Chapter 18

WOUNDS AND SOFT TISSUE INJURIES

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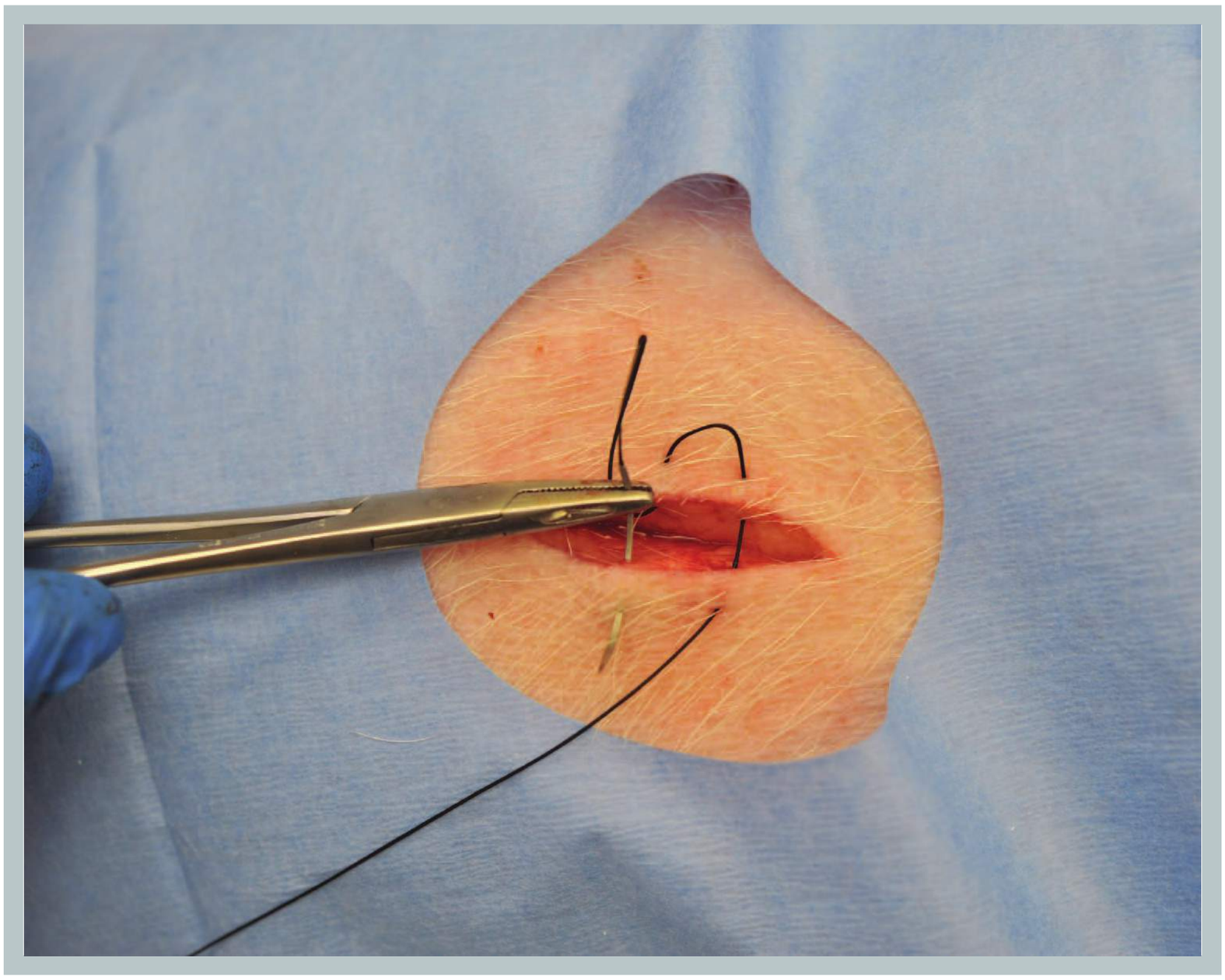


Horizontal Mattress Suture. (Photo contributor: Michael L. Juliano, MD.)

The authors acknowledge Matthew D. Sztajnkrycer and Alexander T. Trott for portions of this chapter written for the second edition.

WOUND CLEANING AND IRRIGATION

Clinical Summary

The goals of minor wound care are to achieve optimal wound aesthetics and infection prevention. For most uncomplicated wounds, irrigation is the most effective means of reducing bacterial count. However, debridement may be necessary in contaminated wounds since devitalized tissue may impair the wound's ability to resist infection.

Management and Disposition

Preliminary wound management begins with assessment, adequate hemostasis, foreign-body removal, and irrigation. Provide adequate analgesia prior to wound cleansing. In simple well-vascularized wounds, tap water is as effective as normal saline or sterile water. In contaminated wounds, povidone-iodine diluted 1:10 with normal saline may help with disinfection. Bacterial-static solutions, such as nonionic surfactant cleaner, may also reduce bacterial inoculum. Solutions containing ionic detergents (eg, Betadine surgical scrub) should not be used as it is toxic to wound tissue. If necessary, wound scrubbing should be done gently to avoid damaging viable tissue.

Irrigation is the most effective means of reducing bacterial inoculum; 500 to 1000 mL of fluid or 60 mL/cm of wound length is adequate for most uncomplicated wounds. The recommended irrigation pressure of 5 to 8 lb per square inch (PSI) can be accomplished by attaching an 18- or 19-gauge intravenous (IV) catheter sheath, or a commercially available splash shield, to a 20- or 30-mL syringe. A bulb system is suboptimal; it generates only 0.5 to 1 PSI. Debris that cannot be irrigated can be scrubbed or sharply debrided. The tissue should appear pink and viable; a scant amount of fresh bleeding indicates good vascular supply. High-pressure irrigation (≥25 PSI) may be necessary in highly contaminated or complicated wounds requiring operating room washout. However, it offers no advantage for routine wounds.

FIGURE 18.1 ■ **Wound Soaking.** Soaking is an appropriate method for loosening debris and coagulated blood but is not a substitute for irrigation. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

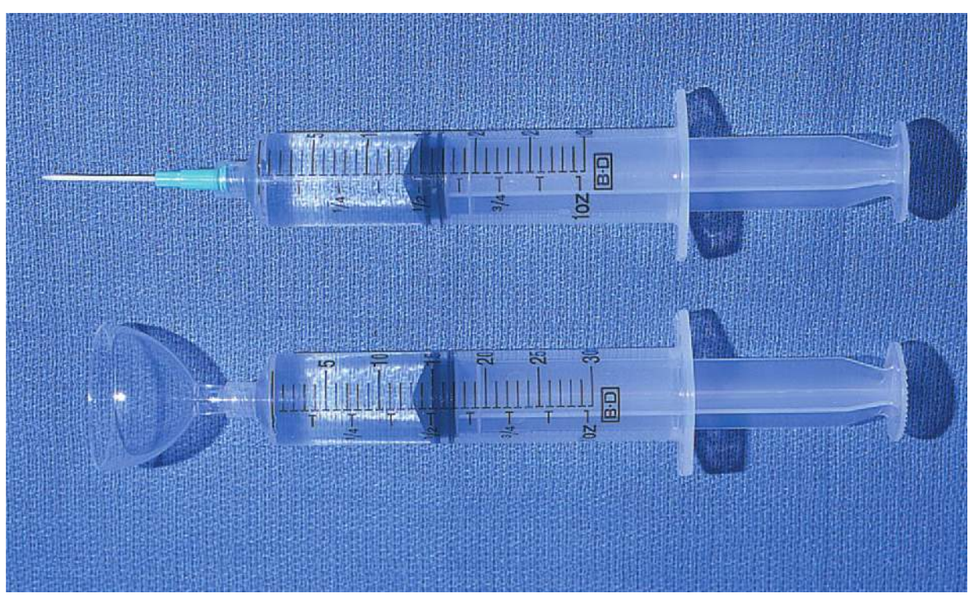


FIGURE 18.2 ■ **High-Pressure Irrigation Devices.** Ideal pressure for routine wound irrigation is 5 to 8 PSI. This can be achieved with a 30-mL syringe attached to an 18- or 19-gauge IV catheter sheath (top) or splash shield (bottom) (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

Pearls

1. Universal precautions, including gloves and face shield, should always be observed.
2. Antibiotics are no substitute for thorough wound cleansing and irrigation.
3. Shaving the eyebrow for wound repair is contraindicated due to the unpredictable pattern of hair regeneration.

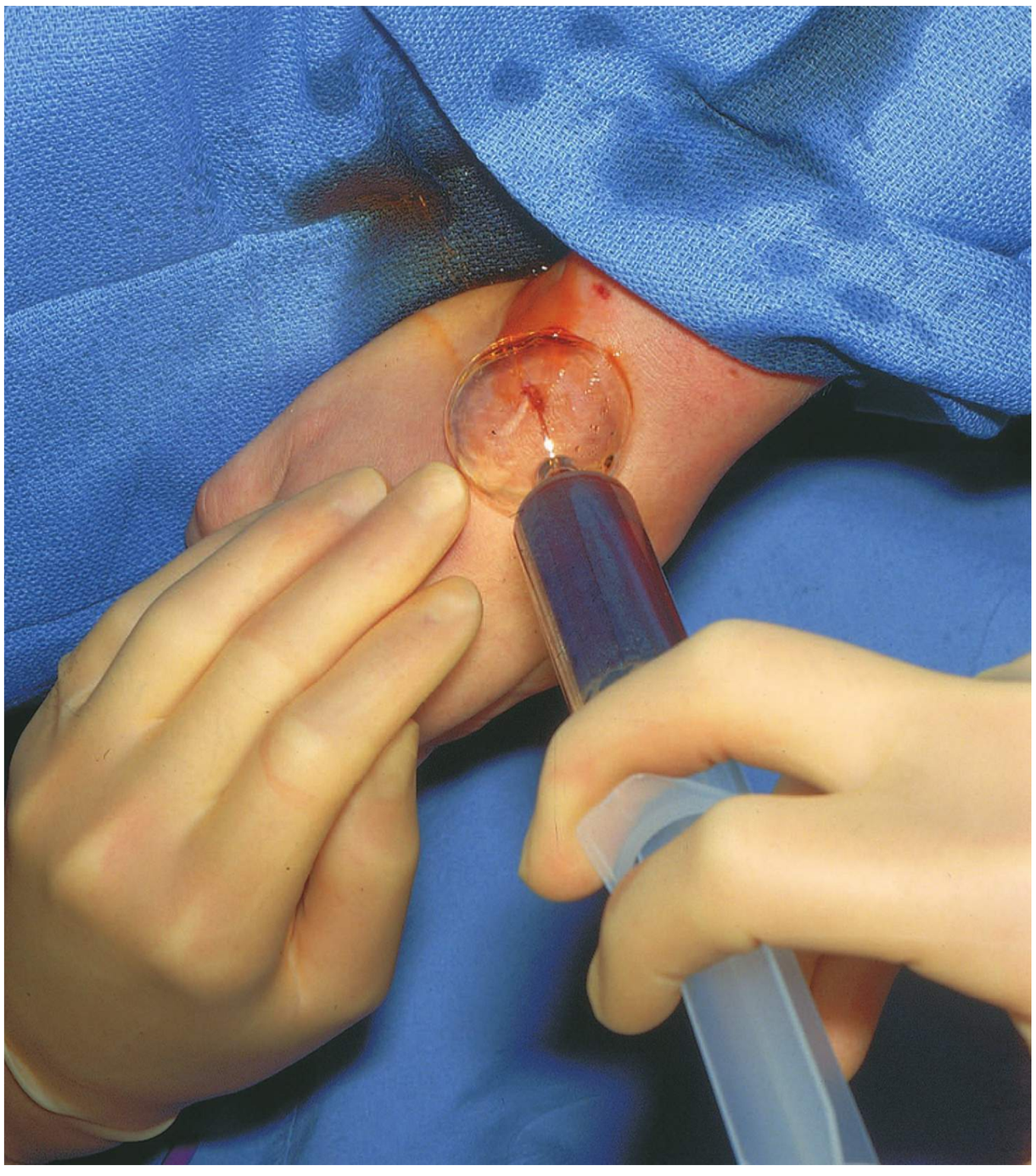


FIGURE 18.3 ■ **Wound Irrigation.** After adequate anesthesia, a wound is thoroughly irrigated. Even with the shield, there can be significant splatter and potential for body fluid exposure. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

Clinical Summary

Proper wound assessment and preparation are essential to good wound management. Always consider the age and mechanism of injury, risk for contamination or foreign body, risk to the nerve, blood vessel and tendon, tetanus status, and identifying comorbid conditions that may affect wound healing.

Management and Disposition

Patient compliance is integral to achieving adequate wound exploration. The use of local or regional anesthesia is usually sufficient. However, procedural sedation may be required in some. Document neurovascular status prior to any anesthetic administration. Application of direct pressure is often the easiest way to achieve hemostasis. Other methods include the use of blood pressure cuff or tourniquet to achieve temporary hemostasis. Anesthetic solution containing epinephrine (1:100,000 dilution) may help constrict small vessels; however, particular caution should be exercised when using any vasoconstrictors in areas of end arterial circulation (eg, fingers, nose, toes, ears, and penis).

While hemostats or other self-restraining devices such as retractors may help achieve adequate exposure, care must be exercised to avoid damaging the dermis and the tissue's vascular integrity. If exposure is still not adequate despite hemostasis and separation, the wound margins may be slightly extended with fine iris scissors or scalpel to allow better visualization. The wound is extended from one end, through the epidermis and dermis only, to avoid further injury to underlying structures. Once the superficial fascia has been exposed, it may be carefully and bluntly dissected using forceps or scissors.

Pearls

1. Never probe a wound blindly or blindly attempt to control bleeding with hemostats.
2. At 1:100,000 dilution, epinephrine-containing anesthetics may be used with caution in areas of end arterial circulation such as the ear, fingers, nose, toes, and penis.
3. If epinephrine-induced tissue ischemia occurs, injection of phentolamine around the area of ischemia may help restore flow.

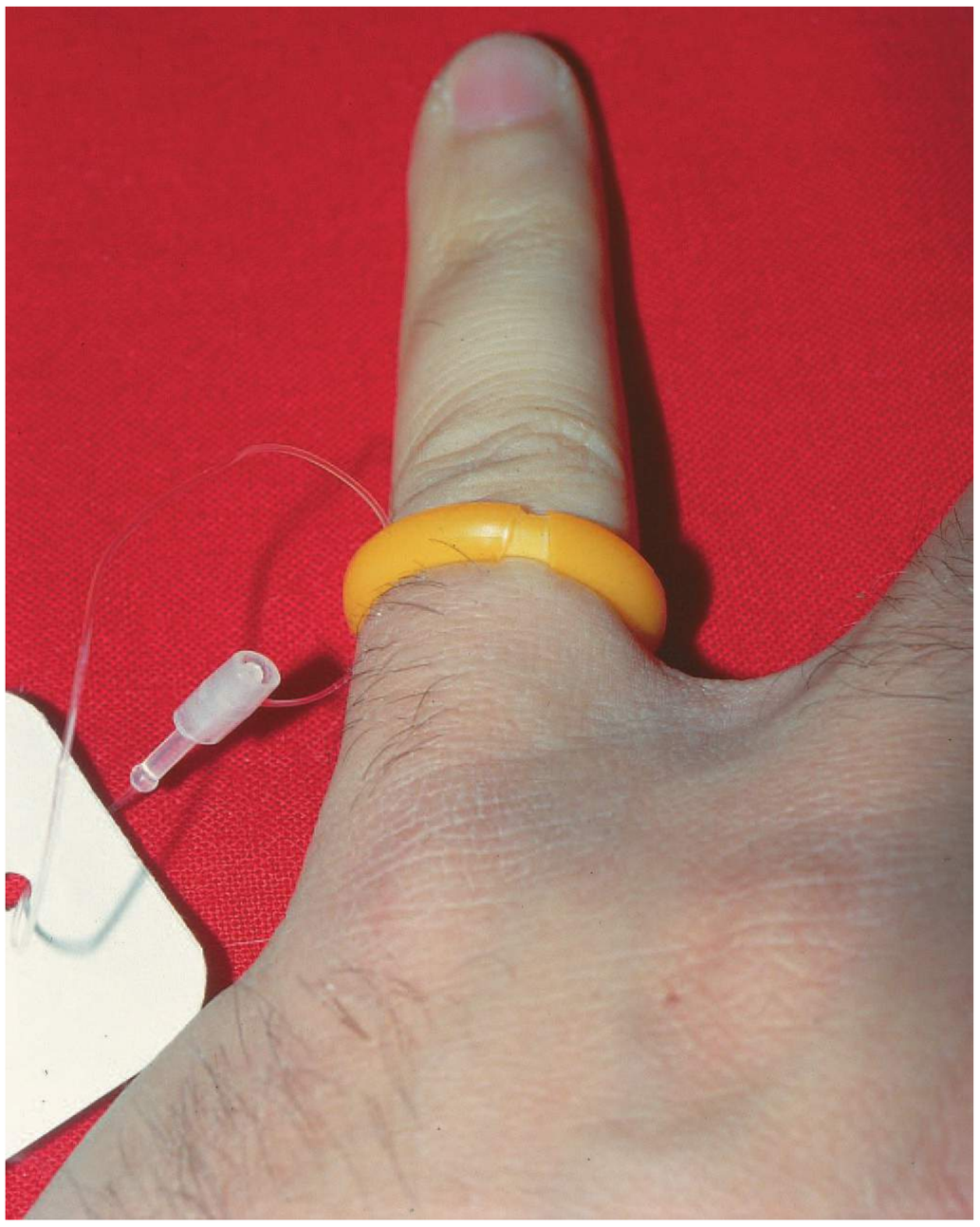


FIGURE 18.4 ■ **Finger Ring Tourniquet.** The ring tourniquet is an effective means of hemostasis. Removal after the procedure is important to prevent finger ischemia. Another effective method of hemostasis involves using a Penrose drain tightened with hemostats. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

FIGURE 18.5 ■ **Epinephrine Injection.** Although epinephrine can be used to help achieve hemostasis, it should be used with caution on areas with poor collateral blood supply. Epinephrine has been injected into the right thumb which shows the pallor of finger ischemia. (Photo contributor: Selim Suner, MD, MS.)

