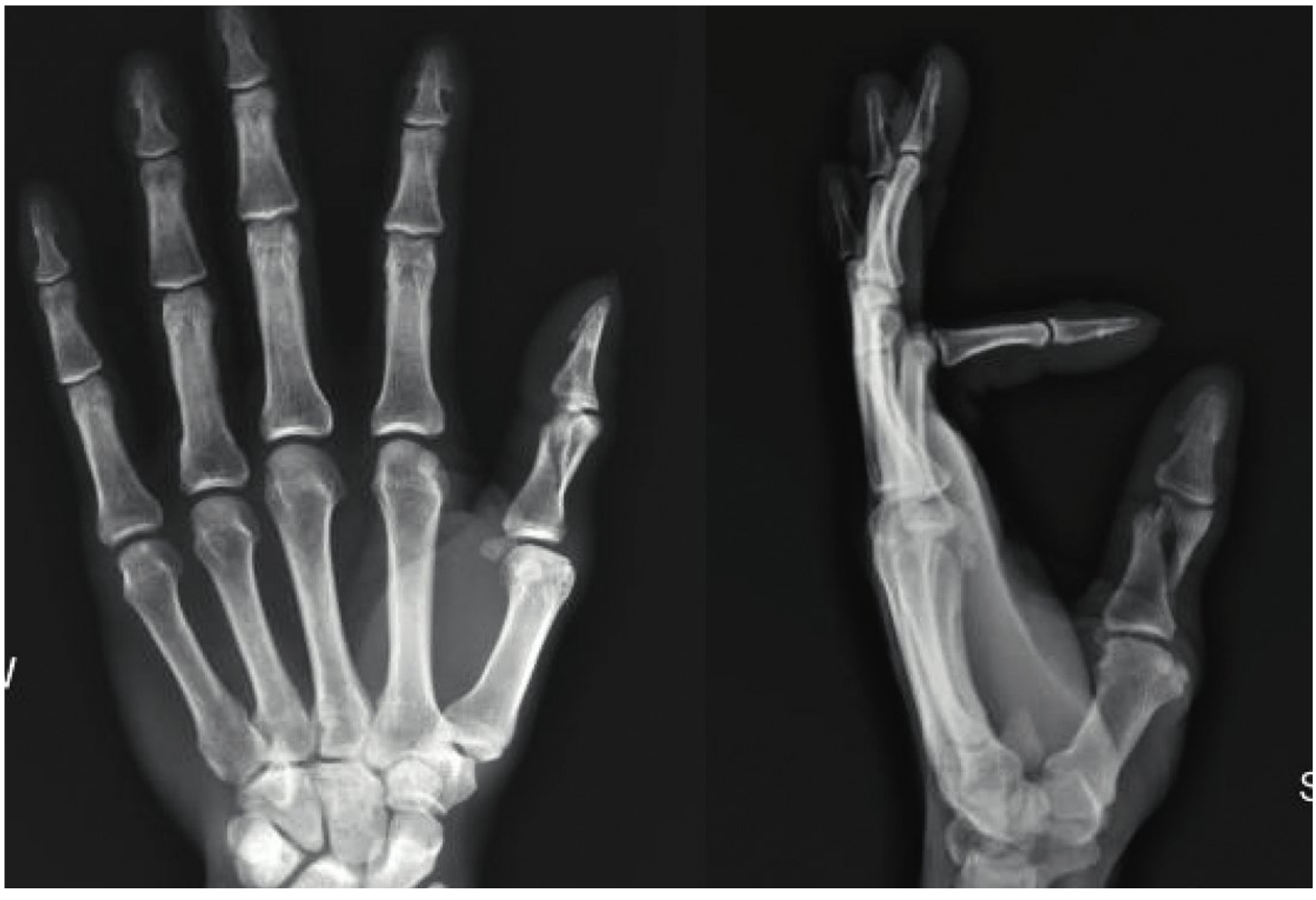


Fig. 14.6 Fracture of the thumb proximal phalanx that requires fixation.

Intra-articular—(occurs when axial load is placed on partially flexed thumb):

- Dorsal base avulsion = mallet thumb.
  - Treat with 6 to 8 weeks of extension splint.
  - Consider ORIF if subluxation present.
- Volar base fracture.
  - Consider avulsion of flexor pollicis longus.
- Fractures of the ulnar base represent avulsion of the ulnar collateral ligament and are also called **skier's thumb** or **gamekeeper's thumb**.
- If the fragment is displaced > 2 mm or > 25% of articular surface, then K-wire fixation versus ORIF.
- In the emergency room, place in a thumb spica splint (see Chapter 13, **Fig. 13.5**).

Thumb Metacarpal Fractures

Head and shaft fractures result from torsional, direct impact, angulatory, or rotary forces.

Extra-articular:

- Fractures up to 30 degrees angulation are acceptable due to compensation by CMC mobility.
- **Head**—rare fracture that requires reduction and K-wire fixation versus ORIF if displaced. Can attempt closed reduction by Jahss maneuver (**Fig. 14.4**).

- **Shaft**—after radial and median nerve block at the wrist, close reduce and splint in thumb spica splint (**Fig. 13.5**).

Intra-articular:

- **Bennett fracture**—occurs when partially flexed thumb is axially loaded.
  - It is defined as intra-articular fracture-subluxation of the base of the first metacarpal. On X-rays, the volar ulnar aspect of the metacarpal base remains stable due to the anterior oblique ligament. However, the rest of the metacarpal moves dorsally, proximally, and radially due to the pull from the abductor pollicis longus.
  - If the bone fragment is > 20% of CMC joint surface, then reduce closed and stabilize with a K-wire.
  - If the fracture cannot be reduced in a closed fashion, then ORIF.
  - In the emergency room, splint in a thumb spica splint and plan operative treatment (**Fig. 13.5**).
- **Rolando fracture**—any comminuted intra-articular fracture of the base of the first metacarpal, but traditionally referred to as **Y**- or **T**-shaped intra-articular fractures.
  - If severely comminuted, use skeletal traction and perform percutaneous fixation.
  - If fracture contains large fragments, only ORIF.
  - In emergency room, place in thumb spica splint (**Fig. 13.5**) and plan operative treatment.

Pediatric Phalangeal and Metacarpal Fractures

Children rarely present with fractures of the hand. When they do, the chance of displacement is less than in adults. This is due to the malleability of the child's bones, as well as the tougher periosteum. Fractures are classified into either nonepiphyseal fractures (66%) or fractures that involve the epiphysis. Epiphysis fractures are categorized by Salter-Harris classification (**Fig. 14.1**). Fractures in children heal twice as fast as those in adults, and the epiphyseal plate compensates for angular deformity of the fractures. However, accurate reduction is crucial in intra-articular fractures.

Extra-articular Fractures

Fractures commonly occur in the middle and proximal phalanx in children.

- If the fracture is not displaced (type I), splint in a safe position.
- If decreased (type II) or no (type III) bone contact between the fragment and remainder of the bone, then use K-wire fixation.

- In the emergency room, splint the hand in a safe position (see Chapter 13, **Fig. 13.2**).
  - In infants, consider including the elbow in flexion along with dorsally placed plaster in the splint.

Intra-articular Fractures

- In cases of displaced fractures, ORIF with miniaturized wires (0.028–0.039 inches) and screws—especially in children > 2 years old.

Epiphyseal Fractures (Fig. 14.1)

Salter-Harris I. This is a fracture through the epiphyseal plate with separation of epiphysis from the metaphysis (shear injury). This usually occurs in early childhood when the plate is thick with a zone of hypertrophying chondrocytes and sparse calcification. The prognosis is good with a wrist block, reduction, and splinting the hand in a safe position (see Chapter 13, **Fig. 13.2**). May require dorsal plaster in young children.

Salter-Harris II. This fracture involves a metaphyseal fragment associated with an epiphyseal fracture. This is the most common Salter-Harris fracture. The prognosis is good with adequate reduction and splinting.

Salter-Harris III. This fracture occurs in children > 10 years of age due to avulsion force. It is an intra-articular fracture through the epiphysis and the epiphyseal plate. Unless accurate reduction is performed, the prognosis is poor.

Salter-Harris IV. This is a rare fracture that occurs at any age. The fracture extends from the articular surface through the epiphysis and through the epiphyseal plate, and it also involves a portion of the metaphysis. It has a poor prognosis unless an accurate reduction is performed.

Salter-Harris V. This is an extremely rare fracture that occurs at any age. It is caused by crushing of the epiphyseal plate by axial load. It has a poor prognosis due to growth arrest.

Dislocations

Phalanx and Metacarpal Dislocations

Proximal Interphalangeal Joint

The PIP joint is stabilized by a combination of thick collateral ligaments, accessory collateral ligaments, and the volar plate. This is a hinge joint with an arc of rotation of 100 to 110 degrees and is the most common

site of ligamentous injury. The direction of dislocation is dependent on the position of the middle phalanx at the moment of joint dislocation.

Types of Dislocation

- **Volar:** Rare, caused by rotary longitudinal compression force on a semiflexed middle phalanx.
- **Dorsal**: Result of longitudinal compression and hyperextension.

Stability

- **Active:** Ask patient to move through full ROM. If subluxation occurs, the patient has severe ligamentous injury. If the patient has full ROM without subluxation, then adequate stability exists.
- **Passive:** Hold finger in full extension and then at 30 degrees of flexion. Test lateral stress on the collateral ligaments. Compare stability to unaffected PIP joint.

Grades

- *Mild:* Joint stable with microscopic tears.
- *Moderate:* Joint with abnormal laxity with moderate degree of tear.
- *Complete:* Collateral ligaments are completely torn.

Treatment

Sprains:

- Splint joint in extension for 2 to 3 days (with aluminum splint).
- If it remains stable, start early motion.

Dislocations:

- **Volar:** Examine finger for the integrity of the central slip. After giving finger block, then reduce using traction and splint 2 to 3 days in extension (aluminum splint or tongue blade). If stable, start early motion.
- **Dorsal:** If dislocation is stable after reduction, then splint in extension for 3 weeks. If dislocation is unstable after reduction, then surgery is required. These dislocations often involve > 40% of the volar articular surface. Plan ORIF or volar plate arthroplasty.
- **Lateral:** Use a combination of buddy taping and extension splinting for 3 weeks. In nearly all cases, the ligaments return to their normal position even though they may have been completely disrupted.

Distal Interphalangeal Joint and Thumb Interphalangeal Joint

Because both flexors and extensors insert on the distal phalanx and help stabilize the joint, distal interphalangeal joint and thumb interphalangeal joint dislocation is rare. Dorsal and lateral dislocations with open wounds are most common.

- Perform a digital block (see Chapter 13, **Fig. 13.1**).
- Although these dislocations are rarely reducible, try to reduce it by using longitudinal traction and direct pressure on the dorsum of the distal phalanx; manipulate the distal phalanx into flexion.
- Immobilize in a dorsal splint for 2 to 3 days with PIP joint free.
- Then buddy tape and start conservative active motion.

Finger Metacarpophalangeal Joint

The condyloid joint is usually dislocated in a dorsal or ulnar direction.

Dorsal dislocation most commonly occurs in the index or small finger, caused by forced hyperextension. In a simple subluxation the volar plate usually stays with the proximal phalanx.

Treatment

Flex the wrist to relax flexor tendons. Then flex the MCP joint by applying distal and volarly directed pressure to the proximal phalanx. Do not apply traction or hyperextend because this will convert the injury to a complex dislocation.

- Complex dislocation: Volar plate is usually jammed into the joint; therefore, flexion and reduction are impossible.
  - ORIF and immobilize for 2 weeks.
- Lateral: Radial collateral ligament is ruptured by the forced ulnar deviation while the MCP joint is flexed.
  - Reduce and immobilize in 30 degrees of flexion for 3 weeks.
  - Buddy tape with motion for 2 to 3 weeks.
- Volar: This is extremely rare.
  - Attempt closed reduction.
  - If reduction not stable, then ORIF.

Thumb Metacarpophalangeal Joint

Gamekeeper's Thumb

Gamekeeper's thumb is the most common thumb MCP joint injury, which occurs when the partially flexed thumb is axially loaded. This is defined as an avulsion fracture from the ulnar base of the proximal phalanx due to disruption of the ulnar collateral ligament.

Treatment

Immobilize the joint for 6 weeks in a thumb spica splint. Surgical exploration is indicated if the joint continues to be unstable or if the ulnar collateral ligament is blocked by interposition of the adductor pollicis muscle (Stener lesion).

Wrist Injuries

The wrist is an anatomically complex structure that plays a vital role in all aspects of human life. The distal radioulnar (DRU) joint is a site at which hand supination and pronation occur as the radius rotates around the ulna. Distal to the DRU joint is the proximal carpal row, which is composed of the scaphoid, lunate, triquetrum, and pisiform. These bones articulate with the distal portion of the radius and ulna and allow for flexion and extension of the hand, as well as ulnar and radial deviation. Distal to the proximal row of carpal bones is the distal carpal row, which is composed of the trapezium, trapezoid, capitate, and hamate. The distal carpal row and the second and third metacarpals form the "fixed unit" of the hand.

A detailed history that includes patient's occupation, hand dominance, and detailed characterization of the mechanism of injury, as well as the location and level of pain, should be the first step of evaluation. Three patterns of wrist injury exist: the perilunate pattern, the axial pattern, and injury from localized force concentration.

Perilunate Injuries

Perilunate injuries occur in an arc emanating from the lunate. The bones involved include the scaphoid, triquetrum, and capitate. If any of these bones are fractured, then the others should be checked for a fracture.

Axial Pattern Injuries

Axial pattern injuries result from anteroposterior compression forces. These forces, generally occurring from an explosion or crush injury, propagate on either the ulnar or the radial side of the capitate.

Carpal Bone Fractures

Single-bone fractures of the carpus usually are the result of a concentrated localized force.

Scaphoid

Most carpal bone fractures occur in the wrist. By articulating with the lunate proximally and the capitate distally, the scaphoid stabilizes the wrist. Upon disruption, the wrist becomes more susceptible to collapse.

The superficial palmar branch and the dorsal carpal branch of the radial artery enter at the distal aspect of the scaphoid. A loss of blood supply due to fractures at the waist or the proximal portion of the scaphoid leads to avascular necrosis of the scaphoid (Preiser's disease) and future pain/instability.

Mechanism

Scaphoid fractures result from a fall on an outstretched hand.

Diagnosis

The patient has tenderness in the anatomical snuffbox and radial wrist pain. Order X-rays in AP, pronation oblique, supination oblique, and lateral views. Additionally, CT scans are useful in establishing the vascularity and degree of displacement. A clenched-fist position may improve view. Look for a radiolucent line radial to the scaphoid on an AP view. If the line is preserved, then the scaphoid is intact. If the scaphoid is fractured, the line is displaced or obliterated (navicular fat stripe sign) (**Fig. 14.7**).

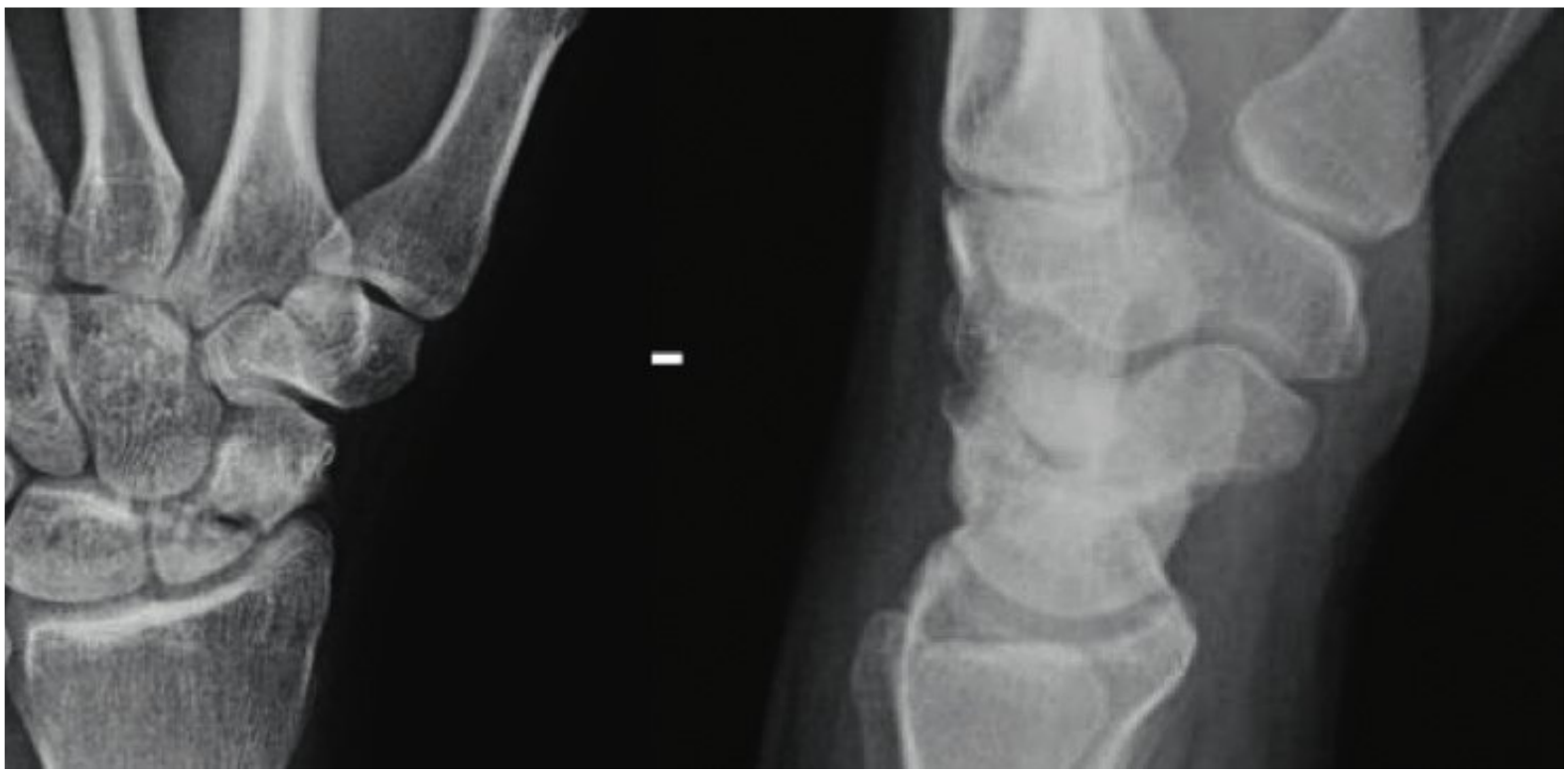


Fig. 14.7 Scaphoid fracture.

Types of Fracture

- **Horizontal oblique:** Fracture of the scaphoid oblique to the longitudinal axis of scaphoid but perpendicular to the long axis of the limb (most common fracture of the scaphoid). Stable and usually treated with closed treatment in thumb spica for 6 to 8 weeks (see Chapter 13, **Fig. 13.5**).
- **Transverse:** Scaphoid fractures that are perpendicular to the longitudinal axis of the scaphoid, but oblique to the limb. Less stable and less common than horizontal oblique fractures, these usually heal with 6 to 12 weeks of closed treatment (thumb spica) (**Fig. 13.5**).
- **Vertical oblique:** Rare and less stable; requires longer casting.

Treatment

- Closed treatment—reserved for suspected fractures and stable fractures with < 1 mm displacement or a scapholunate angle < 60 degrees or radiolunate fractures < 15 degrees.
- Place patient in thumb spica cast (**Fig. 13.5**) for 12 weeks (long arm thumb spica cast for the first 6 weeks and then 6 weeks in short arm thumb spica cast).

Suspected Fractures

- Place the patient in thumb spica.
- Technetium 99m methylene diphosphonate (Tc 99m MDP) bone scan in 2 weeks (time for bone at fracture site to resorb).
  - If negative, then no fracture exists.
  - If positive, order a CT scan for determination of fracture site and further treatment.

Nondisplaced Fractures

- Thumb spica cast until fracture heals.
- Check for union.
- Immobilize in a long arm cast for 6 weeks, then a short arm cast for an additional 6 weeks.

Pediatric

- Rarely displaced; ORIF only in severe displacement.
  - Otherwise, immobilize until skeletal maturity.
- Surgical treatment.
  - For open fractures of the wrist.
  - Failed closed treatment (no healing in 12 weeks or nonunion after 6 months of casting).
  - Displacement > 1 mm or scapholunate angle > 60 degrees or radiolunate fracture > 15 degrees.
  - Very proximal fractures that are prone to avascular necrosis.
  - If a patient has a nondisplaced fracture that cannot be immobilized, an arthroscopic approach can be taken.
    - Complications.
      - Malunion.
      - Avascular necrosis.
      - Nonunion.
      - Arthritis.
      - Carpal instability.
      - Scaphoid advance collapse.

Other Carpal Bone Fractures

Mechanism

Other carpal bone fractures can result from a fall on an outstretched hand.

Diagnosis

The patient has pain in the wrist; X-rays demonstrate fractures of carpal bones. If a fracture is suspected, then the Tc 99m MDP will be positive in 2 weeks. A CT scan can also be performed for diagnosis of fractures, especially in the distal row. Triquetral fractures are caused by wrist hyperextension. Trapezial fractures are seen using the Betts view and often occur in cyclists.

Treatment

- Closed treatment.
  - Nondisplaced carpal bone fractures should be immobilized for 6 weeks.
  - Use a thumb spica for lunate fractures.
  - Splint the hand in a safe position for capitate fractures.
- Surgical treatment.
  - Use for all open fractures and displaced fractures.

Lunate Fracture

Lunate fractures may cause Kienböck's disease by affecting the lunate blood supply and causing avascular necrosis.

Hook of Hamate Fracture

Hook of hamate fractures are associated with racquet sports and golf; patients present with ulnar and volar wrist pain. The fracture occurs on impact with the ball. Nonunion is diagnosed with a CT scan. Removal of the hook relieves the pain.

Pisiform Fracture

Nonunion is a common complication of pisiform fractures. Supinated oblique and carpal tunnel X-rays are most useful in diagnosing the fracture. Pain on nonunion resolves with removal of the pisiforms.

Scaphocapitate Syndrome

Scaphocapitate syndrome results from fractures in both the scaphoid and capitate, along with rotation of the capitate fragment 90 to 180 degrees. Treat with ORIF early. If missed, then treat expectantly. If symptoms persist, perform a wrist arthrodesis.

Dislocations of the Wrist

Dislocations of the wrist range from perilunate ligamentous injuries to lesser arc injuries (dislocations only) to greater arc injuries (dislocation + fracture).

The patient may present with acute onset carpal tunnel syndrome due to pressure from dislocated carpal bone, specifically the lunate. Emergent reduction and decompression are indicated.

Scapholunate Ligamentous Injuries

As the force causing perilunate ligamentous injury increases, there is a predictable pattern of injury. The progression proceeds from scapholunate sprains to scapholunate dislocation, perilunate dislocation, and finally dislocation of the lunate. In severe cases, patients present with extreme dorsiflexion of the wrist.

Diagnosis

- Watson shift test—put thumb on distal pole of scaphoid. Next, move joint radially, ulnarly, into extension and flexion.
  - Assess for pain or subluxation, which may herald instability.
- Stress radiographs.
  - Scapholunate disruption.

On the AP X-ray, a scapholunate disruption can be seen between the scaphoid and lunate > 3 to 4 mm (Terry Thomas/Letterman/gap sign), or as a wedge-shaped lunate (piece of pie sign). If the lunate is rotated dorsally, then the patient has a dorsal intercalated segment instability (DISI) deformity. A volar dislocation of the lunate will be apparent as the spilled teacup sign on lateral view (not associated with scapholunate disruption).

Reduction Techniques

If the patient presents within 3 to 4 days of injury, then attempt closed reduction, which usually requires K-wire for fixation. If the patient cannot be taken to surgery, then one can attempt closed reduction in the emergency room. This must be preceded by a thorough neurovascular examination. A hematoma block or brachial block can be performed.

Perilunate Dislocation

Initially, dorsiflex the wrist, and then slowly flex the wrist volarly while holding the position of the lunate with the thumb of your other hand. Rearticulate the capitate and the lunate using pronation. Use fluoroscopy if needed. If patient presents with acute carpal tunnel syndrome, reduce fracture and release carpal tunnel if symptoms persist.

Lunate Dislocation (Fig. 14.8)

Start with the procedure for perilunate reduction, then stabilize the lunate with your thumb and bring the capitate into palmar flexion. Emergency reduction and decompression of carpal tunnel are indicated if acute carpal tunnel syndrome symptoms exist.

Scapholunate Dislocation

First dorsiflex the wrist and then radially deviate the wrist. If reduction is performed in the operating room (preferred), then K-wire the reduction. If reduction is performed in the emergency room, then attempt to place the patient in a splint in a thumb spica. If reduction does not hold, then you must perform ORIF.

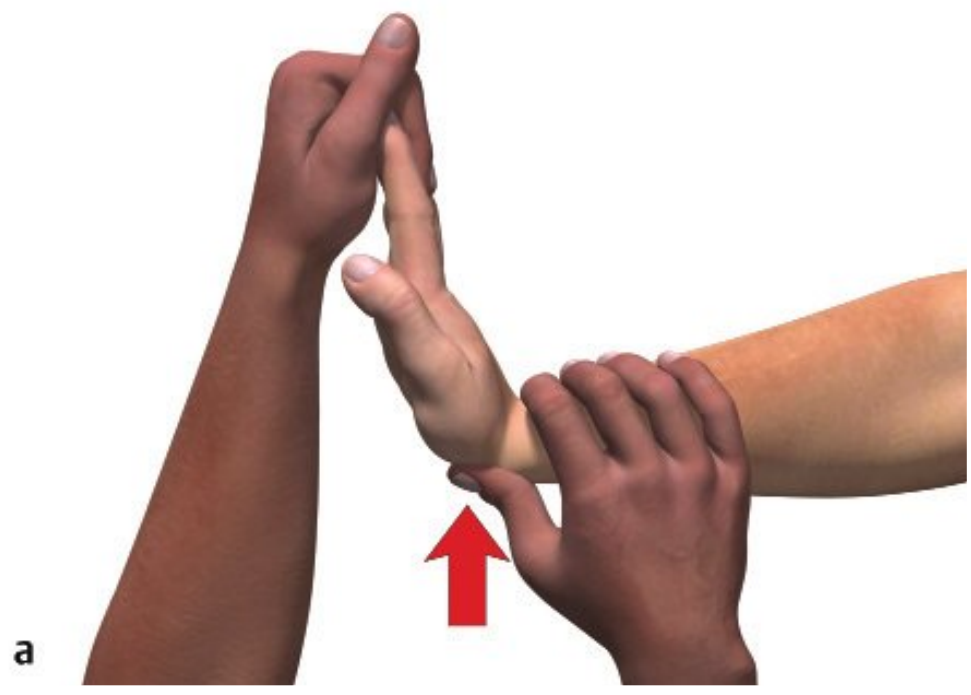
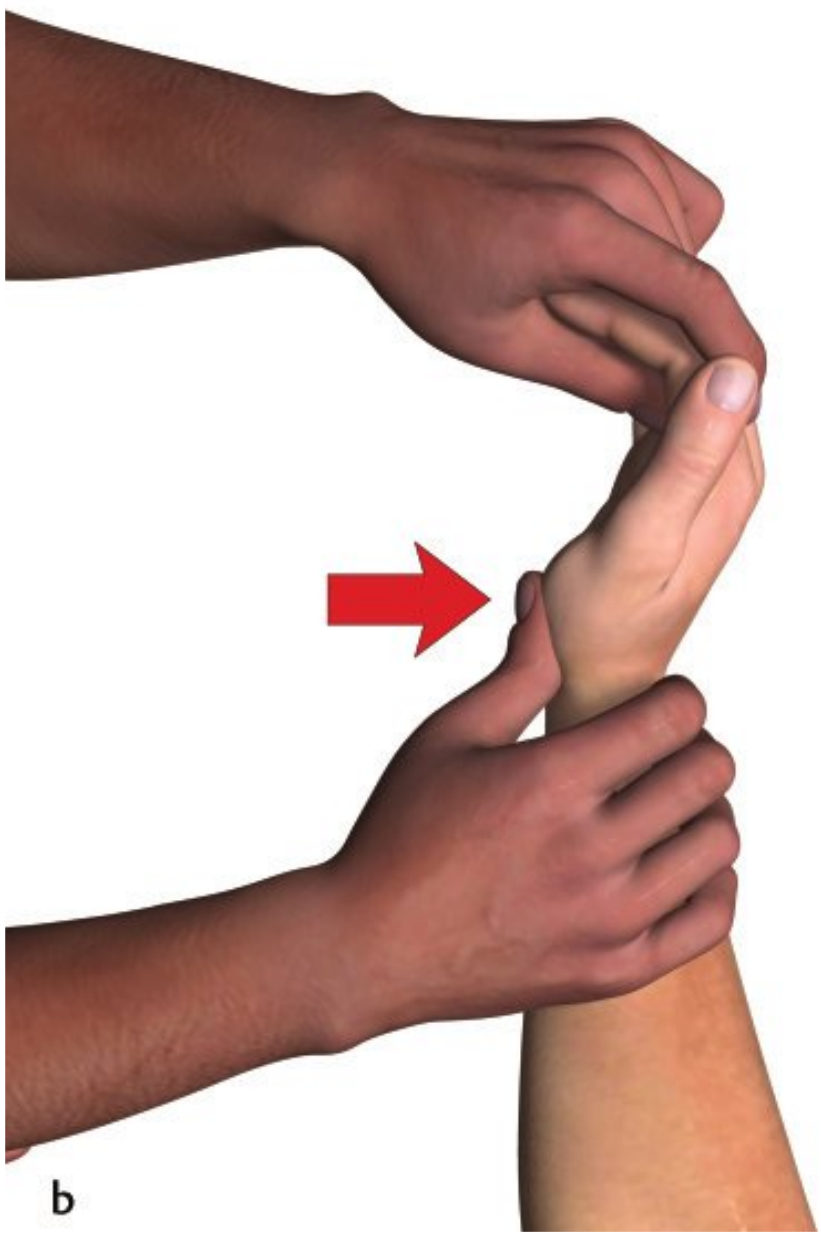


Fig. 14.8 **(a–c)** Reduction of a dislocated lunate.



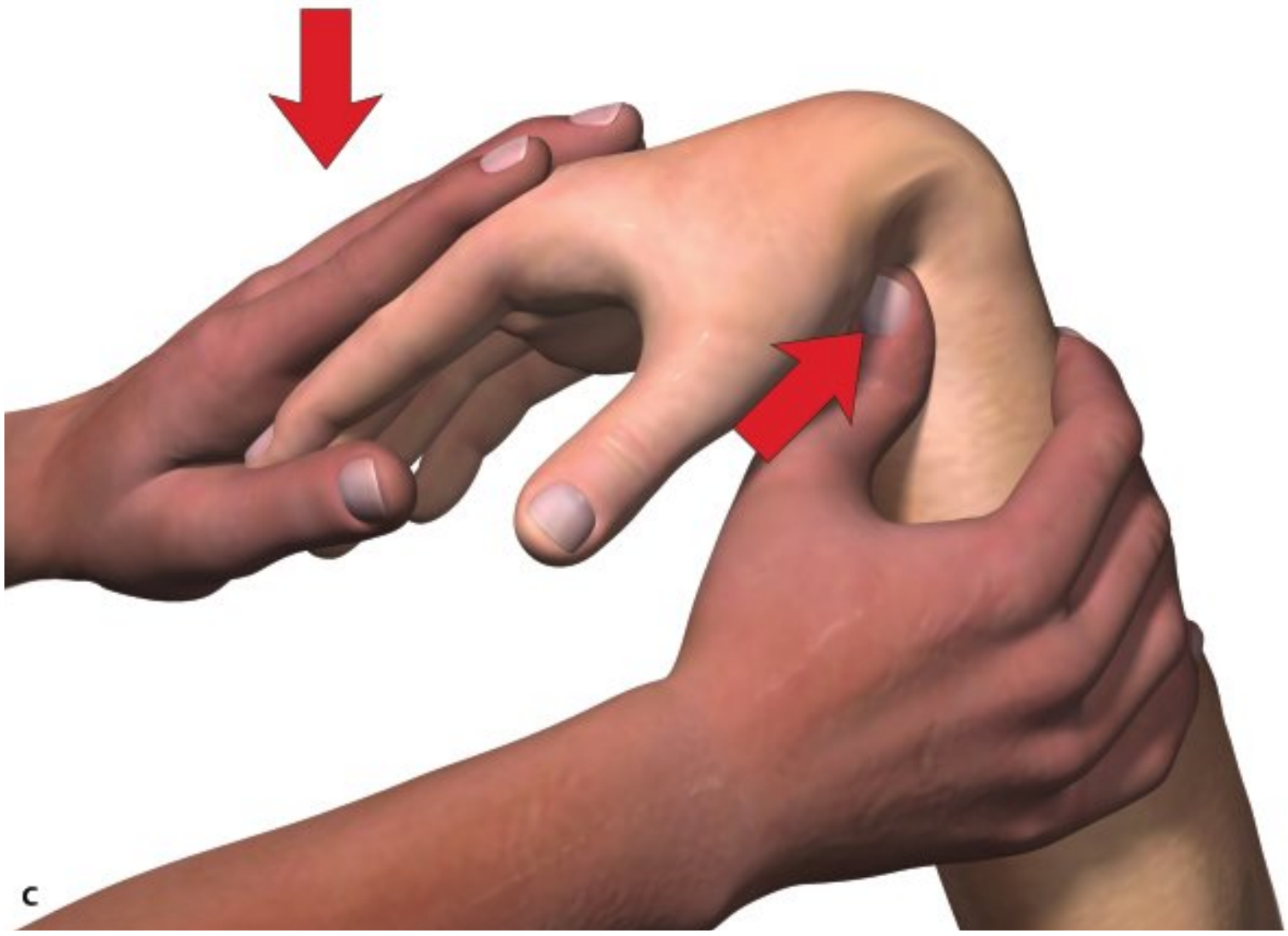


Fig. 14.8 (Continued) **(a–c)** Reduction of a dislocated lunate.