FIGURE 18.6 ■ **Wound Exploration.** A blood pressure cuff is an alternative means to obtain hemostasis before wound assessment. (Photo contributor: Alan B. Storrow, MD.)

WOUND FOREIGN BODIES

Clinical Summary

All foreign bodies can become a nidus for delayed infection. All reasonable attempts should be made to remove them. Radiographic evaluation or ultrasound (see related chapter) may assist in locating foreign bodies not directly visualized. Foreign bodies are characterized as being either reactive (eg, organic materials such as wood, bone, and soil) or nonreactive (eg, glass and metal).

Management and Disposition

Patients are often unaware that a foreign body is present in their wound; a high level of clinical suspicion should accompany any injury pattern at risk for foreign-body penetration. Wounds at increased risk for foreign-body penetration include lacerations caused by broken glass, perioral injuries with loss of dentition, and injuries to the hands and feet involving needles, nails, or splinters.

Suspicion of a retained foreign body mandates local wound exploration and the consideration of radiographic or ultrasound evaluation. Nearly 80% of objects can be identified on plain radiographs. More specifically, approximately 90% of glass fragments greater than 2 mm in size can be identified through the use of plain radiographs; fragments as small as 0.5 mm can be identified in 50% to 60% of cases. In situation where plain radiographs are poor, ultrasound may be considered.

Due to their increased risk for delayed infection and poor wound healing, reactive material must be removed. Nonreactive objects, however, may be left in place if reasonable effort to remove it has been unsuccessful, and no potential for harm to a vital structure exists. Glass, however, has the potential for significant irritation and a removal attempt should be pursued.

Pearls

1. The base of the wound must be visualized as many foreign bodies hide there.
2. Common foreign bodies retained in hand wounds are wood splinters, glass fragments, metallic objects, and needles.
3. Missed retained foreign bodies are a very common source of litigation in emergency medicine.

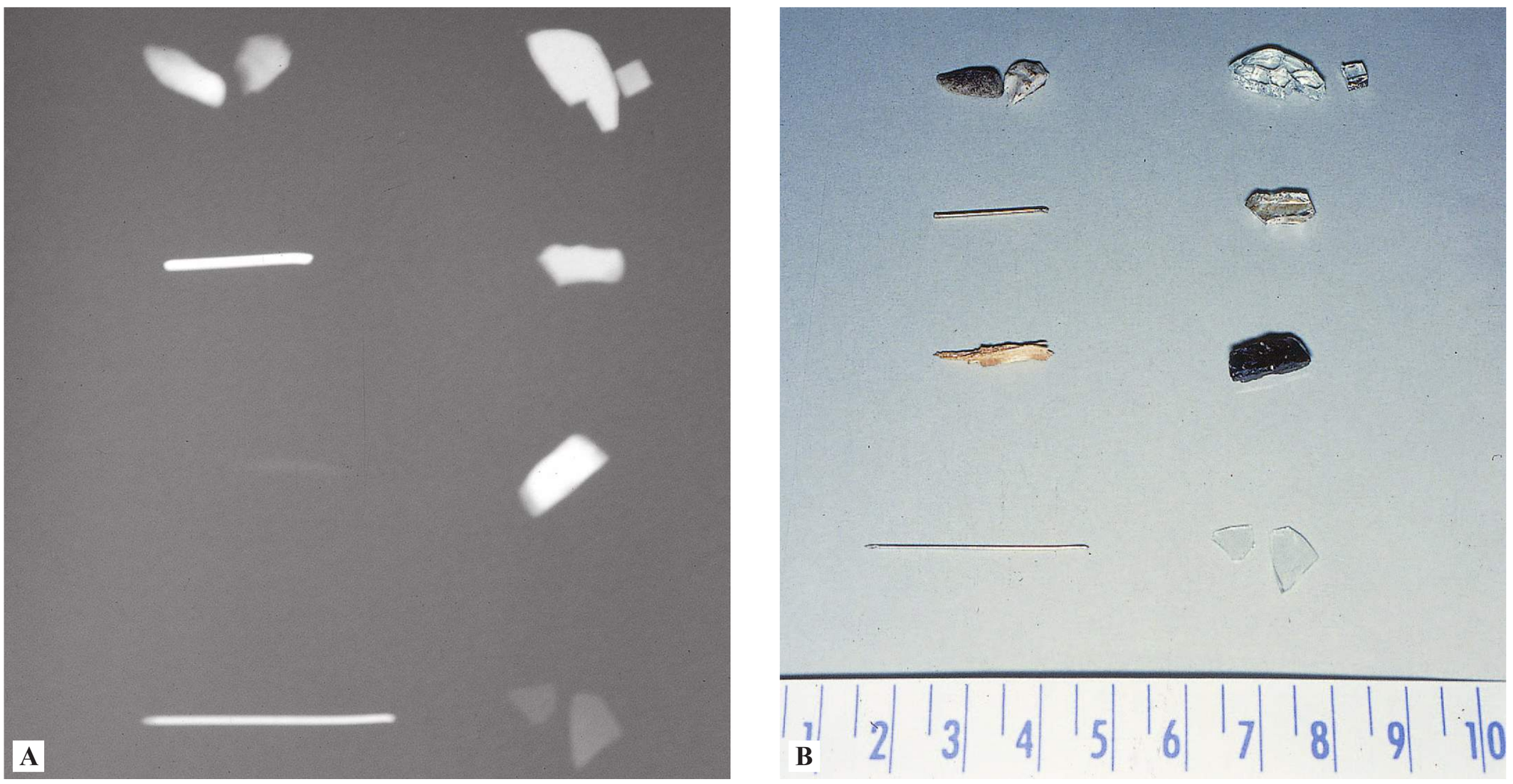


FIGURE 18.7 ■ **Radiodensity of Common Foreign Bodies.** The plain radiograph (**A**) demonstrates the radiodensity of common foreign bodies. Counterclockwise from top left: pebbles, paper clip fragment, wood splinter, hollow needle, light bulb glass, dark ("beer bottle") glass, transparent glass, and automobile windshield glass. Note that, although faint, the wood splinter is visible on plain radiography. Ruler markings (**B**) for the photograph of the corresponding objects are in centimeters. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

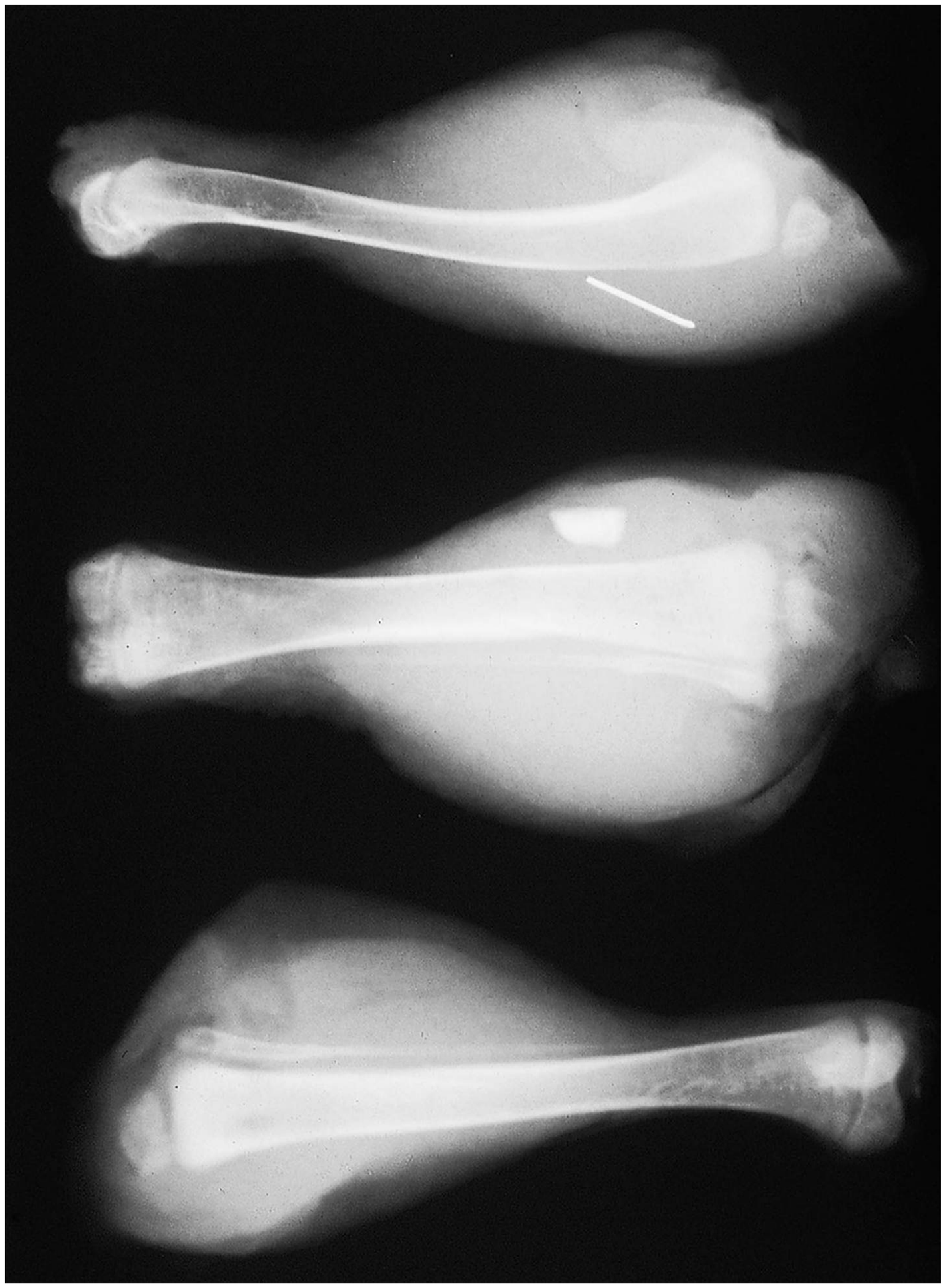


FIGURE 18.8 ■ **Radiodensity of Common Foreign Bodies in Tissues.** The paper clip, dark glass, and wood splinter (top to bottom) imaged in Fig.18.7 were inserted into chicken legs and radiographs taken. The wood splinter is no longer clearly visible within the soft tissue. For purposes of foreign-body localization, a minimum of two radiographic views at 90 degrees to one another are obtained, and the site of the foreign-body entry clearly marked. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

Clinical Summary

Traumatic surface wounds are caused by one of three mechanisms: shearing, tension, or compression. Such a division helps to guide management decisions involving infection risk and scar formation.

Shearing injuries are caused by sharp objects, such as glass shards or knives, which impart low-energy injury and minimal tissue destruction. A majority of uncomplicated shearing injuries (ie, those not involving neurovascular or anatomically important structures) are repaired primarily in the emergency department (ED). The risk of infection is low and scar formation is typically cosmetically acceptable. Puncture wounds occur from sharp objects that pierce the skin and penetrate into deeper tissues. Such wounds are at a higher risk for infection, foreign-body retention, and underlying structural injury.

Tension or partial avulsion injuries occur when an object strikes the skin at a sharp angle creating a triangular flap. This results in potential vascular disruption, greater tissue destruction, and a higher risk for infection and tissue ischemia. During the repair, vascular supply to the flap must be meticulously preserved; otherwise, the flap may become ischemic.

Crush or compression injuries occur when a blunt object strikes tissue at a right angle, imparting a high degree of kinetic energy. This results in significant tissue destruction of the skin and its underlying supportive fascial layers. Crush injuries are typically ragged, with irregular wound edges and a complex laceration pattern. Despite meticulous wound care and careful primary closure, the resulting scars may be cosmetically poor.

Management and Disposition

First update the tetanus status of all patients requiring wound management. Carefully document the time and functional/neurovascular status at initial evaluation, as this may change over time. Upon optimizing wound preparation, appropriate closure tension must be achieved. If open fracture is suspected

FIGURE 18.9 ■ **Linear Laceration.** A large, uncomplicated, linear leg laceration. Given the depth and gaping, it can be sutured using a layered closure to remove surface tension at the wound edges and promote a more cosmetically acceptable outcome. (Photo contributor: Alan B. Storrow, MD.)

FIGURE 18.10 ■ **Linear Laceration.** A long, linear shearing laceration involving the forehead and scalp, with exposed galea. The wound is explored and palpated for evidence of a depressed or open skull fracture. Closure of large galeal lacerations is recommended to prevent infection. Large frontal galeal lacerations are also repaired to prevent a cosmetic deformity of the frontalis muscle. (Photo contributor: Kevin J. Knoop, MD, MS.)

or confirmed, IV antibiotics are administered and orthopedic consultation obtained. Repair of traumatic wounds depends on the depth, complexity, and location. Deep wounds are closed in layers or by using a vertical mattress technique to remove dead space and relieve tension. Superficial wounds may be repaired with staples, simple interrupted sutures, or running sutures. In certain circumstances, the use of adhesive skin closures or adhesive glues may be adequate.

Pearls

1. Simple, uncontaminated, and uncomplicated wounds caused by clean, sharp objects in otherwise healthy patients may undergo primary closure up to 18 hours from the time of injury with good cosmetic result (see Simple Wound Closures).
2. The vascular supply to a flap is often tenuous; improper closure may further compromise the tissue, especially at the tip. A repair using a corner stitch will help minimize further ischemia (see Complex Wound Closures).
3. Crush injuries have an increased susceptibility to infection. Thorough cleansing, copious irrigation, and judicious debridement are required.

FIGURE 18.12 ■ **Puncture Wound.** A variety of linear objects may result in puncture wounds, in this case a bicycle brake. Radiographic evaluation is mandatory to assess for bone and joint involvement. (Photo contributor: Larry B. Mellick, MD, MS.)

FIGURE 18.11 ■ **Puncture Wound.** A puncture wound to the foot with a contaminated garden instrument. Tetanus status must be carefully addressed in such an injury. A radiograph of the foot demonstrated no associated bony injuries. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

FIGURE 18.13 ■ **Partial Avulsion Injury.** A typical partial avulsion laceration from a fall onto the edge of a staircase. Note the triangular "flap" in the upper left wound quadrant. Closure of partial avulsion injuries must be particularly meticulous to reduce any further compromise of the flap tip's vascular supply. (Photo contributor: Alan B. Storrow, MD.)

FIGURE 18.14 ■ **Lower Extremity Degloving Avulsion.** A complex degloving injury after entanglement of the lower extremity in a rope while water-skiing. (Photo contributor: Alan B. Storrow, MD.)

FIGURE 18.15 ■ **Ring Injuries.** Rings being caught and forced proximally are a common cause of finger degloving (**A**). Soft tissue injury due to tourniquet effect of a ring in an elderly woman with diabetic neuropathy (**B**). (Photo contributors: A. Selim Suner, MD, MS and B. Lawrence B. Stack, MD.)

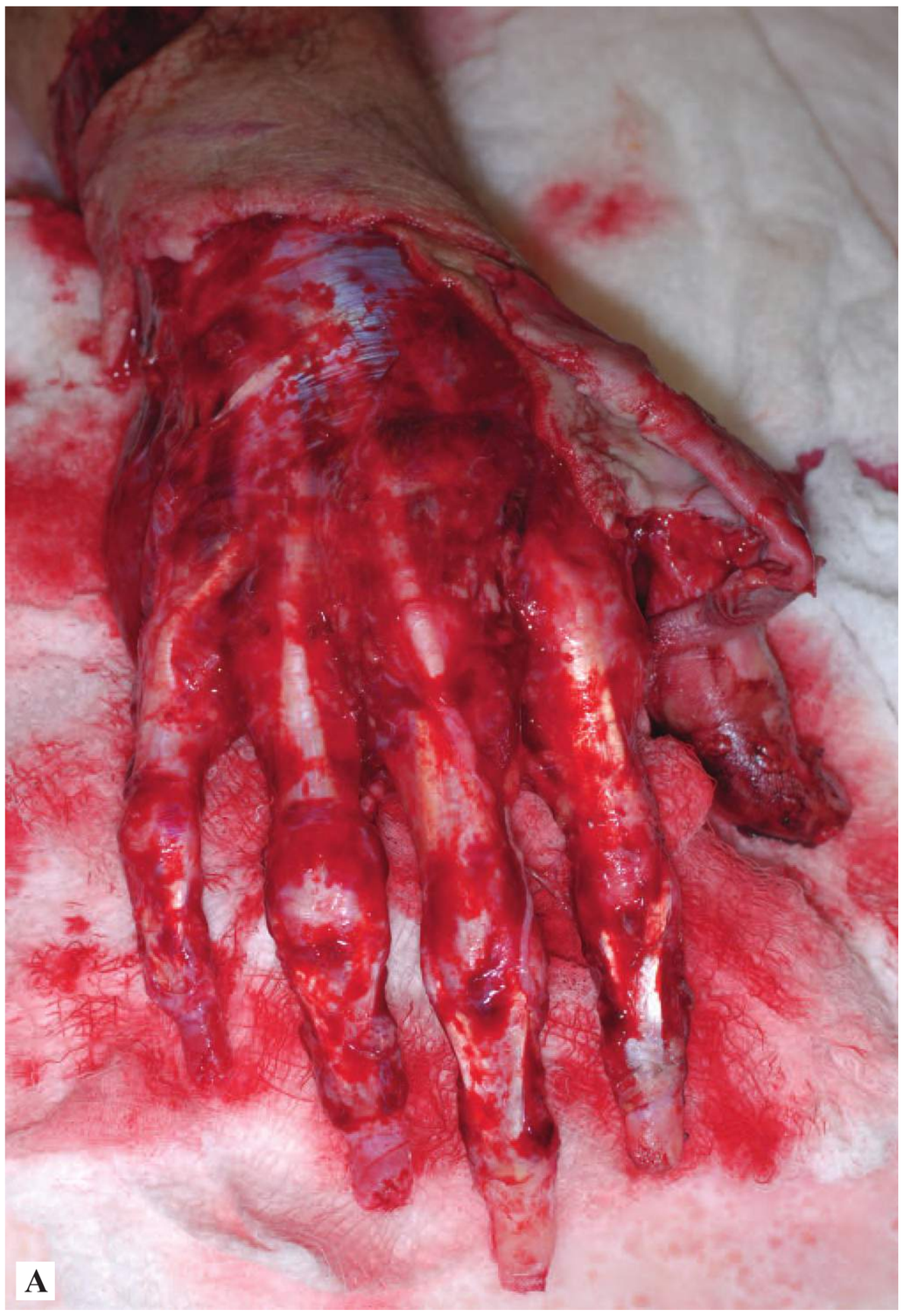


FIGURE 18.16 ■ **Hand Degloving Avulsion.** This extensive degloving (**A**) was caused by an industrial accident and resulted in near-complete removal of the skin of the hand (**B**). (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 18.17 ■ **Crush (Compression) Injury.** Extensive crush injury, with degloving, resulting from an industrial accident. (Photo contributor: David Effron, MD.)

TABLE 18.1 ■ GUIDE TO TETANUS PROPHYLAXIS IN ROUTINE WOUND MANAGEMENT

History of Adsorbed Tetanus Toxoid (Doses)	Clean Minor Wounds		All Other Wounds[a]	
	Tdap or Td[b]	TIG[c]	Tdap or Td[b]	TIG[c]
<3 or unknown	Yes	No	Yes	Yes
≥3 doses	No[d]	No	No[e]	No

[a]Such as (but not limited to) wounds contaminated with dirt, feces, soil, and saliva; puncture wounds; avulsions; and wounds resulting from missiles, crushing, burns, and frostbite.

[b]For children younger than 7 years of age, DTaP is recommended; if pertussis vaccine is contraindicated, DT is given. For persons 7 to 9 years of age, Td is recommended. For persons >10 years, Tdap is preferred to Td if the patient has never received Tdap and has no contraindication to pertussis vaccine. For persons 7 years of age or older, if Tdap is not available or not indicated because of age, Td is preferred to TT.

[c]TIG is human tetanus immune globulin. Equine tetanus antitoxin should be used when TIG is not available. If only three doses of fluid toxoid have been received, a fourth dose of toxoid, preferably an adsorbed toxoid, should be given. Although licensed, fluid tetanus toxoid is rarely used.

[d]Yes, if it has been 10 years or longer since the last dose.

[e]Yes, if it has been 5 years or longer since the last dose. More frequent boosters are not needed and can accentuate side effects.

Source: Table courtesy of the Centers for Disease Control and Prevention. Please see www.cdc.gov for the most current information.

EAR LACERATIONS

Clinical Summary

The ear is composed of a poorly vascularized cartilaginous skeleton covered by tightly adherent skin. Given the paucity of subcutaneous tissue, ear injury that results in hematoma formation can cause pressure necrosis of the cartilage. The goal of repairing an ear laceration involves completely covering the exposed cartilage and preventing hematoma formation.

Management and Disposition

Prior to repair, the area is examined for signs of acute hematoma formation or other associated traumatic injuries. Hemotympanum or Battle sign suggests the presence of a more serious closed head injury, especially basilar skull fracture. Blunt trauma may result in barotrauma resulting in tympanic membrane perforation. Examination can be facilitated by local anesthesia infiltration or, in the case of larger or more complex lacerations, a regional nerve block.

Simple lacerations through the earlobe or the helix can be repaired with interrupted 6-0 nonabsorbable monofilament sutures if the cartilage is not exposed. Simple lacerations that involve the cartilage are primarily repaired by ensuring complete coverage of the exposed cartilage by careful apposition of the overlying skin. The skin generally provides sufficient support, so sutures are not required for the cartilage itself. If the wound is sufficiently irregular and cartilage debridement becomes necessary to avoid undue wound tension, the debridement should be kept to a minimum.

A perichondral hematoma must be drained within 72 hours to prevent potential pressure necrosis which can result in a "cauliflower" ear. Ear wounds are best dressed with a mastoid pressure dressing either primarily or after later hematoma drainage. Such a dressing reduces the chances for future hematoma formation and its complications. Ear sutures are removed in 4 to 5 days.

Pearls

1. Epinephrine-containing anesthetic at a 1:100,000 dilution may be used in ear laceration repair.
2. Hematoma evacuation needs to be rechecked in 24 hours to evaluate for reaccumulation.
3. If cartilage has been exposed or a hematoma drained, antistaphylococcal antibiotic coverage is recommended.
4. Complex lacerations and hematomas of the ear are best cared for in conjunction with a consultant.

FIGURE 18.18 ■ **Ear Laceration.** This patient sustained an uncomplicated, linear pinna laceration. Closure must cover all exposed cartilage. (Photo contributor: Alan B. Storrow, MD.)

FIGURE 18.19 ■ **Ear Laceration.** Complex ear laceration through the helix down through the antihelix. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Lip lacerations may result in significant cosmetic defects if not properly repaired. The lip has two significant anatomic landmarks: the mucosal border, which divides intraoral and external portions of the lip, and the vermilion border, which separates the lip mucosa from the skin of the face. Meticulous alignment of the vermilion border and its associated "white line" is the cornerstone of cosmetic repair. Lip anatomy may be distorted by the kinetic energy of the impact as well as the resultant edema surrounding the wound. Lacerations of the lip's vermilion border may be partial- or full-thickness, compromising the underlying orbicularis oris.

Management and Disposition

Given the high bacterial content of the oral cavity, lip lacerations will not remain clean during repair. The goal of irrigation is to remove clotted blood and gross contaminants such as tooth fragments or dirt. If a fractured tooth is noted, the wound must be explored for fragments. If the tooth or fragment is unaccounted for, then a Panorex or soft tissue radiograph of the face and a chest radiograph should be obtained. Anesthesia for laceration repair is best performed using either an infraorbital (upper lip) or mental (lower lip) nerve block since local infiltration often distorts the tissue and impairs proper alignment of the vermilion border.

If the vermilion border is violated by a superficial laceration, then the first suture, typically 6-0 in size, is placed at the border to reestablish anatomic relationships. Once alignment is judged adequate, simple interrupted sutures are used for completion. If the laceration extends within the oral cavity, absorbable 5-0 sutures are used to close the intraoral component.

With deep or "through and through" lacerations involving the orbicularis oris, the muscle layers are initially approximated with deep, usually 5-0, absorbable sutures. Once the muscle is approximated, the first skin suture is again placed at the vermilion border.

Sutures are removed in 3 to 5 days in children, 4 to 5 days in adults. The patient is advised to eat soft foods, not to apply excessive force to the suture line, and to rinse after eating to prevent the accumulation of food particles.

Pearls

1. Misalignment of the vermilion border by as little as 1 mm may result in a cosmetically noticeable defect.
2. A marking pen may be used to identify landmarks prior to placing the sutures, as suturing itself causes some tissue edema, bleeding, and distortion.
3. Any patient with a lip laceration requires a thorough inspection of the oral cavity for associated trauma, including dental fractures, oral lacerations, and mandibular injuries.

FIGURE 18.20 ■ **Vermilion Border Lacerations.** A complex and a simple vermillion border laceration. (Photo contributor: David Effron, MD.)

TENDON LACERATIONS

Clinical Summary

Tendon injuries are often associated with hand or wrist lacerations. Accurate assessment requires documentation of both motor function and strength. Partial tendon ruptures, including near complete, may still result in normal function.

Management and Disposition

Prior to wound exploration, a thorough exam is performed to assess neurovascular and motor function. All individual flexor and extensor tendons are assessed, including deep and superficial. Abnormal resting posture of the involved extremity can also indicate tendon injury. Tendons are taken through a full range of motion, including re-creation of limb position at the time of insult, in order to detect injuries along the tendon length. Adequate exploration requires excellent hemostasis achieved through direct pressure, or use of a blood pressure cuff or other tourniquet. Initial wound care should include irrigation, exploration for foreign bodies, debridement, antibiotics, and tetanus prophylaxis if indicated.

Partial tendon lacerations are treated conservatively, with splinting in neutral position and follow-up. Isolated extensor tendon lacerations may be repaired in the ED with subsequent specialist follow-up. Flexor tendon lacerations generally require consultation with a hand surgeon or orthopedic surgeon.

Pearls

1. While extensor tendon repair may be accomplished by an emergency care physician with appropriate training and experience, flexor tendon lacerations are a challenging orthopedic problem and require referral.
2. Flexor tendons are weakest approximately 3 weeks after repair.
3. Inability to flex the distal phalanx with intact proximal phalanx extension suggests a flexor digitorum profundus disruption.

FIGURE 18.21 ■ **Flexor Tendon Laceration.** This patient presented after sustaining a laceration to his third and fourth digits (left). The injury was associated with an inability to flex these two digits. Wound exploration revealed the distal segment of the transected flexor tendon apparatus (right). (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

FIGURE 18.22 ■ **Extensor Tendon Lacerations.** Note the laceration over the third and fourth metacarpals (**A**). Inability to extend the long fingers is evidence of complete extensor tendon disruption. Another extensor tendon laceration associated with inability to extend the fifth digit. (**B**). (Photo contributors: A. Selim Suner, MD, MS and B. David Effron, MD.)

FIGURE 18.23 ■ **Flexor Tendon Laceration.** Wrist laceration and associated complete tendon rupture. (Photo contributor: David Effron, MD.)

ANIMAL BITE WOUNDS

Clinical Summary

Bite wounds (abrasions, lacerations, and punctures) are a frequent emergency care challenge, although most are minor. Dog bites account for the vast majority, followed by cats, humans, and, rarely, other animals (raccoons, foxes, livestock, etc).

Bite wound microbiology is frequently polymicrobial with mixed aerobic and anaerobic bacteria. Pasteurella multocida (a gram-negative anaerobe) has been cultured from up to 80% of cat bites and 25% of dog bites. Eikenella corrodens has been recovered from human bites. Capnocytophaga canimorsus (formerly known as DF-2), a virulent organism which can be normal oral flora in dogs (16%) and cats (18%), can lead to sepsis, disseminated intravascular coagulation, gangrene, or death in susceptible individuals (eg, immunocompromised, asplenic, or diabetic).

The primary site of a bite wound in adults is on the extremities. The hand is at highest risk for developing infection, while the face is the most infection-resistant. Puncture wounds, especially from cats, are also prone to infection. Simple abrasions, regardless of animal, are unlikely to become infected with proper wound care.

Management and Disposition

Management depends on location, animal involved, time elapsed before presentation, and wound type. All wounds should be thoroughly cleaned and debrided. Radiographs should be obtained to exclude bony injury or retained dentition. Contusions and superficial abrasions can be treated with local wound care.

Recommendations vary regarding timing of wound closure. Closure of facial and head wounds can be performed up to 12 hours (and in some reports up to 24 hours). Puncture wounds (especially if caused by a cat), hand lacerations, or high-risk wounds (wounds > 12 hours or clinically infected) should be considered for delayed primary closure. While a linear incision over a puncture wound may facilitate cleaning and exploration, efficacy of this is questioned. Human bites to areas other than the face and head should generally be left open and considered for delayed primary closure. Hand injuries caused by human bites are left open and managed in consultation with a hand specialist (see Chapter 11, Extremity Trauma, Clenched Fist Injury). One should maintain a high index of suspicion for human bite in any laceration near the metacarpals.

All sutured bite wounds should be reevaluated by a healthcare provider within 24 hours. Closed wounds which appear infected on reevaluation should be opened, irrigated, and allowed to close by secondary intention. Cyanoacrylate adhesives should never be used to close a bite wound.

Antibiotic therapy recommendations for bite wounds differ widely. Antibiotics are not recommended for minor wounds. They are recommended for cat bites, hand wounds, and in persons with chronic diseases (eg, diabetes). Empiric therapy is started with broad-spectrum antibiotics such as ampicillin-sulbactam, cefoxitin, or ceftriaxone. Alternatively, ciprofloxacin (or trimethoprim-sulfamethoxazole in children) and clindamycin can be used. Infection by P multocida classically becomes apparent within 24 hours of the bite and is marked by prominent pain, erythema, and swelling.

FIGURE 18.24 ■ **Dog Bite.** Extensive facial wounds secondary to a dog bite. (Photo contributor: David Effron, MD.)

FIGURE 18.25 ■ **Dog Bite.** This patient sustained multiple avulsion injuries to her hand and forearm from a pit bull attack. (Photo contributor: Selim Suner, MD, MS.)

Amoxicillin-clavulanate is the suggested regimen to cover polymicrobial infections and infections caused by P multocida or C canimorsus. Cultures are not recommended for initial treatment. However, they should be obtained in any wound worsening during antibiotic therapy. Tetanus immunization should be appropriately updated.

Rabies infection is rare in the United States; worldwide, dogs are the number one reservoir (see Chapter 21, Tropical Medicine, Rabies). Carnivorous animals (eg, raccoons, skunks, foxes, dogs, or bats) are more likely to be infected. If an animal is suspected as rabid, it should be detained and observed for any signs of rabies; occasionally this requires euthanasia and necropsy. Bats require special attention as many people do not recall being bitten. Healthcare professionals should have a low threshold for instituting rabies prophylaxis in persons with a bat exposure or in whom the animal is not available. Local animal control offices can be contacted for recommendations on appropriate rabies prophylaxis measures.

Pearls

1. All patients with bite wounds from susceptible animals (especially bats) should be assessed for rabies exposure.
2. Given cosmetic concerns and low infection risk, dog bites to the head and face can generally be sutured up to 24 hours after appropriate irrigation.
3. Bites which become infected within 24 hours, especially from cats, should be assumed to be caused by P multocida.

FIGURE 18.26 ■ **Cat Bite.** Note the swelling and erythema to the index finger indicating infection, possibly with P multocida (a prominent and fastidious organism seen commonly with feline bites). (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Fishhook barbs often prevent backing the hook out of the puncture site. Several different methods have been described for removal.

Management and Disposition

Adequate anesthesia, usually local, is essential for removal. Procedural sedation may be needed if a child has a fishhook embedded in a sensitive area (eg, eyelid).

The method used to remove the hook depends primarily on the location of the barb relative to the skin surface and the body part embedded. The most common removal technique is the "push-through and cut." This is recommended when the tip of the fishhook is close to breaking through the skin surface after being embedded (see figures). Care should be taken when performing this maneuver in the hand or face as pushing the fishhook forward may damage nearby structures.

Superficially embedded hooks or hooks with small barbs may be removed in a retrograde fashion, by exerting pressure on the fishhook shaft toward the barb and backing the hook out through the original site of penetration. This technique can be performed manually or with the use of a string.

Once fishhooks are removed, the wound should be cleaned, irrigated, and left open. Antibiotics are usually not necessary; however, treatment (doxycycline) for Vibrio species (especially Vibrio parahaemolyticus) should be considered in wounds contaminated with saltwater.

Pearls

1. Hooks embedded in cartilaginous structures, such as the ear or nose, are best managed with the push-through methods.
2. Hooks that penetrate joint spaces or bone should be managed in consultation with orthopedics.
3. Fishhooks that penetrate the globe of the eye are left in place and emergent ophthalmologic consultation is obtained. The patient is placed in the semirecumbent position and the globe is protected with an eye shield. Pressure patches are contraindicated, as they may extrude intraocular contents.

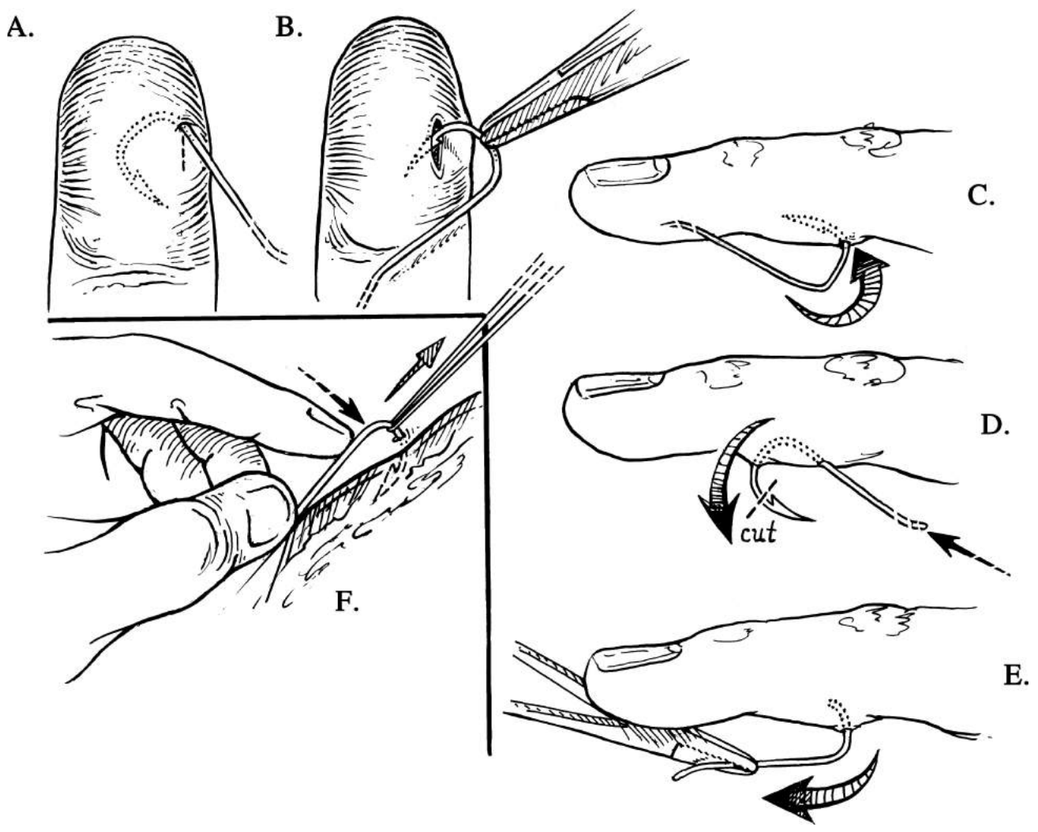


FIGURE 18.27 ■ **Fishhook Removal.** Hooks with small superficially embedded barbs may be carefully backed out through the original puncture site (**A** and **B**). This may require a small incision, made in line with the concavity of the hook curve. The push-through technique is useful for large barbs or those more deeply embedded. The hook is pushed out through the skin, the barb removed, and the remainder of the hook subsequently removed through the original penetration site (**C, D,** and **E**). The traction (string) technique provides an alternative for removing hooks with small barbs. While pressing down on the shaft of the hook, traction is applied with 0 silk or umbilical tape. A swift yank of the cord in the direction opposite the barb will dislodge the hook (**F**). Care is taken to warn bystanders of the potential for the fishhook to fly across the room.