Fracture-Dislocations of the Wrist (Major Arc Injury)

The most common type of fracture-dislocation of the wrist is the trans-scaphoid perilunate fracture-dislocation. Use X-rays taken in traction for diagnosis. These usually require ORIF.

Ulnar-Sided Ligamentous Injuries

The patient presents with tenderness on the ulnar side of the wrist (over lunotriquetral ligament) with possible avulsion fractures of the triquetrolunate ligament.

Diagnosis

- Ballottement test (Reagan test)—displacement of the triquetrum dorsally and volarly on the lunate with painful crepitus.
- Lichtman test—subluxation and pain with axial loading and deviation of wrist ulnarly.
- X-ray—AP view demonstrates volar intercalated segment instability (VISI) with volar-flexed scaphoid. Lunate is volar-flexed and triangular.

Treatment

Immobilize for 6 weeks in short arm cast.

Triangular Fibrocartilage Complex (TFCC) Tears

The TFCC is a ligamentous and cartilaginous structure, which stabilizes the distal radioulnar joint and is the articulating surface for the ulnar carpus.

Diagnosis

- When patient grasps an object, wrist pain worsens.
- X-ray.
  - Ulnar positive variance on X-ray.
  - Arthroscopy versus MRI.

Treatment

- Short arm cast for 6 weeks.
- Consider arthroscopy and débridement of tears.

15 Hand Infections and Injection Injuries

Hand Infections

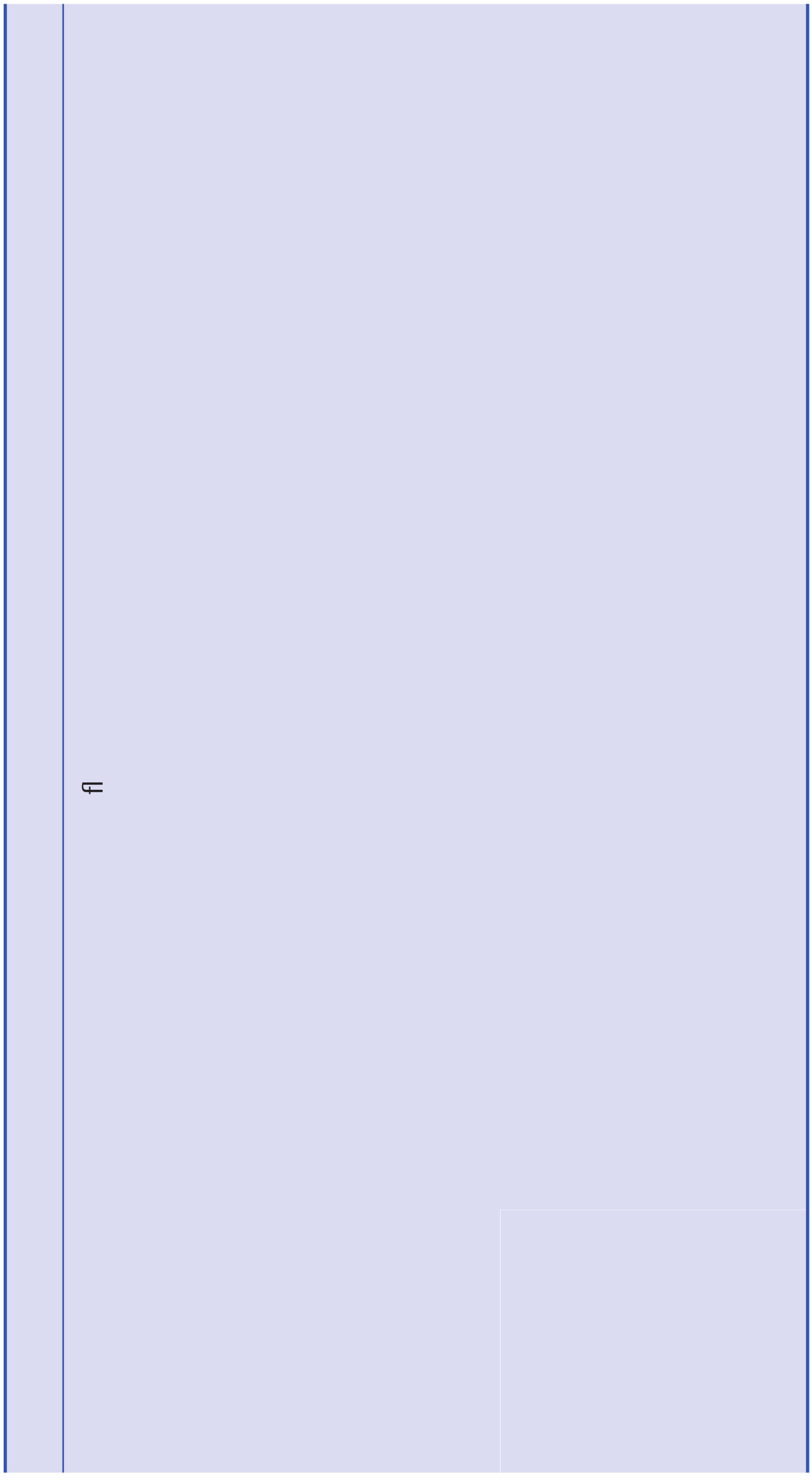
Hand infections are classified as superficial, deep, acute, subacute, or chronic. Hand infections range from superficial cellulitis to osteomyelitis. Cellulitis is a superficial inflammation of the dermal/epidermal components of the skin secondary to bacterial contamination. Deep to the dermis, infection in the subcutaneous tissue manifests as an abscess. Continuous deep infectious penetration will affect the fascia or the synovial sheaths of the flexor and extensor tendons, particularly in the hand and forearm. These deeper infections warrant rapid evaluation and treatment to prevent necrotizing inflammation within the deep tissue planes and erosive extension into the hand and forearm.

History and Physical Examination

- Ascertain the cause of the infection, anatomical location, and length of duration.
- Inspection and palpation to determine location and to assess the depth of the infection.
  - Remove all jewelry (watches, rings, etc.) to prevent secondary vascular constriction from a tourniquet effect when edema develops.
- Assessment of neurovascular status.
- Passive ROM assessment of all joints.
- Obtain radiographs of the involved hand/digit—three views.

Management

The antimicrobial treatments for common hand infections are presented in **Table 15.1**. The antimicrobial therapies delineated in **Table 15.1** represent empirical recommendations until definitive culture results are available for specific therapy.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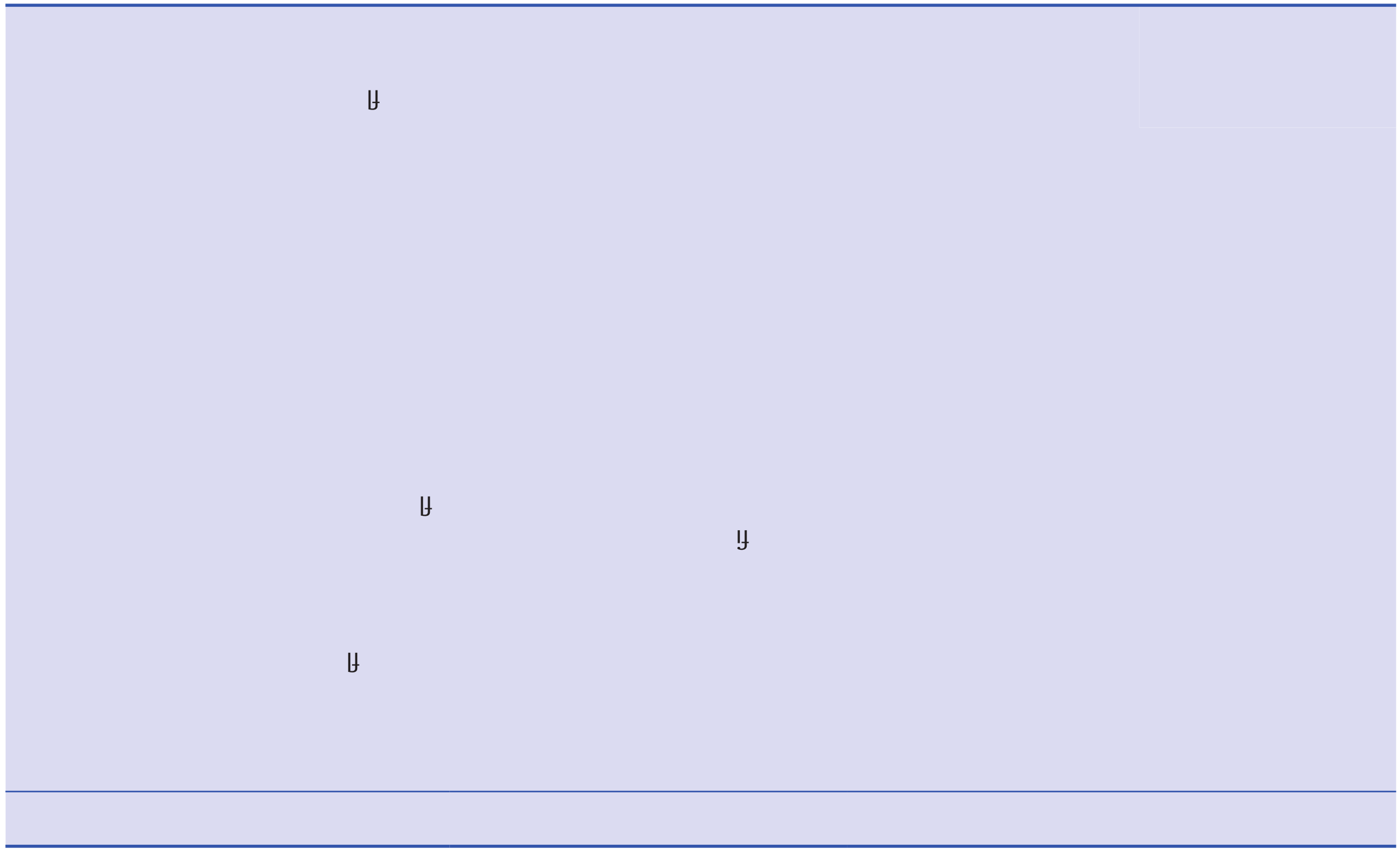


Types of Hand Infections

Acute Paronychia

Acute paronychia is an infection that involves the eponychial or paronychial fold or the nail matrix. This process usually begins under the skin of the lateral nail fold, causing erythema and edema (paronychia). Persistent disease may cause extension into the eponychium (eponychia) or under the nail sulcus to the contralateral fold (runaround infection or horseshoe infection).

Etiology

- Poor fingernail hygiene.
- Minor trauma.
- Nail biting.
- Finger sucking.
- Manicures.
- Artificial nails.
- Hangnails.

Treatment

An early infection without evidence of fluctuance can be treated conservatively:

- Warm soaks three times a day with a 1:1 solution of 3% hydrogen peroxide and normal saline.
- Oral antibiotics for 1 week; consider anaerobic coverage (clindamycin 450 mg by mouth four times a day) for associated nail biting or finger sucking etiologies (human bite).
- Elevation.
- Short-term follow-up.

A paronychia that has developed a collection of purulence requires drainage:

- Perform a digital block (see Chapter 13, **Fig. 13.1**).
- Use the incision technique (**Fig. 15.1a**).
  - Incise along the lateral nail fold with a scalpel.
  - Elevate the nail fold using small elevator from the paronychial/eponychial junction to the proximal nail edge.
  - Drain the purulent material.
  - Excise the distal third of the nail to evacuate purulent material and, if needed, for additional exposure.
- Excision technique (**Fig. 15.1b**).
  - Begin with a longitudinal incision along the lateral nail fold with a 15-blade scalpel beveled away from the nail.

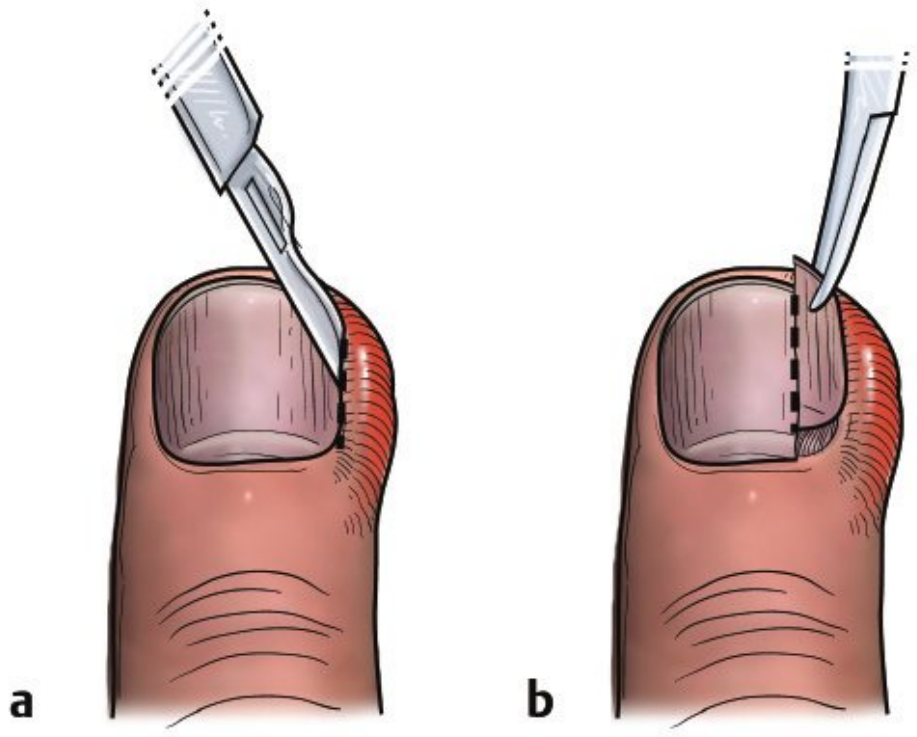


Fig. 15.1 **(a)** Incision and drainage of a paronychial infection. **(b)** Partial nail excision technique.

- Repeat bilaterally if both sides are involved.
- Drain purulent material at the base of the nail by elevating the eponychial fold.
- Excise a longitudinal strip of the nail adjacent to the fold with an edge of the eponychium for drainage.
- Extensive eponychial and subungual infections require removal of the nail plate and stenting of the fold with iodoform gauze.

Postoperative Care

- Warm soaks three times a day with a 1:1 solution of 3%hydrogen peroxide and normal saline.
- Oral antibiotics for 1 week.
- Elevation.
- Short-term follow-up.
- Avoid nail biting and trimming nails too closely.

Chronic Paronychia

Chronic paronychia is defined as an infection that involves the eponychial or paronychial fold or the nail matrix lasting longer than 6 weeks. Fungi are most commonly associated with these infections. C. *albicans* is the primary infectious organism. However, atypical mycobacteria are implicated in hand infections of persons chronically exposed to water. Generally, these infections are found in swimmers, dishwashers, and housekeepers who have prolonged exposure to moist environments and repeated exposure to chemical irritants.

Treatment

- Topical miconazole twice a day or terbinafin twice a day.
- Oral ketoconazole 200 mg by mouth daily or fluconazole 100 mg by mouth daily for 4 weeks.
- Consider biopsy to rule out squamous cell carcinoma for recalcitrant disease.
- Marsupialization.
  - Perform a digital block.
  - Apply finger tourniquet.
  - Using a 15-blade scalpel, incise along the proximal and distal edge of the eponychium in a crescent fashion.
  - Excise the eponychial skin and infected tissue, leaving the germinal matrix intact.
  - Irrigate the exteriorized germinal matrix, then pack the region with iodoform gauze.
  - Remove the nail plate if grossly deformed.
- Change the dressing every day until complete epithelialization has occurred.

Felon

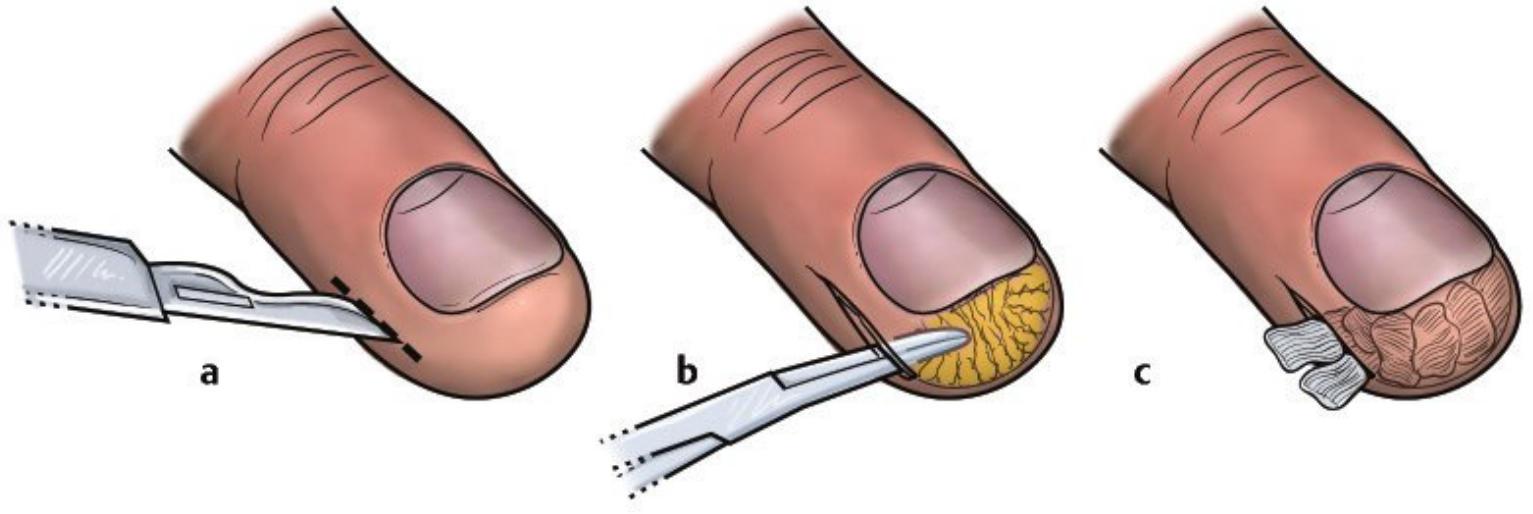
The volar pad of the distal phalanx is divided into 15 to 20 fibrous fascial compartments by vertical fibrous septa extending from the dermis to the distal phalanx. Infections in this area are compartmentalized, causing the formation of small abscesses. *S. aureus,* streptococci, and anaerobes cause most felons. Evaluation should rule out the presence of a foreign body, which occasionally can be detected radiographically. Persistent disease will result in extension to the distal phalanx and possibly the tendon sheath of the flexor digitorum superficialis (FDS), causing osteomyelitis or flexor tenosynovitis, respectively.

Treatment

The volar pad septa must be completely obliterated, while minimizing damage to the neurovascular bundle:

- Perform a digital block.
- Apply a finger tourniquet.
- Mark the nondominant side of the finger for the incision. This is usually the ulnar side of the index finger, long finger, or ring finger. For the thumb and little finger, release via an incision on the radial side of the digit.

- Incisions.
  - High lateral incision (**Fig. 15.2**).
  - Fish mouth incision.
  - Palmar longitudinal incision.
- Obtain culture.
- Spread through septa-disrupting all of the compartments.
- Irrigate thoroughly.
- Pack with iodoform gauze.



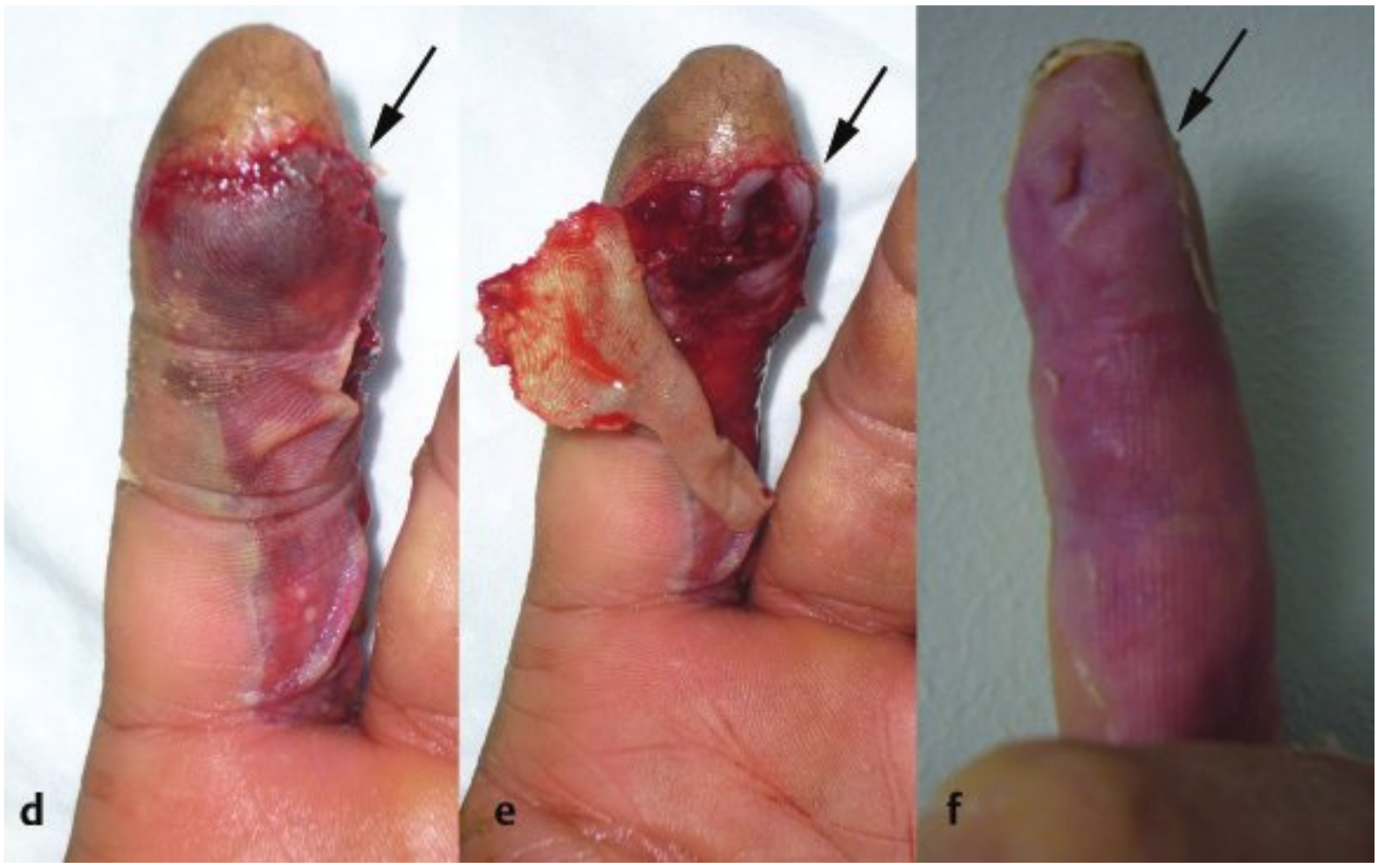


Fig. 15.2 Incision and drainage of a felon. **(a)** High lateral incision avoiding neurovascular bundle. **(b)** Disruption of the ventral fibrous septa. **(c)** Packing of the space with iodoform gauze after thorough irrigation. **(d)** Complicated felon demonstrating epidermolysis extending to the ulnar position of the index finger. **(e)** Débridement of the detached epidermis and ulna-based incisions and drainage of felon. **(f)** Follow-up 1 month after surgery. The arrow indicates site of felon.

Postoperative Care

- Warm soaks three times a day with a 1:1 solution of 3% hydrogen peroxide and normal saline.
- Oral antibiotics for 1 week.
- Elevation.
- Short-term follow-up.

Herpetic Whitlow

Herpes simplex is the causative organism associated with vesicular eruption of the distal digits. The viral contamination is usually secondary to exposure to oral secretions. Health care workers, particularly dentists and anesthesiologists, are at increased risk. Physical examination reveals clear vesicles that progress to ulceration within 14 days. The volar pad is edematous, but soft and painful to palpation. Diagnosis can be confirmed by viral cultures and a Tzanck smear that demonstrates multinucleated giant cells. The ability of the virus to live in the dorsal root ganglion promotes the recurrence of this disease.

Treatment

- Do not attempt incision and drainage.
  - This a self-limited disease that resolves in 10 to 14 days.
- Cleanse wound twice a day to prevent a bacterial superinfection.
- Cover wound with loose dressing.
- Oral antiviral drugs decrease the clinical course and recurrence.
  - Acyclovir 200 mg by mouth every 4 hours × 10 days (recurrence × 5 days), suppression 400 mg by mouth twice a day.
  - Valacyclovir 1 g by mouth twice a day × 10 days (recurrence 500 g by mouth twice a day), suppression 500 g by mouth daily.

Flexor Tenosynovitis

Synovitis of the flexor tendon sheath occurs from inflammatory and infectious etiologies. In the acute setting, suppurative stenosing infection of the flexor tendon sheath requires rapid evaluation and treatment to prevent extension to the forearm. Infections of the flexor tendon sheath result from either direct extension from a subcutaneous abscess (e.g., felon, midpalmar space abscess) or direct inoculation from penetrating trauma. A patient who presents with flexor tenosynovitis will exhibit the four Kanavel signs (**Fig. 15.3**).

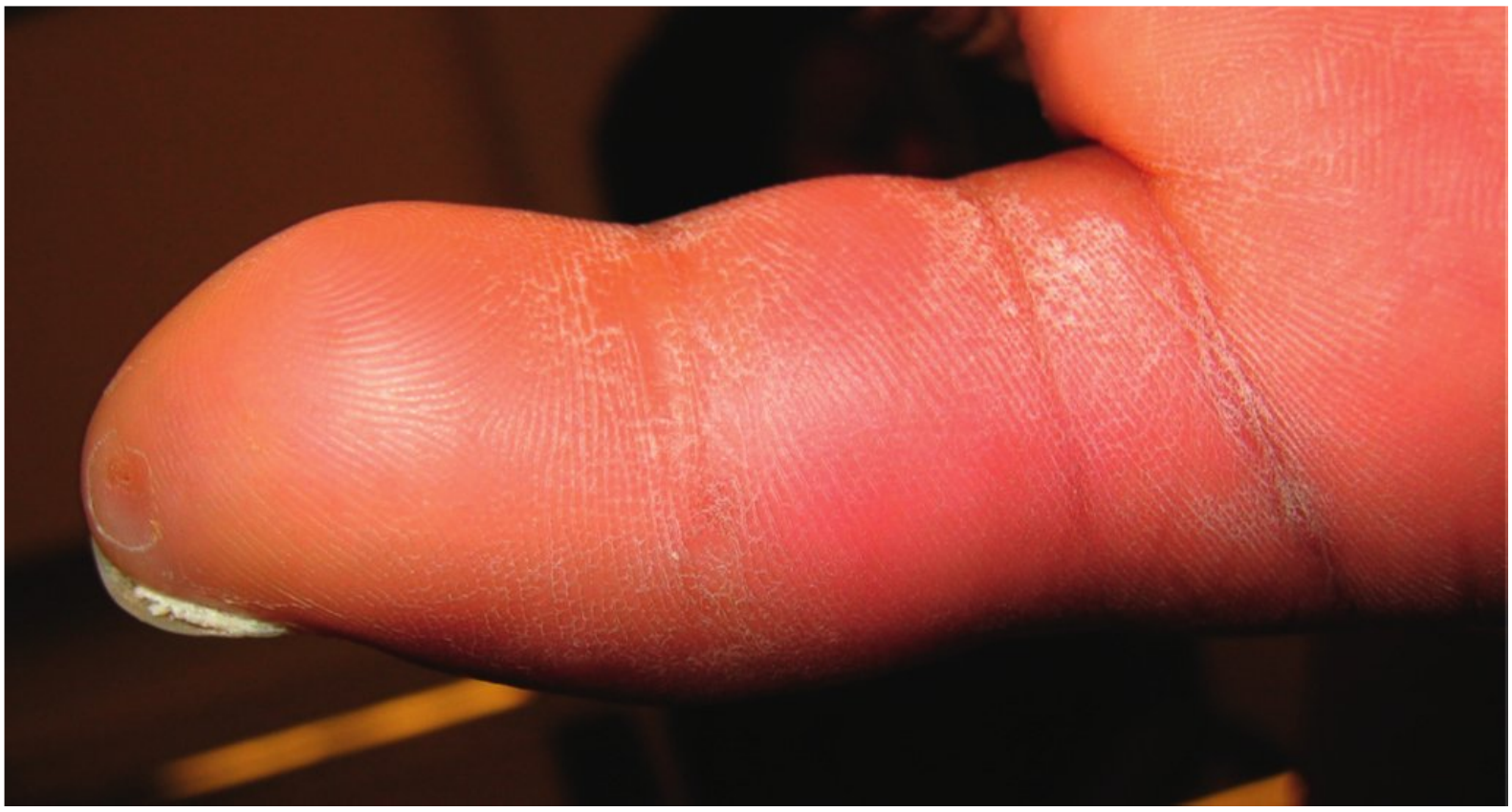


Fig. 15.3 Presentation of flexor tenosynovitis of the thumb illustrating Kanaval signs.

Hallmark of Flexor Tenosynovitis is Pain—on passive extension

Patient complaints of severe pain, paresthesia, and swelling of the hand may indicate flexor tenosynovitis. These symptoms can be explained by Kanavel signs.

Kanavel Signs
Pain over tendon sheath
Fusiform swelling of digit
Finger held in flexion
Pain on passive extension (hallmark sign)

The tendon sheath is a closed space from the DIP to the A1 pulley. The thumb and small finger tendon sheaths communicate with the radial and ulnar bursae, respectively, and continue into the wrist. The radial and ulnar bursae communicate via the Parona space. The intricate architecture and proximity of the tendon sheaths and bursae allow extension of the infection to the hand proximally. Additional potential complications include carpal tunnel syndrome, tendon necrosis, and tendon adhesions.

Treatment

Patients with flexor tenosynovitis require hospital admission, broad-spectrum antibiotics, and urgent operative exploration.

- Bring the patient to the operating room.
- Place tourniquet and exsanguinate arm by elevating arm for 2 minutes and occluding the brachial artery. Raise tourniquet to 100 mm Hg greater than the SBP. Do not exsanguinate using mechanical exsanguination techniques.
- Limited incision and catheter drainage (**Fig. 15.4d**).
  - Incise the midaxial border of the involved distal phalanx. (Avoid contact and pressure surfaces of the digit).
  - Make a separate transverse incision at the level of the A1 pulley.
  - Through these incisions, expose the flexor tendon sheath.
    - Evacuate purulence.
    - Obtain culture.
    - Thoroughly irrigate both wounds.
  - Insert a small catheter for irrigation into the tendon sheath—6 French pediatric feeding tube.
    - Irrigate.
  - Keep catheter in place for continuous irrigation.
    - 500 mL normal saline + 1 g vancomycin.
    - Infuse at 20 to 50 mL/hour, depending on patient tolerance.
  - Remove catheter in 48 hours.
- Extensive exploration and débridement—required for delayed diagnosis and extensive soft tissue necrosis.
  - Mark the nondominant side of the finger for the incision. This is usually the ulnar side of the index finger, long finger, and ring finger. For the thumb and little finger, release via an incision on the radial side of the digit.
  - Make Brunner (**Fig. 15.4b**) zigzag incisions from the distal phalanx to the palm.
    - Take care not to damage the neurovascular bundles or cross the flexion creases volarly at right angles.
  - Evacuate purulence.
  - Obtain culture.
  - Remove necrotic debris.
  - Thoroughly irrigate both wounds.
- Close skin loosely over a small catheter for continuous catheter irrigation using a 6 French pediatric feeding tube.
  - 500 mL normal saline + 1 g vancomycin.
  - Infuse at 20 to 50 mL/h, depending on patient tolerance.
  - Remove catheter in 48 hours.
- Splint in safe position.
- Begin ROM protocols after catheter is removed to decrease adhesions.

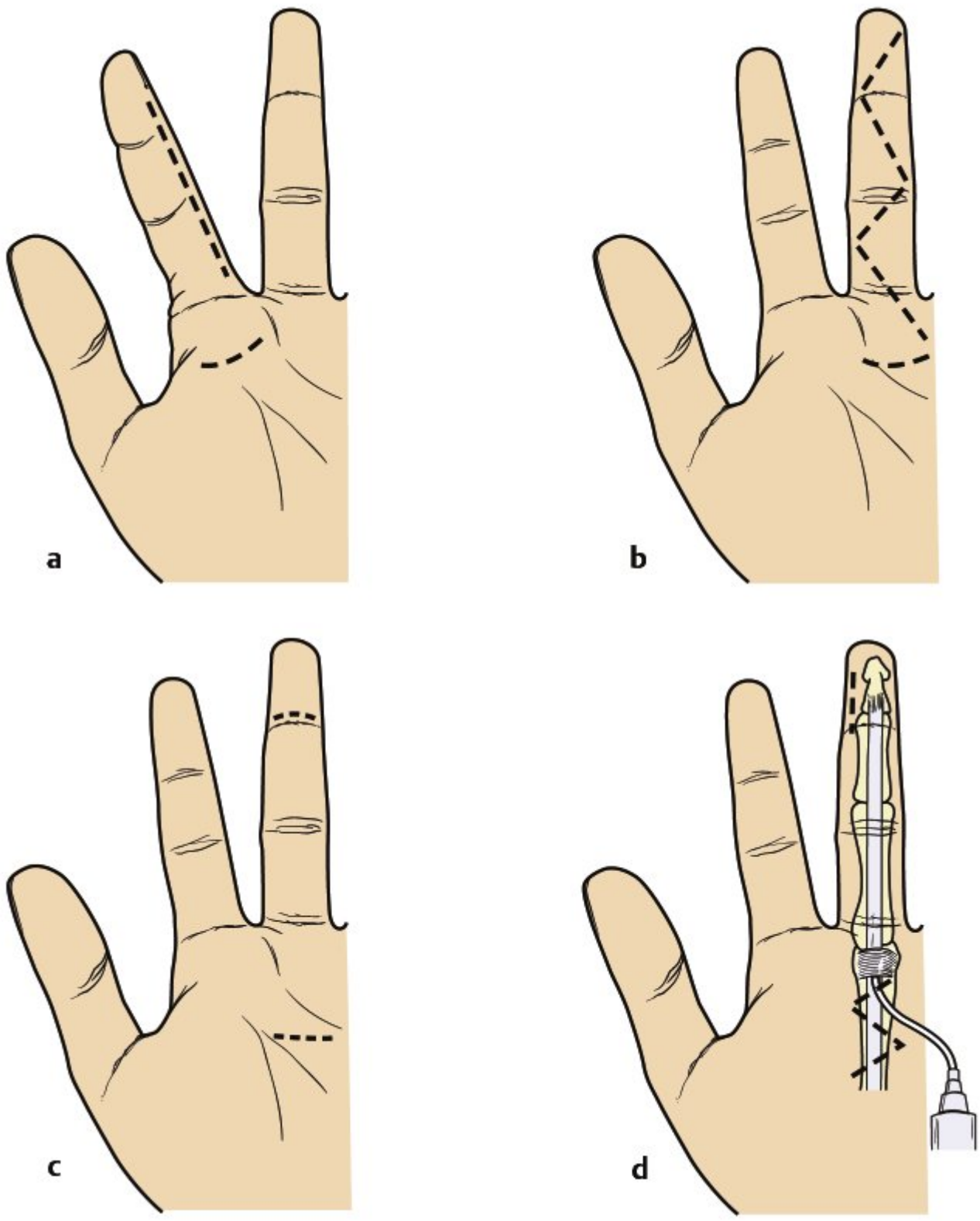


Fig. 15.4 **(a–d)** Incision techniques for drainage of the flexor tendon sheath.