FIGURE 18.28 ■ **Multiple Pronged Fishhook.** Care must be taken when removing this three-pronged hook due to the multiple sharp tips and potential for damaging surrounding structures. (Photo contributor: Selim Suner, MD, MS.)

FIGURE 18.29 ■ **Fishhook Injury and Removal**. The push-through technique was used to remove this hook. Use of a ring cutter proved unsuccessful; a bolt cutter was eventually required to remove the distal portion of this large hook. (Photo contributor: Alan B. Storrow, MD.)

SIMPLE WOUND CLOSURES

Clinical Summary

Most ED lacerations are uncomplicated and can be repaired primarily. Complications (dehiscence, infection, improper healing, scarring) can be minimized by proper suturing techniques, including good wound edge approximation and decreasing wound tension. Wound edge eversion is also essential for proper healing as the edge will flatten with time.

Management and Disposition

Suture techniques must be individualized depending on physician's skill, laceration type, and location. Suture material, size, and duration before removal are determined by anatomic site (Table 18.2).

Deep interrupted absorbable sutures are used, if necessary, to reduce wound tension before superficial repair. Start the deep suture from the bottom of the wound, continue across the top (subdermally), and return to the bottom in finishing. This will leave the knot at the bottom of the wound, decreasing the chance suture material will exit at the surface.

Simple interrupted closures involve single nonabsorbable sutures, each independently tied. The needle should penetrate the skin at 90 degrees and exit the other side of the wound at 90 degrees.

Staples are a rapid means of closing linear wounds. They should not be used on the hands, feet, face, or over joints. Wound edge eversion is also critical to obtain the best outcome.

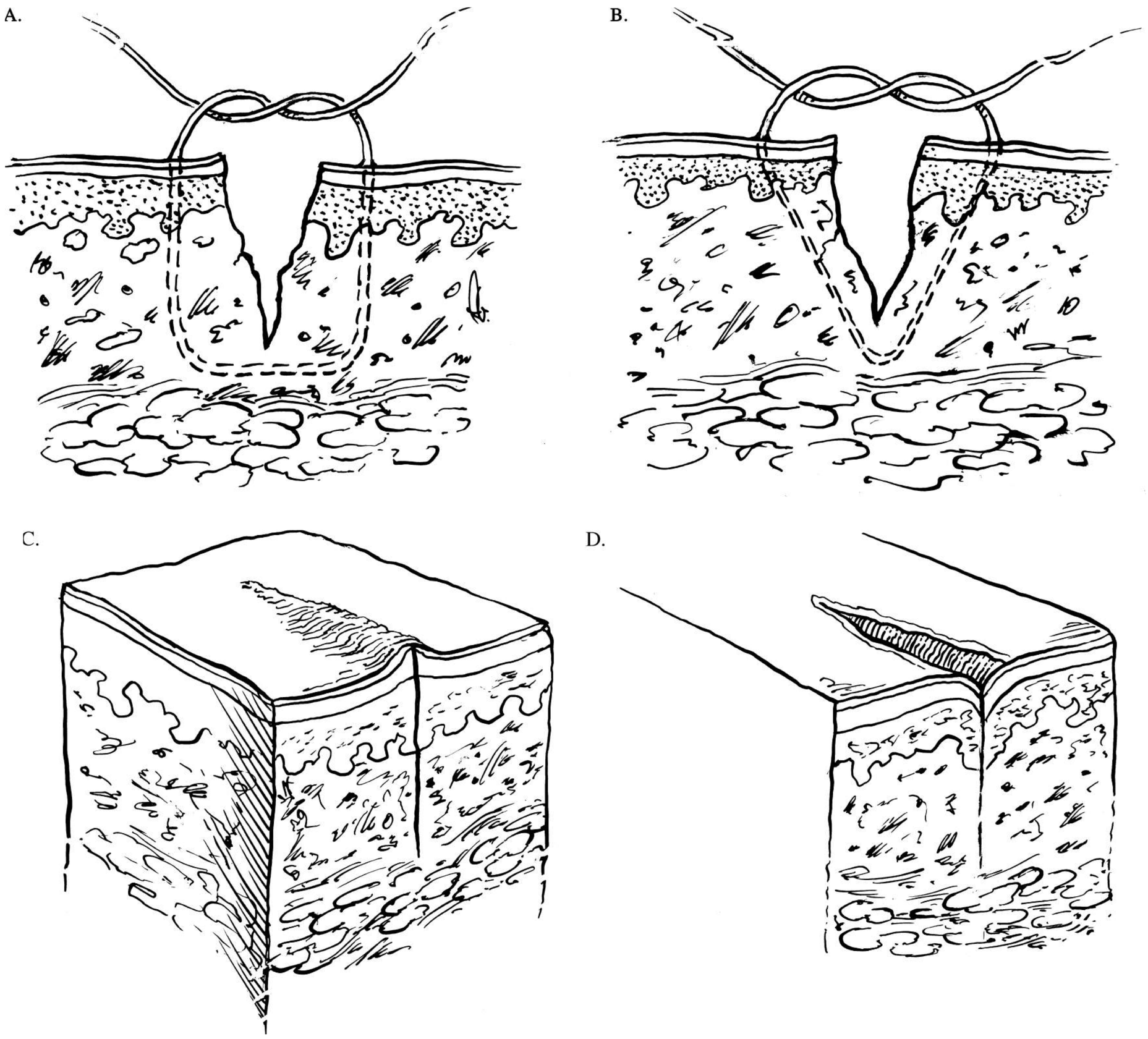


FIGURE 18.30 ■ **Wound Edge Eversion.** Eversion of wound edges is critical for optimal wound healing. For proper eversion, the needle point should enter the epidermis at a 90-degree angle, generating a square or bottle-shaped suture configuration (**A**). This results in a slight rise of the skin edges above the skin plane (**B**). Such eversion will flatten at the level of the skin plane during healing. Entry at a shallower angle (**C**) often leads to wound edge inversion, eventual contraction of the wound edges below the skin plane, and subsequent scar formation (**D**).

Running closure is a rapid technique using several bites along the length of a wound without tying individual knots. Knots are tied only at the beginning and end.

Liquid wound adhesives (eg, cyanoacrylate) have advantages due to speed and lack of a repeat visit for suture removal. Anesthesia may not be required. Small linear lacerations are ideal. The wound must be dry and free of active bleeding for proper adhesion. Antibiotic ointment should not be applied as this will dissolve the adhesive.

Elastic wound closure strips have the same advantages and indications as adhesives. They can be used alone or in conjunction with a tissue adhesive (benzoin resin) applied to the skin on either side of the wound. The edges will curl up over time and can be trimmed by the patient as needed, until they fall off in 2 to 3 weeks. They can also be used after suture removal to give the wound more time to gain tensile strength. Wound closure strips are not recommended in children as they have a tendency to remove them prematurely.

Pearls

1. The bites on both sides of a wound should be equidistant for both optimum wound healing and cosmetic outcome.
2. In gaping wounds, surface tension should be reduced with deep sutures.
3. Antibiotic ointment may be used to remove the adhesive from areas unintentionally glued together by cyanoacrylate.

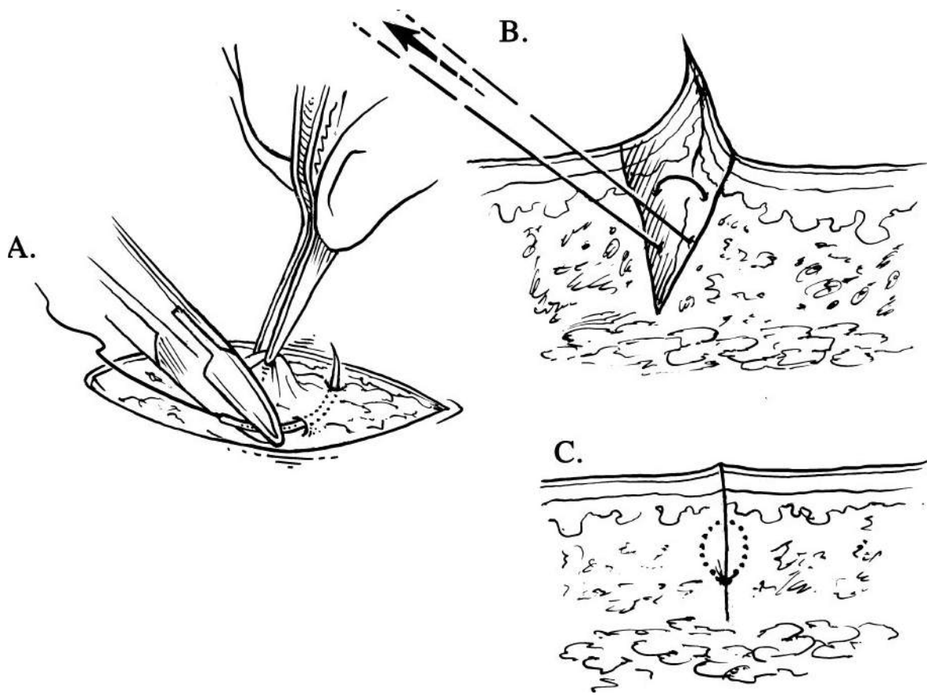


FIGURE 18.31 ■ **Deep Sutures.** Judicious placement of deep sutures allows approximation of the dermis, reduces tension on the wound edges, and may facilitate final superficial closure. The needle is driven from deep within the wound to a superficial level (**A**). On the opposite side of the wound, the needle is driven from superficial to deep (**B**). By having the leading and trailing suture come out on the deep and same side of the superficial cross suture, the knot is buried within the wound (**C**).

FIGURE 18.32 ■ **Simple Interrupted Wound Closure.** An uncomplicated linear laceration generated by a sharp object. For anesthesia and hemostasis, the wound edges are infiltrated with lidocaine-containing epinephrine. The wound is subsequently closed with simple interrupted sutures. Attention is paid to obtaining a degree of wound edge eversion. (Photo contributor: Alan B. Storrow, MD.)

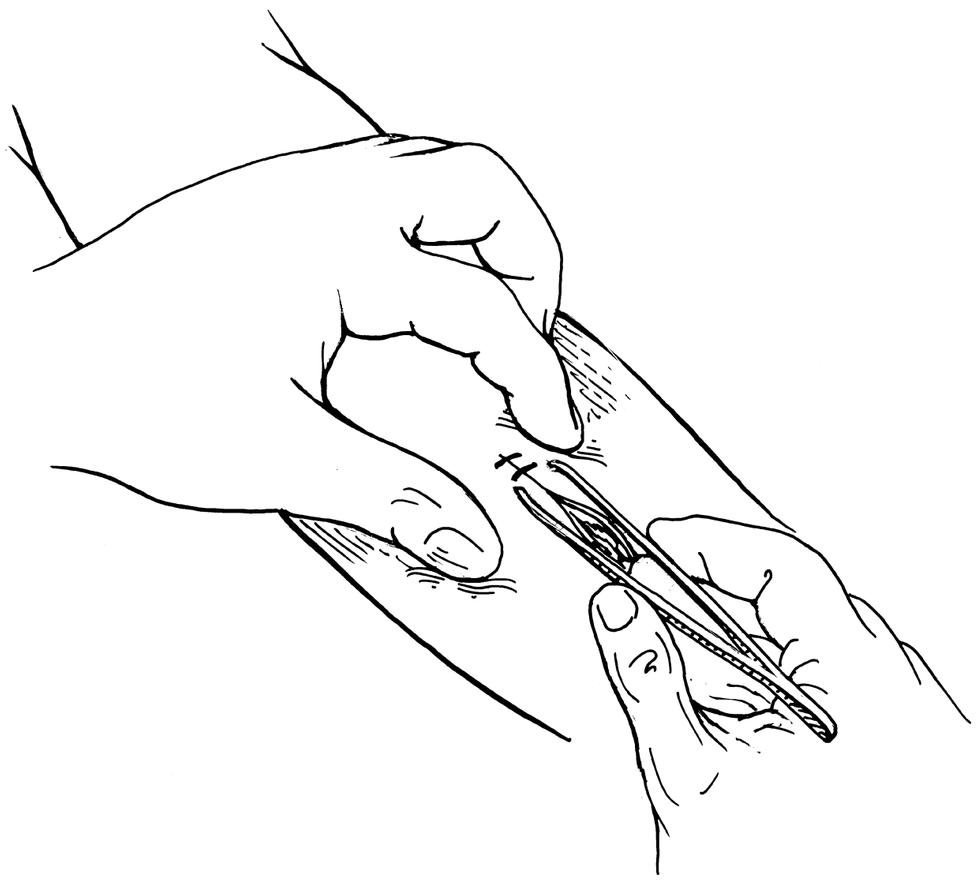


FIGURE 18.33 ■ **Staple Closure.** Meticulous care must be taken when using staples to properly approximate and evert wound edges. This can be facilitated through the use of forceps during staple closure.

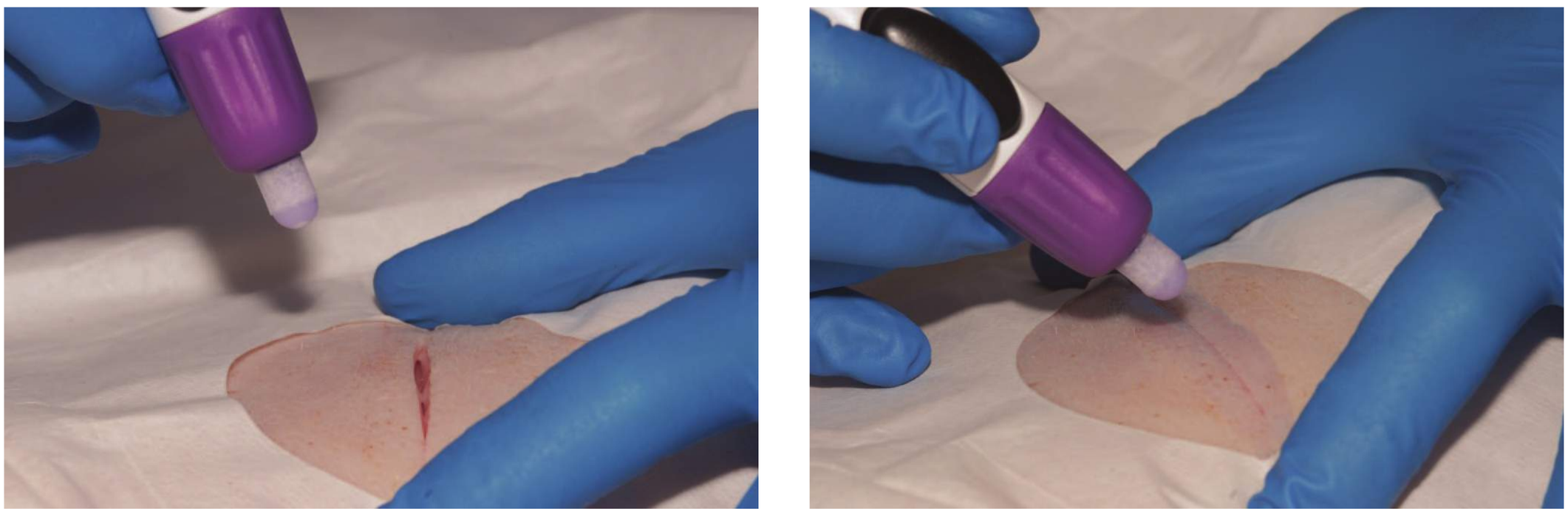


FIGURE 18.34 ■ **Liquid Wound Adhesive Closure.** Wounds that are ideal for this type of closure are small, linear, and hemostatic. Care must be taken to ensure the wound edges are correctly approximated before applying the adhesive. (Photo contributor: Michael L. Juliano, MD.)

TABLE 18.2 ■ SUTURE MATERIALS, SIZE, AND DURATION BY ANATOMIC SITE

Anatomic Site	Skin	Deep	Duration
Scalp	5-0, 4-0 monofilament	4-0 absorbable	6-8 days
Ear	6-0 monofilament	N/A	4-5 days
Eyelid	7-0, 6-0 monofilament	N/A	4-5 days
Eyebrow	6-0, 5-0 monofilament	5-0 absorbable	4-5 days
Nose	6-0 monofilament	5-0 absorbable	4-5 days
Lip	6-0 monofilament	5-0 absorbable	4-5 days
Oral mucosa	N/A[a]	5-0 absorbable	N/A
Face/forehead	6-0 monofilament	5-0 absorbable	4-5 days
Chest/abdomen	5-0, 4-0 monofilament	3-0 absorbable	8-10 days
Back	5-0, 4-0 monofilament	3-0 absorbable	12-14 days
Arm/leg	5-0, 4-0 monofilament	4-0 absorbable	8-10 days
Hand	5-0 monofilament	5-0 absorbable	8-10 days[b]
Extensor tendon	4-0 monofilament	N/A	N/A
Foot/sole	4-0, 3-0 monofilament	4-0 absorbable	12-14 days

[a]Not applicable.
[b]Add 2 to 3 days for joint extensor surfaces.
Source: Adapted with permission from Trott AT. Wounds and Lacerations: Emergency Care and Closure. 2nd ed. St. Louis, MO: Mosby-Year Book; 1997.

Clinical Summary

Horizontal and vertical mattress sutures, as well as the corner stitch (see figures for technique), may be used to manage wounds unable to be closed with simple techniques.

Management and Disposition

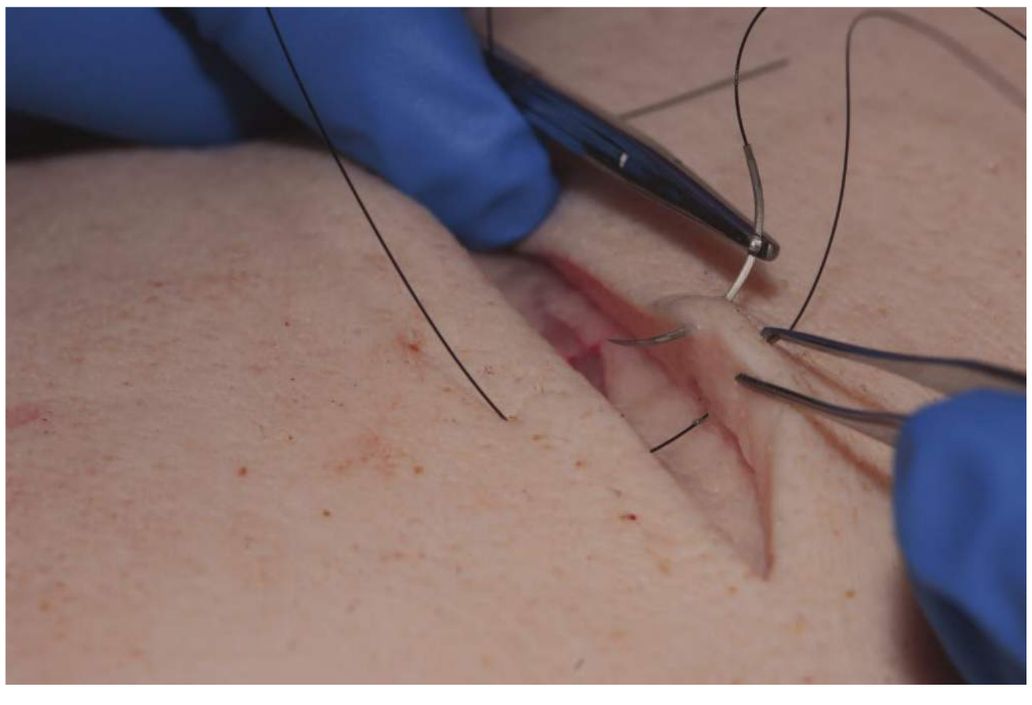
Vertical mattress suture is a useful technique for deep wounds. It reduces wound tension by acting as both a deep and superficial suture.

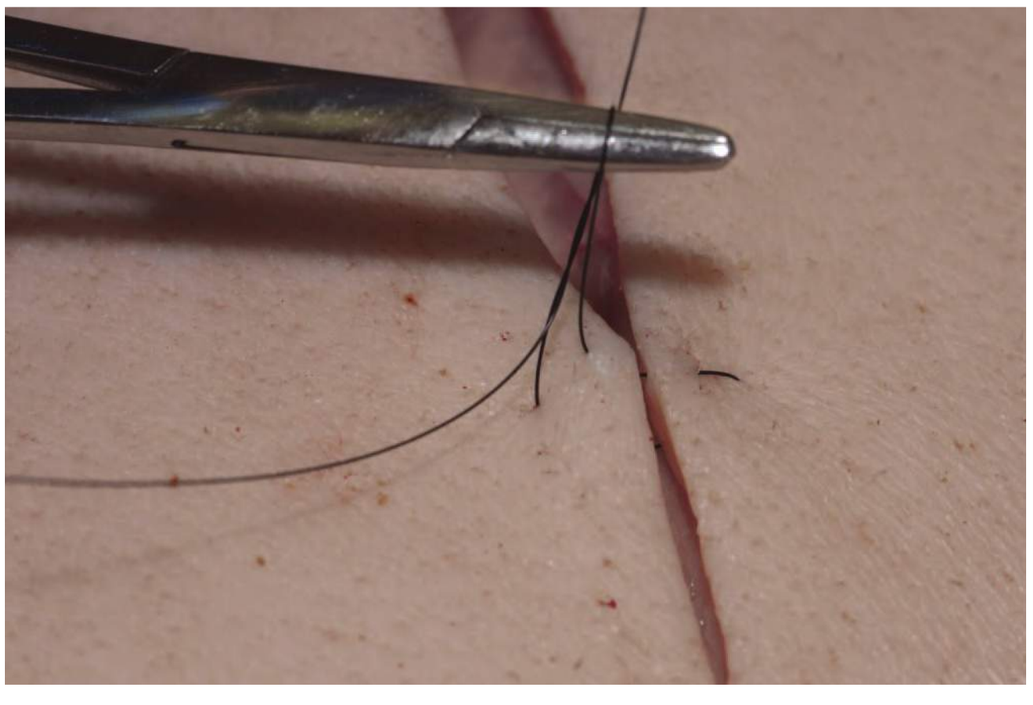
Horizontal mattress suture is best used for wide, gaping wounds with a risk of increased wound tension after closure (eg, lacerations overlying a joint).

Corner stitch is used to close triangular wounds or flaps. A simple interrupted suture cannot be placed to approximate the point of the flap due to a tenuous blood supply and increased chance of dehiscence. Once the corner is secured, simple sutures are used to repair the rest of the wound, with care taken to place the sutures far enough from the tip to optimize circulation.

Pearls

1. Utilization of a mattress suture can aid in wound edge eversion and tension reduction. They are particularly useful in areas where the deep subcutaneous tissues are too fragile for deep sutures (eg, over a joint or shin).
2. A single corner stitch may be used to close several corners of a stellate wound.





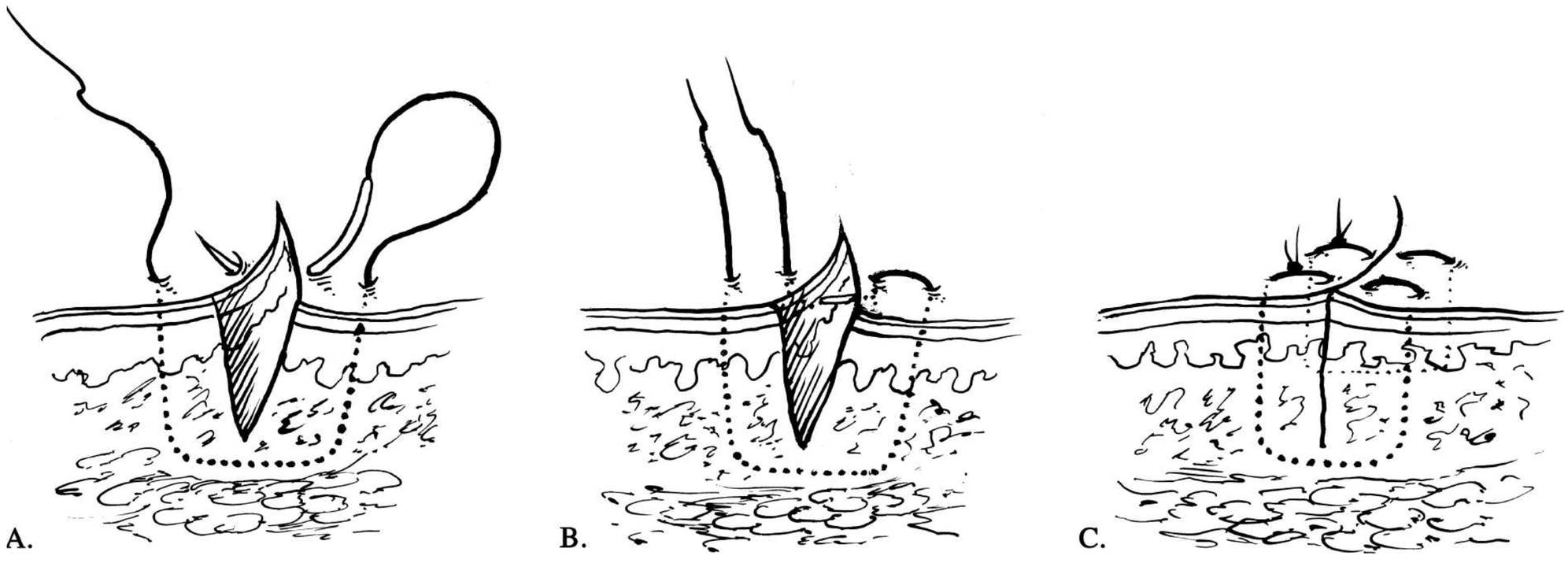
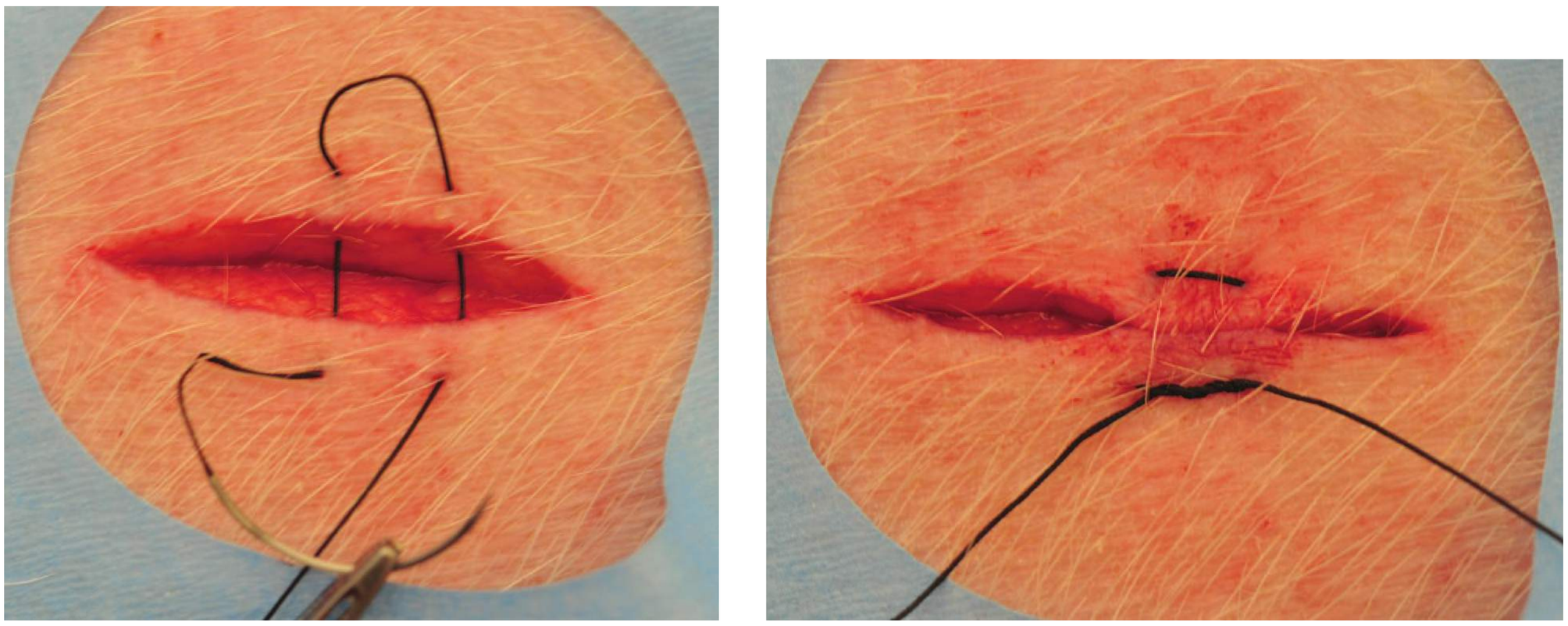


FIGURE 18.35 ■ **Vertical Mattress Suture.** The suture is placed by first taking a large deep bite of tissue approximately 1 cm away from the wound edge and exiting at the same location on the other side of the wound. A second small superficial bite is then performed in the reverse direction (**A**). When the bites are complete (**B**), tying results in nice apposition of the wound edges (**C**). This technique is especially useful in areas of lax skin, such as the elbow or dorsum of the hand. (Photo contributor: Michael L. Juliano, MD.)



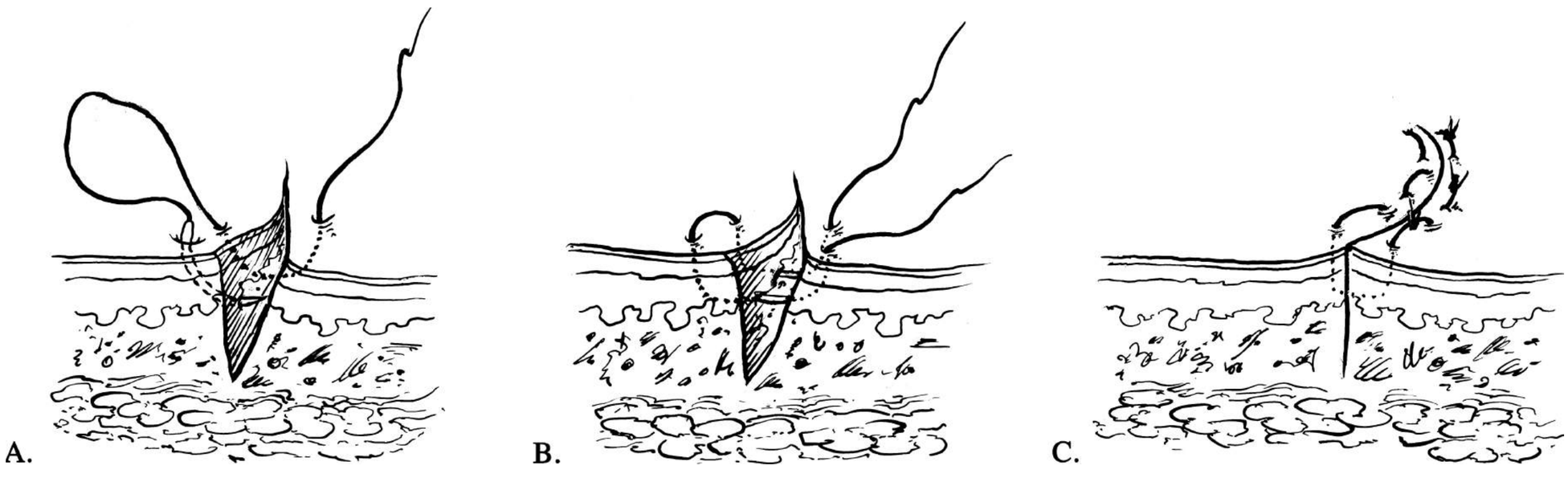


FIGURE 18.36 ■ **Horizontal Mattress Suture.** Useful in achieving wound edge eversion, the horizontal mattress suture begins with a standard suture throw. A second bite is taken approximately half a centimeter from the first exit (**A**) and brought through at the original starting edge, half a centimeter from the original entry point (**B** and **C**). (Photo contributor: Michael L. Juliano, MD.)