- If there is significant soft tissue destruction, whirlpool therapy as an adjunct is useful for débridement of the devitalized soft tissues after operative drainage.

Deep Fascial Space Infections

Deep space infections begin from penetrating wounds of the hand or by extension of a superficial infection (**Fig. 15.5**). The complex anatomical relationship of the deep fascial spaces in the hand is illustrated in **Fig. 15.6**; **Fig. 15.7** shows the incisions for deep palmar abscesses.

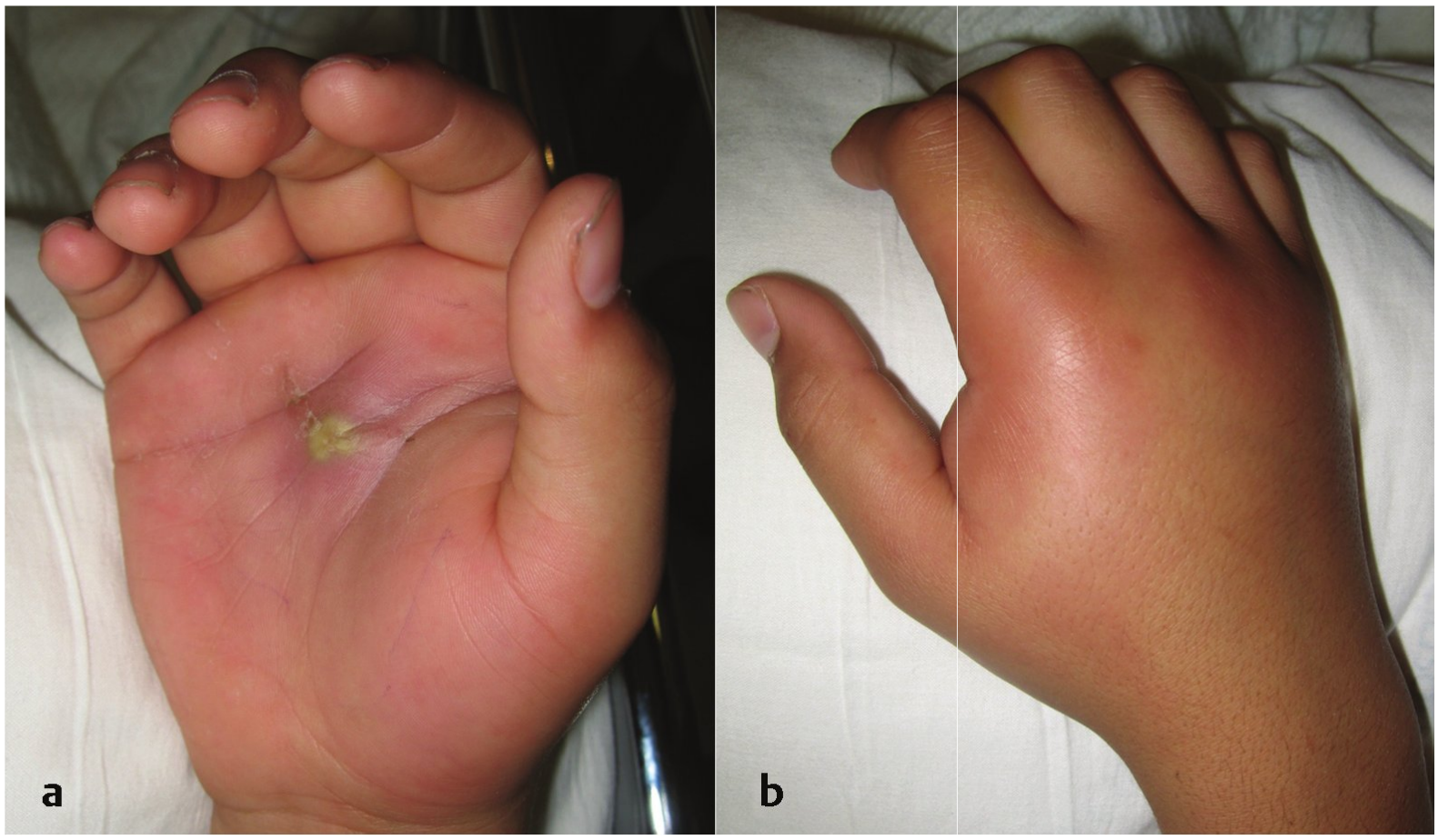


Fig. 15.5 **(a,b)** Presentation of a midpalmar space infection with dorsal cellulitis.

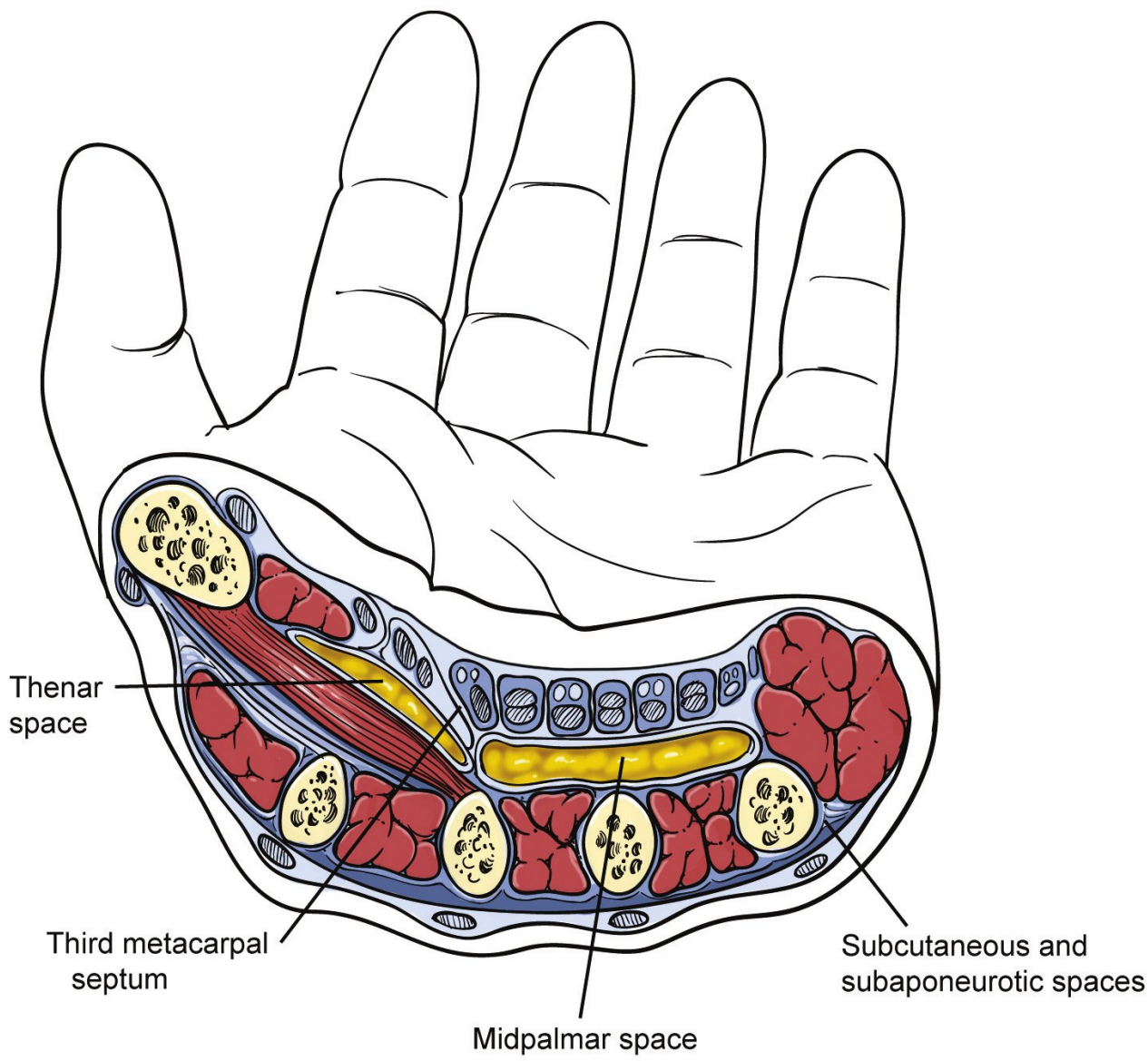


Fig. 15.6 Deep fascial spaces of the hand.

Treatment

- Place tourniquet and exsanguinate arm by elevating arm for 2 minutes and occluding the brachial artery. Raise tourniquet to 100 mm Hg greater than the SBP.
- Incision and drainage.
  - The wound is left open.
  - Wound packed with iodoform or a Penrose drain is placed.
  - Daily dressing changes are performed.
- Small dorsal abscesses can be safely drained in the emergency room. Make incisions in between extensor tendons to avoid injury to the tendons.
- Volar abscesses are explored in the operating room.
- Antibiotics.
- Whirlpool therapy twice a day.
  - Aids with débridement of devitalized tissue.
  - Edema may be increased throughout the duration of therapy.

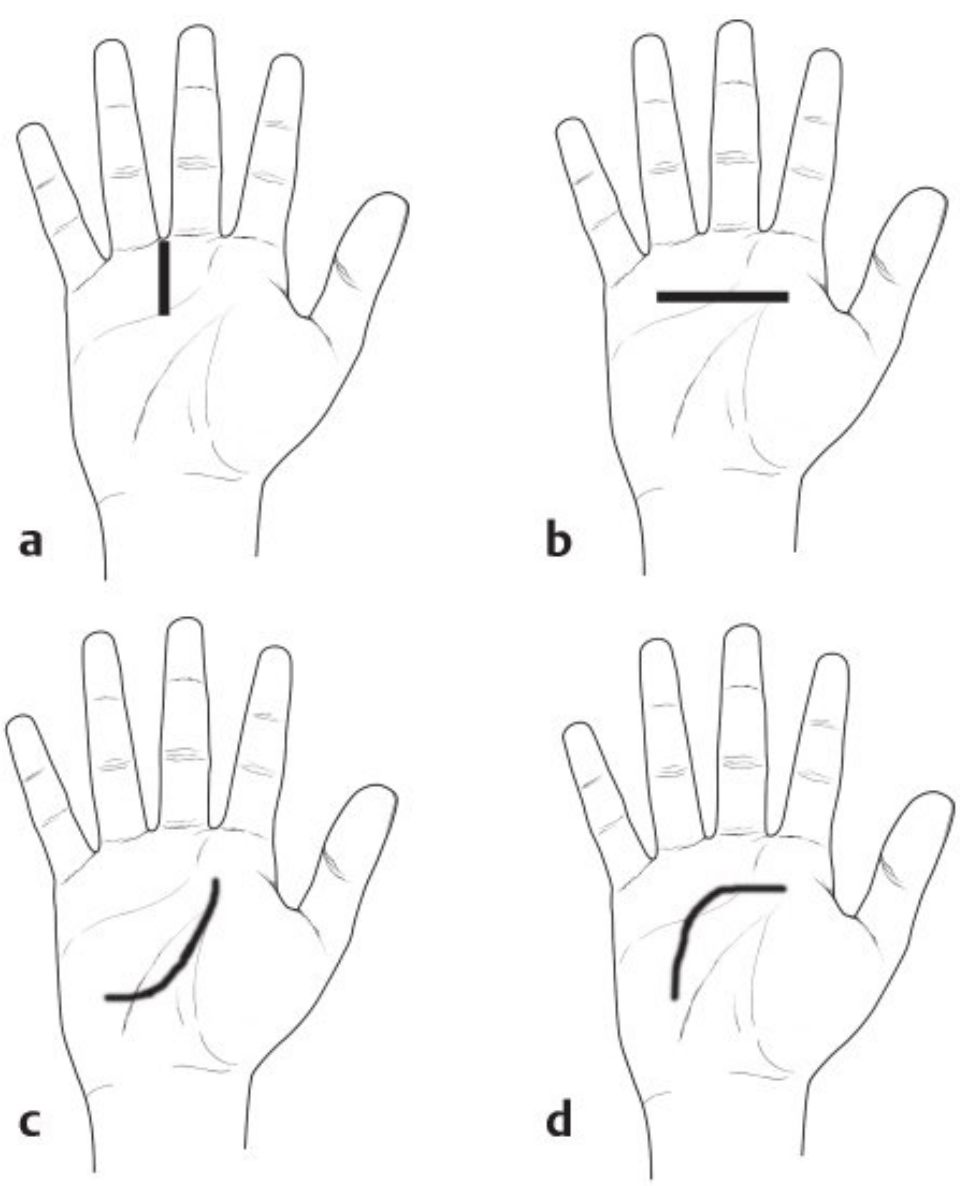


Fig. 15.7 (a–d) Incision techniques for drainage of deep hand abscesses. Avoid damage to neurovascular structures.

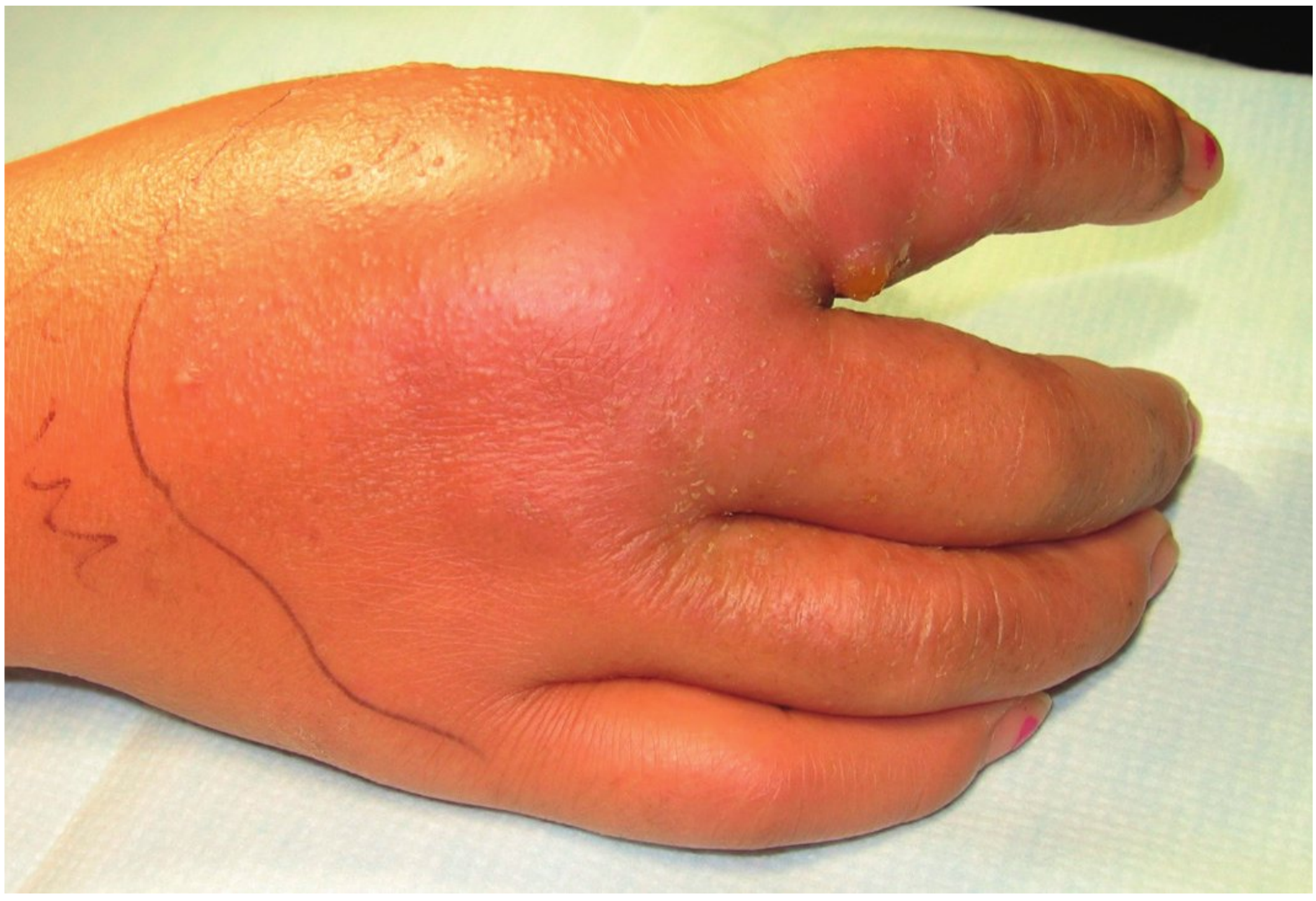


Fig. 15.8 Presentation of a collar button abscess of the second web space with abduction of the digits and dorsal cellulitis.

Collar Button Abscess

A purulent infection of the web space is referred to as a collar button abscess (**Fig. 15.8**). Fissures and blisters are commonly implicated as etiologies. Patients present with an hourglass configuration at the base of the digit in an abducted position. Collar button infections are drained through two longitudinal incisions on the dorsal and volar surfaces of the web space (**Fig. 15.7a**).

Dorsal Subaponeurotic and Subcutaneous Abscesses

Beneath the extensor tendons on the dorsum of the hand, an infection may reside in the subaponeurotic space, which is differentiated from an infection above the extensor tendons (subcutaneous space). Drainage is performed through longitudinal incisions. A suspected subaponeurotic infection is approached through longitudinal incisions over the index and small finger metacarpals (avoid damage to extensor tendons).

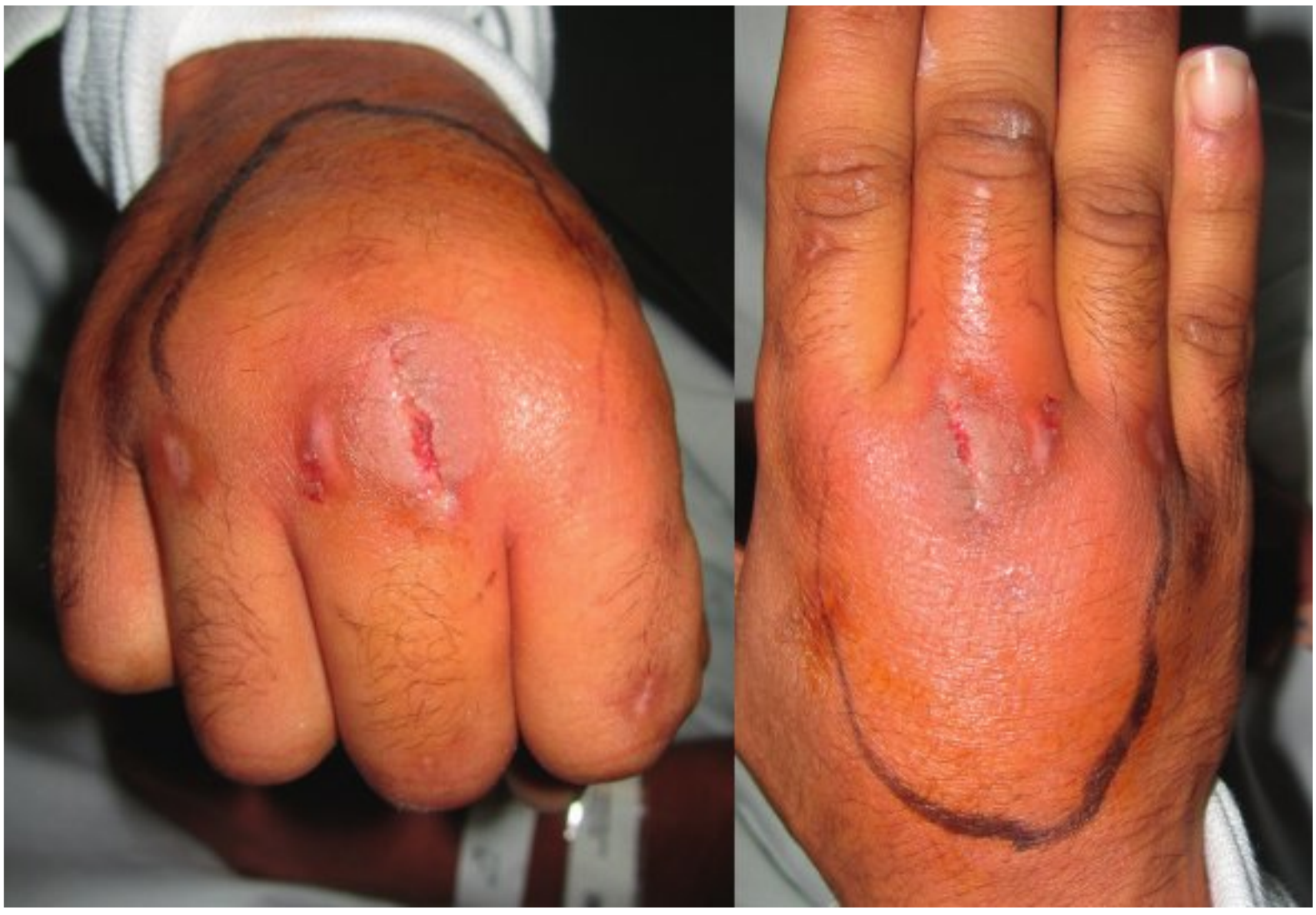


Fig. 15.9 Presentation of a fight bite injury with associated abscess and MCP joint contamination. A fracture of the metacarpal head is often associated with these injuries.

Fight Bite

Closed fist injuries occurring during fisticuffs may result in inoculation of the MCP joint with oral anaerobes from the assaulted. When this occurs there should be the consideration of a concomitant fracture to the metacarpal head. Patients often neglect these injuries due to the inciting circumstance and present in a delayed fashion (**Fig. 15.9**). All patients with this mechanism of injury should have thorough irrigation of the joint space and antibiotic therapy. Patients with more advanced presentations (cellulitis and abscess formation) should be admitted, irrigated in the operating room, and placed on IV antibiotics (see Chapter 14 for a detailed management strategy for fight bite injuries).

Thenar and Midpalmar Space Abscess

Thenar space infections occur in the volar soft tissues of the thumb and first dorsal interossei. The thumb is held in abduction and pain is elicited with adduction. Drainage is performed with an incision that is parallel to the thenar crease.

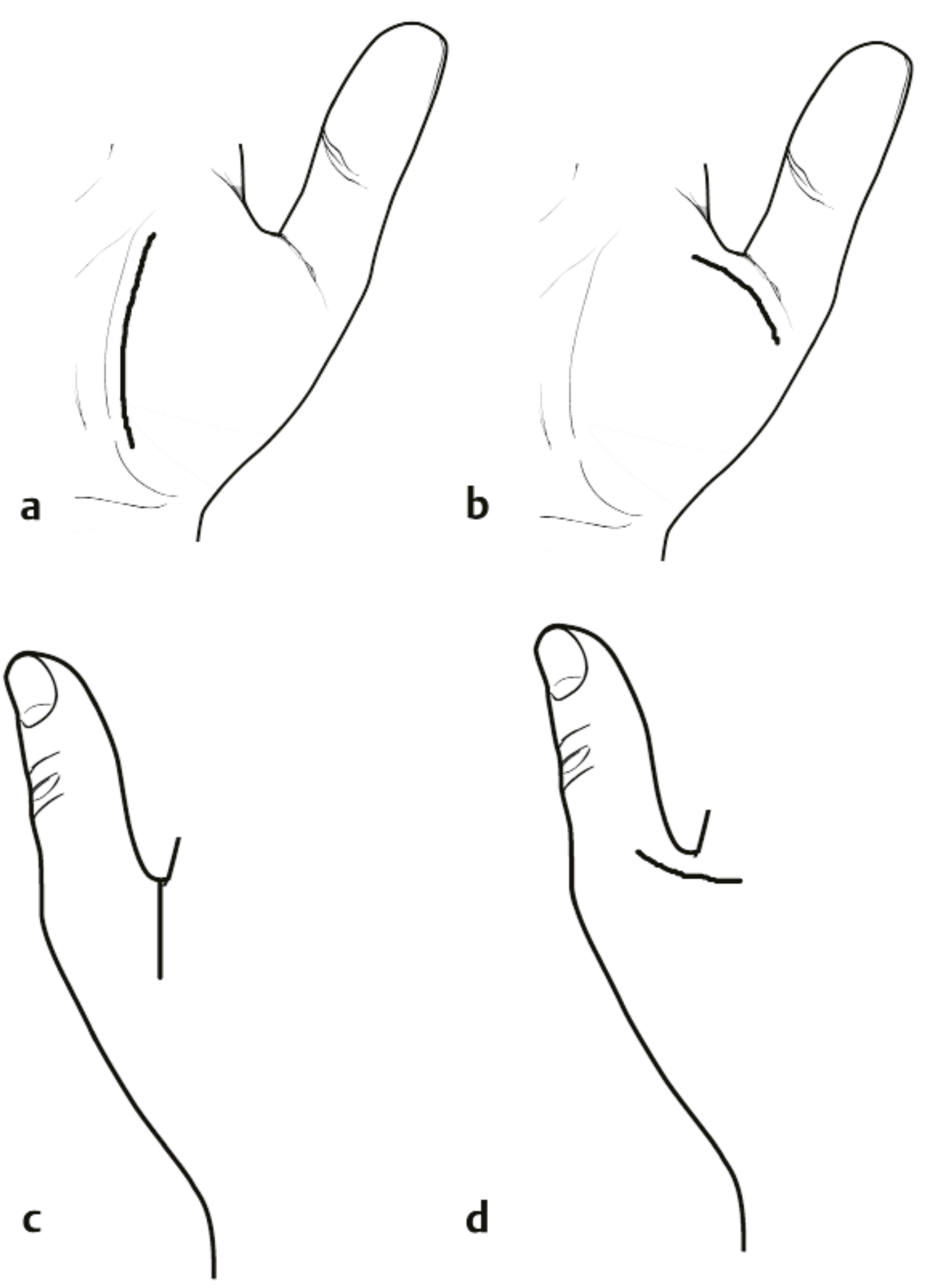


Fig. 15.10 **(a–d)** Incision techniques for drainage of thenar abscesses.

The midpalmar space comprises an area deep to the flexor tendons. Patients present with fluctuance, erythema, and palpable tenderness in the midpalm. Transverse or oblique volar incisions are utilized to explore the flexor tendons and midpalmar space (**Fig. 15.10**). After drainage, the wounds are closed loosely over a continuous irrigation catheter. Patients are admitted and IV antibiotics are administered.

Osteomyelitis and Septic Arthritis

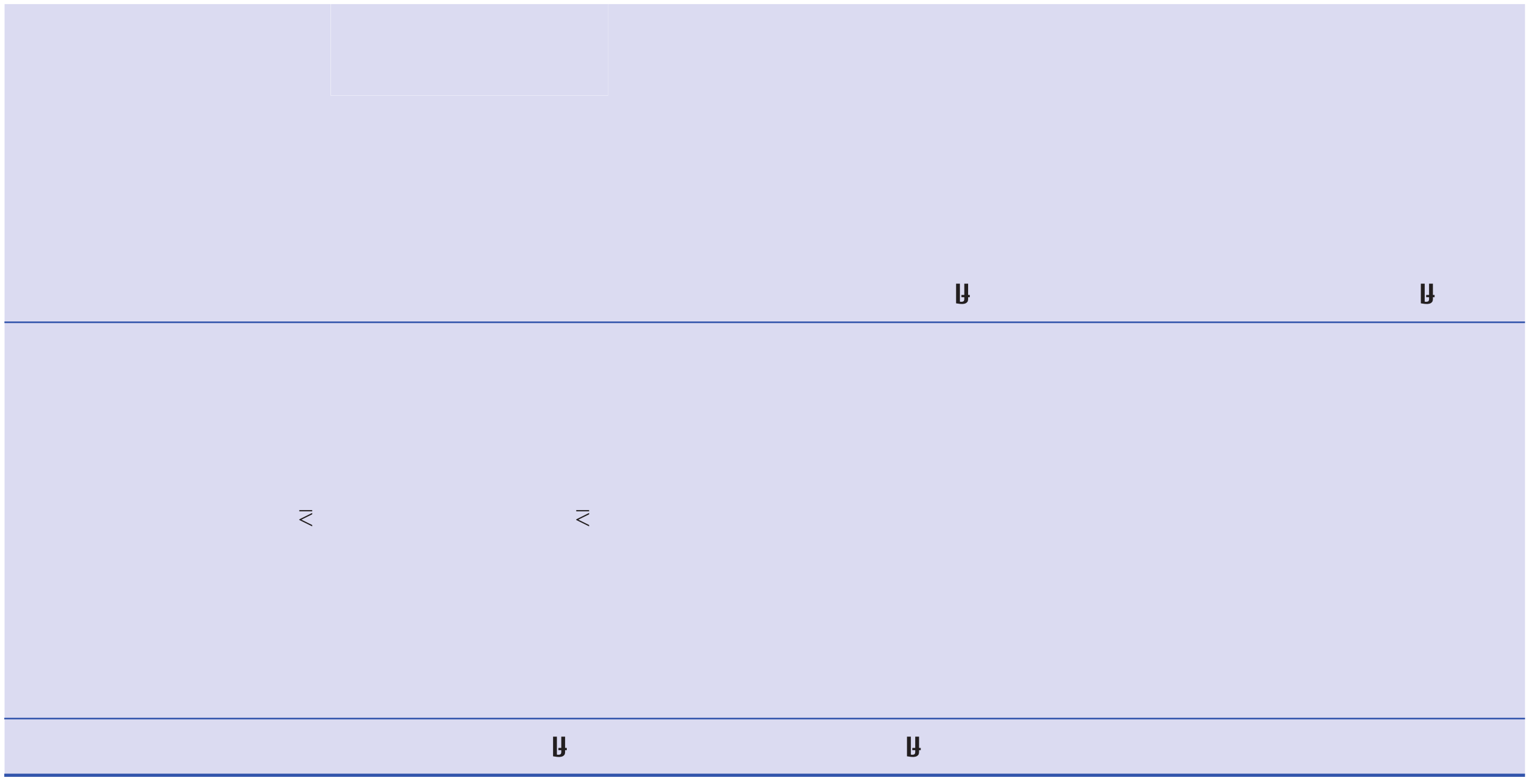
Infections of the joint and bone are usually the result of open fractures, an extension of chronic soft tissue infections, or secondary to direct inoculation from a penetrating object (e.g., a tooth). Septic arthritis and osteomyelitis are less commonly the result of hematogenous contamination from a distant focus. This mechanism is particularly common in immunosuppressed and pediatric patients secondary to the intrinsic vascular architecture at the physis/epiphysis. Diagnosis is based on history and radiographic evaluation. Patients have a history of chronic infection, nonhealing wounds, or nonunions. Elevated CRP and ESR are nonspecific when acute inflammatory conditions occur simultaneously. Plain radiographs will reveal bony erosion and periosteal elevation. MRI is more specific than plain films.

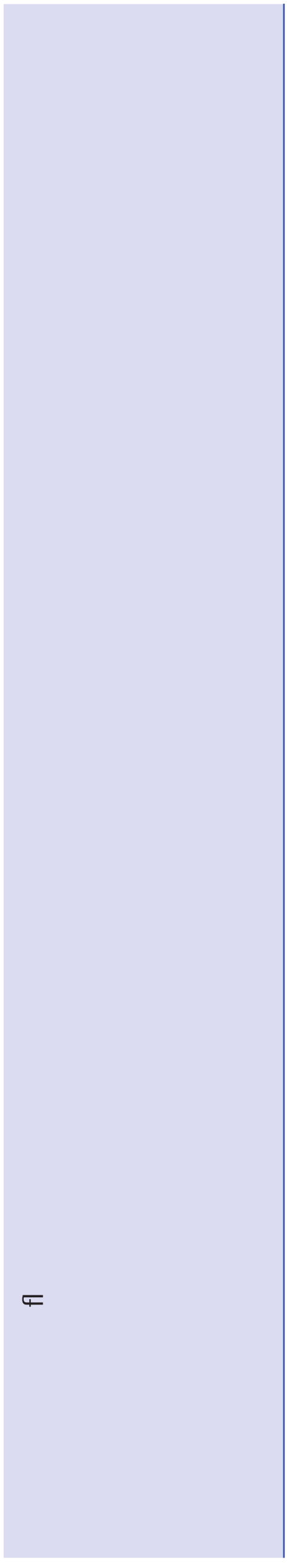
Osteomyelitis is treated with débridement of the infected bone and removal of the sequestra and sinus tracts. Bone specimen is sent for pathology and culture. Antimicrobial treatment is instituted for 6 weeks.