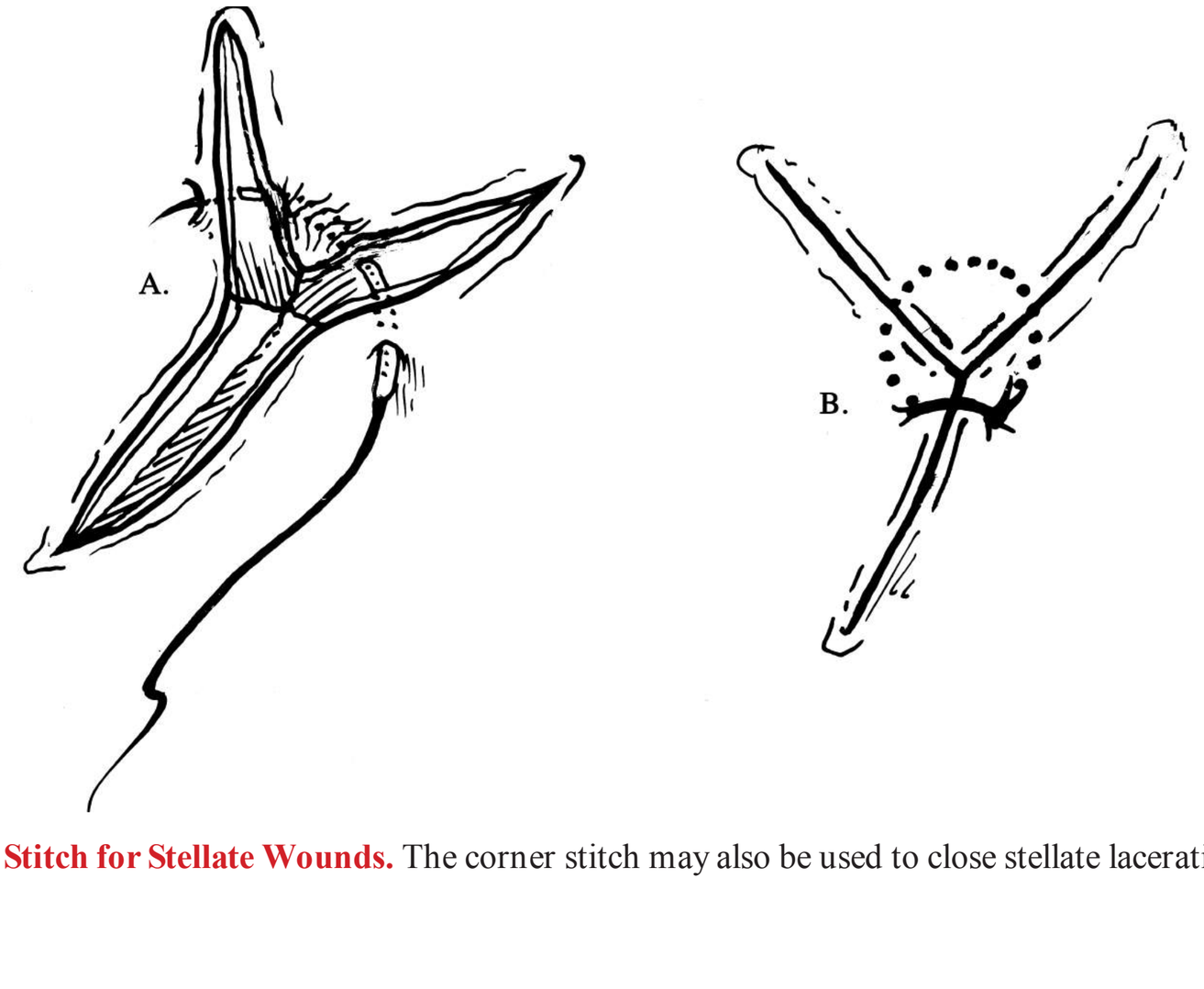
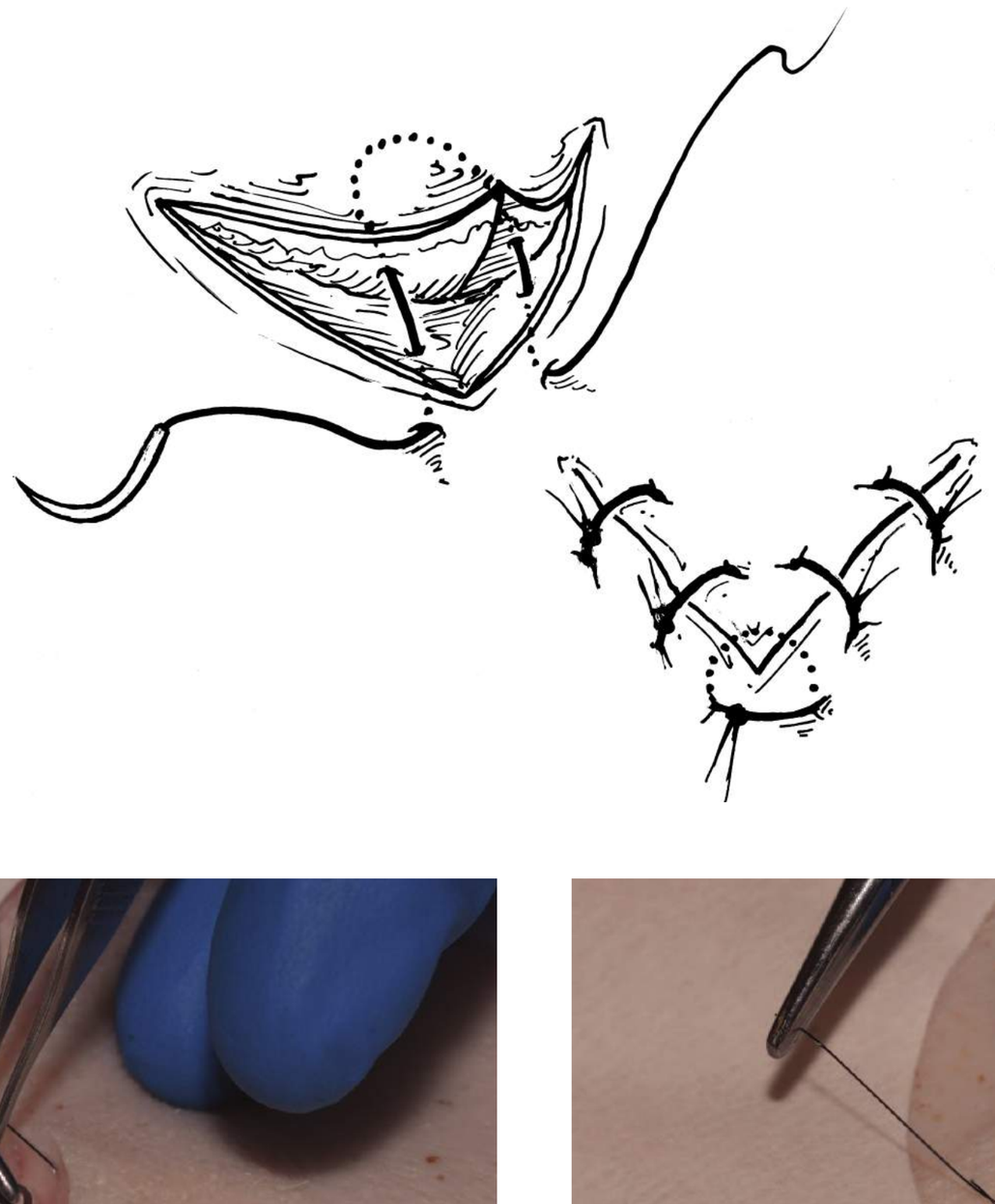
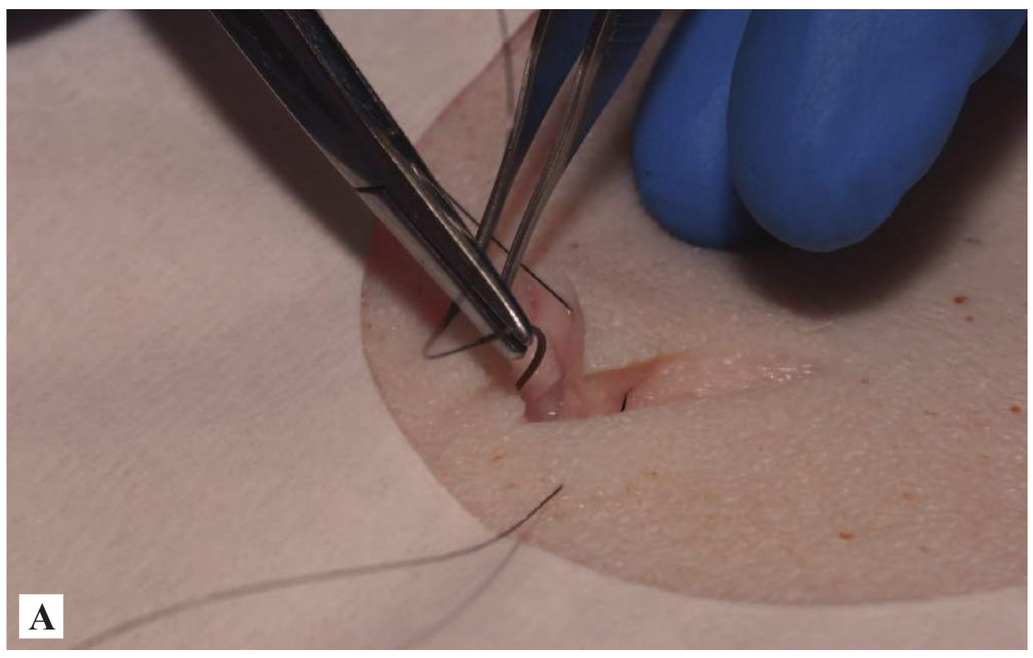
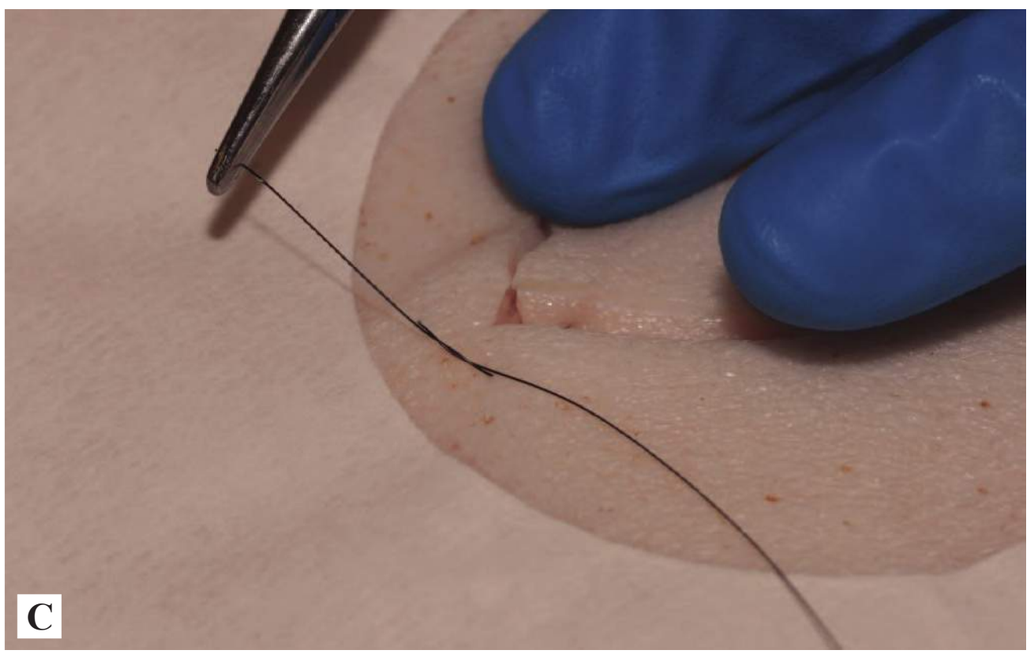
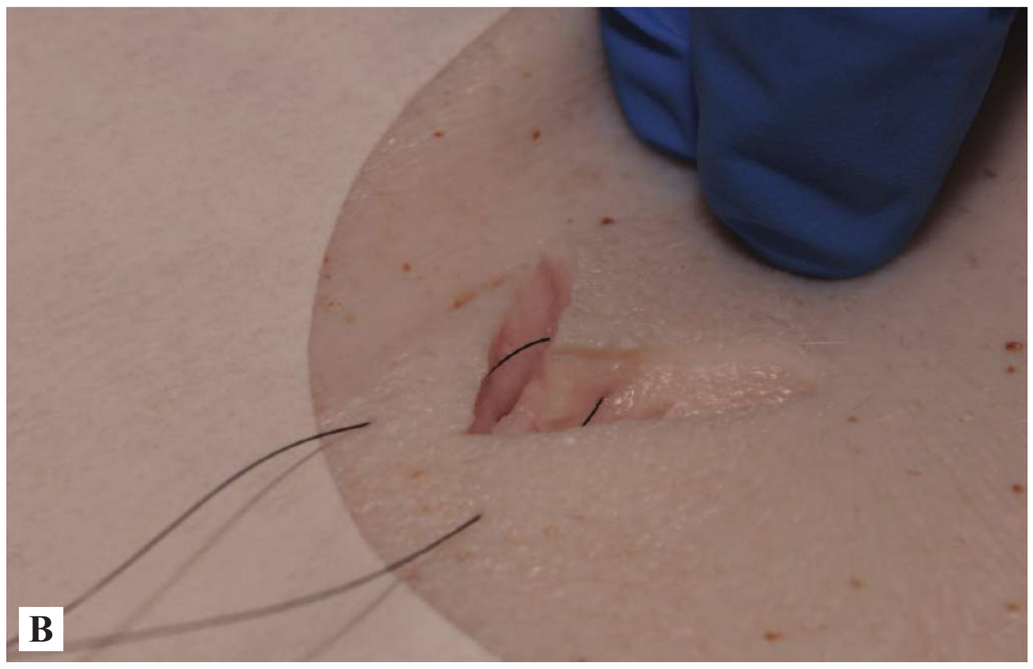


FIGURE 18.37 ■ **Corner Stitch for Stellate Wounds.** The corner stitch may also be used to close stellate lacerations.









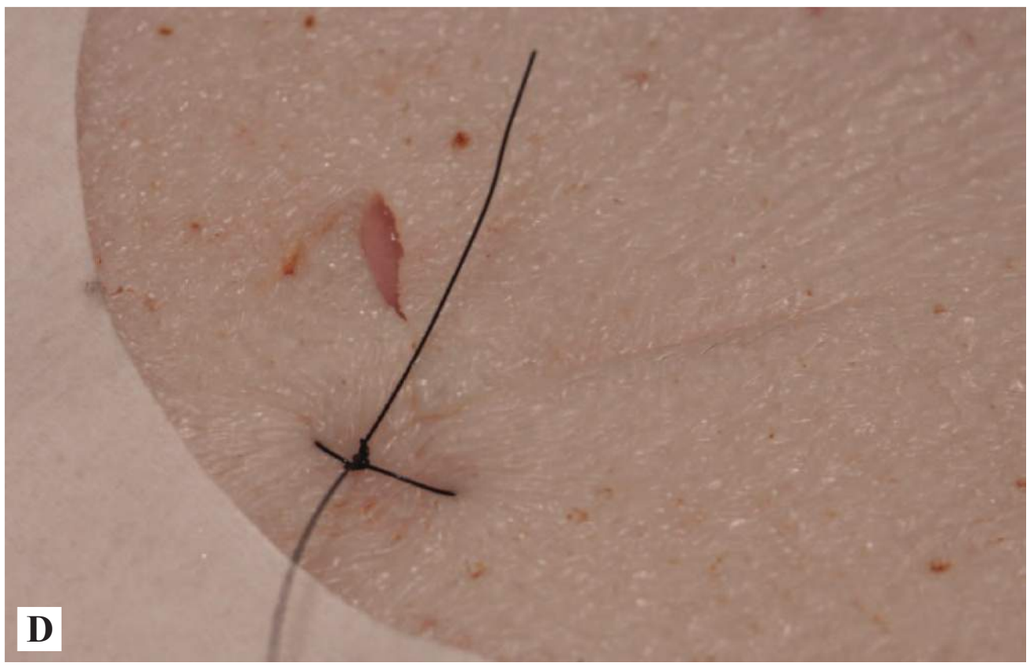


FIGURE 18.38 ■ **Corner Stitch.** Flaps generated by partial avulsion injuries must be repaired with care to avoid compromising the tenuous blood supply of the flap. The corner stitch is performed through the use of a half-buried horizontal mattress suture. The suture begins percutaneously away from the wound corner. The suture needle is then brought horizontally through the corner at the level of the dermis and back out through the epidermis at the opposite noncorner portion of the wound (**A**). This technique avoids placing suture material near the apex of the flap (**B**). Tying (**C**) results in approximation of the corner (**D**). (Photo contributor: Michael L. Juliano, MD).

Clinical Summary

Infection is suggested by pain, warmth, erythema, edema, and purulent drainage. While dehiscence can occur at any time, 7 to 10 days after repair a wound is at its weakest (this also closely coincides with suture removal). Impaired wound healing, primarily from infection, medications (especially corticosteroids), foreign bodies, advanced age, poor nutritional status, diabetes mellitus, and peripheral vascular disease, contributes to dehiscence. Elastic wound closure strips can be applied after suture removal and may reduce dehiscence.

Some degree of scarring is inevitable, but not considered a complication of wound repair. This should be discussed with the patient or caregiver. Wound myiasis is infestation by fly larvae (maggots, see Chapter 21, Tropical Medicine, Myiasis) that invade necrotic tissue.

Management and Disposition

Infections are treated with suture removal, thorough irrigation, and low threshold to explore for missed foreign bodies. A 7-day course of a first-generation cephalosporin or antistaphylococcal penicillin is appropriate; however, if methicillin-resistant Staphylococcus aureus (MRSA) is suspected, the antibiotic choice should be adjusted (eg, trimethoprim-sulfamethoxazole or other current recommendations for your area). For animal bites, other antibiotics (eg, amoxicillin/clavulanate) may be more appropriate. Sepsis, advanced infections, or infections in persons with chronic medical problems (eg, diabetes, immunocompromised) should be managed with parenteral antibiotics and possible inpatient admission.

Wound dehiscence is managed conservatively by treating the underlying causes and allowing healing via secondary intention. Dehiscence of wounds in cosmetically sensitive areas is best managed in conjunction with a consultant. Myiasis is treated with wound cleaning and irrigation.

Pearls

1. All accidental wounds are considered contaminated and treated as such. Thorough irrigation and cleansing is of paramount importance in preventing wound infection.
2. Expedient ED wound care is important since bacterial contamination increases over time.

FIGURE 18.39 ■ **Postoperative Wound Infection.** A patient with increasing pain and redness at the site of his staple closure. The area was erythematous, tender, warm, and had scant purulent drainage around some of the staples. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

FIGURE 18.40 ■ **Wound Dehiscence.** After suture removal, the patient returned with dehiscence. The wound had a clean base of granulation tissue and was allowed to close by secondary intention. (Photo contributor: Alan B. Storrow, MD.)

FIGURE 18.41 ■ **Keloid.** The degree of excessive scar bulk extending beyond the original wound margins may be dramatic and cosmetically significant. (Photo contributor: Thea James, MD.)

FIGURE 18.42 ■ **Wound Myiasis.** Necrotic tissue can be invaded by fly larvae (maggots). (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Burns can be caused by heat, electricity, chemicals, friction, or radiation. Skin barrier damage can lead to infection, fluid loss, and electrolyte abnormalities. Long-term consequences include permanent scarring, loss of sensation, and in severe cases loss of extremities due to inadequate circulation.

Burns are assessed by determining the percentage of body surface area (BSA) involved, the depth, and the area of the body involved. A common system used to estimate BSA is following the "rule of nines." This system breaks up the body into zones that each equate to 9% of BSA (see figure). Some clinicians use the palm of the patient's hand as an equivalent to 1% BSA and measure the area involved by using this method.

First-degree burns only involve the epidermal layer. They are red, painful, and heal in approximately 1 week. Second-degree burns are subdivided into two categories, superficial partial thickness and deep partial thickness. Superficial second-degree burns extend from the epidermis to the superficial dermis. Pain, skin blistering, and intact capillary refill are characteristic. Deep partial thickness burns extend into the deep (reticular) layer of the dermis and damage hair follicles, sweat glands, and sebaceous glands. Blisters may occur, the exposed dermis is pale white to yellow in color, and capillary refill is absent. The entire thickness of the skin is compromised in third-degree burns. They appear pale, feel leathery, and are painless. Fourth-degree burns extend through the layers of the skin and involve muscle or bone.

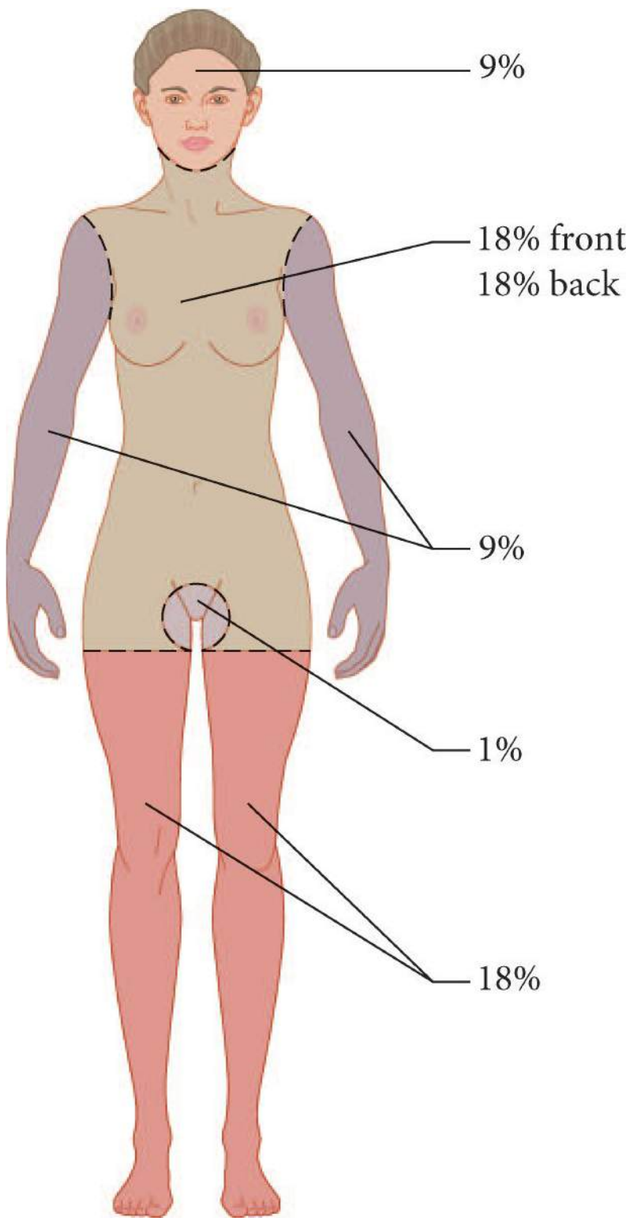


FIGURE 18.43 ■ **Rule of Nines.** Rule of nines is used to estimate the percentage of burn. (Reproduced with permission of Tintinalli JE, Stapczynski JS, Ma OJ, Cline DM, Cydulka RK, Meckler GD. Tintinalli's Emergency Medicine: A Comprehensive Study Guide. 7th ed. McGraw-Hill; 2011: Fig. 210-2, p. 1375.)