Patients with pyogenic arthritis present with the involved joint held in a distracted position to maximize volume. Tenderness and erythema is localized over the joint. Pain is elicited with passive ROM. A subset of patients with inflammatory arthritides (e.g., gout, rheumatoid arthritis) may present with joint signs and symptoms similar to those of septic arthritis. In these cases, a careful history with serology will assist in making the diagnosis. Joint aspiration is indicated for diagnosis (**Table 15.2**) and for purposes of culture identification (**Table 15.1**). Incision and drainage of the MCP joint is performed through dorsal incisions proximal to the sagittal band. The thumb MCP joint is approached through a midaxial ulnar incision. The IP joints are also approached through a midaxial incision and irrigated with a butterfly needle.

Hand Injection Injuries

These injuries occur most commonly secondary to industrial guns loaded with paints, grease, or fuels. Pressure of approximately 100 psi (7 kg/cm^2) is required to penetrate the epidermis, but industrial guns

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can inject with a power of 10 to 100 times greater than this pressure. The nondominant index finger is the most affected site.

Although the site of injection and penetration of fluids under pressure may seem small initially, most such injuries require wide surgical incision of the hand, meticulous lavage, and débridement.

Symptoms are initially subtle, at times only a small pinpoint puncture in the skin. After several hours, however, patients complain of increasing edema, pain, dysesthesia, and discoloration. If left untreated, this will progress to necrosis, gangrene, lymphangitis, and bacterial infections.

Examination

- Determine the precise location of injury, the time of injury, type of fluid injected, and tetanus vaccination status.
- Examine the entire upper extremity because the superficial appearance of the injury often belies the extent of tissue damage.
- Neurovascular evaluation and documentation are essential.
- Check X-rays to rule out fractures and visualize radiographically opaque material.

Treatment

- Tetanus vaccine.
- Broad-spectrum antibiotic coverage (vancomycin 1 g IV every 12 hours + cefepime 1 g IV every 12 hours or Zosyn 3.375 mg IV every 6 hours).
- Elevate limb.
- Immediate surgical exploration.
- Evaluate the need for fasciotomy if injury presents late.

Operative Treatment

Use an upper extremity tourniquet without using mechanical exsanguination. Instead, raise the arm for 3 minutes while compressing the brachial artery prior to inflation of the tourniquet. Use an axillary block for anesthesia.

Perform Brunner (**Fig. 15.4b**) or midlateral (**Fig. 15.4a**) incisions in the fingers. Brunner incisions are made by first cutting along a diagonal line from the lateral nail bed site to the flexion crease on the opposite

side. The incision is then zigzagged back diagonally to the next flexion crease on the opposite side. It can be carried proximally across the palm. A midlateral incision is first marked by flexing the finger. Next, a line is drawn that interconnects the most dorsal aspect of the flexion creases to each other and to a point lateral to the nail plate. The nondominant side of the finger is usually the ulnar side of the index finger, long finger, and ring finger. For the thumb and little finger, release via an incision on the radial side of the digit.

Culture any purulent material that may be present. The most common infecting agent in these wounds is *Staphylococcus epidermidis,* and polymicrobial infections are common. It is important to débride all nonviable tissues while preserving neurovascular structures that are not affected. Depending on the spread of the material, a carpal tunnel release may be required. In addition, open all involved tendon sheaths and the radial and ulnar bursae. Irrigate all involved structures thoroughly. Do not attempt to neutralize any chemicals because the neutralizing chemicals often cause damage. Pack the wound with wet (saline-soaked) gauze, and be prepared for further débridement in the operating room if necessary 24 to 48 hours later. Splint the involved extremity in a safe position (see Chapter 13). If the injected material was radiopaque, then a postoperative X-ray may help to determine if all material was removed.

Postoperative Care

Ensure the wound is clean and clear of devitalized tissue through as much débridement as is necessary. Whirlpool therapy may be used postoperatively to provide additional débridement. Elevate the involved extremity when the patient is in a splint. Attempt to start activity in the extremity as soon as possible to decrease the amount of contractures. In subsequent surgeries, attempt to close parts of the wound that are clean and granulating. Use a skin graft or synthetic/acellular dermal matrix and a skin graft to close wounds. Alternatively, severely contaminated wounds can be closed by secondary intention. Begin the patient on hand/occupational therapy as soon as possible, because therapy is a major determinant of the patient's ultimate level of function.

16 Hand and Forearm Tendon Injuries

Injuries of the distal upper extremity range from simple lacerations to complex open blast injuries involving destruction of vital soft tissue, nerve, and vascular structures (**Fig. 16.1**). Effective evaluation and treatment requires detailed attention to the mechanism, time, and level of injury. The initial evaluation of an injured hand or forearm consists of a complete assessment for bony, vascular, and soft tissue injuries. Complex injuries mandate prioritizing reconstruction. First, the osseous structures are stabilized with internal or external fixation methods. Following rigid stabilization of the extremity, soft tissue repair is undertaken to protect and provide minimal tension over delicate vascular and nerve reconstructions. When loss of soft tissue is extensive, priority is placed on bony stabilization and revascularization. Soft tissue coverage is then employed, and the soft tissue is allowed to stabilize and heal for 3 weeks. Tendon and nerve reconstruction is delayed until soft tissue coverage has stabilized.

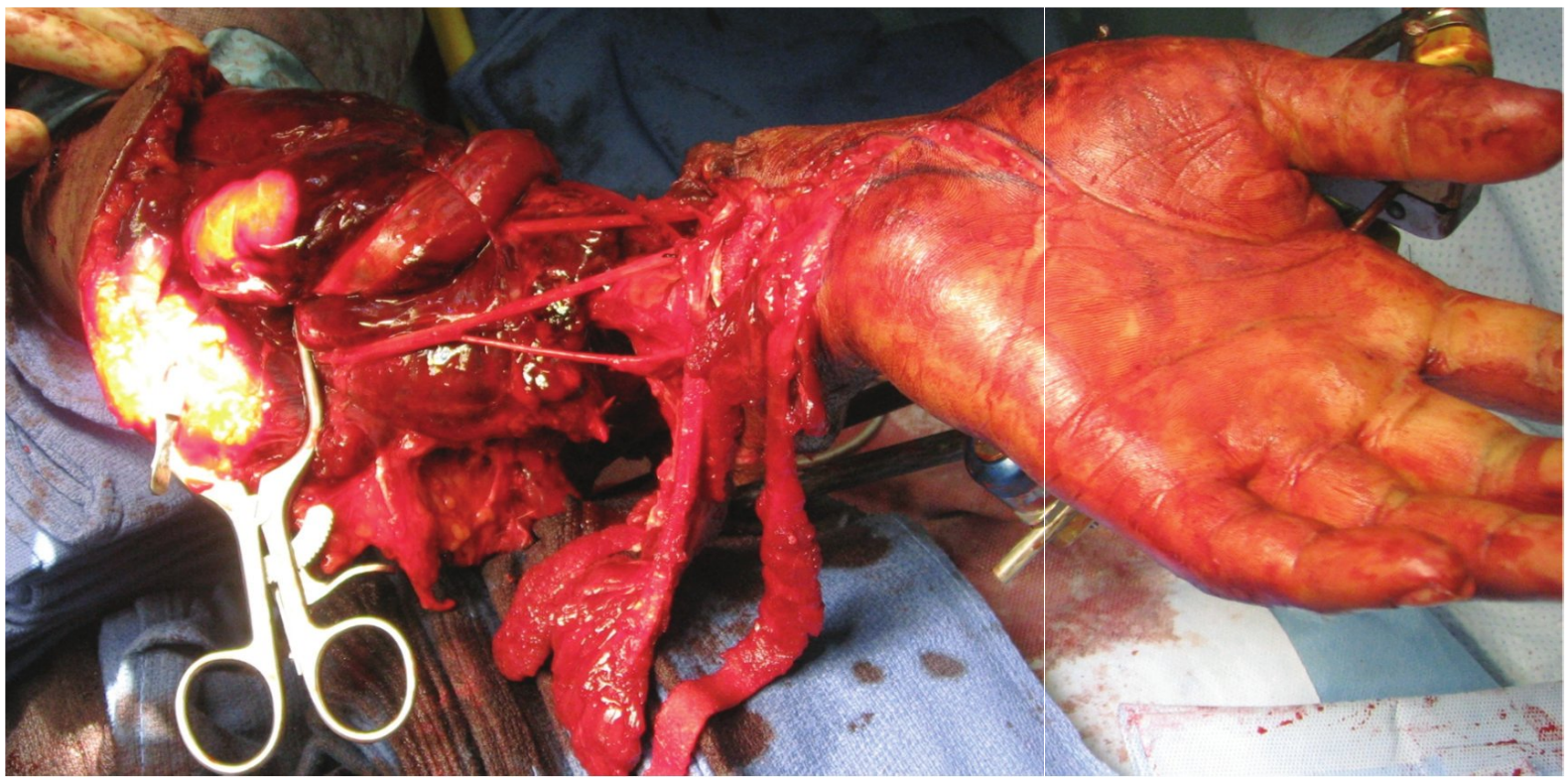


Fig. 16.1 Complex forearm injury involving tendons, neurovascular structures, muscle, and bone.

Tendon injuries of the forearm and hand range from a simple incomplete laceration of a single tendon to laceration and structural loss of multiple musculotendinous units. The mechanism of injury dictates the method of repair. Simple tendon lacerations are repaired directly with any familiar tendon repair techniques (**Fig. 16.2**). The repair of tendon lacerations that are the result of blast and avulsion injuries is usually delayed because the extent of tendon damage is not immediately known. A delay of 3 to 4 weeks allows the determination of viable tendon, at which point, tendon reconstruction with tendon grafts is performed. Over the course of the delay, the tendon path and muscle length can be preserved utilizing Silastic tendon rods sutured to the injured tendon ends.

Extensor Tendon Injuries

Patients with extensor tendon injuries present with obvious lacerations over the involved tendon or palpable pain in the region of a closed injury. The resting hand position will display the involved digit in flexion secondary to the loss of the counterbalancing extensor. The patient will also be unable to extend the involved digit actively while the palm of the hand is face down on a flat surface (**tabletop test**).

Extensor tendon injuries are commonly the result of open lacerations, but they also occur secondary to a variety of closed etiologies. Closed traumatic rupture of the extensor tendon includes but isn't limited to rupture of the extensor tendon at distal insertion of the distal phalanx (**mallet finger**), central slip from the dorsum of the middle phalanx (**boutonnière deformity**), and rupture of the extensor pollicis longus associated with radius fractures. Patients with rheumatoid arthritis develop attrition ruptures at multiple levels that can also present in a similar fashion.

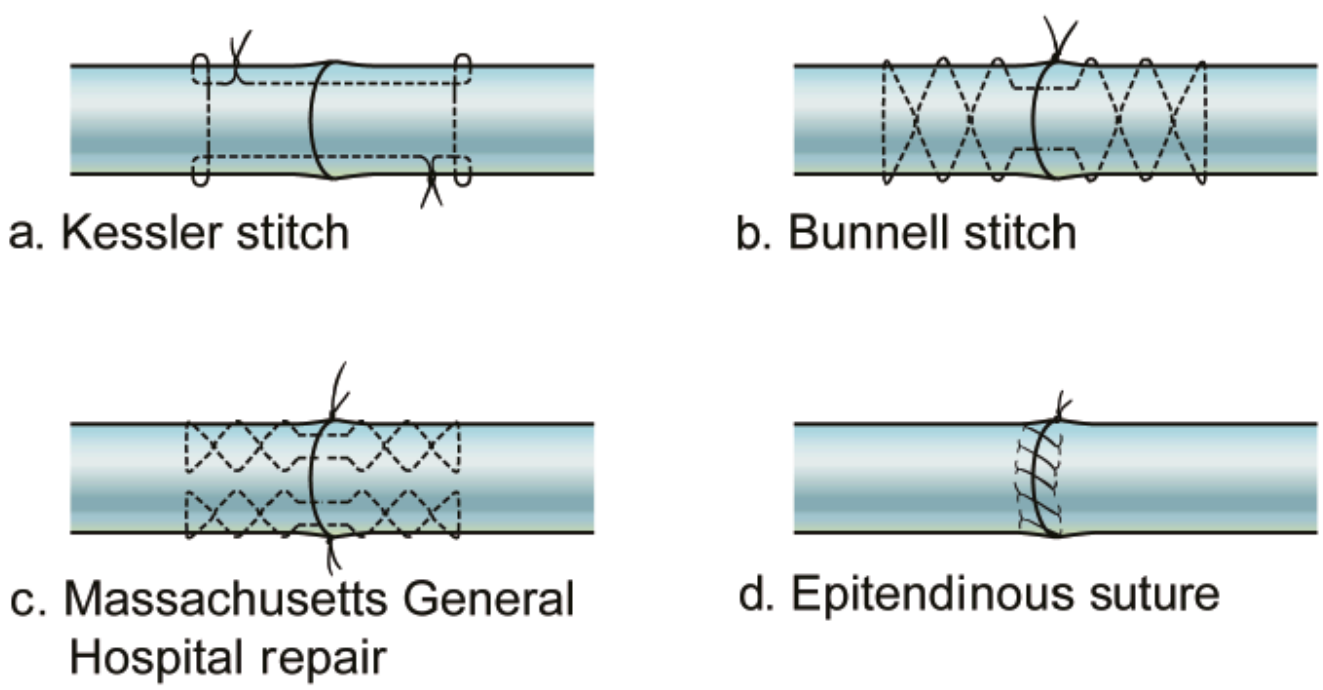


Fig. 16.2 Tendon injury repairs. **(a)** Modified Kessler stitch. **(b)** Bunnell stitch. **(c)** Massachusetts General Hospital repair. **(d)** Epitendinous suture.

Anatomy

The extensor compartments of the hand are separated into six dorsal compartments:

1. Abductor pollicis longus and extensor pollicis brevis.
2. Extensor carpi radialis longus and brevis.
3. Extensor pollicis longus.
4. Extensor indicis and extensor digitorum communis.
5. Extensor digiti minimi (quinti).
6. Extensor carpi ulnaris.

Longitudinally the extensor tendon is divided into nine zones from its course from the muscle to insertion into the distal phalanx (**Fig. 16.3**):

- *Zone I:* Distal interphalangeal joint.
- *Zone II:* Middle phalanx.
- *Zone III:* Proximal interphalangeal joint.
- *Zone IV:* Proximal phalanx.
- *Zone V:* Metacarpophalangeal joint.
- *Zone VI:* Metacarpal.
- *Zone VII:* Carpus.
- *Zone VIII:* Distal forearm.
- *Zone IX:* Musculotendinous junction proximal forearm muscle.

The thumb has only five zones (**Fig. 16.3**):

- *Zone I:* Interphalangeal joint.
- *Zone II:* Proximal phalanx.
- *Zone III:* Metacarpophalangeal joint.
- *Zone IV:* Metacarpal joint.
- *Zone V:* Carpus.

The extensor tendon zones are a useful tool for describing the level of injury; they also correlate to functional prognosis. For ease of reference, the odd-numbered zones are located over the joints and the even-numbered zones are located over the bone.

In the proximal forearm, the extensor digitorum communis tendons arise from a common muscle belly and disallow independent extension of the middle and ring fingers. The exceptions are the index and small fingers, which have their respective independent extensors (extensor indicis proprius, extensor digiti quinti). In zone VI, the juncturae tendinum connect the long, ring, and small finger tendons, allowing approximately 30 degrees of extension of the MCP joint even if the specific extensor digit to that tendon is cut. This anatomical configuration may confuse physical examination findings when obvious lacerations suggest proximal injury.

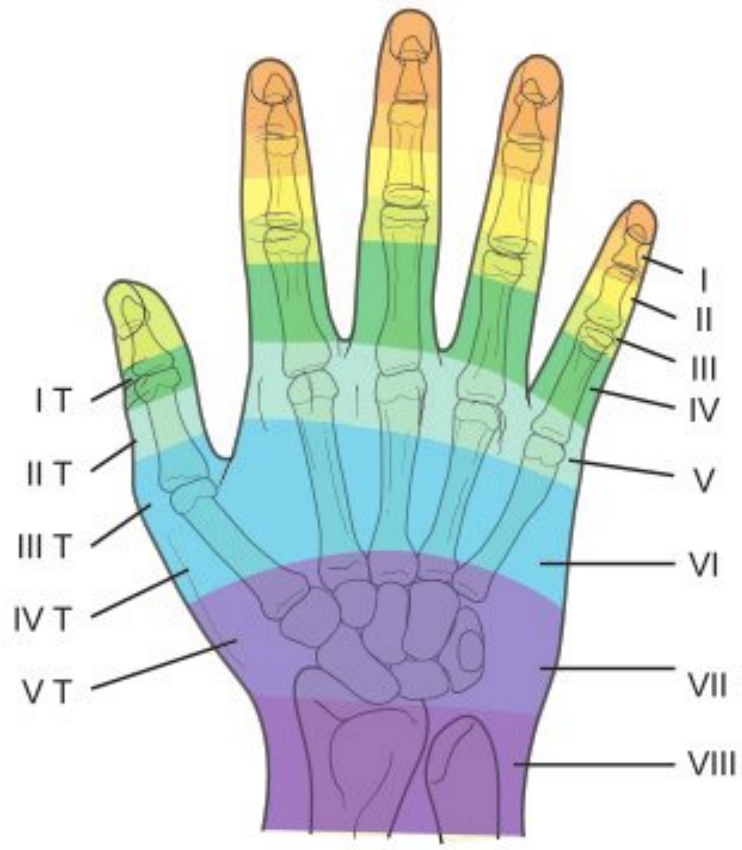


Fig. 16.3 Extensor tendon zones.

Timing of Repair

The timing of a repair depends primarily on the extent of the injury. Simple extensor tendon lacerations may be repaired easily in the emergency room with adequate local anesthesia. However, complex injuries of multiple tendons at many levels or with gross contamination require repair in the operating room, which allows the patient the benefit of sustained anesthesia in a tourniquet-controlled environment. Tendon injuries that are not associated with ischemia-related injury to the hand or digit are repaired within 1 week. If it is not feasible to repair the tendon at the initial evaluation, the wound is irrigated and temporarily closed and the patient is placed in a volar splint with wrist, MCP, and IP joints in slight extension.

Treatment

Due to the complex architecture of the extensor tendon, the treatment regimen and aftercare are dependent on area of injury. Generally, complete open lacerations are repaired acutely, while closed and partial (< 50%) injuries can be treated with appropriate splinting to allow healing.

Zone I

Mallet deformities are classified into four types:

- *Type I:* Closed laceration with or without fracture of the distal phalanx (less than one-third of the articular surface).
- *Type II:* Open injury without fracture of the distal phalanx.
- *Type III:* Open injury with loss of skin and subcutaneous cover.
- *Type IV:* Fracture of the distal phalanx involving one third or more of the articular surface.

Closed mallet deformities (type I) are treated by splinting the DIP joint in extension for 6 weeks. The splint is isolated to the DIP joint and spares the PIP joint. Open injuries are repaired by dermatotenodesis. The skin and tendon are repaired in a composite fashion with mattress or continuous 4–0 monofilament nonabsorbable sutures.

The addition of K-wire fixation of the DIP joint in extension for 6 weeks is advocated in type III/IV injuries. K-wire fixation of the DIP joint should also be considered in all pediatric zone I extensor tendon injuries because of the high incidence of noncompliance with splinting.

Zone II

Dermatotenodesis is also recommended in zone II, with mattress or continuous 4–0 monofilament nonabsorbable sutures for open injuries.

Zone III

Injuries at the PIP joint level involve the central slip and lateral bands. Disruption of the central slip causes volar displacement of the lateral bands. This results in a configuration in which the PIP joint is fixed in flexion and the DIP joint is fixed in extension—**the boutonnière deformity**. Closed injuries at this level may not be clinically apparent in the initial period after injury and usually develop 2 to 3 weeks after central slip rupture secondary to progressive migration of the lateral bands.

Closed acute boutonnière deformity is treated with either splinting of the PIP joint in full extension or K-wire fixation of the PIP joint in extension. Splintage places the PIP joint in maximum extension with the MCP and DIP joints free for 6 weeks. Active and passive ROM is encouraged in the DIP joint, while the PIP joint is held static in extension. Open injuries of the central slip or lateral bands are repaired directly. The lateral bands are repaired with 5–0 or 6–0 monofilament nonabsorbable

mattress sutures. Complete laceration of the central slip is repaired with 4–0 monofilament nonabsorbable modified Kessler or Bunnell sutures. The patient is then splinted with the wrist in 15 to 30 degrees extension and the MCP and PIP joints in full extension.

Placing K-wires obliquely across the PIP joint is a reliable way to hold the joint in firm extension for closed injuries or in complex cases of soft tissue loss. K-wires are utilized for 3 weeks, after which they are removed and the patient is placed in a PIP joint extension splint with the MCP and DIP joints free, similar to that described above, for an additional 3 weeks.

Zones IV and V

The extensor tendon over the MCP joint and the proximal phalanx is composed of the central slip and the sagittal bands. At this level, injuries of the extensor tendon are associated not only with open injuries, but also with closed injuries secondary to forceful flexion or extension. This is most common in the middle finger and is usually secondary to a tear of the radial sagittal band. Rupture of the radial or ulnar sagittal bands causes contralateral subluxation of the central slip. Physical examination reveals incomplete finger extension with unilateral displacement of the tendon.

The central slip is repaired primarily with 4–0 monofilament nonabsorbable modified Kessler or Bunnell sutures. The sagittal bands are repaired with 5–0 monofilament nonabsorbable horizontal mattress sutures. In cases in which there is loss of substance of the sagittal band mechanism, the tendon should be centered on the MCP joint by either suturing the transverse fibers to the joint capsule or tethering the tendon with the juncturae tendinum or a retrograde slip of the tendon. Splinting in these zones after repair is with the wrist in 45 degrees extension, the MCP joint in 15 degrees flexion, and the PIP joints in full extension.

Open injuries in zone V are also associated with human bites, the so-called **fight bite** wound. In this case, the contaminated wound should be explored and the joint inspected if the capsule is violated. The wound is then cultured, thoroughly irrigated, and left open. The associated tendon laceration is repaired secondarily in 5 to 7 days, depending on the status of the surrounding soft tissue. The patient is treated with Augmentin 875 mg twice a day (clindamycin for penicillin-allergic patients) for 10 days. Patients that present with an obvious infection after a human bite injury are admitted and placed on Unasyn. Refer to Chapters 14 and 15 regarding management of these injuries.

Zones VI and VII

Tendon injuries in zones VI and VII are usually secondary to open lacerating injuries. Fortunately, these injuries have the best prognosis due to the well-defined tendon substance and nourishing paratenon. Tendons are repaired in these regions with four-strand core sutures with the knots buried and an epitendinous suture. A modified Kessler suture with a 3–0 looped Supramid (S. Jackson, Inc.) suture will facilitate four-strand core sutures with one knot. The epitendinous repair is performed with a 6–0 continuous nylon suture. In zone VII, the extensor retinaculum is partially excised longitudinally over the repair to provide adequate excursion and to prevent formation of adhesions. Repairs in this zone are splinted with the wrist in 45 degrees extension, the MCP joint in 15 degrees flexion, and the PIP joints in full extension.

Zones VIII and IX

Proximal and distal forearm injuries to extensor compartments of the forearm occur from the extensor origin at the lateral epicondyle to the wrist. In the proximal forearm, lacerations involve the muscle belly of the involved digit extensor. These injuries commonly include laceration of the radial sensory nerve and significant hematoma. Penetrating wounds in this region are explored under tourniquet and irrigated, and all hematoma is evacuated. Repair of the muscle belly is done with 3–0 PDS figure-eight sutures.

In the distal forearm, lacerations occur in the distal muscle belly, in the musculotendinous junction, or just proximal to the wrist. At the junction of the tendon and muscle, the fascial margins are identified within the muscle and sutured to the distal tendon end using a 3–0 looped Supramid-modified Kessler suture. The fascial margins are repaired around the junction with a 4–0 PDS suture. Injuries more distal to this region are repaired similarly to zone VI and VII lacerations.

The extremity is splinted in an elbow-immobilizing fashion after repair for 4 weeks. The elbow is placed in 90 degrees flexion, the wrist in 45 degrees extension, MCP joints in 15 degrees flexion, and IP joints in full extension.

Flexor Tendon Injuries

Patients who present with flexor tendon damage will have disruption of the normal resting arcade. The loss of one or both flexor tendons will result in unbalanced extension of the involved digit. Usually lacerations will give clues to the level of injury. Deep lacerations of the volar surface of the finger and hand not only place the superficialis and profundus tendons at risk, but also indicate that neurovascular injury should be suspected. A thorough examination of these patients includes radiographs, sensory evaluation, and isolated motor testing of the superficialis and profundus tendons (see Chapter 12, **Fig. 12.1** and **Fig. 12.2**.). Isolated injury to one flexor tendon may still allow flexion of the digits at the PIP joint. Therefore, to test the integrity of the profundus tendon, the PIP joint is held in extension while flexion of the DIP joint is initiated.

Flexor Tendon Injury Zones

The volar hand area is divided into five zones that describe flexor tendon injuries (**Fig. 16.4**):

- *Zone I:* Distal to the insertion of the flexor digitorum superficialis (FDS).
- *Zone II:* **No man's land;** distal palmar crease to zone I.
- *Zone III:* Distal edge of the transverse carpal ligament to the distal palmar crease.
- *Zone IV:* The carpal tunnel.
- *Zone V:* Distal portion of the forearm.

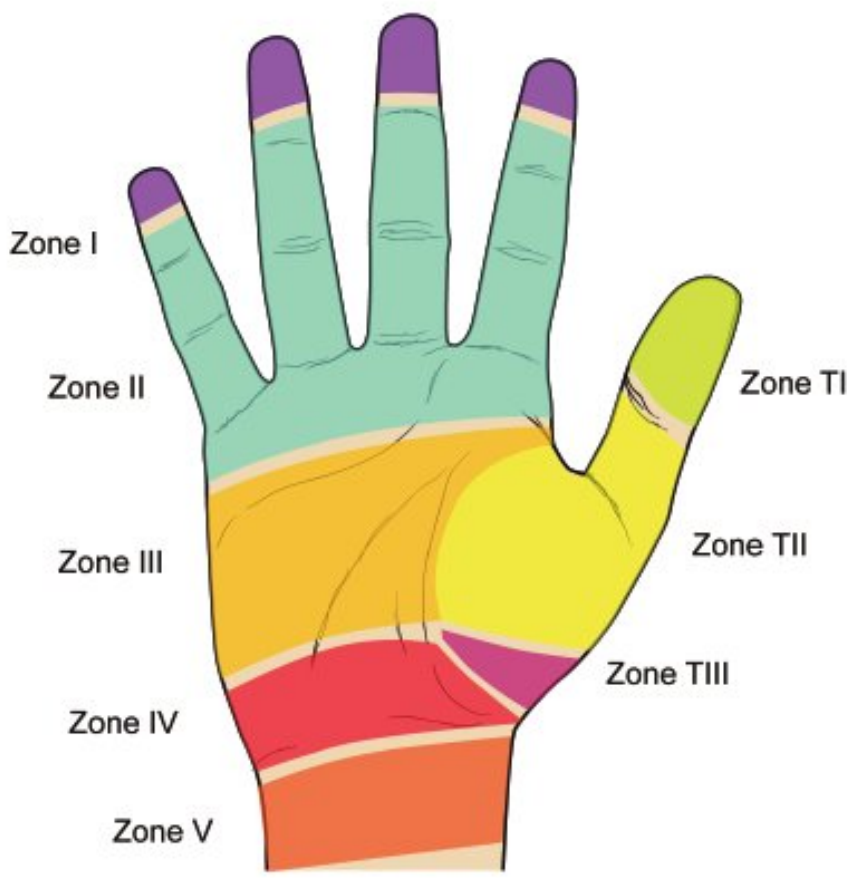


Fig. 16.4 Flexor tendon zones. TI, TII, and TIII are the flexor tendon zones of the thumb.

Flexor Tendon Repair (Fig. 16.1)

- Perform repair within 48 to 72 hours.
- Acutely place patient in extension block splint (see Chapter 13, **Fig. 13.6**).
- Delay tendon repair until bony stabilization and soft tissue decontamination.
- Delay tendon repair when infection is present.
- Perform tendon repair in the operating room to allow greater exposure for tendon retrieval.
- Repair both the FDS and the flexor digitorum profundus (FDP) when injured.
- Extension block splint after repair.

Techniques for tendon retrieval:

- Flex wrist and MCP joint to advance tendon.
- Mobilize proximal end with a suction catheter.
- Massage proximal end into wound using an Esmarch bandage or manually.
- Grab end with 18-gauge needle or skin hook.
- Suture tendon end to rubber catheter to pass under pulleys or into tendon sheath.

Zone I Repair

Repair of the FDP tendon distal to the insertion of the FDS takes into consideration the status of the insertion of the tendon onto the base of the distal phalanx. If an avulsion fragment is present with the FDP attached, the repair is performed with composite pin fixation of the tendon to the distal phalanx. When the FDP is detached, the distal portion is secured to the distal phalanx with a bone suture anchor (3–0 or 4–0) or pulled through the distal phalanx using a double-armed 3–0 Supramid suture and tied over a button.

In the repair of more proximal zone I injuries, the proximal tendon is retracted into the region of the middle phalanx. The distal stump that is attached to the distal phalanx is exposed by dissection of the A5 pulley. Care should be taken not to disrupt the A4 pulley. A core suture is placed in the proximal tendon end (3–0 Supramid), and the needle is passed under the A4, pulled, and sutured into the distal tendon stump.

Zone II Repair

Due to the technical difficulty of repair and poorer functional outcomes, zone II is colloquially known as **"no man's land."** Exposure for repair of zone II flexor tendon injuries requires wide exposure with proximal and distal Brunner incisions, as well as dissection of the flexor tendon sheath and pulley system. The A2 and A4 pulleys should be preserved during dissection. Once the proximal tendon end is identified, a core suture (3–0 Supramid) is placed and pulled under the pulleys so that repair can be performed between a pulley sheath window. The proximal end can be held tension free by placing an 18-gauge needle in the end through the pulley and the sheath. Repair is then performed with four-strand core sutures and an epitendinous 6–0 running Prolene. Extreme care is taken in zone II to provide a repair that is flat without fraying to avoid tethering during tendon excursion. Partial resection of the adjacent pulley is acceptable to allow excursion of the repair.

Zones III, IV, and V

Injuries proximal to zone II have an improved prognosis with good functional recovery. However, in these zones there is a higher propensity to injure tendons of multiple digits and major nerve and vascular structures of the hand. Repair in these zones is aided by extending palmar and forearm incisions for exposure and tendon retrieval. Place a tourniquet on the patient's upper extremity to achieve hemostasis. The tourniquet should be raised to 100 mm Hg above the systolic blood pressure. It can be left on for 2 hours, but needs to be deflated for 20 minutes prior to reinflation for 2 more hours (5 minutes of deflation for every 30 minutes of inflation).

Always flex the interphalangeal joints, the MCP joint, and the wrist to deliver the distal ends of the lacerated tendons into the operative field. The lacerated ends of the wound can be extended proximally and distally to allow for exposure of the tendons, nerves, and vessels. Any bone fixation should precede any repair of soft tissues, as described in Chapter 17. Identify all the flexor tendon ends. Oftentimes, tendons are hidden in a small hematoma in the tendon sheath. Dissect out all the tendons and identify their function by pulling on the ends and noting their action. Next, tag them by performing tendon repair on the distal end of the tendon, with looped Supramid sutures (**Fig. 16.1**; we use the modified Kessler technique). Once all the ends are accounted for, match them to the proximal ends of the tendons, based on the position of the tendon ends in the proximal forearm. Having accounted for all the distal tendons and having matched them to their

counterparts in the forearm, begin repairing the tendon by completing the repair from the deepest tendon to the most superficial. Remember to perform epitendinous repair with 6.0 Prolene suture (**Fig. 16.1**) after the core suture repair. If required, perform revascularization of the hand by anastomoses of the severed ends of the ulnar or radial artery along with the cephalic vein or the venae comitantes. Finally, repair the median or ulnar nerve injuries. These repairs should involve lining up the fascicles and vasoneurium in the nerve and performing epineural repair using 9–0 nylon suture. The repair can be wrapped with 3- to 4-mm neurotube tubing, if desired. All attempts should be made at primary repair, since this technique heralds the best prognosis. Mobilize the nerve to allow tension-free repair.

17 Hand Vascular Injuries and Digit Amputations

Vascular injuries of the hand and digital amputations require immediate attention by a specialist to ensure proper revascularization and optimum functional results for the patient.

Vascular System of the Hand

The hand is supplied with blood by the ulnar artery and the radial artery. The radial artery is dominant 57% of the time, whereas the ulnar artery is dominant 21.5% of the time (the two arteries are codominant the remaining 21.5% of the time). The radial artery divides into a small superficial palmar artery and large dorsal radial branch. The ulnar artery divides into superficial and deep branches. Next, the dorsal radial branch gives off the princeps pollicis and radial digital index branch and then anastomoses to the deep branch of the ulnar artery to form the deep palmar arch. The superficial branch of the radial artery anastomoses to the superficial ulnar artery to make the superficial palmar arch. The common digital arteries arise from the superficial arch. The digital branches arise from the common digital arteries. The vascular supply is regulated by metabolic demands, sympathetic tone, hormonal factors, and environmental factors.

Physical Examination

Basic tenets of the vascular examination of the hand include the following:

- Test each digit for capillary refill, sensation, edema, color, gangrene, and petechiae.
  - If you are having difficulty performing a capillary refill test on the digits due to ecchymosis or avulsion of skin, you may use Doppler ultrasonography to determine the integrity of the digital vessels.
  - For continuous monitoring of the perfusion to a finger, a pulse oximeter may be used on the involved finger.
- Test the proximal blood supply by taking the blood pressure in both arms for a difference comparison.

- Perform an Allen test (see Chapter 12).
  - If you cannot feel a pulse, then check the wrist for radial and ulnar Doppler ultrasound signals and use Doppler ultrasonography to perform an Allen test. Test the integrity of the palmar arches.
- Utilize angiography when needed. If you are unable to perform the above exam or if the zone of injury to the vessels is in question, then an angiogram may be helpful.
- Finally, make sure to palpate the compartments of the forearm/hand as well as measure the compartment pressures with a Stryker needle or arterial line to rule out compartment syndrome and the need for a fasciotomy (see Chapter 19).

Arterial Injuries

- *Presentation:* Pallor, lack of capillary refill or pulse distally, pulsatile bleeding. Intimal damage may present with late thrombosis.
- *Mechanism:* Crush, stab, or avulsion injuries.

The indications for repair of **radial or ulnar artery** injuries are

- Absolute indication: Hand or digital ischemia.
- Relative indications: To improve cold intolerance, to provide better circulation for wound healing.
- Indication for digital vessel repair: Digit ischemia.

Treatment

Forearm and Hand Injuries

In cases of sharp injuries to the artery, direct repair can be performed. When there has been a crush injury to the artery or an avulsion, resection of the injured portion and the use of vein grafts for reconstruction are required. Signs of vessel damage include telescoping of the vessels, petechial hemorrhages on the vessel wall, vessel thrombosis, cobwebs in the vessels, or poor flow from the proximal end of the injury. In these cases, reversed vein grafts can be used. The dorsal hand veins can serve as a good donor site. If the patient requires extensive forearm fracture reduction prior to repair of the artery, Silastic shunts can be used as a temporary way to establish perfusion to the hand until the fracture is stable in the operating room. Then arterial reconstruction is performed

at that setting. A forearm fasciotomy should be performed if the patient has compartment syndrome or compartment syndrome is anticipated.

Digital Vessels

Only one digital artery is required for adequate perfusion. Therefore, injuries to both arteries require repair of at least one artery. Venous outflow is also critical; intact skin bridges may maintain capillary venous outflow. However, if repair of the digital veins cannot be performed, then venous outflow and signs of congestion should be followed, and outflow should be augmented with leeches if necessary.

Cannulation Injuries

A vessel injury can often occur from cannulation of the radial artery with an arterial line or arterial blood gas sampling. This can result in pseudoaneurysm formation, thrombosis, or arteriovenous fistula formation. The rate of thrombosis in the vessel is directly correlated to the duration of cannulation. If the patient has a loss of radial pulse, but does not have any digital ischemia, surgery is not needed. This can be managed conservatively. Anticoagulation should be considered in these cases. The treatment of these injuries entails surgical exploration and thrombectomy with direct arterial repair. Significant vessel gaps are repaired via reversed vein grafts harvested from the cephalic or saphenous veins. An arteriogram may be beneficial preoperatively or intraoperatively to identify the level and character of occlusion.

Hypothenar Hammer Syndrome

Hypothenar hammer syndrome, the most common cause of thrombosis in the upper extremity, results from repetitive trauma to the ulnar artery caused by the patient using the hand as a hammer. This syndrome usually occurs in laborers in their 50s who smoke. There is damage to the elastic lamina of the ulnar artery, and a thrombus and/or aneurysm can result. The thrombus may embolize.

The patient should refrain from the activity causing the trauma. Smoking could exacerbate the condition; therefore, smoking cessation is paramount. Hypothenar hammer syndrome may be treated medically with thrombolytics (urokinase, streptokinase, TPA). Alternatively, throm-

bectomy or resection of the involved portion of the vessel is required with either primary closure or reconstruction using vein grafts from the forearm veins or dorsal foot. In some cases, ligation of the artery allows for vasodilation and collateral flow. Keep a high index of suspicion for compartment syndrome.

Digit Amputation

Replantation

Whereas revascularization of a digit refers to restoration of blood supply to an incompletely severed digit, replantation refers to reattachment of and restoration of blood supply to a completely severed digit.

Indications for Replantation

- Amputation of the thumb.
- Multiple digit amputation.
- Partial hand amputation.
- Wrist or distal forearm amputation.
- Above-the-elbow amputation.
- Any amputation in a child < 12 years old.

Relative Indication

- Single-digit amputation distal to the insertion of the flexor digitorum superficialis (FDS).

Contraindications for Replantation

- If the severed digit has undergone warm ischemia for > 12 hours or cold ischemia for > 24 hours.
  - In the case of amputations proximal to the wrist, only a warm ischemia time of 6 hours and a cold ischemia time of 12 hours can be tolerated.
  - If the patient is not medically stable enough to undergo a long operation.
  - Relative contraindications are mentally unstable patients, smokers, and patients with diabetes mellitus.

Crush and avulsion injuries can be expected to have a higher failure rate for replantation. If a patient presents with a severely mangled digit with multiple levels of injury, replantation will likely be unsuccessful. Another predictor of poor outcome is the presence of a red line on the skin and on the neurovascular bundles.

Peri-Operative Considerations

The amputated digit or part should be transported to the emergency room wrapped in a saline-soaked sponge, placed in a plastic bag, and placed on top of ice. Do not let the finger freeze or be submerged in ice because frostbite will result. Take X-rays and photographs of the hand along with the amputated digit to determine the level of injury. Prior to replantation, the amputated part should be examined under a loupe or microscopic magnification to establish the integrity of the involved vessels. Use this information to determine if replantation is feasible.

Provide good fluid resuscitation for the patient and discuss the risks and benefits of the surgery with the patient so he or she can understand the procedure and the need for rehabilitation, and have realistic expectations.

In the operating room, first, the bone is shortened and fixed with a K-wire, and then the repair is undertaken in the following order: extensor tendons, dorsal veins, dorsal skin, flexors, arteries, and nerves. The sequence of veins, arteries, and flexor tendons is controversial. Vessel repair and anastomoses should be performed outside of the zone of injury. The liberal use of vein grafts and venous flow-through flaps will allow microsurgical repair in a region with minimal inflammation. If multiple digits are replanted at the same time, replantation should proceed part by part instead of finger by finger (i.e., same replantation step for each finger at the same time).

Postoperatively, splint the injury, place the patient in a comfortable warm room, and elevate the extremity. Leeches can be used to aid with venous congestion by providing the local anticoagulant hirudin and removing blood. Usually the leeches are placed on the fingertip and they are engorged in 30 minutes. The therapy is performed for 5 to 7 days. Prophylactic antibiotics such as third-generation cephalosporins or gentamicin or Bactrim can be used to avoid infection with *Aeromonas hydrophila*.

The best results are achieved with thumb, wrist, and distal FDS replants. Overall viability is reported at 80 to 90%

Complications

- Cold intolerance.
- Nonunion.
- Malunion.
- Joint contractures.
- Infection.

Fingertip Injuries

Tip avulsions and amputations are a subset of injuries that occur distal to the terminal arborization of the digital vessels. In this region of the distal phalanx, the digital arteries and veins are unable to be repaired microsurgically. Additionally, these injures commonly occur with concomitant nail avulsion and distal phalangeal fractures (tuft fractures). Repair of tip injuries requires attention to fracture reduction, nail repair, and soft tissue restoration.

Nail Repair

Nail anatomy is depicted in **Fig. 17.1**. Commonly after injury, patients will present with subungual hematomas that indicate disruption of the sterile matrix. Small subungual hematomas (<40% of the nail) are treated with aspiration of the subungual space and subsequent irrigation with an 18- or 20-gauge needle. When severe damage to the sterile matrix is suspected or larger subungual hematomas are present, removal of the nail plate and direct repair of the matrix is appropriate.

Complete nail plate avulsion injuries are repaired first by direct closure of the sterile matrix with 6–0 plain gut suture. Next, the germinal matrix is stented with a piece of fine gauze, foil, or the native nail plate with two vertical mattress 5–0 chromic sutures. Preservation of the germinal matrix will prevent synechia and allow growth of a new nail plate (**Fig. 17.2**). Patients should be counseled that with time the stent will be replaced by the growth of the new nail plate from beneath.

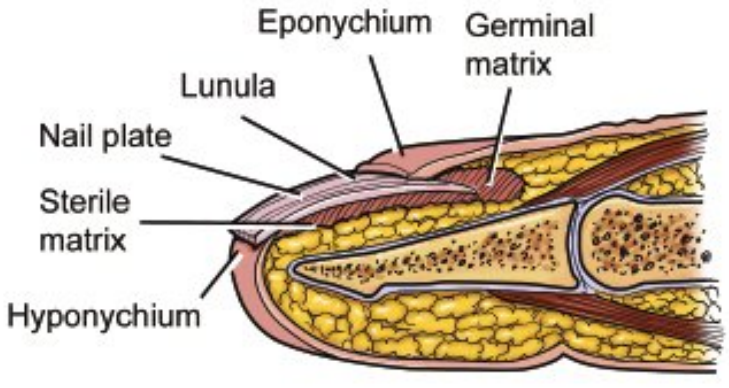
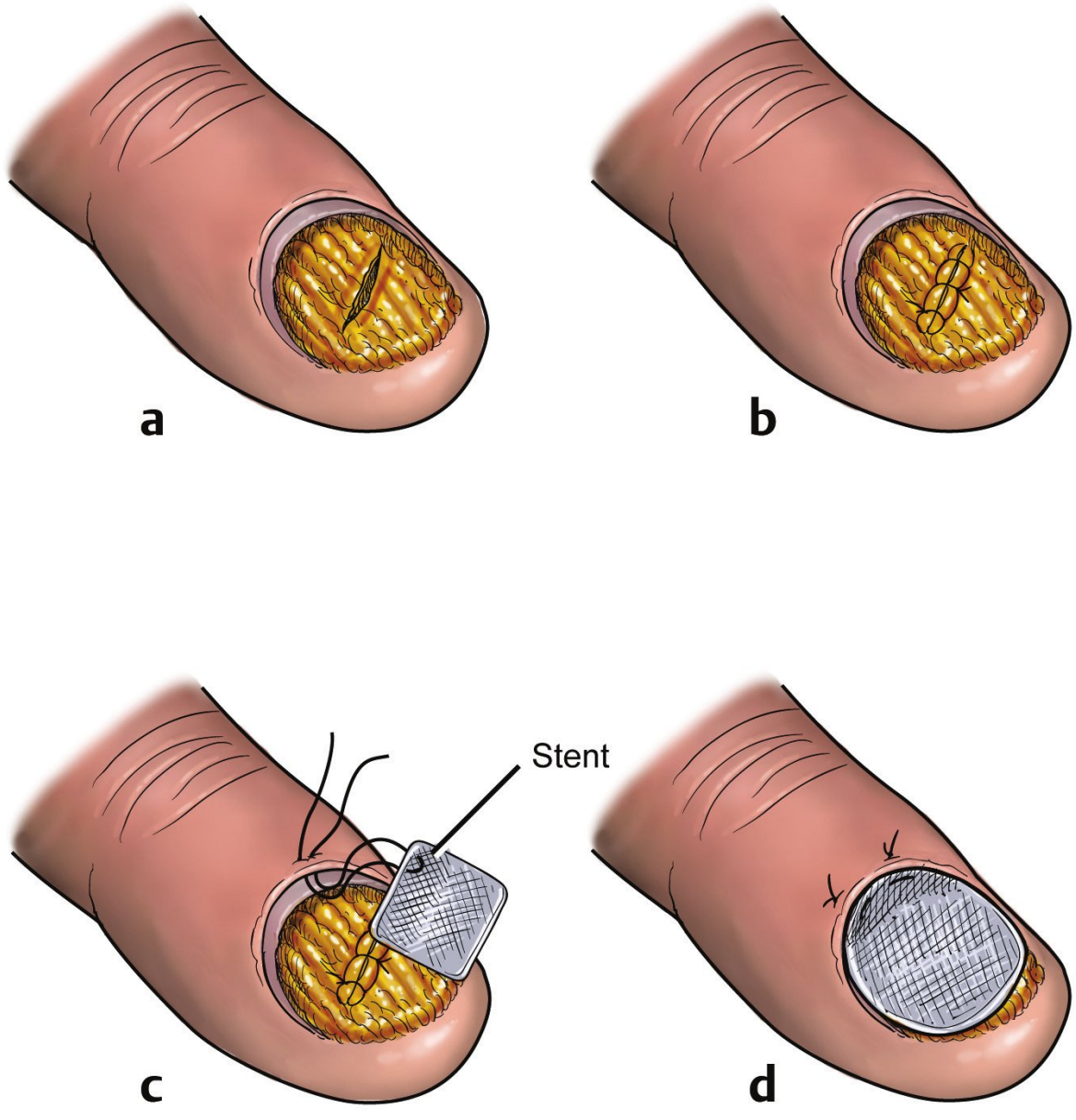


Fig. 17.1 Normal fingertip anatomy.



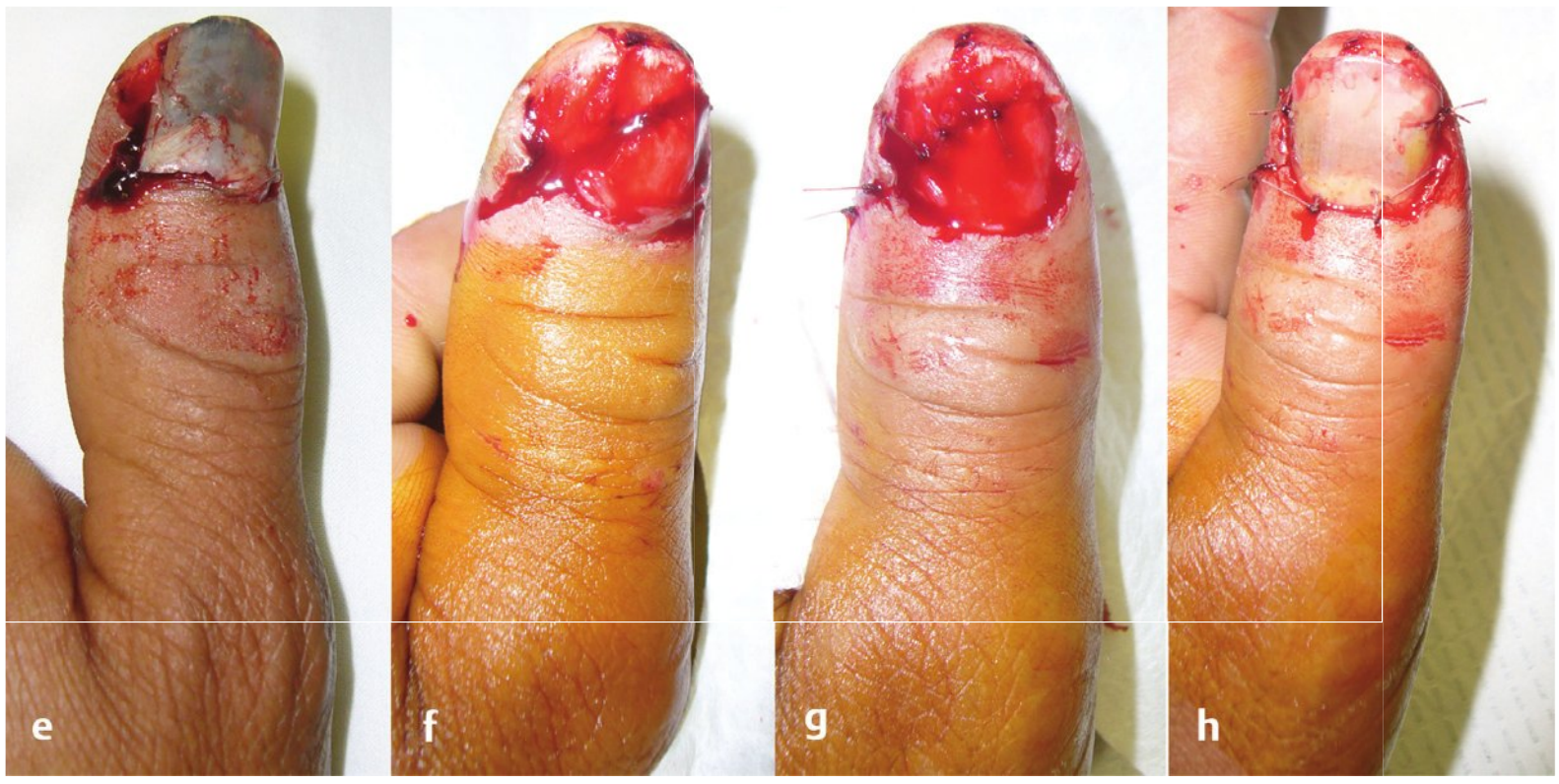


Fig. 17.2 Nail bed repair. **(a,b)** Suture repair of sterile matrix laceration. **(c,d)** Germinal matrix stenting. **(e–h)** Repair of nail bed in a patient with distal tuft fracture.

Tuft Fractures

The management of distal phalanx fractures is discussed extensively in Chapter 14. Tuft fractures occurring with tip injuries are simple and uncomplicated. Restoration of the normal soft tissue architecture by suturing and subsequent immobilization with an aluminum finger splint is adequate for stabilization. The insertion of an axial K-wire or 20-gauge needle is also useful to reduce the fracture fragments.

Soft Tissue Repair

Repair of the fingertip soft tissues depends on the degree of injury (amputation versus avulsion) and the availability of the amputated part. When avulsion of the tip is apparent, assess the avulsed fragment to determine its viability. If the avulsed fragment is cyanotic or ischemic, the fragment is amputated. If the avulsed fragment is viable, it is because the intact arterial capillary plexus between the tip and the fragment is providing perfusion. In these cases, the avulsed fragment is repaired by a suture to the tip with 4–0 nylon (5–0 chromic in children).

A primarily or secondarily amputated tip can be salvaged by removing the subcutaneous tissue from the overlying glabrous skin. The skin is then sutured to the tip as a full-thickness skin graft (**Fig. 17.3**). The injury is splinted and protected with an aluminum splint in place for 5 days.

If the amputated tip fragment is unavailable, repair is dependent on the size of the defect and exposure of the underlying structures. Small defects of the tip (< 1 cm) without exposed distal phalanx heal well by secondary intention. These injuries are dressed with Xeroform gauze, and patients are counseled to perform dressing changes twice a day. Large defects are closed with full-thickness skin grafts from either the hypothenar eminence or the forearm. When the distal phalanx is exposed, the wound is thoroughly irrigated and local flaps can be employed for closure if the surrounding soft tissue is not significantly devitalized (**Fig. 17.4, Fig. 17.5**).

Otherwise, the wound is dressed with Xeroform gauze and local wound treatment is followed until declaration of the viability of the surrounding soft tissues. Short-term follow-up allows for assessment of the patient's wound for closure with local or regional flaps.

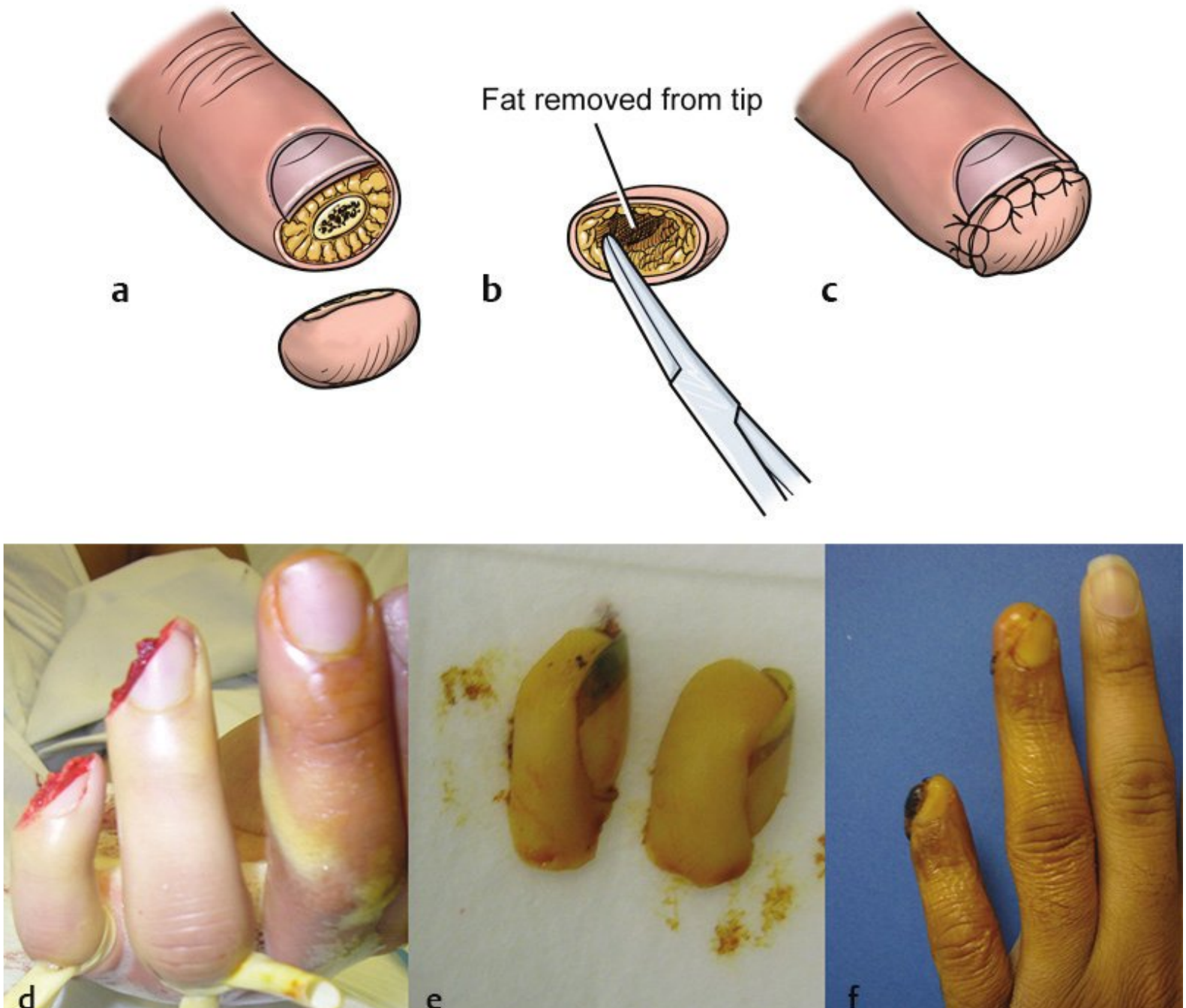


Fig. 17.3 **(a–c)** Composite repair of tip amputation with amputated portion placed as a full-thickness skin graft. **(d)** Presentation of distal amputation. **(e)** Amputated distal tuft. **(f)** Follow-up 1 month after repair.

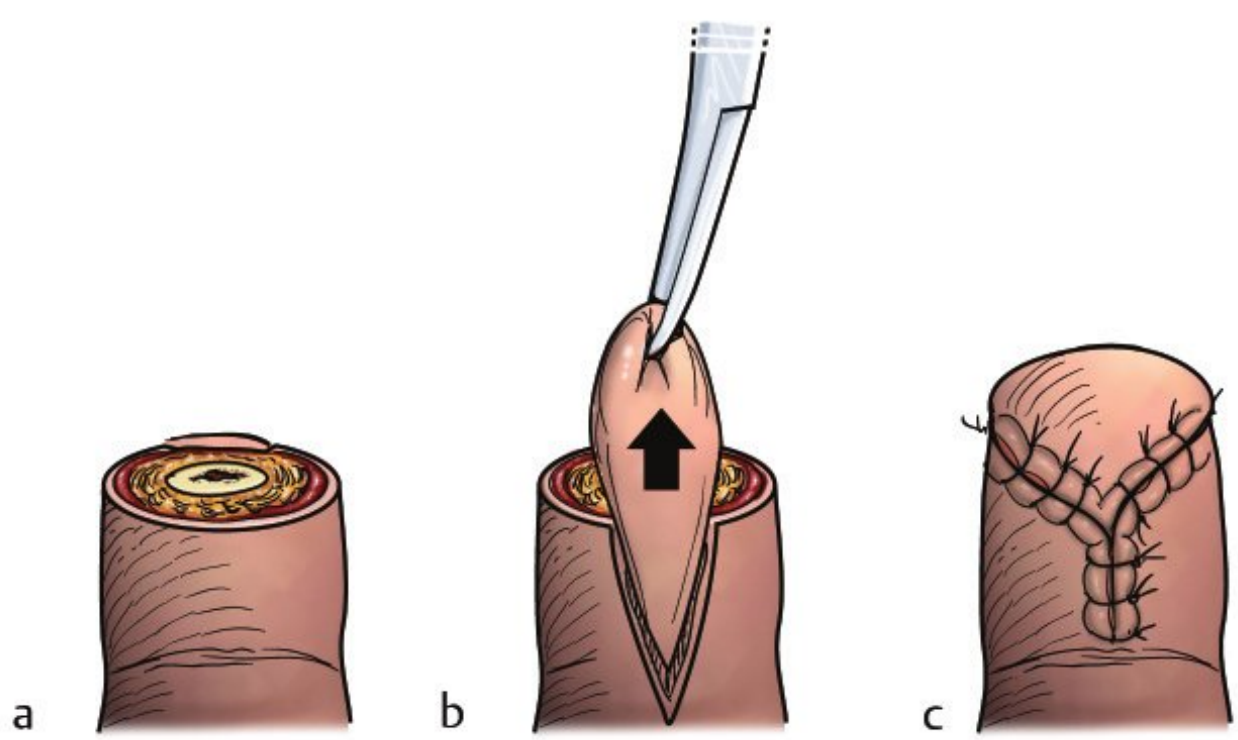


Fig. 17.4 **(a–c)** Volar V-Y advancement coverage of transverse tip injury.

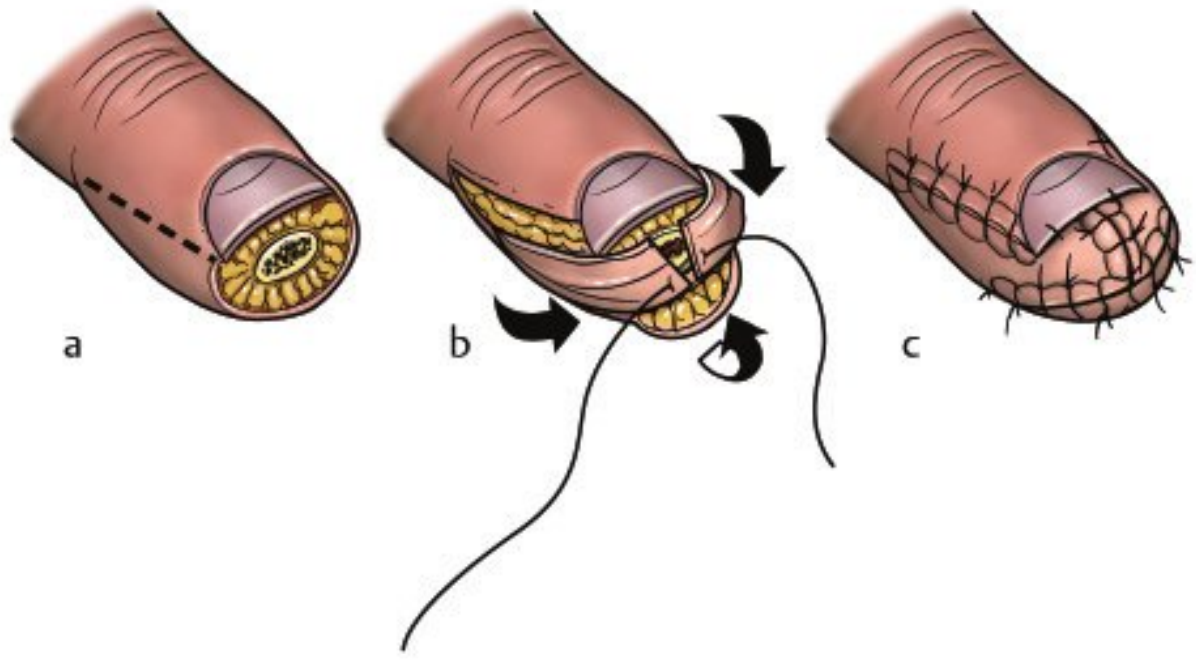


Fig. 17.5 **(a–c)** Kutler lateral V-Y advancement flap coverage of tip defect.

18 Upper Extremity Peripheral Nerve Injuries

Nerve injuries in the upper extremity occur as the result of a blast, crush injury, a penetrating blow, or due to an injury caused by a sharp object. Management is predicated on establishing nerve continuity in an environment that will allow nerve growth and regeneration. Wallerian degeneration of the axons occurs at the time of injury. **Reinnervation of the motor end plates before 18 months will prevent muscular atrophy and subsequent deformity.** Therefore, appropriate initial management of these injuries will confer successful results with minimal functional morbidity.

Classification of Injury (Fig. 18.1)

First Degree: Neuropraxia

First-degree injuries occur secondary to crushing, compressing, or stretching of the nerve. In these scenarios, the nerve architecture is not disrupted and there is nerve continuity. Conservative management including splinting of the involved extremity and physical therapy is appropriate. The nerve should recover in 3 months; otherwise, a second-, third-, or fourth-degree injury should be suspected, which would require operative intervention.

Second-, Third-, and Fourth-Degree Injuries

Injuries that disrupt the internal architecture of the nerve may consist of isolated axonal derangement of nerve fascicles with subsequent scar formation (axonotmesis/second degree). However, there may exist a scar conduction block at the fascicular level or across the entire nerve (third and fourth degree). An axonotmetic lesion will heal without surgical intervention if it allows nerve growth through the intact sheath at 1 mm per day or 1 inch per month. Injuries that heal with a scar block cause incomplete conduction across the nerve. These lesions may require internal neurolysis or excision and direct repair, depending on the conduction drop across the scar. Differentiation of the degrees of these lesions is determined by EMG and nerve conduction studies done at some time interval after the injury, if no nerve function returns.

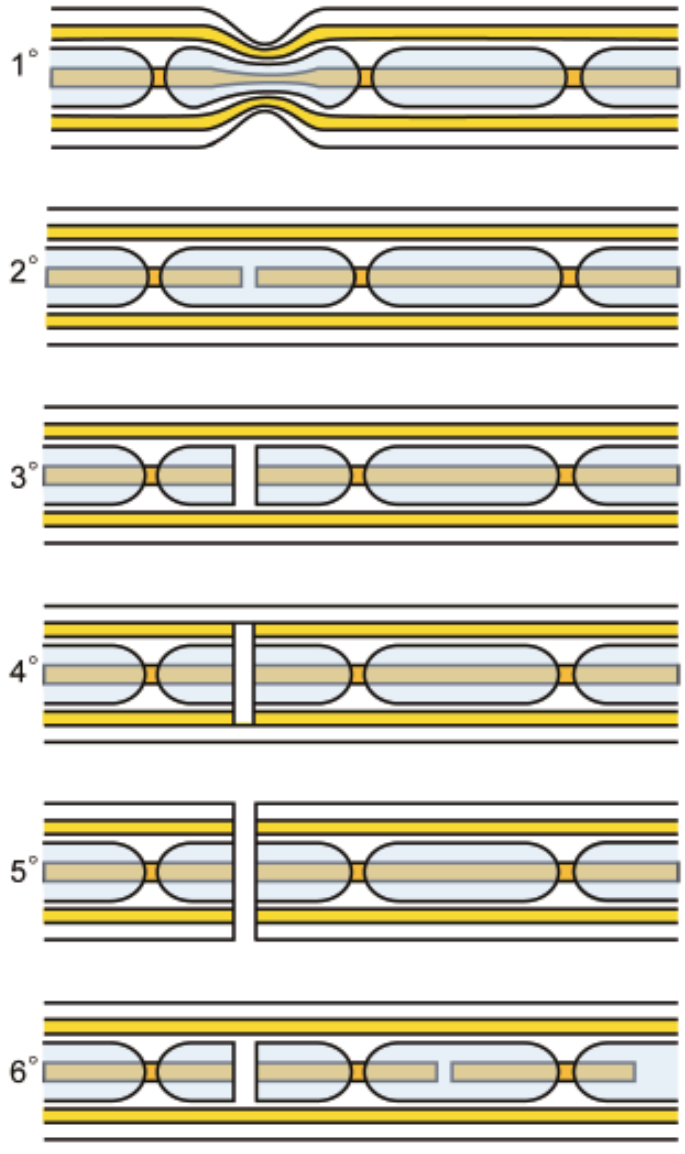


Fig. 18.1 The Seddon-Sunderland-Mackinnon classification of nerve injury.

Fifth- and Sixth-Degree Injuries

A complete disruption of the nerve is referred to as neurotmesis. Neurotmesis is repaired by direct coaptation or via nerve conduit grafts. The sixth-degree injury is multiple lesions along the length of the nerve. Due to the potential for longitudinal scar formation, these lesions require operative intervention.

Management

The location of the injury is obvious in patients who present with peripheral nerve injuries. Commonly, the injury is open and associated with specific trauma to an extremity. Nevertheless, a thorough physical examination is warranted to determine the degree of function loss. The motor examination should include assessment of all involved muscle groups with documentation of their strength. Sensory examination includes the assessment of two-point discrimination and response to light touch and vibratory stimulus.

The specific management of these injuries depends on the degree of injury and the mechanism. Generally, closed injuries—neuropraxic or axonotmetic—are managed conservatively. Recovery of function is

expected within 3 months. Open injuries are repaired primarily if the nerve and surrounding soft tissue do not have the potential for devitalization. Sharp lacerations of the nerve should be explored and repaired at the time of injury after the wound bed is decontaminated. **Repair is delayed in blast, crush, and avulsion injuries.** With these lesions the nerve is considered "stunned," with the potential for devitalization of nerve and tissues in the subacute period. Often these injuries are open. Therefore, the nerve should be explored and examined. If the nerve ends are in close proximity, the nerve is repaired. Otherwise, nerve ends are tagged for delayed repair in 4 to 6 weeks. Primary nerve grafting is not recommended in blast, crush, and avulsion injuries. In open injuries, nerve repair is performed after repair of bony and vascular damaged structures.

Brachial Plexus Injuries

Injury to the brachial plexus is suspected in patients who present with high-velocity wounds or direct penetration in the region of the cervical roots. These patients will present with gross loss of sensation and weakness of the involved upper extremity. Care must be taken to rule out associated injury to the cervical spine, thoracic outlet vessels, and shoulder girdle.

Evaluation

Physical examination is performed to determine the location of the lesion based on knowledge of the brachial plexus anatomy (**Fig. 18.2**). Examination includes assessment of sensory loss, motor function, and vascular integrity. In addition to the physical examination, radiographic evaluation of the cervical spine and involved upper extremity is performed. CT scans of the cervical region would reveal a cervical spine injury and assist in evaluation for root avulsion.