FIGURE 18.44 ■ **Second-Degree Burn.** This patient has sustained a second-degree burn to his hand from hot oil. (Photo contributor: Suzanne Dooley-Hash, MD.)

FIGURE 18.45 ■ **Facial Burn, Second Degree.** Ensuring that the patient has a protected airway in this type of injury is crucial. (Photo contributor: David Effron, MD.)

Management and Disposition

After resuscitation and stabilization, referral to a burn unit should be for partial thickness burns that involve greater than 10% BSA, third-degree burns, or involvement of the hands, feet, face, or perineum. Electrical burns, chemical burns, inhalation injuries, and patients with significant comorbidities should also be considered for a burn unit. Clinicians should cover the burned areas with a clean, dry sheet, administer aggressive pain control, and address fluid resuscitation. The Parkland formula is commonly used to estimate fluid requirements. The patient's weight in kilograms is multiplied by the percent BSA involved; this number is multiplied by 4 mL of lactated Ringer solution. Half of this amount is given during the first 8 hours from time of initial injury and the remaining amount is given over the next 16 hours. It is recommended to keep urine output approximately 0.5 to 1.0 mL/kg/h. In order to monitor for effects of cell breakdown, urinalysis, creatine kinase, and an EKG should be obtained. Circumferential burns of the extremities may compromise circulation. If compartment syndrome is suspected, an escharotomy should be considered.

For minor burns, provide pain control, cleansing of the area, and application of topical antimicrobials (eg, 1% silver sulfadiazine, bacitracin, or triple-antibiotic ointment). Dressing changes should occur daily and patients must be instructed to watch for signs of infection. Follow-up with a burn-care expert needs to be arranged within a few days of discharge for deep partial thickness or third-degree burns.

Pearls

1. Minor burns can be managed in the ED. Pain control, irrigation, antimicrobial ointments, and dressing changes are the mainstays of therapy.
2. Criteria for burn center referral should be reviewed for each patient (especially the young and elderly).

FIGURE 18.46 ■ **Extensive Burn Injury.** Burns can be of varying depths; this patient has both second- and third-degree burns. Distal pulses should be closely monitored to guarantee that there is adequate circulation. (Photo contributor: David Effron, MD.)

FIGURE 18.47 ■ **Abdominal Burn.** Mostly second-degree abdominal burn. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

Electricity generates heat through tissue resistance or directly by the current on cells. Many factors affect injury severity: type of current (DC or AC), intensity, duration, tissue resistance, and pathway through the body. When electricity traverses the tissues, it may cause contact burns, thermal injury, muscular tetany, or severe contraction. Sudden death (asystole, respiratory arrest, ventricular fibrillation), myocardial damage, myoglobinuria, compartment syndrome, and various metabolic disorders have also been described.

High-voltage DC or AC current typically causes a single violent muscular contraction and throws the victim from the source; blunt trauma and blast injuries may occur. Low-voltage AC currents (as from a household outlet) typically cause muscular tetany, forcing the victim to continue contact with the source.

Management and Disposition

After initial stabilization, consider cervical spine immobilization, oxygen administration, cardiac monitoring, and IV crystalloid infusion. A Foley catheter will help monitor urine output and is especially important if rhabdomyolysis is suspected. Diagnostic testing to consider includes: electrocardiogram (ECG), complete blood count (CBC), urinalysis, CPK, CPK-MB, electrolytes, BUN, creatinine, and coagulation profile.

Severe or high-risk injuries should be admitted to a burn unit, or trauma center with burn consultation. Patients with minor, brief, low-intensity exposures, with a normal ECG, normal urinalysis, and no significant burns or trauma may be considered for discharge after a period of observation.

Pearls

1. Toddlers are at increased risk of labial injury from chewing on electrical cords.
2. High-risk features include high-voltage exposure (>600 V), deep burns, neurologic injury, dysrhythmias, abnormal ECG, evidence of rhabdomyolysis, suicidal intent, or significant associated trauma.

FIGURE 18.48 ■ **Electrical Burn.** This patient grabbed a high-voltage power line with his hand. Exit wounds occurred where the patient was grounded, through his feet. This patient with transthoracic injury should have cardiac monitoring. (Photo contributor: Alan B. Storrow, MD.)

FIGURE 18.49 ■ **Electrical Flash-Over Burn**. A patient struck by flash over electricity while repairing urban outdoor electric lines. (Photo contributor: Selim Suner, MD, MS.)

FIGURE 18.50 ■ **Electrical Burn.** Extensive electrical burn from a high-voltage power line. (Photo contributor: David Effron, MD.)

FIGURE 18.51 ■ **DC Thermal Ring Injury.** This middle-aged man sustained a circumferential thermal finger burn. His ring was heated when a wrench he was holding simultaneously contacted both terminals on a car battery. (Photo contributor: Aubrey Mowery, MPH, CHES.)

DECUBITUS ULCERS

Clinical Summary

Decubitus ulcers develop when soft tissue is compressed between a bony prominence and a hard external surface. Tissue compression results in decreased blood flow, tissue ischemia, and cell death. Pressure, shearing forces, friction, and excessive moisture contribute to formation. Areas commonly affected include the sacrum and heels. Immunocompromised, nursing home, neurologically impaired, and immobilized trauma patients are at high risk.

Stage I ulcers are characterized by an area of nonblanchable erythema over intact skin. Stage II appears as a shallow, open sore with a pink wound base. When the wound is full thickness with no muscle, tendon, or bone exposed, it is defined as stage III. If muscle, tendon, or bone is exposed, it is stage IV. Some wounds may have an area of black eschar over them; these wounds cannot be categorized since injury depth cannot be determined.

Management and Disposition

Prevention is key. All patients who have decreased mobility, such as trauma and nursing home patients, should have their entire skin surface regularly checked for skin breakdown and should be repositioned frequently. Backboards, c-collars, and other immobilizing devices should be removed as soon as possible.

Treatment includes pain relief, keeping the affected area clean, and keeping pressure off the area to prevent further tissue destruction. There are multiple commercial products available (hydrocolloid dressings) to use for the treatment of pressure ulcers. Frequent monitoring is required and consultation with wound care specialists or a surgeon may be necessary for advanced ulcers. The main complications of decubitus ulcer formation include infection and skin dehiscence.

Pearls

1. Decubitus ulcers are divided into four stages. If an eschar is present, the ulcer cannot be categorized.
2. Remove hard external surfaces and immobilizing devices as soon as possible.

FIGURE 18.53 ■ **Stage III Decubitus Ulcer.** Stage three decubitus ulcers extend into the subcutaneous tissue. (Photo contributor: David Effron, MD.)

FIGURE 18.52 ■ **Stage II Decubitus Ulcer.** Partial thickness loss of dermis presenting as a shallow open ulcer with a red-pink wound bed. (Photo contributor: Suzanne Dooley-Hash, MD.)

FIGURE 18.54 ■ **Stage IV Decubitus Ulcer.** Full-thickness tissue loss with exposed muscle. (Photo contributor: Lawrence B. Stack, MD.)

Chapter 19

CLINICAL FORENSIC MEDICINE

William S. Smock
Lawrence B. Stack

Skin Foreign Body. A bullet from a remote gunshot wound came to the skin surface of the right posterior chest wall. The bullet was removed (inset) at the patient's request. (Photo contributor: Lawrence B. Stack, MD.)

GUNSHOT WOUNDS

Clinical Summary

Gunshot injuries can be accurately identified and classified as entrance, atypical entrance, exit, or atypical (grazing) wounds based upon their physical characteristics. **Wounds are not classified based upon their size**. Physical findings in and around these wounds may offer evidence as to the actual mechanism of injury, supporting or refuting the initial history given to the provider. As most physical findings are transient in nature (cleaned, debrided, or eventually healed), the emergency physician must be diligent in recognizing and documenting them at the time of presentation. The physician's failure to accurately document the physical characteristics of the wounds or correctly interpret the physical findings can compromise the legal process and obstruct justice.

Entrance Wounds

Gunshot wounds of entrance are divided into four categories based on their range of fire: distant, intermediate, close, and contact. Range-of-fire is the distance from the gun's muzzle to the victim's skin or clothing.

The size of the entrance wound bears no relation to the caliber of the inflicting bullet. Entrance wounds over elastic tissue will contract around the tissue defect and have a diameter much less than the caliber of the bullet.

Distant Wounds: The distant wound is inflicted from a range sufficiently distant that the bullet is the only component expelled from the muzzle that reaches the skin. There is no visible tattooing or soot deposition associated with a distant entrance wound. As the bullet penetrates the skin, friction between it and the epithelium results in the creation of an "abrasion collar" (Fig. 19.1). The width of the abrasion collar will vary with the angle of impact. Elongated abrasion collars from projectiles that enter on an angle may produce a collar with a "comet tail" (Fig. 19.2). Most entrance wounds will have an abrasion collar; however, gunshot wounds to the palms and soles are exceptions—their entrance wounds appear slit-like. Bullets that pass through an intermediate object, a door or windshield for example, will become deformed or misshapen (Fig. 19.3). A misshapen bullet creates an irregular abrasion collar (Fig. 19.4) as compared to the smooth abrasion collar created by a nondeformed bullet (Fig. 19.1).

Intermediate-Range Wounds: Tattooing is pathognomonic for an intermediate-range gunshot wound and presents as punctate abrasions from contact with partially burned or unburned grains of gunpowder (Fig. 19.5). This tattooing cannot be wiped away. Clothing and hair, as intermediate objects, may prevent the gunpowder grains from making contact with the skin. Tattooing can, but rarely does, occur on the palms and soles owing to the thickness of their epithelium.

Tattooing has been reported with a range of fire as close as 1 cm and as far away as 4 feet. The density of the abrasions

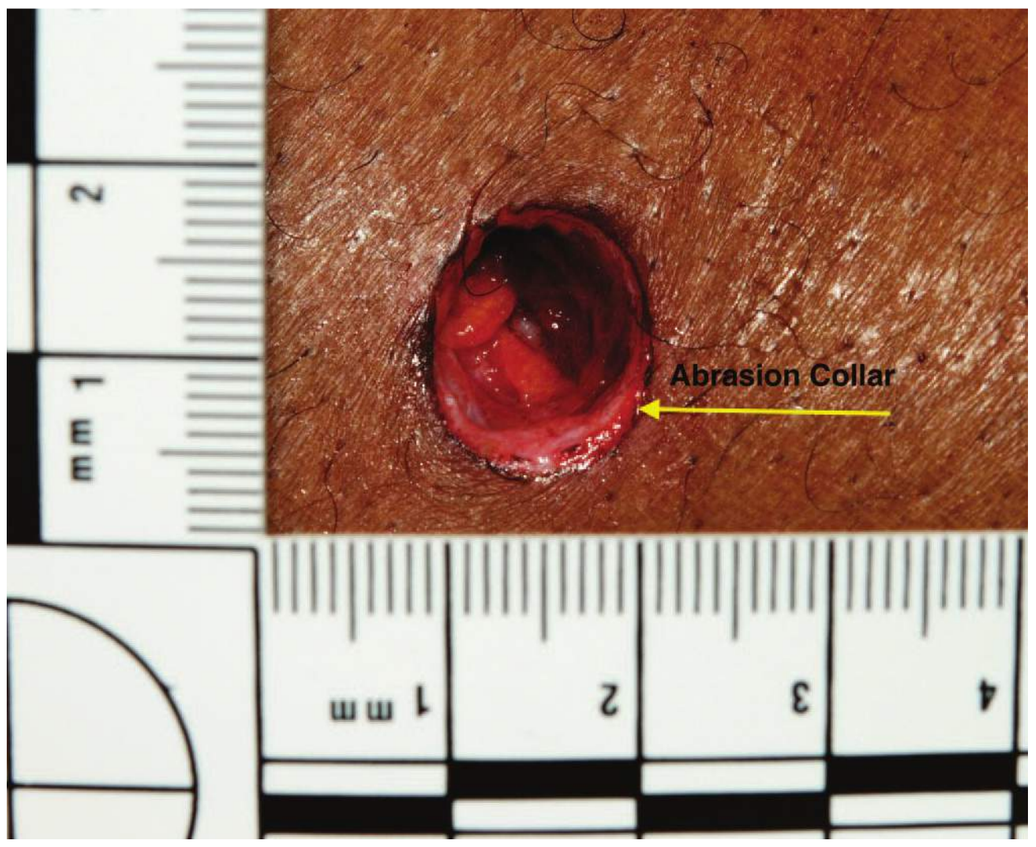


FIGURE 19.1 ■ **Distant Gunshot Wound.** An abrasion collar surrounds the wound defect and is created from the friction of a bullet passing through the skin. All entrance wounds will have an abrasion collar with the exception of entrance wounds to the palms and soles. The lack of soot, seared skin, or gunpowder tattooing confirms this is a "distant" range of fire. (Photo contributor: William S. Smock, MD.)

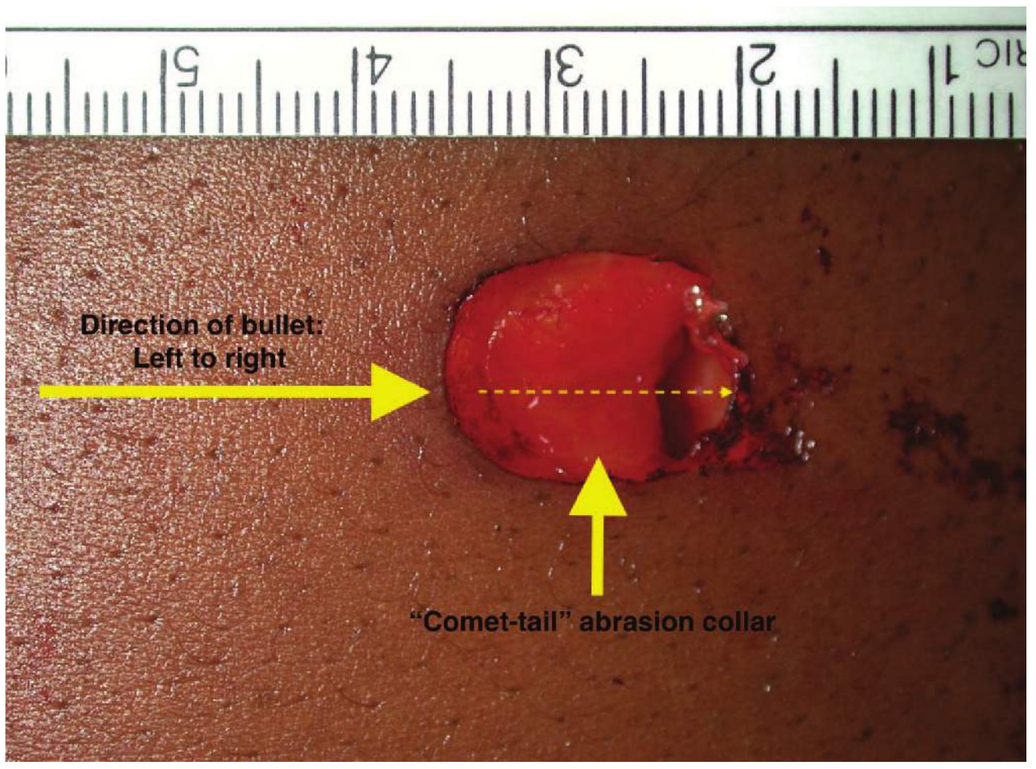


FIGURE 19.2 ■ **"Comet-Tailed" Abrasion Collar.** The "comet tail" abrasion collar located on the lateral aspect of the wound indicates that the bullet entered the wound at an angle. The "comet tail" also indicates the bullet's direction of travel: from left to right. (Photo contributor: William S. Smock, MD.)

FIGURE 19.3 ■ **Deformed Bullet.** This bullet passed through a car windshield and deformed prior to striking the suspect in the arm. The bullet's irregular shape created the atypical abrasion collar associated with the entrance wound seen in Fig. 19.4. (Photo contributor: William S. Smock, MD.)