Management

Management of brachial plexus injuries is dependent on whether the lesion is an avulsion or a rupture outside of the spinal cord. Avulsion lesions will require delayed reconstruction after a thorough assessment of the functional anatomy of the plexus by EMG/NCS and SSEP studies. In the acute setting, these patients are treated conservatively with splinting and rehabilitation. The upper extremity is placed with the elbow in flexion and the hand and wrist in the safe position.

Lesions outside of the CNS (ruptures) are managed similar to isolated peripheral nerve injuries (**Fig. 18.3**).

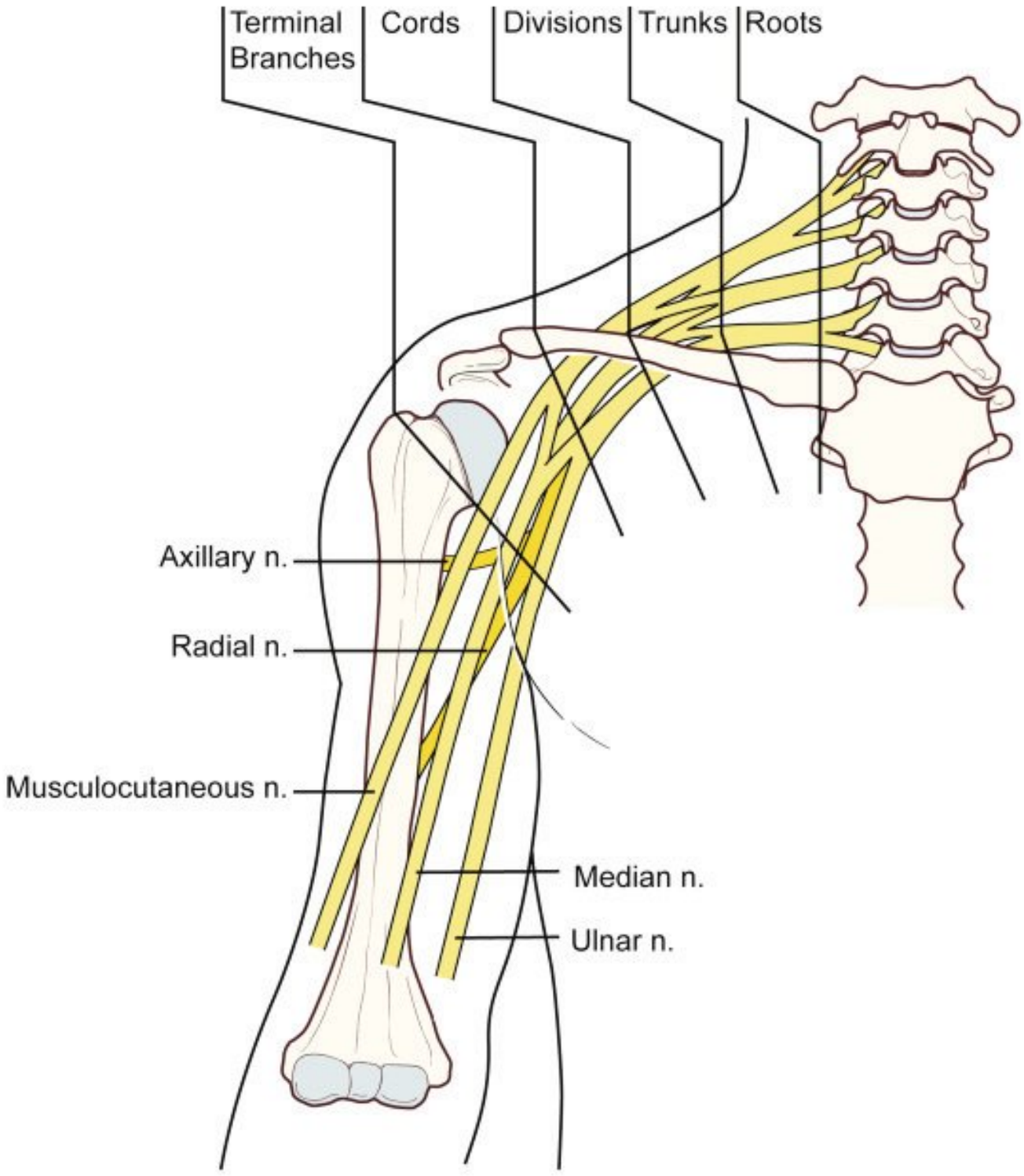
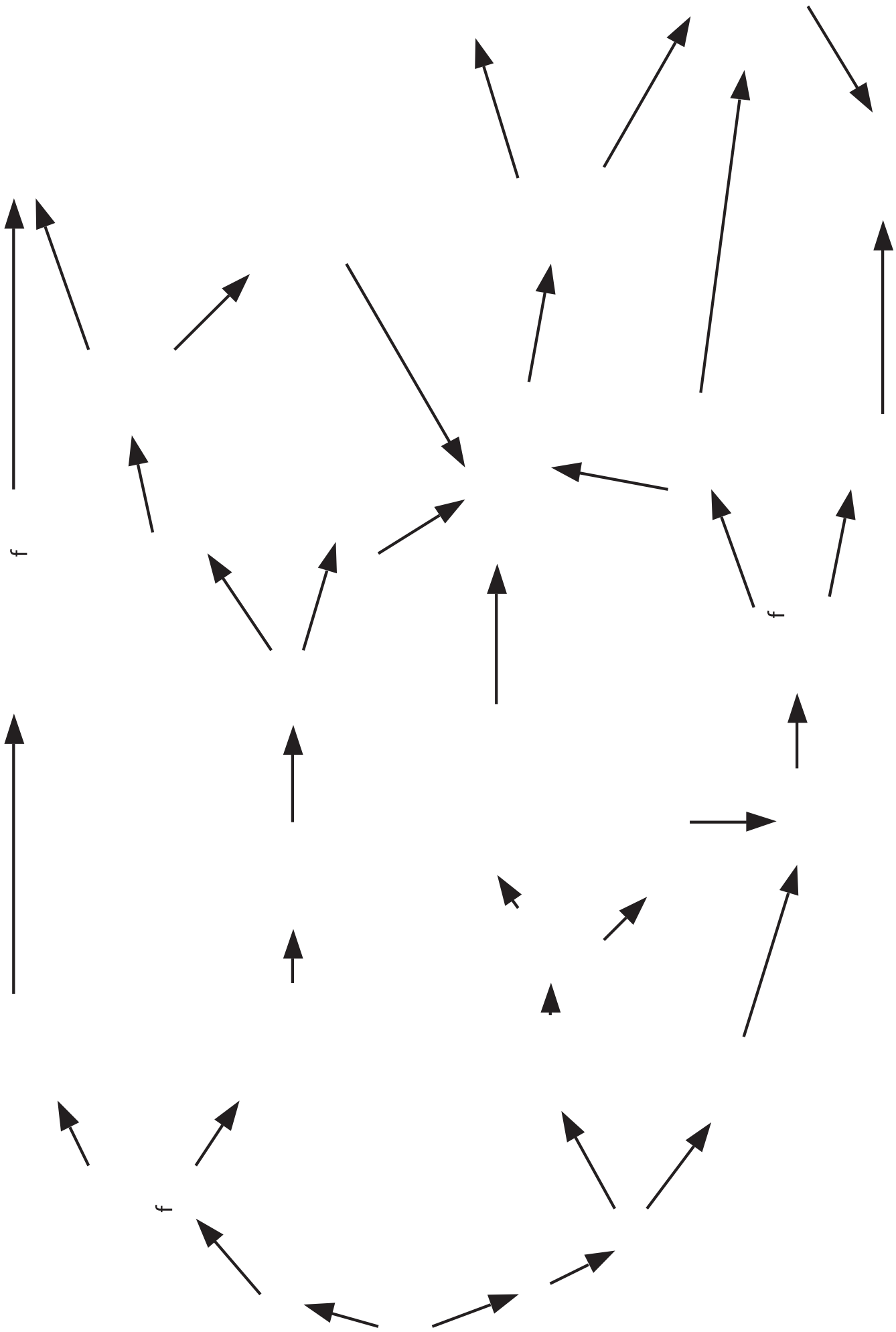


Fig. 18.2 Anatomy of the brachial plexus.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19 Upper Extremity Compartment Syndrome

Blunt crushing trauma is the most common cause of upper extremity compartment syndrome. Although less common, compartment syndrome can also occur in the hand and fingers. When confronting upper extremity injuries, it is important to closely monitor the patients for tissue ischemia and to correctly diagnose those who develop a true compartment syndrome. Delay in surgical intervention leads to devastating consequences. Compartment syndrome of the upper extremity requires urgent care due to its immediate sequela of muscle ischemia and long-term sequela of Volkmann ischemic contracture.

Increased compartment content or decreased compartment size leads to increased compartment pressures that cause tissue ischemia. Pay special attention to compartment pressures in cases of crush injury, severe soft tissue damage, fractures, intravenous infiltration, injection injuries, arterial insufficiency, burns, snakebites, patients lying on the limb, and *tight casts or splints*.

Diagnosis

The diagnosis of compartment syndrome is primarily a clinical one. The patient will have persistent pain that becomes worse with passive muscle stretching (a hallmark) or active flexion. The patient complains of diminished sensation, muscle weakness, and pain on palpation of the compartments. The presence of palpable pulses or Doppler ultrasound signals does not exclude increased intracompartmental pressures and compartment syndrome.

Cardinal Signs

- Persistent, progressive pain unrelieved with immobilization and elevation.
- Pain with passive extension.
  - Passive muscle stretch test.
    - Forearm.
      - Dorsal compartment: Finger, thumb, and ulnar wrist extensors—test with passive wrist flexion.

    - Mobile wad: Extensor carpi radialis longus, extensor carpi radialis brevis, brachioradialis—test with passive wrist flexion.
    - Volar compartment: Flexors of the fingers, thumb, and wrist—test by passive extension of the fingers, thumb, and wrist.
  - Hand.
    - Intrinsic compartments: Keep MCP joints in full extension and PIP joints in flexion. Pain with passive abduction and adduction of the fingers is diagnostically significant.
    - Thumb adductor compartment: Pull and abduct the thumb.
- Diminished sensation.
- Tense, tender forearm or hand.

Although a cool, pale, and pulseless extremity is often described in compartment syndrome, these are considered secondary signs and are often not present until late. Their absence should not delay surgery if cardinal signs are present. After 8 hours, the effects of muscle/nerve ischemia are irreversible.

Pressure Measurement

Use a Stryker needle (**Fig. 19.1**) or arterial line (**Fig. 19.2**) to measure compartment pressure. Forearm compartment pressures can be measured in the mobile wad and volar compartments with a Stryker needle:

- Pressure < 25 mm Hg: Normal—clinical observation; if situation worsens, repeat measurements.
- Pressure 25 to 30 mm Hg: Suspicious—observation with repeat measurements every 2 hours.
- *Normotensive patients with positive clinical findings and pressure >30 mm Hg for 8 hours:* Diagnostic for compartment syndrome.
- Altered mental status and pressure > 30 mm Hg for ≤ 8 hours: Highly suspicious for compartment syndrome.
- Hypotensive patients with compartment pressure < 20 mm Hg below diastolic blood pressure for ≤ 8 hours: Highly suspicious for compartment syndrome.

Fasciotomy

Perform a fasciotomy when the above symptoms are present or compartment pressures > 30 mm Hg or if compartment pressures are within 20 mm Hg of diastolic pressures. Perform an immediate fasciotomy if (1) the time of onset of signs and symptoms is unknown, or (2) the patient is

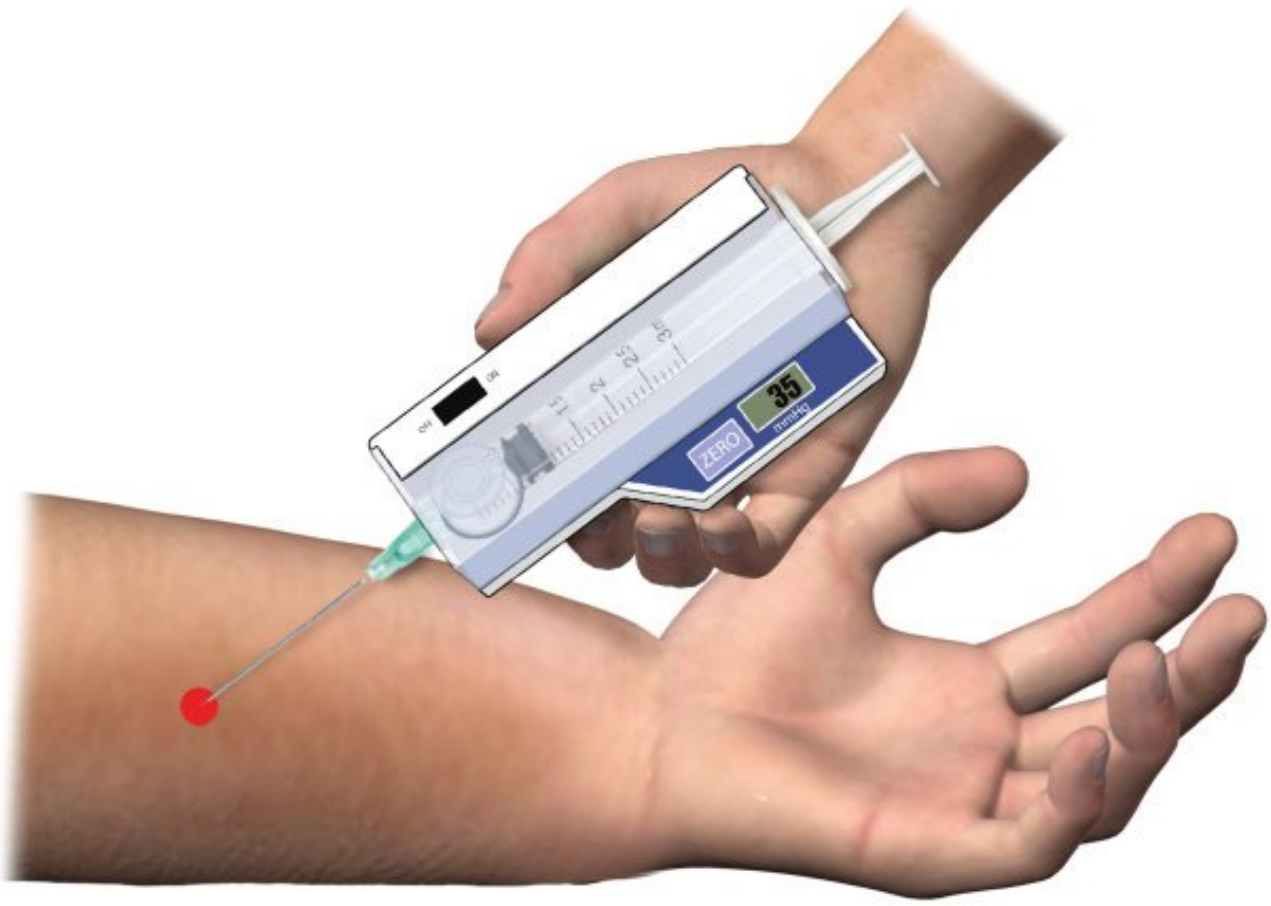

Fig. 19.1 Stryker measurement of compartment pressure. Needle is placed in forearm compartment. Measure pressure of normal arm as control. Detailed instructions are on the back of the Stryker needle device.

obtunded or unconscious. A prophylactic fasciotomy is performed if an arterial injury with ischemic time of 4 to 6 hours exists.

Hand compartment pressures are difficult to assess, and measurements of these pressures are often inaccurate. Rely on a clinical examination in making a diagnosis of compartment syndrome.

Management

Fasciotomy and release of the compartments is the only treatment for compartment syndrome. *Do not elevate* an extremity that has not been decompressed, because the decreased perfusion causes an increase in ischemic damage. Elevation of the extremity after decompression is appropriate.

- *Number of compartments:* 4 in forearm and 10 in the hand.
  - *Forearm:* Volar superficial and deep, dorsal, and mobile wad.
  - *Hand:* Dorsal interossei × 4, volar interossei × 3, hypothenar, thenar, and adductor pollicis.

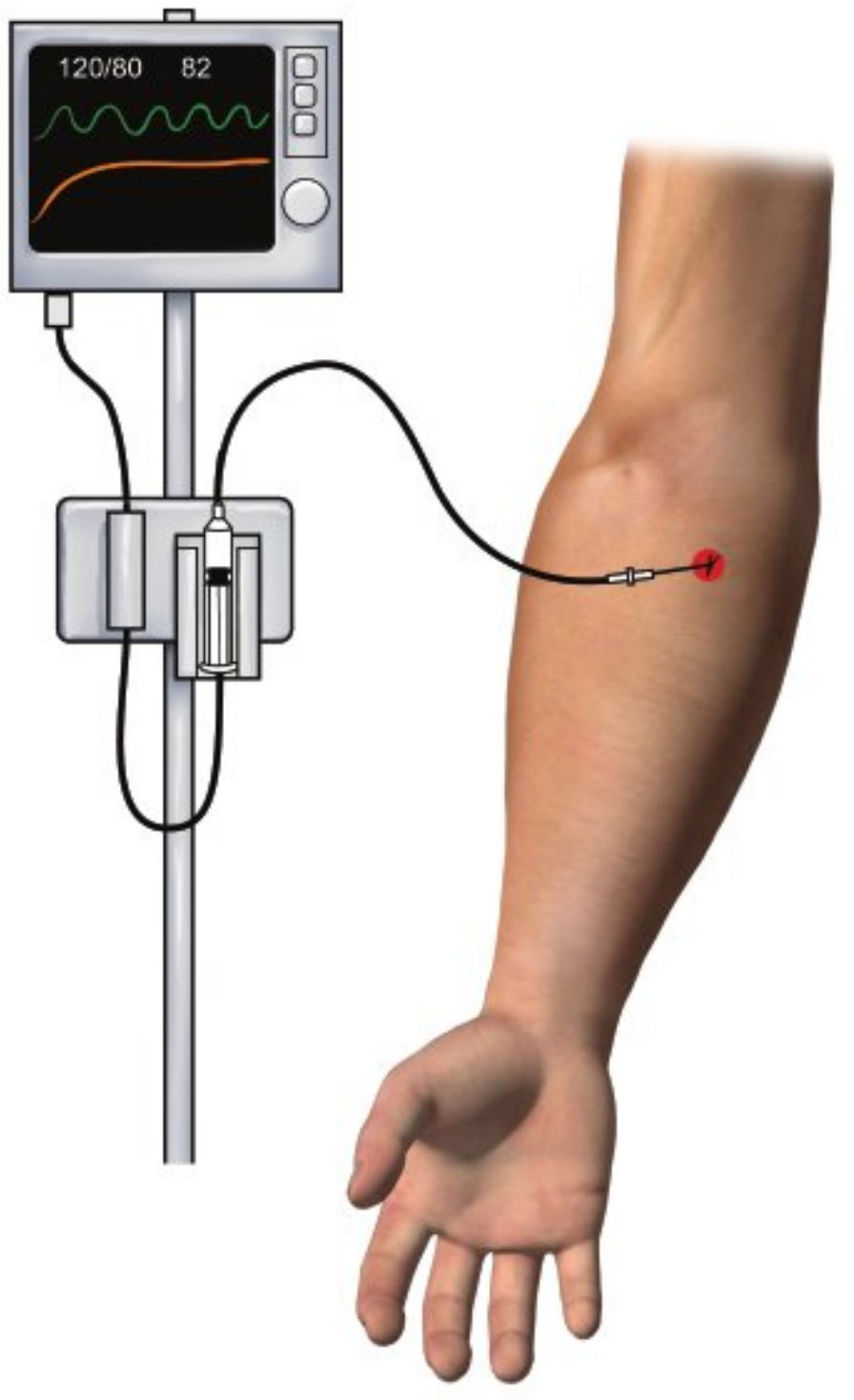

Fig. 19.2 Measurement of compartment pressure with arterial line setup. Needle is placed into forearm compartment. Zero the pressure at the level where needle is placed prior to entering the compartment.

Forearm Fasciotomy (Fig. 19.3, Fig. 19.4)

Release the median nerve, the ulnar nerve, and all three volar compartments. Check muscle bellies in superficial and deep volar compartments. Perform an epimysiotomy if necessary. The incision is started between the thenar and hypothenar eminences (similar to a carpal tunnel incision). At the wrist crease, the incision is carried transversely in the flexion crease directly to the Guyon canal, and the ulnar nerve is released. Avoid transecting the palmar branch of the median nerve, and make straight incisions perpendicular to the wrist crease. Next, carry out the incision approximately 5 cm proximal to the wrist crease on the ulnar side of the forearm to create a flap for median nerve coverage. Next, curve the incision radially. The incision should reach its radial apex approximately one-half to two-thirds of the way up the forearm. The incision is then made in the ulnar direction to

a point just radial to the medial epicondyle, where it can be carried up to explore the brachial artery and avoids a straight incision across the antecubital fossa. The incision should be extended above the elbow where the lacertus fibrosus is released. If muscles appear necrotic, do not débride them, because the extent of the injuries cannot be determined at the time of initial fasciotomy. Cover the median nerve with the small wrist skin flap. The mobile wad is released at the apex of the radial portion of the incision. After release of the superficial volar compartment, the deep volar compartment must be released in an interval between the sublimis tendons and the flexor carpi radialis. This will prevent ischemic contraction of the muscles of the deep volar compartment. Release of the volar compartments significantly decreases the tension of the dorsal forearm. However, if significant tension in this area persists after complete release of all volar compartments, an incision is made along the midpoint of the dorsal compartment.

Hand Fasciotomy

Release the dorsal interossei, volar interossei, and adductor pollicis through incisions on the dorsum of the second and fourth metacarpals (**Fig. 19.3**). On either side of the metacarpal, release the interossei fascia and expose the muscles. Next, release the first volar interossei and the adductor pollicis, and

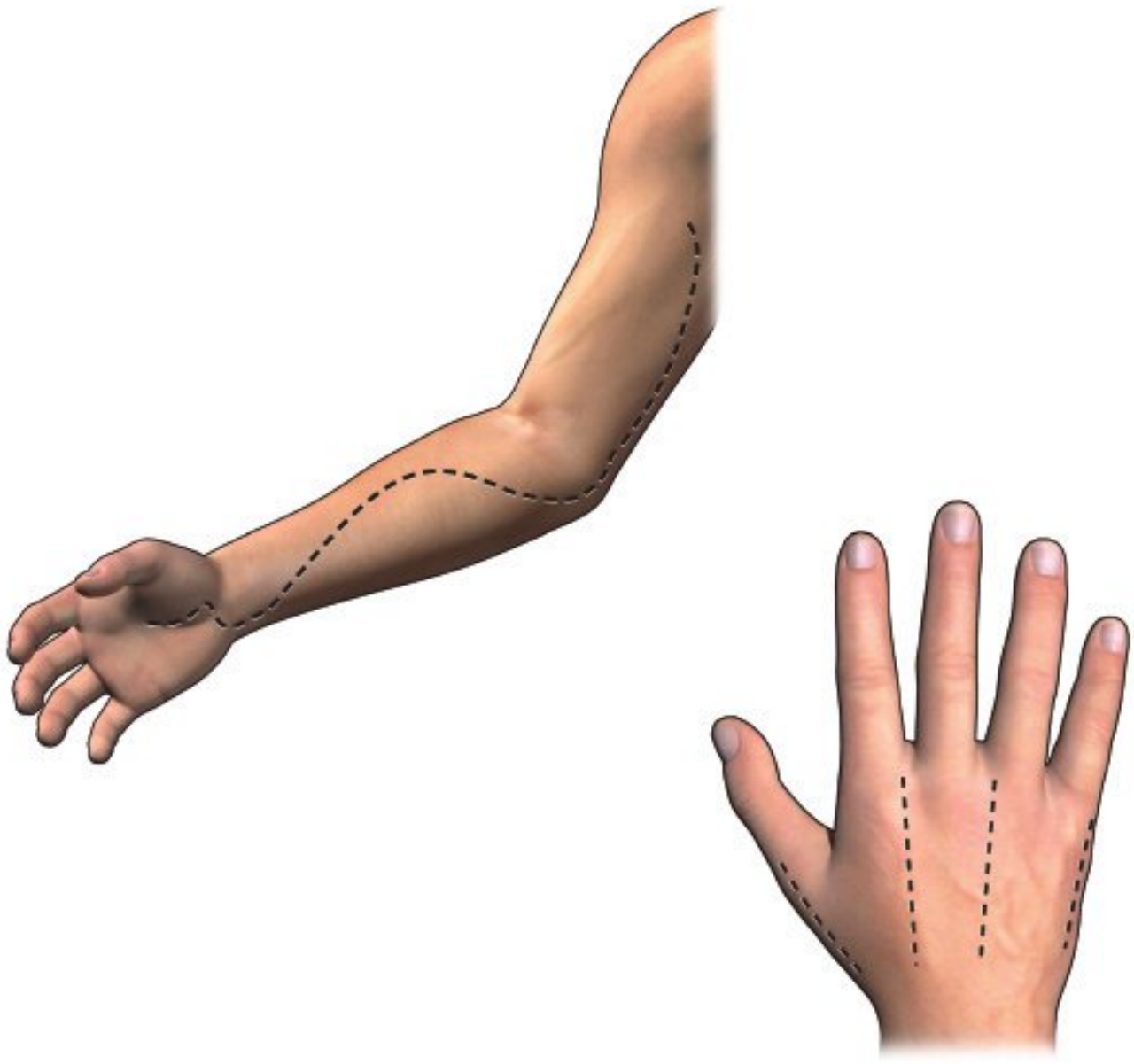

Fig. 19.3 Rowland incision for forearm or hand fasciotomy.

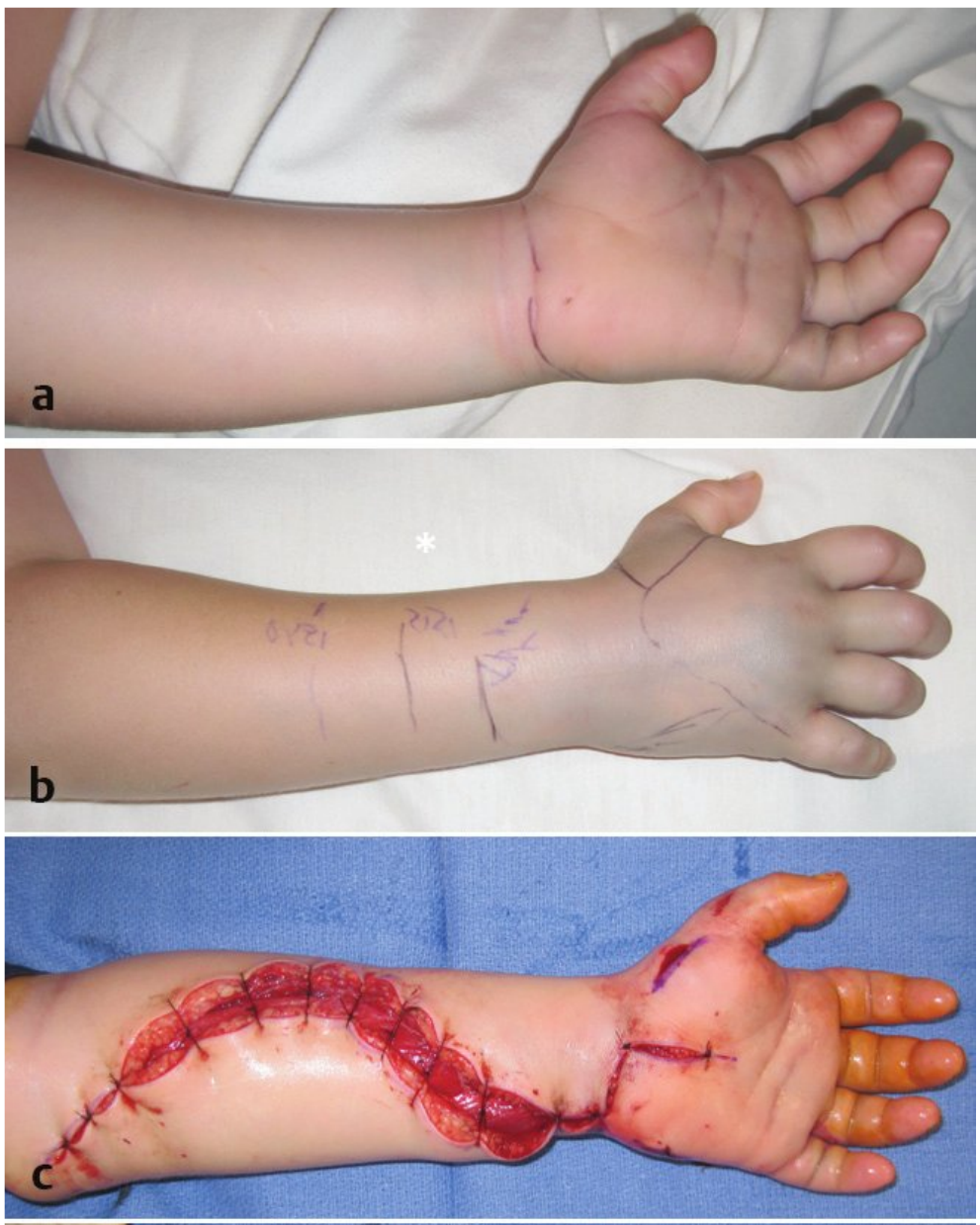

Fig. 19.4 (a,b) Clinical evidence of compartment syndrome of the forearm and hand. **(c,d)** After release of the volar compartments of the forearm and the carpal tunnel, hypothenar, thenar, and dorsal compartments of the hand.

spread with blunt tip scissors along the ulnar side of the second metacarpal. In similar fashion, release the second and third volar interosseous muscles by spreading along the radial sides of the fourth and fifth metacarpal.

Finally, release the thenar and hypothenar compartments using longitudinal incisions along the radial side of the first and the ulnar side of the fifth metacarpal, respectively.

Digits

In severe injuries or burns to the digits, one performs a finger fasciotomy/escharotomy. Use a midaxial incision along the nondominant side of the finger. The midaxial portion of the finger is marked by first flexing the finger. Next, a line is drawn that interconnects the most dorsal

aspects of the flexion creases to each other and to a point lateral to the nail plate. The nondominant side of the finger is usually the ulnar side of the index finger, long finger, and ring finger. For the thumb and little finger, release via an incision on the radial side of the digit. Then, release the flexor tendon by dissecting along the volar side of the flexor tendon sheath and releasing all vertical connections. Carry out the incision across the midline at the tip of the finger.

Perioperative Medical Management

Patients should be monitored for crush syndrome and the systemic sequela of massive myonecrosis. This is done by analysis of serum potassium, creatine kinase, and myoglobin. Additionally, renal protective strategies are warranted. These include bladder catheter drainage and aggressive hydration to ensure a urine output between 0.5 and 1 mL/kg/h. Additionally, assessment of the urine pH and myoglobin should be done. In cases of significant myoglobinuria, the urine should be alkalized with acetazolamide (250 mg by mouth every morning or 5 mg/kg IV every 24 hours) and mannitol (50 to 100 g of 5% solution IV over 2 hours, repeat dose to maintain urine output with a maximum dose of 200 g/d). Serial assessment of serum K^+, CK, and urine myoglobin parameters allows one to follow the course of disease and dictates resolution of disease, as well as termination of treatment.

Postoperative

Splint the wrist in extension, MCP joints at 90 degrees, thumb in abduction, and IP joints at 180 degrees. Elevate the extremity after decompression. No efforts should be made at skin closure; however, skin should be loosely closed over exposed nerves and arteries. Dress open areas with Xeroform or Adaptic (Johnson & Johnson, Inc.) gauze followed with Kerlix and a volar splint. Elevate the extremity postoperatively. Maintain neurovascular checks in a monitored care unit to assess for adequate decompression. In severe cases, plan for a second-look operation in 24 to 48 hours to débride necrotic tissue.

Close wounds definitively by 10 days. If skin cannot be closed, then place split-thickness skin grafts or temporary skin substitutes on the wound. Prior to closure, all necrotic tissue should be débrided. Alternatively, Silastic vessel loops can be used to slowly close the wound with tightening daily.

20 Postoperative Evaluation of Free Flap Reconstructions

In addition to the basic postoperative approach to any surgical patient, the patient with a free flap reconstruction requires specific attention to detect and prevent a potentially compromised flap. An important rule of thumb is to physically inspect a flap when there is any question of a change in status. Unless you are very experienced, any examination of a *flap* with a suspected change in status should be reported to the attending surgeon responsible for the *flap*.

Assessment

Vital Signs

Heart rate monitoring is important for assessing pain control, intravascular volume status, and possible arrhythmias. Inadequate pain control is a frequent cause of tachycardia. Be sure to ask how comfortable the patient is and assess whether additional pain medication is needed. Watch for bradycardia, which can result from heart blocks or overuse of antihypertensive medications such as β-blockers and analgesics. Most free flap patients will spend the first night in a monitored care unit on telemetry; therefore, attention should be paid to the tracing to rule out atrial fibrillation, flutter, or other arrhythmias. It is important to control these arrhythmias, not only for the safety of the patient, but to maintain the viability of the flap. Sudden fluctuations in blood pressure can lead to turbulent flow across the microvascular anastomosis or within the flap, which could lead to compromised perfusion of the flap.

Blood pressure should be monitored very closely in the postoperative period. Most free flap patients should have a MAP > 90 and SBP > 120. Of paramount importance is keeping the patient from becoming hypotensive. **Hypotension can result in recipient artery spasms and venous stasis that can lead to thrombosis.** Due to the prolonged surgery, insensible losses, and postoperative third spacing, free flap patients are usually intravascularly depleted and often require fluid supplementation in the acute postoperative period. Intravascular fluid status is most accurately represented by the patient's urine output. **Free flap patients should produce at least 0.5 mL/kg/h of urine (35 mL/h for a 70-kg patient), but preferably 50 to 100 mL/h.** Patients suspected to

be intravascularly depleted should receive LR or normal saline boluses. After the first 24 hours, D5 1/2NS at maintenance rate is used for the stable patient.

Patients should never **receive diuretics** to induce urine output unless there are clear signs of renal compromise in a well-hydrated patient. Likewise, **avoid use of pressors** to treat hypotension. Pressors should be a last resort and only used when absolutely necessary (profound hypotension).

Hypertension (> 180/100) can lead to bleeding in a fresh postoperative patient. Elevated blood pressure is most commonly a sign of inadequate pain control. Extremes in hypertension unresponsive to analgesics should be managed with low-dose antihypertensives (hydralazine 10 mg IV, or labetalol 5 to 10 mg IV as needed) to prevent rapid decreases in the MAP that can ultimately be detrimental to a flap.

Oxygenation should also be assessed with a pulse oximeter to keep the blood oxygen saturation > 93% In replants, the oximeter is a useful tool for monitoring the replanted digit. When the sensor is placed on the reattached part, loss of the signal indicates arterial compromise, whereas progressively declining saturations are suggestive of venous congestion.

Hypothermia is avoided to prevent vasospasm. The patient's room should be kept above 70°F (21°C), with heating units, lamps, or Bair Hugger warmers (Arizant Healthcare) used liberally if the room temperature cannot be adequately controlled.

Drain output should be closely monitored. Although drainage may be high in the immediate postoperative period due to expected oozing, a drop in output followed by a sustained increase may be indicative of venous thrombosis. Extensive drainage should prompt immediate evaluation of the flap.

Clinical Observation

Although refinement of microvascular techniques has brought failure rates down, it is the **early recognition of flap compromise followed by immediate surgery that prevents total flap loss.**

Always notify the attending surgeon of any potential flap compromise and keep the patient NPO in case there is any need for operative exploration.

In assessing a flap, good clinical observation techniques are essential. To begin, **always turn on all the lights** in the patient's room and evaluate the flap's general appearance. Healthy flaps should be pink, warm, and soft with a capillary refill of approximately 2 seconds (**Fig. 20.1**). Any other appearance is worrisome (**Table 20.1**). **Fig. 20.2** demonstrates a congested free flap. Note that *pedicled* flaps are often congested postoperatively; this usually resolves with time (**Fig. 20.3**). A sign of arterial

Table 20.1 Important clinical signs that differentiate arterial versus venous problems while monitoring a flap

	Arterial	Venous
Color	Pale, white	Blue, purple
Capillary refill	Sluggish (> 2 s)	Brisk (< 2 s)
Tissue turgor	Prunelike	Tense, swollen
Dermal bleeding	Scant	Rapid dark oozing
Temperature	Cool	Cool
Doppler	Absent	Can still be present

compromise is a pale flap that is cool, with poor tissue turgor. Check to see if the flap blanches and for capillary refill (2 seconds). If an inflow problem is suspected, an 18- or 20-gauge needle can be used to prick or scratch the flap to assess for bleeding. Check distally and proximally, but avoid the pedicle. Always approach the flap at a shallow angle to avoid deeper vascular structures. Signs of poor venous outflow are a tense flap with increased turgor pressure and purple color; the flap can be warm or cool (**Fig. 20.4**). The flap will usually be oozing around the edges with venous blood, and if the flap blanches, the capillary refill is usually brisk. A pinprick to the flap will also result in venous dark bleeding.

Doppler Signal

Doppler is used to measure the velocity and rate of blood flow through a vessel. Normal flow dynamics should possess three distinct audible phases. The first phase is heard during systole with the forward flow of blood distending the vessels. Early diastole represents the second phase, where the elastic vessel rebounds and there is a momentary reversal of flow. The last phase is associated with late diastole and atrial contraction, when there is once again a forward flow.

Therefore, any Doppler signal can be described as being monophasic, biphasic, or triphasic. Sounds should be clear and distinct. Triphasic signals are what one expects in a healthy flap. In the early postoperative period, the signal is often initially biphasic, representing the fact that the flap has been cold and ischemic for a period of time. As the flap warms and perfuses, the third phase will become audible. **A monophasic or "jackhammer" type of sound is indicative of venous occlusion**. Any

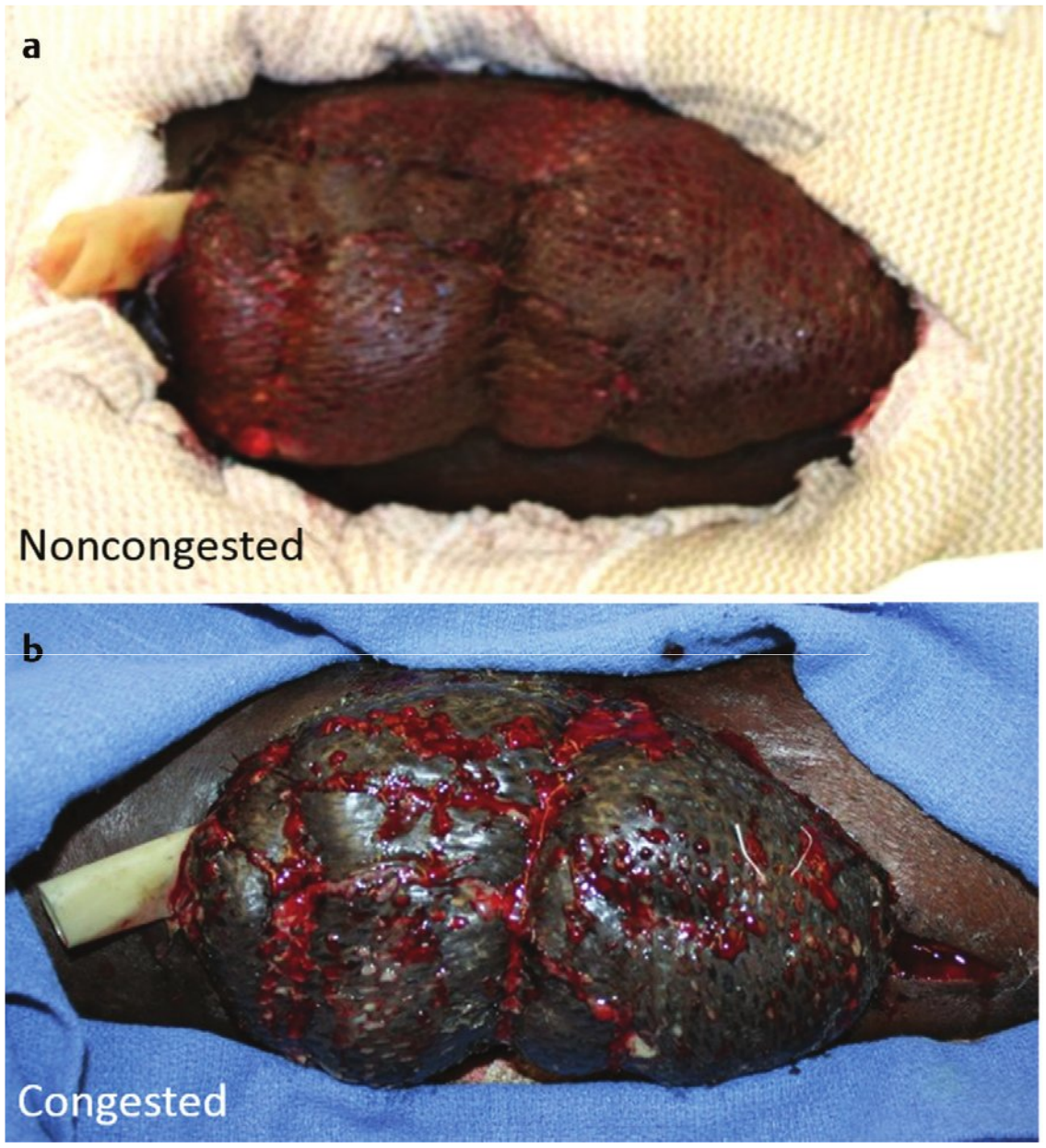

Fig. 20.1 **(a)** A normal muscle flap with a skin graft. **(b)** A congested muscle flap.

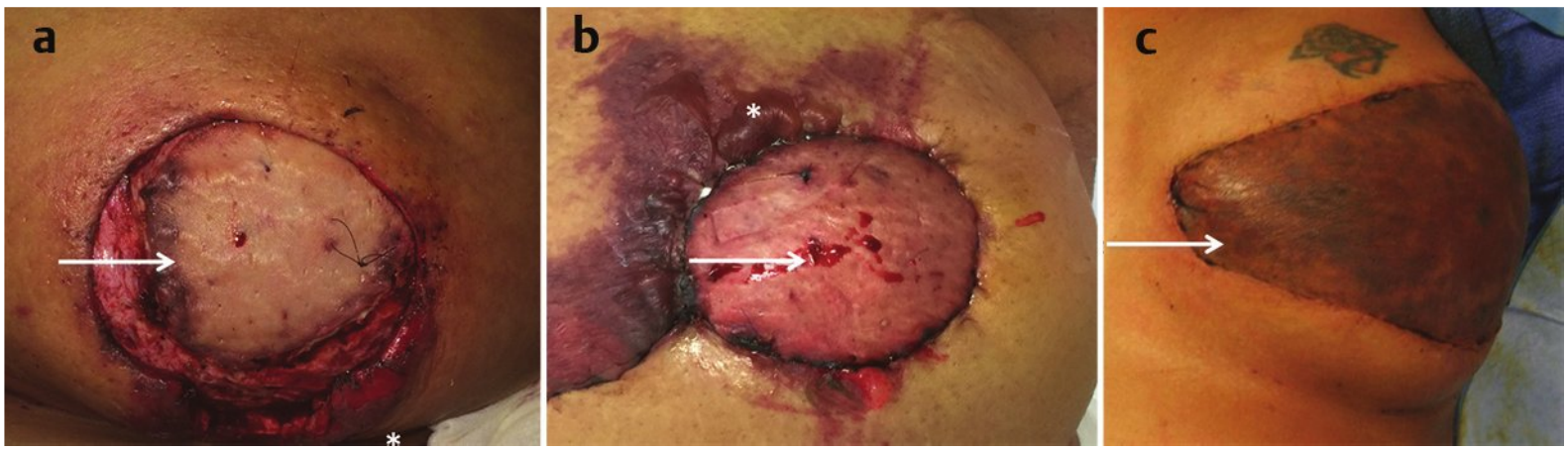

Fig. 20.2 Fasciocutaneous flap congestion. **(a)** Early congestion. **(b)** Moderate congestion. **(c)** Late congestion.

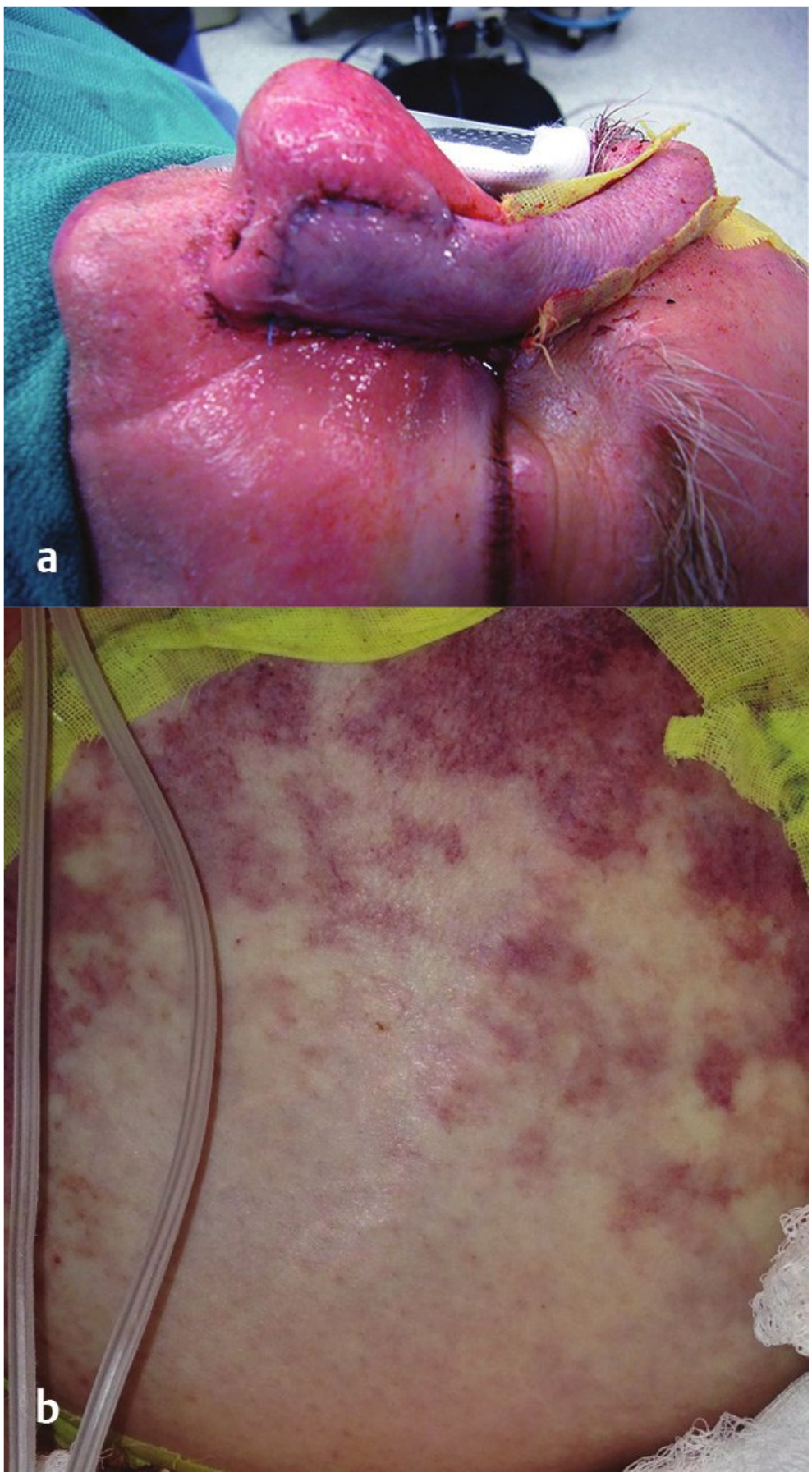

Fig. 20.3 **(a)** A congested pedicle paramedian forehead flap. **(b)** A congested pedicle TRAM flap.

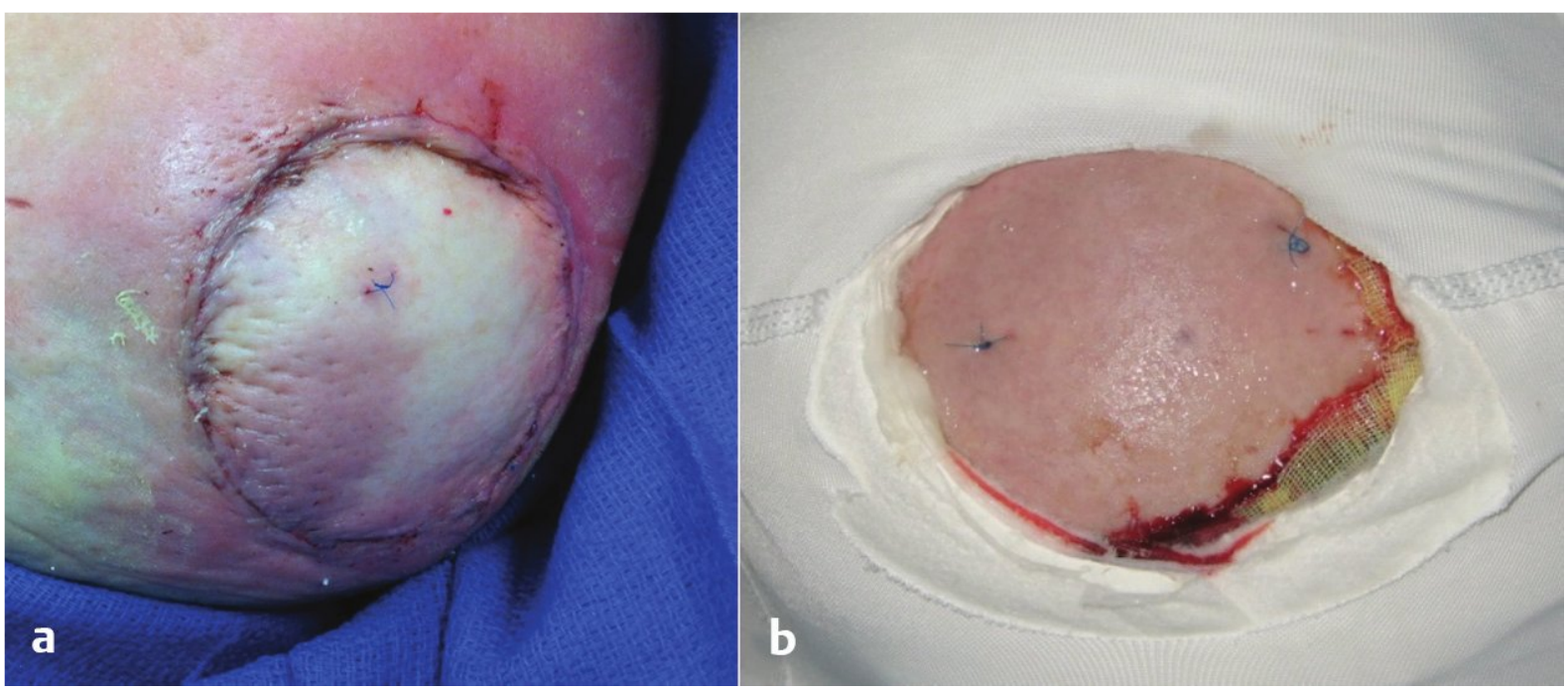

Fig. 20.4 **(a)** An ischemic DIEAP flap. **(b)** A congested DIEAP flap.

change in the signal should prompt a very careful examination of the flap status. The batteries of the Doppler device should also be checked.

Implantable Doppler devices are usually used with buried free flaps, with which there is no skin paddle to monitor externally. The Doppler cuff can be placed on the arterial or venous pedicle, but is more frequently placed on the vein. This is because venous compromise is more common and because an arterial signal can still be transmitted even in cases of complete thrombosis. A loss of signal is most frequently due to displacement of the cuff; however, a clinical exam should still be undertaken to assess the true status of the flap.

Recent advances in free flap monitoring have resulted in the venous flow coupler by Synovis (**Fig. 20.5**). The monitoring device is incorporated into the already popular venous coupler and eliminates the need for wire placement onto the vessels. The flow coupler allows for easy monitoring with a soft flexible wire, similar to the Cook implantable Doppler, and yields the same information. The venous outflow can be continuously monitored with less failure due to positional changes because of the integrated design.

Venous oxygen tissue saturation monitoring (ViOptix) is often employed on flaps with skin paddles. A transcutaneous monitor is placed on the flap skin, and through fiber optics, the monitoring device measures the tissue venous saturation.

- The monitor has two indicators (**Fig. 20.6**):
  - Signal quality.
  - Venous saturation.

The signal quality must be above 80 to accurately assess the venous saturation. The signal quality is related to the contact the transcutaneous monitor has with the skin. If there is poor contact or a barrier between the skin and the sensor (blood, Doppler gel), then this may cause the signal quality to be low and lead to inaccurate readings of the oxygen saturation. The venous oxygen saturation curve of a healthy flap should be a constant flap line with minimal fluctuations. Venous oxygen saturation levels above 30%are acceptable. Levels that are below 30%indicate poor perfusion. Large fluctuations in saturation over short periods of time may indicate arterial spasm or kinking of the pedicle. Downward-trending saturations with changes >20%in an hour may represent impending flap compromise due to venous congestion, caused by either thrombosis of the venous anastomosis or progressive thrombosis of the pedicle or arterial anastomosis.

The venous oxygen tissue saturation is also affected by systemic factors including blood pressure, hemoglobin level, and oxygen saturation. Therefore, interpretation of the venous oxygen saturation of the flap must take into account the overall condition of the patient.

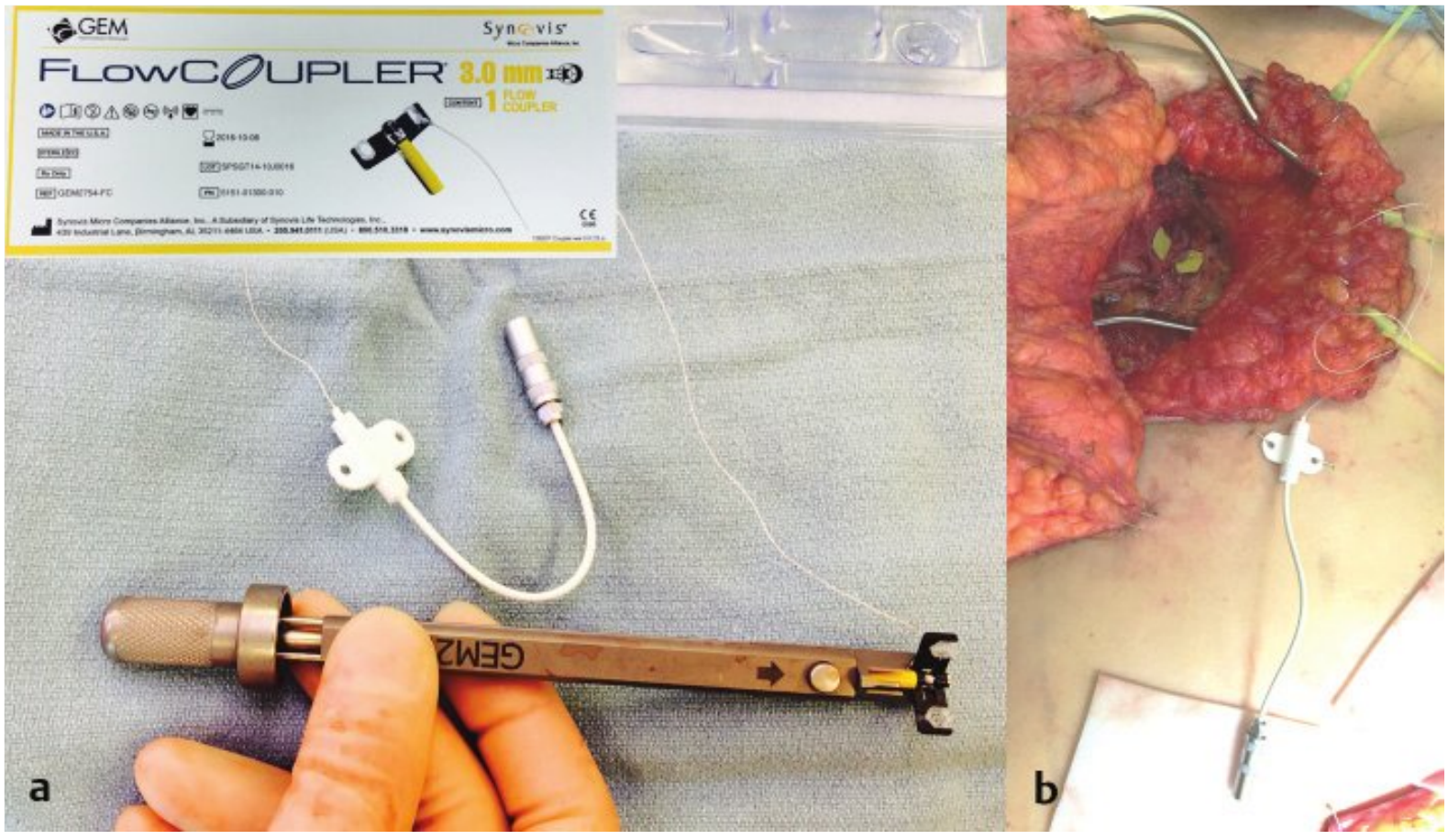

Fig. 20.5 Flow Coupler device. **(a)** a Doppler probe is integrated into the venous coupler that is used to create a venous anastomosis. **(b)** After completion of the venous anastomosis, the flexible wire is directly attached to an external monitoring device.

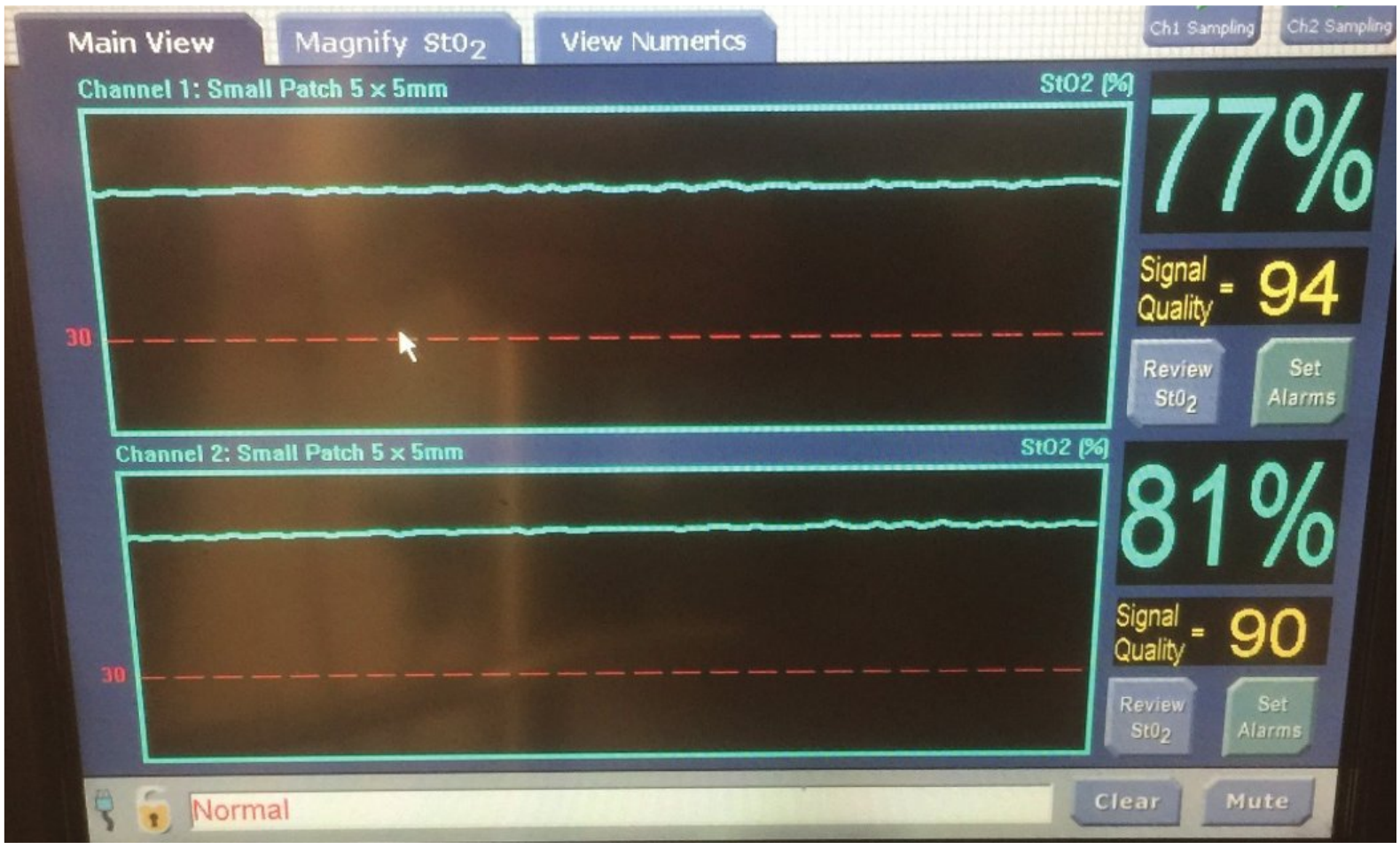

Fig. 20.6 ViOptix flap monitoring. Venous oxygen saturation curves of two free flaps (bilateral breast). Signal quality must be above 80 to ensure accurate reading. A healthy flap should have a venous oxygen saturation that is steady and above 30%

Preventive and Salvage Techniques

Local factors possibly compromising the flap should always be considered initially.

- Loosen all constrictive dressings to avoid any unnecessary pressure or compression on the flap (remove sutures immediately if they appear to place undue tension on the flap). Sutures can be removed to help evacuate a hematoma, or to relieve postoperative edema and congestion affecting flap perfusion.
- Strip all drains to relieve clots that may prevent evacuation of a hematoma.
- Reposition the patient to correct any potential kinking or compression of the pedicle. This may sometimes lead to immediate flap viability and relief of compromised inflow or outflow.
- Elevate extremities when possible to prevent inadequate venous drainage from the limb and to reduce postoperative edema, both of which can lead to tamponade (extremity free flaps and replants).

Anticoagulation

Leech therapy is usually used in digital replants or with mildly congested free flaps (**Fig. 20.7**). Leeches secrete the peptide hirudin in their saliva, which causes the flap to bleed. They can salvage a flap by relieving congestion. Leeches attach for approximately 30 minutes and actively suck blood from the flap. After they fall off, the bite wound continues to ooze due to the hirudin, which accounts for most of the blood loss. When using leeches, the wound is more susceptible to *aeromonus hydrophila* species and prophylactic antibiotics (Bactrim DS by mouth twice a day or ciprofloxacin 500 mg by mouth or IV twice a day) should be utilized. If the leeches do not adhere, a 20-gauge needle can be used to initiate bleeding from the flap. This should facilitate leech feeding. After one feeding, leeches are usually sacrificed. Leeches can be obtained from the pharmacy or from other local hospitals, or they can be emergency delivered from Leeches USA Ltd. (telephone 800-645-3569). Although leeches may relieve mild congestion temporarily, a free flap that is congested should return to the OR for evaluation of the venous anastomosis.

Heparin is usually not used postoperatively, but it can be indicated for some replants and anastomotic revisions. Full heparinization in the immediate postoperative period is associated with a fairly high rate of significant bleeding. In compromised flaps, a heparin bolus of 3,000 to 5,000 U can be helpful in preventing propagation of a clot while a patient with a compromised flap is being prepared for the operating room.

Depending on surgeon preference, 10%dextran can be routinely used postoperatively. It not only acts as a volume expander, but also has anti-

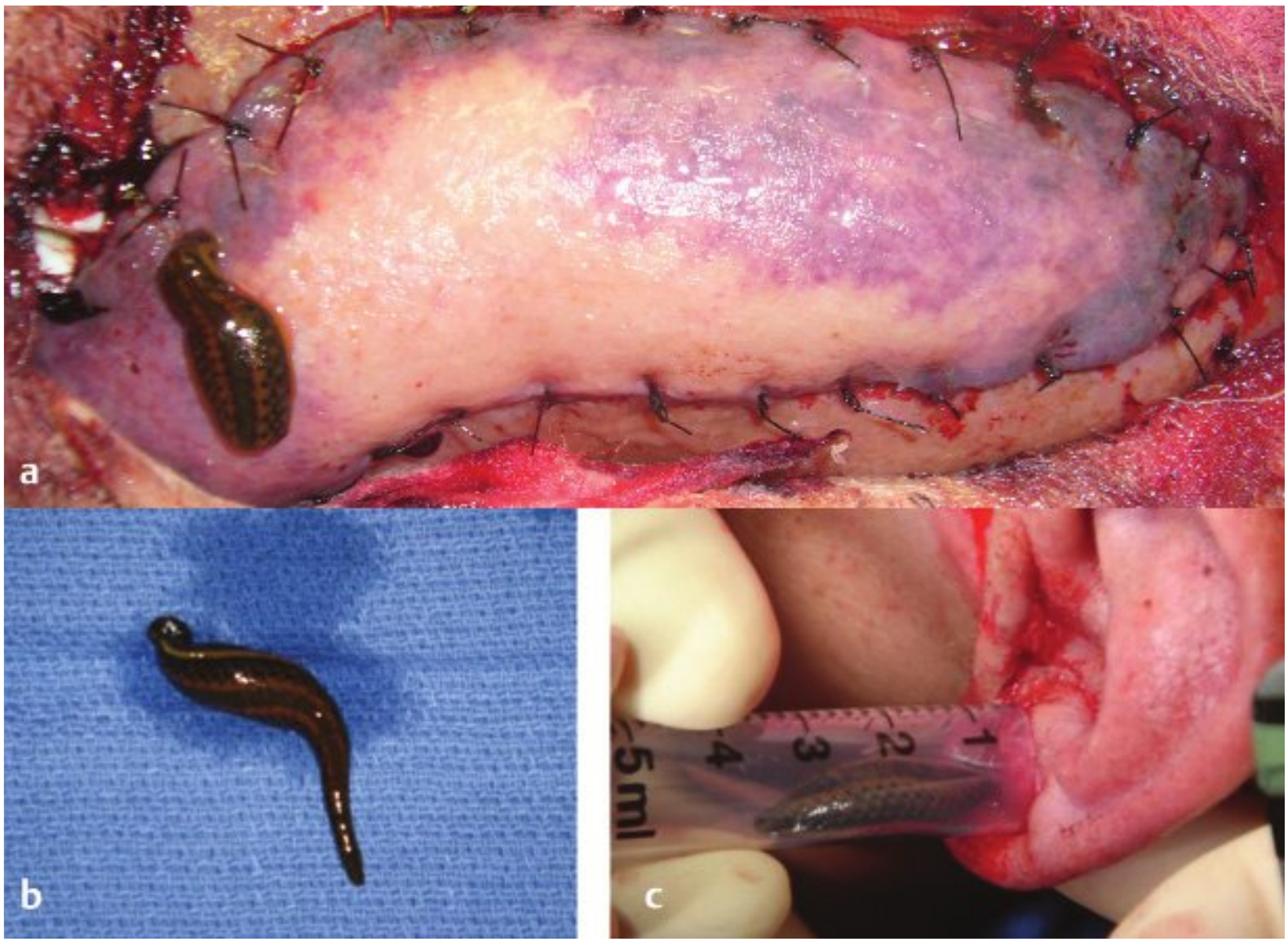

Fig. 20.7 **(a)** Leech therapy for a congested flap. **(b)** A medicinal leech (Hirudo medicinalis). **(c)** Use of a cut syringe for placement of the leech in the desired position.

platelet properties. A 5-mL test dose is usually given, and then empirical therapy is started at 25 mL/per hour per day for 3 to 5 days or 40 mL/h x 12 hours for 3 to 5 days. Side effects include congestive heart failure, volume overload, renal toxicity, and allergic reactions.

The use of thrombolytics has been effective in laboratory protocols; however, their results in the clinical setting have been mixed and controversial. They have been described as being effective in flaps with venous compromise in lysing thrombus within the flap. Streptokinase (500,000 to 750,000 U) or 2 to 4 mg of TPA is infused into the arterial pedicle once the vein has been cut to prevent systemic administration.

Salvage rates can approach 50 to 75% if problems are diagnosed early. The evaluator must be astute when assessing the free flap and keep in mind all the potential causes of flap failure (thrombosis, intimal flap, back walled anastomosis, kinked pedicle, tight skin closure, edema, hematoma, external pressure, vasospasm, hypothermia, hypovolemia).

One should not wait until the flap is purplish blue and cold and has no Doppler signal. At that time, the flap is likely beyond salvage. Always consult with the attending surgeon or a surgeon of senior experience after a thorough examination of the flap and be prepared to return the patient to the operating room if necessary.

21 The Postoperative Aesthetic Patient

Postoperative evaluation of the cosmetic surgery patient involves evaluation for early signs of complications, patient's comfort level, questions, and desires. Always check the patient's vital signs. A high heart rate, low blood pressure, and decreased urine output can herald an impending complication. In addition, ignoring high blood pressure due to pain can result in a hematoma formation. Hematoma formation not only may lead to life-threatening anemia, but also will compromise skin flaps and may lead to functional morbidity. Ask the patient if there is a difference in the pain with respect to laterality. This can often indicate a hematoma or infection. When there is an indication of change, excessive pain, or any other concerns, dressings should be removed and the wounds checked (always remove dressings of patients who have had an otoplasty and complain of asymmetric severe pain). Assessing the patient accurately and proposing a concise and appropriate plan to the primary surgeon should be done prior to any intervention.

Abdominoplasty

Considerations

- Check vital signs for high or low blood pressure, pulse oximetry, and heart rate.
- Jackson-Pratt (JP) drain's output.
  - If the JP drain output is high and bloody and does not turn serous, consider a hematoma.
- Beware of low drain output and enlarging/painful masses under the abdominal skin flaps. Consider a hematoma. The drain may be clotted. A clot in the drain, low pressure, low urine output and tachycardia are consistent with a hematoma and potentially ongoing bleeding.
- Keep patient in "lawn chair" or flexed position.
  - Put a sign above the patient's bed to alert caregivers about the desired position.
  - Unplug the bed controls.
- Incentive spirometry.
  - Reduce atelectasis.

- Get patient out of bed (OOB) with walker ± physical therapy postoperative day (POD) 1.
  - Use sequential compressive devices on the lower extremities.
  - Consider starting pharmacologic DVT prophylaxis—Lovenox (Aventis Pharmaceuticals) 40 mg SC daily or SC heparin 5,000 U twice a day.
- Abdominal binder will assist with patient comfort, as will participating in activity.
  - Caution—excessively tight binders and garments can cause pressure necrosis and ischemia-related changes to the abdominal skin flap. Therefore, consider periods of garment relief, especially when the patient is resting.
- Umbilicus viability.
  - Small areas of delayed wound healing will eventually heal through secondary intention.
  - Keep the umbilicus clean.

Hematoma

- Diagnosis.
  - Asymmetric pain or asymmetric bulging of incision/abdomen.
  - Increasing heart rate, decreasing BP, decreasing urine output, high drain output, or clots in the drain.
  - Dropping hemogram.
- Treatment.
  - Strip drains and check serial hemoglobin and hematocrit (every 6 hours).
  - Bolus fluids (NS 500 mL) and increase fluid rate appropriately (beware of patients with cardiac history—overresuscitation could cause pulmonary edema and heart failure).
  - Hold all anticoagulants.
  - Crossmatch blood type and hold PRBCs in preparation for transfusion.
  - Prepare for exploration in the operating room.

Respiratory Distress

Pulmonary Embolus

- Diagnosis.
- Clinical signs and symptoms—dyspnea, pleuritic pain, hypoxia, tachycardia, hemoptysis, orthopnea, JVD.

  - Arterial blood gasses (ABGs).
    - Look for hypoxemia, hypercapnia, and respiratory alkalosis.
    - High probability when low PaO_2 and dyspnea.
- Elevated D-dimer.
- ECG changes—AFib, RBBB, Q waves in leads II, III, and aVF.
- **Order a CT scan of the chest, pulmonary embolism protocol.**
- Check for calf pain and swelling—if DVT is suspected, then request a duplex ultrasound.
- Treatment.
  - If you have a very high suspicion of pulmonary embolism, then start heparin drip,
  - Start patient on heparin or Lovenox,
    - Heparin—load with 80 U/kg bolus and then 18 U/kg/h infusion; check PTT every 6 hours and keep PTT between 60 and 90.
    - Lovenox 1 mg/kg every 12 hours SC (Lovenox has a longer half-life than heparin).

Hemodynamically unstable patients should be transferred to the ICU and may require support for circulatory collapse.

Pulmonary Edema

- Diagnosis.
- Dyspnea, hypoxia, wheezes and crackles in auscultation, palpitations, anxiety.
- History of volume overload, laryngospasm, or heart failure.
  - CXR—bilateral infiltrates.
  - Check CVP if available; if above approximately 12, patient is volume overloaded.
- Treatment.
- Apply supplemental oxygen.
  - Start Lasix (Aventis Pharmaceuticals) 20 mg IV.
  - Check urine output to keep intakes/outputs (I/Os) negative.
  - Redose Lasix as needed.
  - Monitor electrolytes.

Flash pulmonary edema or negative pressure pulmonary edema may result in acute global pulmonary compromise that may require pressure support ventilation or intubation and mechanical ventilation.

Overaggressive Plication

- Pulmonary dysfunction—decreases in functional residual capacity.
  - Significant in patients with a history of asthma or COPD.
  - Treatment.
    - First employ conservative management by changing the patient's position and by respiratory care to include incentive spirometry and bronchodilators.
    - Exploration in the operating room.
- Abdominal compartment syndrome (ACS)—increased intra-abdominal pressure leading to organ dysfunction. This may be exacerbated by overresuscitation.
  - Diagnosis.
    - Hypoxemia, dyspnea, low urine output, increased abdominal distention, and pain.
    - Measure intra-abdominal pressure—a bladder pressure > 20 mm Hg may indicate ACS.
  - Treatment.
    - Limit fluid administration, use colloid resuscitation if necessary.
    - Nasogastric decompression.
    - Plan for the possibility of surgical decompression in the operating room.

Dehiscence

Small area

- Reinforce the nondehisced areas with Steri-Strips.
- Superficial small areas can be acutely repaired with simple suture techniques (3–0 or 4–0 nylon).
- Deeper areas of delayed wound healing will require local wound care with wet to dry dressing changes and future revision.

Large Area

- Operating room débridement and closure or vacuum-assisted closure therapy and tertiary closure.

Breast Augmentation

Hematoma (Fig. 21.1)

- Diagnosis.
  - Unilateral pain, swelling, bruising, and occasional fever.
  - Ultrasound may be helpful in patients who are obese or have large native parenchymal volumes.
- Treatment.
  - Strip drains if present.
  - Discontinue anticoagulants and antiplatelet therapy.
  - Small hematomas—observe if the patient is asymptomatic.
    - Apply circumferential compressive wrap or garment.
  - Large hematomas—evacuation in an operating room.

Infection

- Usual presentation is POD 5 to 10.
- Assess patient for either superficial skin or deep implant infection.
- Diagnosis.
  - Leukocytosis.
  - Warmth, erythema, and edema of the breast or incisions.
  - Rule out periprosthetic infection.
- Order ultrasound/CT.
  - Look for fluid collection or stranding/inflammation around implant.
- Treatment.
  - Superficial.
    - Cellulitis can be treated with antibiotics.
    - Mild cases can be treated as an outpatient with oral antibiotics.
      - Minocycline 100 mg by mouth twice a day or clindamycin 300 mg by mouth four times a day.
    - More severe manifestations or failure of outpatient therapy will require IV antibiotics.
      - Clindamycin 900 mg IV every 8 hours or vancomycin 1 g IV every 12 hours + cefepime 1 g IV every 12 hours for severe infections; also consider antibiotic therapy with equal oral or IV bioavailability (e.g., linezolid, Levaquin, and moxifloxacin).

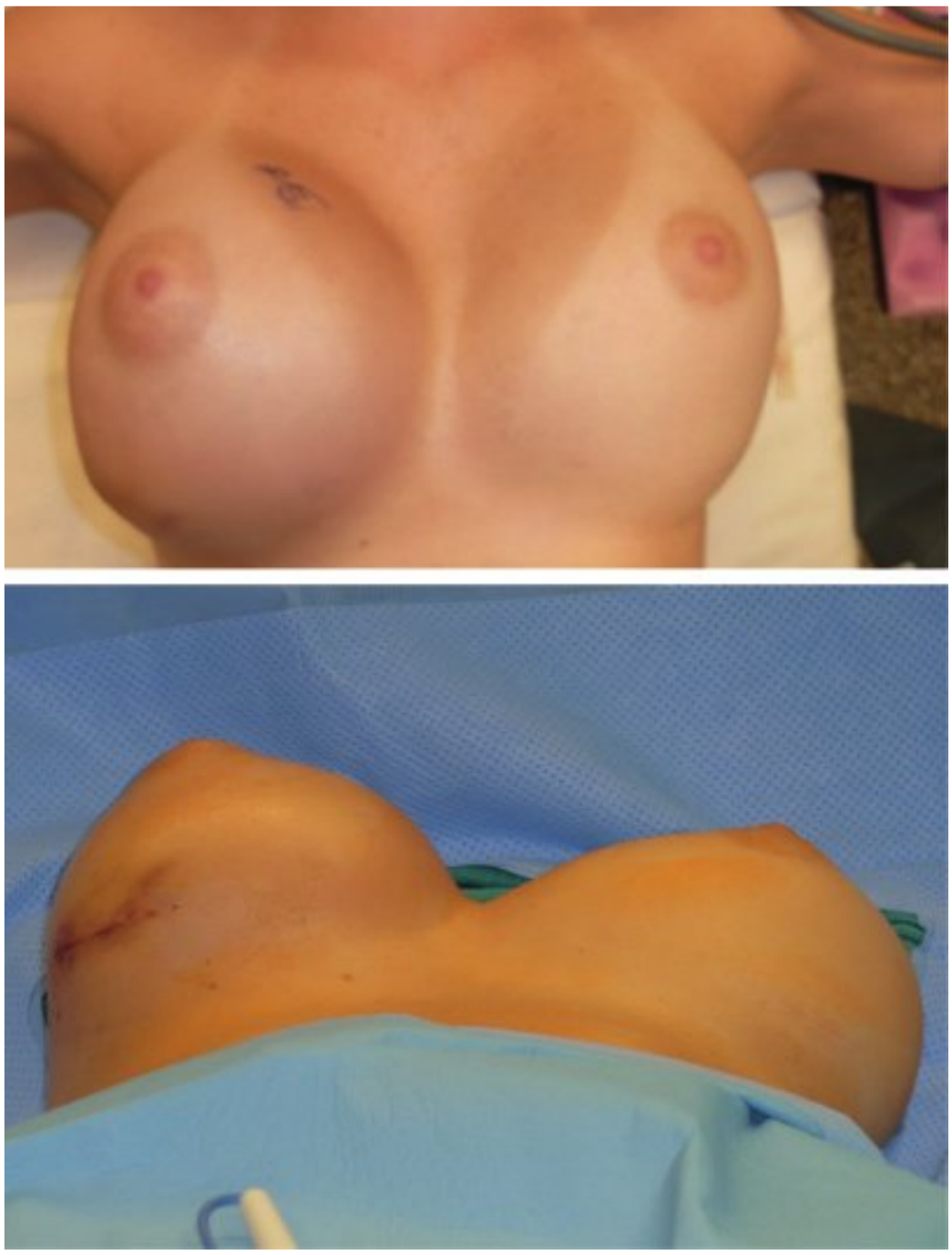

Fig. 21.1 Right breast hematoma after breast augmentation.

  - Exposed implant.
    - Minor contamination without infection.
    - IV antibiotics.
    - Local wound care—Betadine paint.
    - Plan for explantation with device change.
    - Capsulectomy and pocket débridement.
    - ± Site change or flap coverage in reconstruction cases.
  - Infected implant.
    - With evidence of periprosthetic fluid collection and reaction, start IV antibiotics.
      - Plan for explantation and removal of contaminated prosthesis.
      - Capsulectomy and irrigation.
      - Intraoperative evaluation of the pocket.
        * In mild cases, the new prosthesis can be placed with a drain.
        * In severe cases, close the wound over a drain and delay implant placement for 3 to 6 months.
        * Institute an extended course of culture-specific antibiotics postoperatively.

Rhinoplasty

Airway Obstruction

- Nasal packing or intranasal splint aspiration.
  - Evaluate the nasal and oral airways and clear potential obstacles to air movement.
- Assess for aspiration of blood causing laryngospasms.
  - Prepare for airway protective measures, suction, oxygen supplementation, and possible intubation.

Visual Impairment

- Vasospasm from local anesthetic vasoconstriction.
- Thromboembolism causing ophthalmic artery ischemia.
- Treatment.
  - Urgent ophthalmology consult.

Hemorrhage

- Localize source.
- Treatment.
  - Packing.
    - Gauze.
    - Surgicel.
  - Endoscopic cauterization.
  - If all else fails: Posterior nasal packing (see Chapter 6, **Fig. 6.4b**).

Septal Hematoma

- Treatment.
  - Aspiration.
  - Incision, drainage, and packing.
  - Antibiotic coverage to prevent septal abscess,
  - Augmentin 875 mg by mouth twice a day.

Infection

Local

- Cellulitis.
- Abscess.
- Treatment.
  - Augmentin 875 mg by mouth twice a day.

Toxic Shock from Nasal Packing

- Postoperative fever, vomiting, diarrhea, hypotension without obvious blood loss, and an erythematous sunburnlike rash.
- The supertoxin toxic shock syndrome toxin-1 (TSST-1), produced by the organism *S. aureus,* causes this syndrome.
- Treatment.
  - Removal of nasal packing and acquisition of nasal cultures.
  - Appropriate β-lactamase-resistant antistaphylococcal IV antibiotics.
    - Unasyn 3 g IV every 6 hours.
  - Aggressive hemodynamic resuscitation.

Intracranial Infections

- Meningitis.
- Subdural empyema.
- Cerebral abscess.
- Cavernous sinus thrombosis.
  - Diagnose with CT and treat with broad-spectrum antibiotics.
- Acute and/or chronic sinusitis.
  - Treat with Augmentin 875 mg by mouth twice a day.

Neurosurgery Consult

Edema

- Treatment.
  - Head elevation.
  - Cold compresses.
  - Blood pressure control.

Blepharoplasty

Retrobulbar Hemorrhage

- Pain, proptosis, ophthalmoplegia, ± blindness (see Chapter 6, **Fig. 6.6**).
- Treatment.
  - If patient has visual changes.
    - At bedside, open sutures and perform lateral canthotomy emergently.
    - Decadron (Merck & Co., Inc.) 10 mg IV.
    - **Plan immediate exploration in the operating room.**
  - If patient does not have visual changes.
    - Plan immediate exploration in an operating room.
    - Steroids controversial.
  - Control hypertension.
  - IV 20% mannitol (1 g/kg) and acetazolamide (500 mg IV initially, then 250 mg IV every 6 hours) can be used to decrease the intraocular pressure.
  - If access to the operating room is delayed and patient starts to lose visual acuity, then lateral canthotomy and cantholysis may be performed at bedside (**Fig. 6.6**).

Corneal Abrasion

- Diagnosis.
  - Pain, tearing, and sensation of foreign body in eye.
  - Diagnosis with slit lamp by ophthalmologist.
- Treatment.
  - Rule out a foreign body.
  - Maxitrol (Alcon Laboratories) eyedrops.
  - Lacri-Lube (Allergan, Inc.).
  - Ophthalmic bacitracin ointment.
  - Resolves in 24-48 hours.
  - Pressure dressing with eye closed for 24 hours.

Edema

- Treatment.
  - Elevation of head.
  - Swiss Therapy eye mask (Invotec) (cold compress).

Rhytidectomy

Hematoma

- Most common complication, usually resulting from high systolic blood pressure, aspirin or nonsteroidal anti-inflammatory drug (NSAID) intake, or nausea and vomiting.
- Symptoms.
  - Pain, agitation, hypertension, neck/facial swelling, buccal mucosa ecchymosis, and skin ecchymosis.
  - Can lead to skin necrosis.
  - May present with *respiratory distress* when the neck is involved from pressure on the airway.
- Treatment.
  - Large hematomas.
    - Require immediate surgical drainage in the OR to avoid flap necrosis and external airway obstruction.
  - Small hematomas.
    - Evacuate at bedside by expression or serial needle aspirations and pressure dressing.
  - Control blood pressure.

Nerve Injury

- Assess the patient's facial symmetry by asking him or her to raise eyebrows, smile, and pucker lips.
- Most motor nerve paralysis in the acute postoperative patient is due to local anesthetic effect, excessive traction of the superficial musculoaponeurotic system (SMAS), infection, or hematoma.
- The most common nerve injured is great auricular nerve—provides sensation to the inferior ear and ear lobule.
- The most common motor nerve injury is to the buccal branch of the facial nerve.
- Treatment.
  - Nerve paralysis immediately after surgery should be treated with observation. Notify the surgeon of specific physical findings to help determine the origin/treatment of the facial nerve paralysis.

Skin Flap Necrosis

- May first present as cyanosis or congestion that may be reversible.
- Assess for hematomas, seromas, or infection and treat appropriately.
- Partial skin flap necrosis.
  - Apply moist gauze or antibiotic ointment.
  - Treat full-thickness injury with conservative débridement and healing by secondary intention.
- If patient presents with skin ulcers around the mouth, this may indicate a herpes outbreak and the patient should be started on Valtrex (GlaxoSmithKline) 500 mg twice a day.

Liposuction

Fluid Balances

- Large volume liposuction (> 4 L) can have large fluid shifts.
  - Monitor urine output closely with Foley catheter
  - Calculate fluid balance in terms of total in and out during the procedure.
    - Input = IVF + wetting solution.
    - Output = aspirate + urine output.
- Fluid replacement.
  - Small volume < 2,500 mL aspirate.
    - Maintenance IVFs only.

  - Larger volume > 2,500 mL aspirate.
    - Fluid replacement guideline below.
- General guideline for fluid replacement.
  - Total IVF supplement (mL).
    - Perioperative IVFs + postop IVFs + wetting solution = 2× aspirate (mL).
    - Postop fluid replacement = 2×aspirate – [perioperative fluid + wetting solution].
  - Titrate to urine output.
  - Aggressive hydration will cause a hypervolemic state and subsequent cardiopulmonary morbidity.

Blood Loss

- Blood loss is calculated based on the wetting technique (**Table 21.1**).

Hematomas/Seromas

- Treat with compression garments.
- Consider further padding with foam or bulky surgical dressings.
  - Large fluid collections that cause excessive skin tension and ischemia requiring operative intervention.
- Seromas may be aspirated at the bedside or under ultrasound guidance.
  - Consider percutaneous placement of a catheter for large or recurrent seromas.
    - Ultrasound guided with assistance of interventional radiology.
    - Seldinger placement of a seroma catheter (SeromaCath [Greer Medical, Inc.]) (**Fig. 21.2**).

Table 21.1 Wetting technique to calculate blood loss

Technique	Infiltrate	EBL
Dry	None	20–40%
Wet	200–300 mL/area	8–20%
Superwet	1 mL infiltrate: 1 mL aspirate	1%
Tumescent	2–3 mL infiltrate: 1 mL aspirate	1%

Abbreviation: EBL, estimated blood loss.

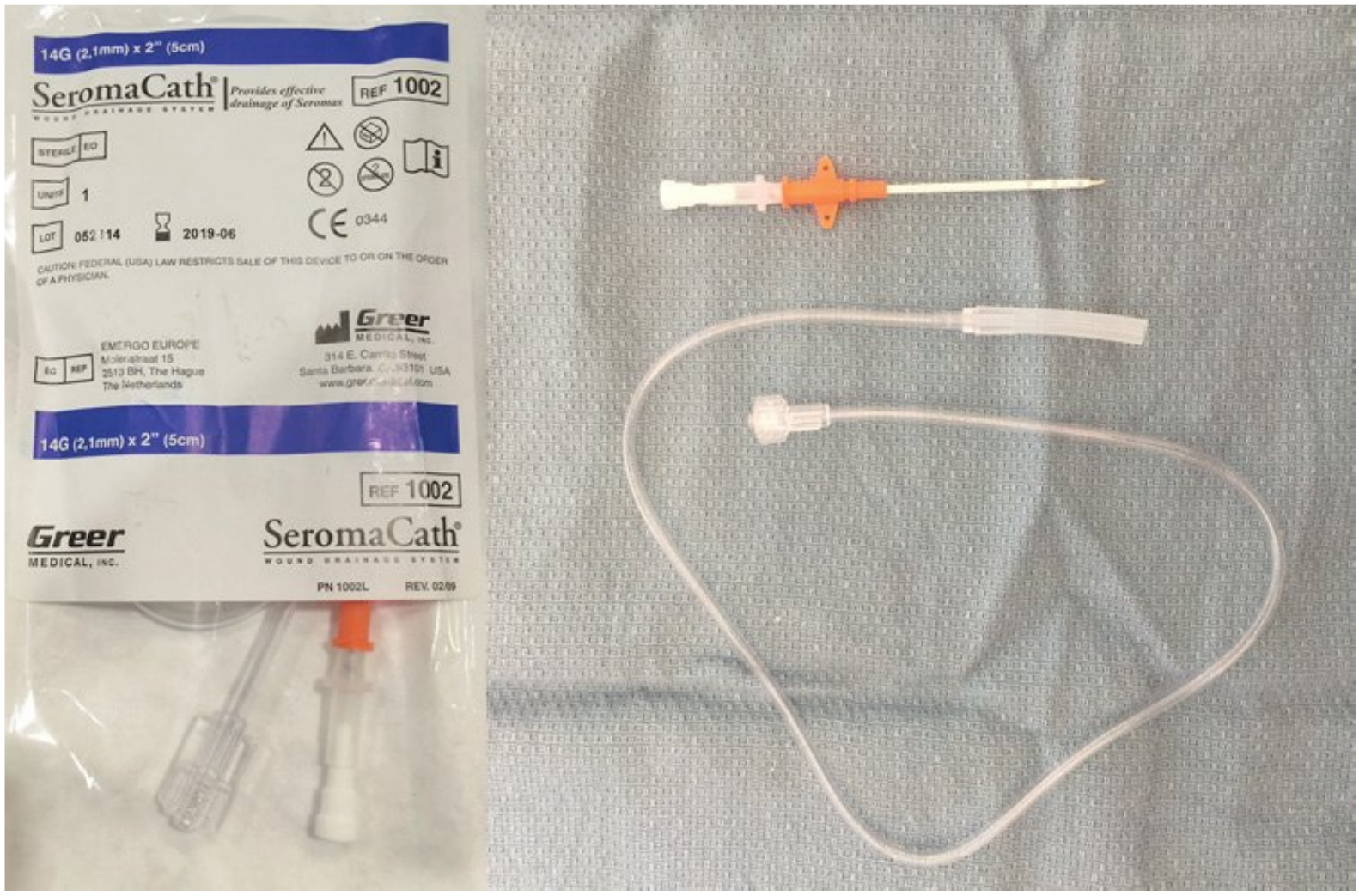

Fig. 21.2 Seroma catheter. A seroma catheter can be placed, using the Seldinger technique, into the suspected cavity and connected to an active suction device (bulb suction).

Lidocaine Toxicity

Recommended dose when used at 0.05%= 35 mg/kg in wetting solution.

- Diagnosis.
  - Circumoral numbness.
  - Metallic taste.
  - Tinnitus.
  - Lightheadedness, dizziness.
  - Impaired concentration.
  - Visual disturbance.
  - Headache.
  - Sedation.
  - Tremors.
  - Seizures.
  - Greater levels of toxicity may lead to coma or cardiopulmonary arrest.
- Treatment.
  - Supportive care.
  - Oxygen/hydration.
  - Maintenance of airway.
  - Intralipid infusion: 1.5 mL/kg of 20% IV bolus, then 0.25 mL/kg/min for 30 minutes. May repeat 1 to 2 times if patient fails to improve.

  - Benzodiazepines for seizure prophylaxis.
    - Diazepam 5 to 10 mg or thiopental 50 to 100 mg.

Hypoesthesias

- Common and transient—sensation returns to normal within 6 months.

Respiratory Distress

- Fat embolism syndrome.
  - Intravenous fat deposits that cause pulmonary compromise and may lead to acute respiratory disease syndrome.
- Physical examination.
  - Tachycardia, tachypnea, dyspnea.
  - Hypoxemia due to ventilation-perfusion abnormalities.
  - Bradycardia.
  - High spiking fever.
  - Petechiae over the trunk.
  - Subconjunctival and oral hemorrhages.
  - Agitated delirium.
  - Stupor, seizures, or coma.
  - Retinal hemorrhages.
- Diagnostic studies.
  - ABGs—hypoxemia, increased pulmonary shunt fraction.
  - Thrombocytopenia.
  - Anemia.
  - Hypofibrinogenemia.
  - Urinary fat stains—fat globules in the urine.
- Treatment.
  - Supportive therapy.
  - Monitored care environment.
  - Continuous oxygen and pulse oximetry.
  - Hydration.
  - DVT prophylaxis.
  - Gastrointestinal stress prophylaxis.
  - Steroids.
    - Decadron 4 mg IV every 8 hours.
- Pulmonary embolism and pulmonary edemas should also be considered in the differential diagnosis.
  - See Abdominoplasty.

22 Sternal Wounds

Early and aggressive intervention and treatment of sternal wound infections is required to avoid catastrophic complications such as severe mediastinitis and death. These wounds find their genesis in cardiac surgery, which may leave the sternum with a suboptimal vascular blood supply, ultimately leading to a chronic contaminated wound or sternal osteomyelitis.

Sternal wounds can be divided into three categories (**Table 22.1**).

- **Class I** wounds include wounds with drainage, with or without sternal instability, that present within the first week postoperatively.
- **Class II** wounds present with cellulitis, purulent mediastinitis, drainage, and sepsis (fevers), usually in the second or third week postoperatively.
- **Class III** wounds present with osteomyelitis and serosanguineous or seropurulent drainage months to years after cardiac surgery (**Fig. 22.1**).

Obtain a thorough **history** that contains the following:

- Type of cardiac surgery performed.
- Determine whether the internal mammary arteries had been harvested (and on which side).
- Elucidate whether the cardiac surgeons engaged in undermining of the pectoralis myocutaneous flap or harvest of the omentum.
- Determine if there is history of intra-abdominal surgery.
- Review concurrent anticoagulation therapy and coagulation profile.

Physical Examination

- Clicking of the sternum indicating loose wires.
- Presence of purulent drainage, location of the drainage.
- Presence of cellulitis should be noted.

Work-up

- Cultures should be taken from the wound.

- CT of the chest with contrast for evaluation of fluid collections above or beneath the sternum.
- Check WBCs, C-reactive protein, and ESR.

Table 22.1 Pairolero and Arnold classification system for sternal wounds.

Class	Presentation time postoperatively	Location of infection	Diagnosis	Treatment
I	1 wk	Superficial	Drainage +/- stable sternum	Débridement, tighten wires, pectoralis flaps; single stage
II	2–3 wk	Mediastinum	Fluid collection and an unstable sternum, high WBC, purulent mediastinitis, sepsis and positive cultures	Débridement, remove wires, pectoralis flaps, omental flap, +/- hardware; one or two stages
III	Chronic	Sternum/ wires	Osteomyelitis, draining fistula, localized cellulitis, rare mediastinitis	Débridement, remove wires, pectoralis flaps, omental flap; single stage

Abbreviation: WBC, white blood cell.

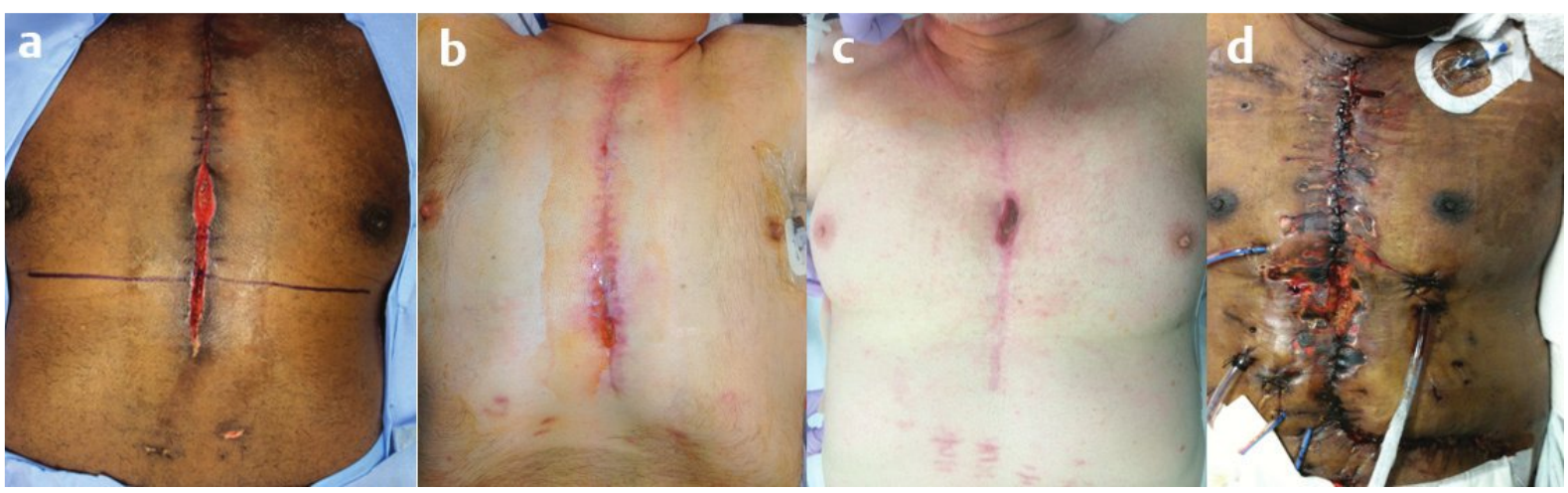

Fig. 22.1 Clinical examples of sternal wounds. **(a)** Class I sternal wound. **(b)** Early class II sternal wound. **(c)** Late class II sternal wound. **(d)** Class III sternal wound.

- PT/PTT/INR.
- If the patient is anticoagulated on warfarin, he or she should be converted to a heparin drip prior to extensive débridement.
- Communication with the cardiovascular surgery, cardiology, and infectious disease teams is an absolute requirement.

Treatment Paradigm

The patient will require operative débridement to decontaminate the wounds and remove necrotic tissue, and thorough exploration of hardware with determination of sternal stability. In the perioperative setting and in the postoperative setting, culture-driven intravenous antibiotics are a requirement. Based on the type of infection that the patient has, treatment may be urgent or elective. In the case of class II purulent mediastinitis, it is imperative for the patient to return to the operating room urgently for débridement of the wound and clearing of the infection, since graft rupture or thrombosis can occur. Class I wounds may be treated by débridement and drainage in the suprasternal area with tightening of the wires or placement of sternal plates or devices. Class II wounds are treated with meticulous débridement of the subcutaneous tissue, the sternum, involved ribs, and all necrotic tissue. Classically, class II wounds are also treated as a staged operation, with return to the operating room as necessary to remove all foreign bodies and necrotic bone or tissue. Class III wounds are treated with removal of infected bony sequestra or wires, followed by immediate flap coverage. If good bone stock is available after a meticulous, thorough débridement of all necrotic tissue, and if the wound is completely clear of infection and purulent drainage, the sternum can be fixated with new plates or sternal fixation devices, either in one stage or in two separate stages. If staged, dressing changes can be performed with Xeroform and wet to dry dressing changes on top of the Xeroform. Alternatively the Vac can be used on top of a layer of Xeroform with very low pressure settings (25 mm Hg). Care must be taken not to place the VAC directly over any vascular structures; these must be protected.

Once the wound is clean, coverage can be provided with pectoralis advancement flaps (based on the pectoral branch of the thoracoacromial vessel), which are excellent for coverage of the superior two-thirds of the sternum. In the pediatric population, pectoralis advancement flaps can cover the entire sternum. The lower portion of the sternum can be covered either with a pectoralis turnover flap (based on the internal mammary perforators) using the whole pectoralis or a split pectoralis, or with the omental flap harvested from the abdomen (based on the gastroepiploic vessels). If the pectoralis and omentum are not available,

the rectus abdominis muscle (or myocutaneous flap) can be used to cover the lower portion and the upper portion of the sternum. It is of the utmost importance to determine the harvest of the internal mammary arteries prior to performing a pectoralis turnover flap or a rectus abdominis muscle flap. The rectus abdominus can be based on the eighth intracostal artery. If none of these flaps are available, the latissimus dorsi flap can be used to cover the sternum.

In patients who do not have enough healthy sternum or rib bone to allow for fixation of the chest wall, flaps can be used by themselves for coverage of the heart and vessels. In most all patients without pulmonary compromise, the inflammatory reaction caused by the infection results in sufficient rigidity for the chest wall. Radiation has the same effect. Once coverage of vital structures has been provided, skin can be closed over the muscles by elevation of fasciocutaneous flaps from the chest wall. Flaps can usually be raised up to the anterior axillary line; care must be taken in elevation of the large flap in female patients with **macromastia** because the blood supply of skin edges may become compromised.

Postoperative Care

- Drain the subcutaneous and subflap regions with large closed suction drains that are sequentially removed over 2 to 3 weeks.
- Mammary support or binder can be used in female patients.
- Anticoagulation can usually be started within the first 5 days postoperatively depending on indications for anticoagulation.
- Long-term antibiotics, as determined by the infectious disease team and extent of infection, are required.
- Physical therapy and rehabilitation.
- Close follow-up over the first month; it is essential to ensure that the purulent mediastinitis has resolved.

Index

A

B

C

D

E

F

G

H

I

J

K

L

M

N

O

P

R

S

T

U

V

W

X

Z