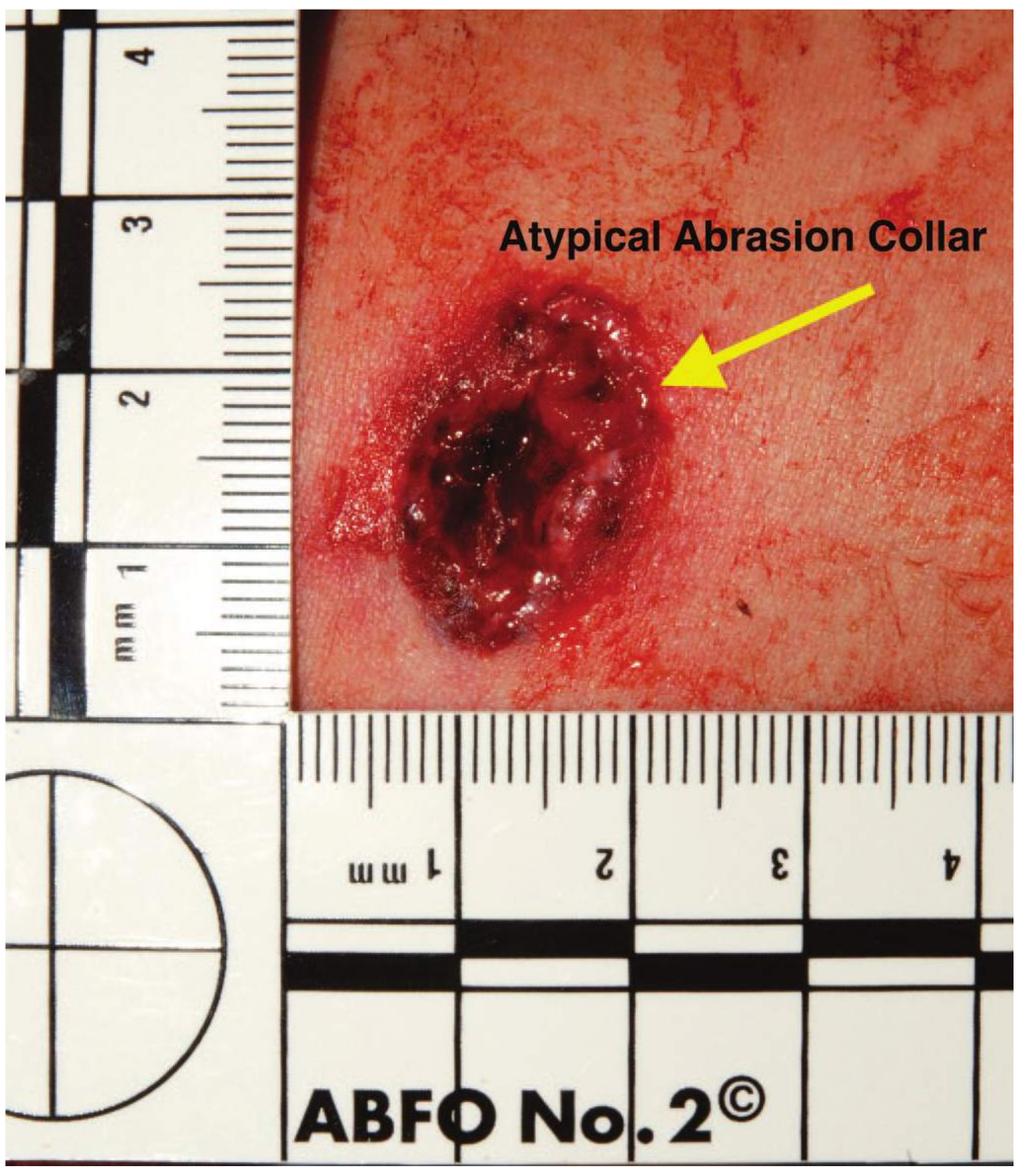


FIGURE 19.4 ■ **Atypical Abrasion Collar.** When a bullet passes through an intermediate object, ie, a door or windshield, it can become deformed or misshapen with an irregular surface. When a misshapen bullet (Fig. 19.3) passes through skin, the abrasion collar it generates appears irregular and jagged when compared to the smooth one caused by the nondeformed bullet (Figs. 19.1 and 19.2). (Photo contributor: William S. Smock, MD.)

and the associated pattern will depend upon the barrel length, muzzle-to-skin distance, type of gunpowder (ball, flattened ball, or flake), presence of intermediate objects, and caliber of the weapon. Spherical powder travels farther and has greater penetration than flattened ball or flake powder. "Pseudotattooing" (Fig. 19.6) is punctate abrasions from fragments created when bullets pass through intermediate objects like wood or glass.

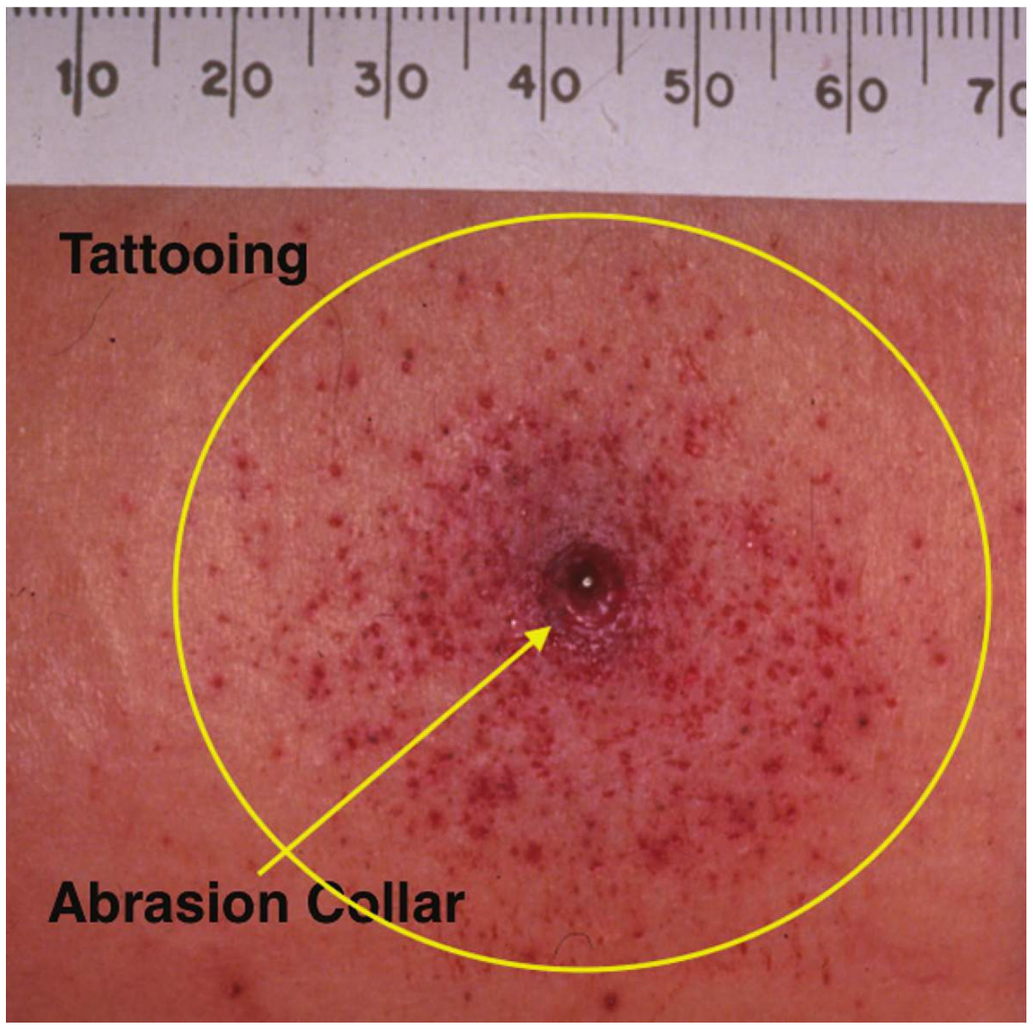


FIGURE 19.5 ■ **Intermediate-Range Gunshot Wound.** Punctate abrasions present on the skin are the result of impact from gunpowder that is either wholly or partially unburned. This phenomenon is termed tattooing or stippling. Tattooing is pathognomonic for intermediate-range (less than 48 inch) gunshot wounds. (Photo contributor: William S. Smock, MD.)

FIGURE 19.6 ■ **Pseudotattooing.** Punctate abrasions present on the forearm are the result of impacts with glass fragments. The fragments were produced from bullets penetrating the windshield. (Photo contributor: William S. Smock, MD.)

Close-Range (Near-Contact) Wounds: "Close range" is defined as the maximum range at which soot is deposited on the clothing (Fig. 19.7) or wound (Fig. 19.8) and typically is a muzzle-to-victim distance of 6 inches or less. On rare occasions, however, soot has been found on victims as far as 12 inches from the offending weapon. The concentration of soot will vary inversely with the muzzle-to-victim distance and its appearance will be affected by the type of gunpowder and ammunition used, the barrel length, the caliber, and the type of weapon.

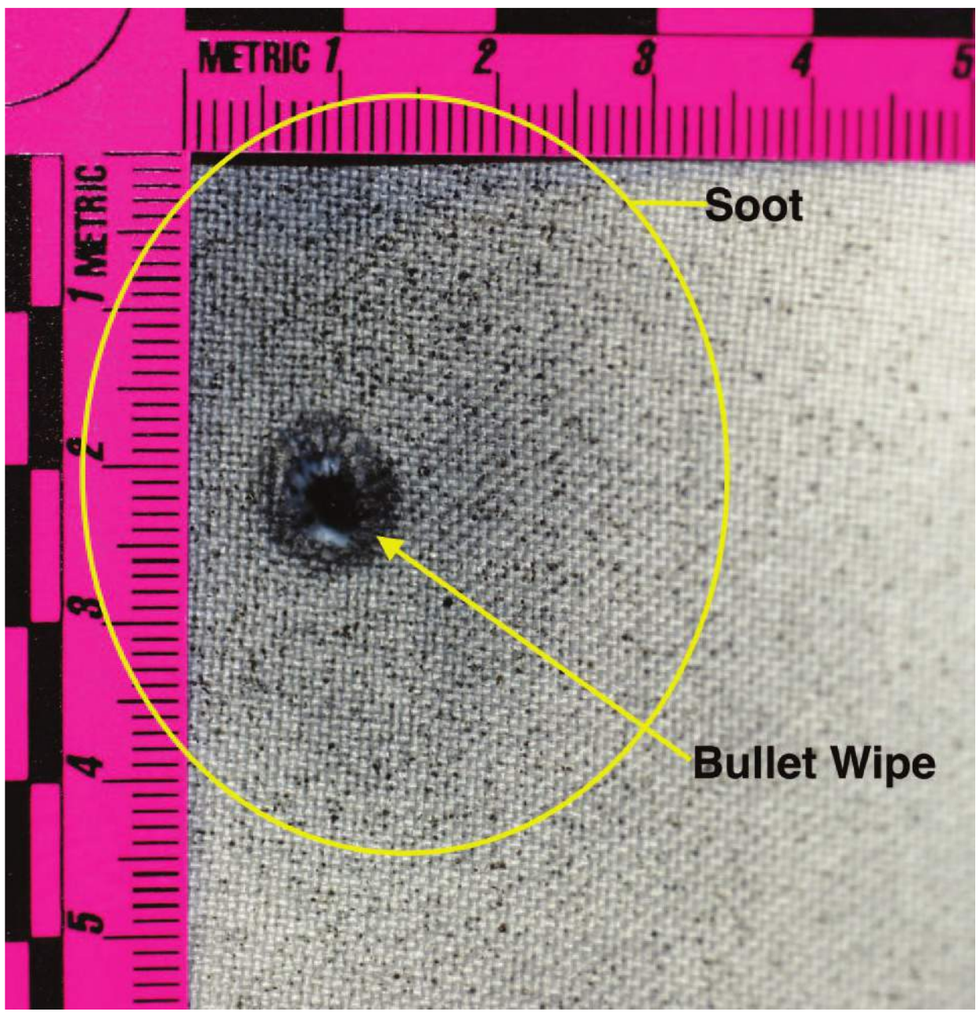


FIGURE 19.7 ■ **Soot and Bullet Wipe.** Soot is the carbonaceous residue from the burning of gunpowder. Soot is associated with close-range wounds, 6 inches or less. Bullet wipe is residue and/or lead from the surface of the bullet that is transferred to clothing or skin. Bullet wipe can be seen at any range of fire. Clothing should be collected and packaged in separate paper bags for submission to the crime laboratory. (Photo contributor: William S. Smock, MD.)

Contact Wounds: A contact wound occurs when the barrel or muzzle is in contact with the skin or clothing as the weapon is discharged. Contact wounds can be described as tight, where the muzzle is pushed hard against the skin, or loose, where the muzzle is incompletely or loosely in contact with the skin or clothing. Wounds sustained from tight contact with the barrel can vary in appearance from a small hole with seared, blackened edges (from the discharge of hot gases and an actual flame), to a gaping, stellate wound (from the expansion of the skin from gases) (Figs. 19.9 and 19.10). Large stellate wounds are often misinterpreted as exit wounds based solely upon their size and without adequate examination of the wound characteristics.

In a tight-contact wound, all materials—the bullet, gases, soot, incompletely combusted gunpowder, and metal fragments—are driven into the wound. If the wound overlies thin or bony tissue, the hot gases will cause the skin to expand to such an extent that it stretches and tears. These tears will have a triangular shape, with the base of the tear overlying the entrance wound. Larger tears are associated with ammunition of .32 caliber or greater, or magnum loads.

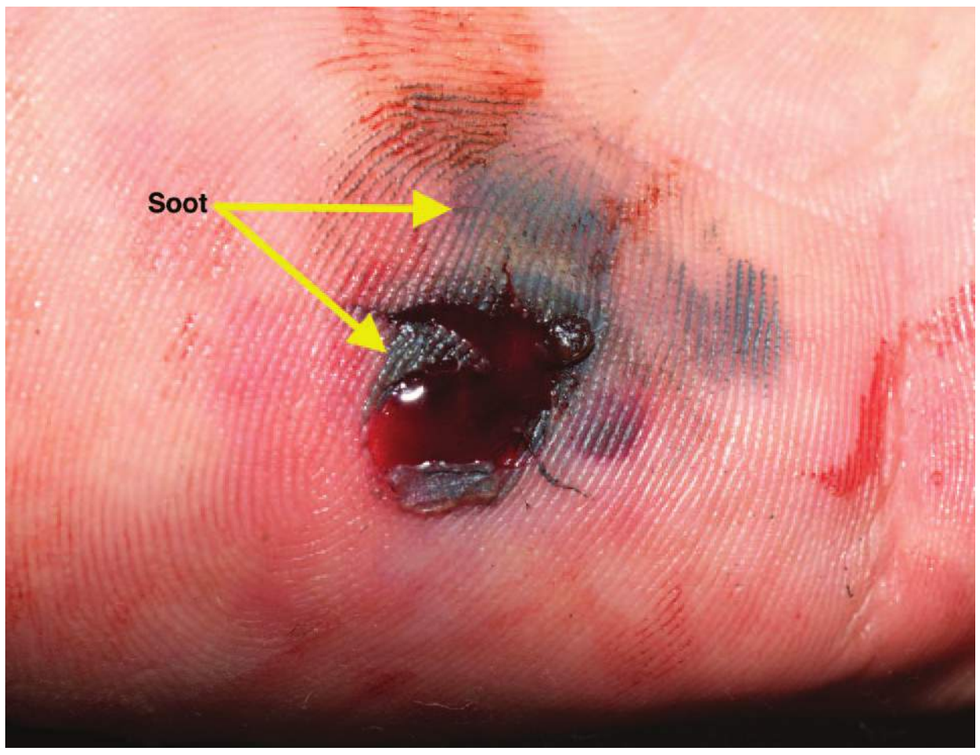


FIGURE 19.8 ■ **Close-Range Gunshot Wound.** The deposition of carbonaceous material or soot is seen on the palm from a close-range or near-contact gunshot wound. Soot is short-lived evidence and its presence surrounding a wound should always be documented. (Photo contributor: William S. Smock, MD.)

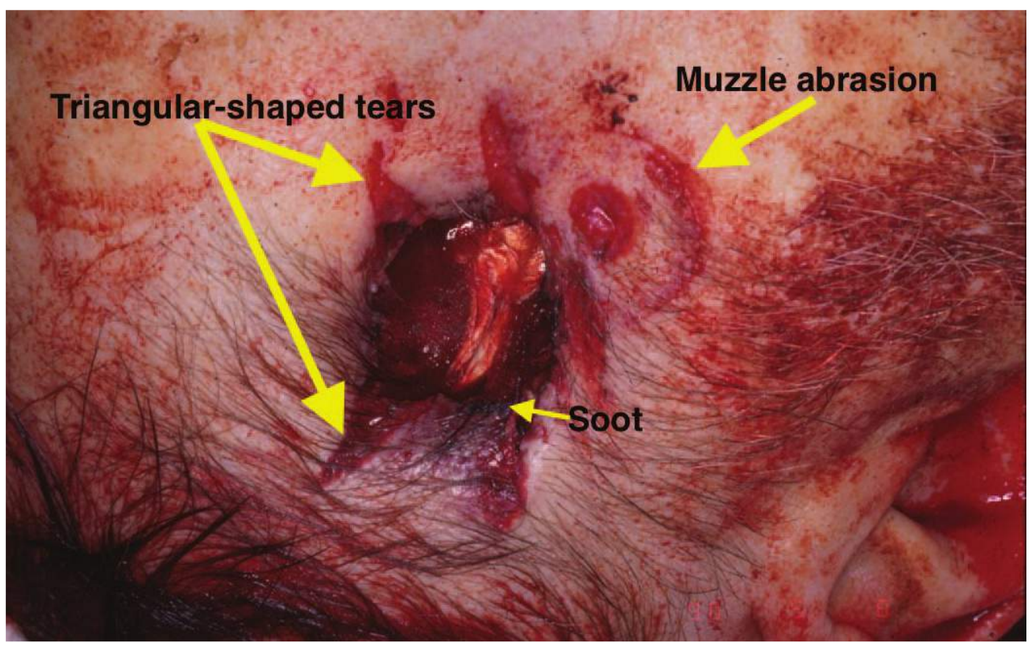


FIGURE 19.9 ■ **Contact Gunshot Wound with Muzzle Abrasion.** A contact gunshot wound to the right temple from a 9 mm semiautomatic handgun. Note the triangle-shaped tears, soot, seared wound margins and a muzzle-abrasion at the 2-o'clock position. The muzzle abrasion or muzzle imprint was the result of the injection of gases into the skin, causing a rapid and forceful expansion of the skin against the barrel. (Photo contributor: William S. Smock, MD.)

FIGURE 19.10 ■ **Contact Gunshot Wound.** A contact wound to the forehead from a .38 caliber handgun. The wound margins display triangle-shaped tears, searing, and soot deposition. (Photo contributor: William S. Smock, MD.)

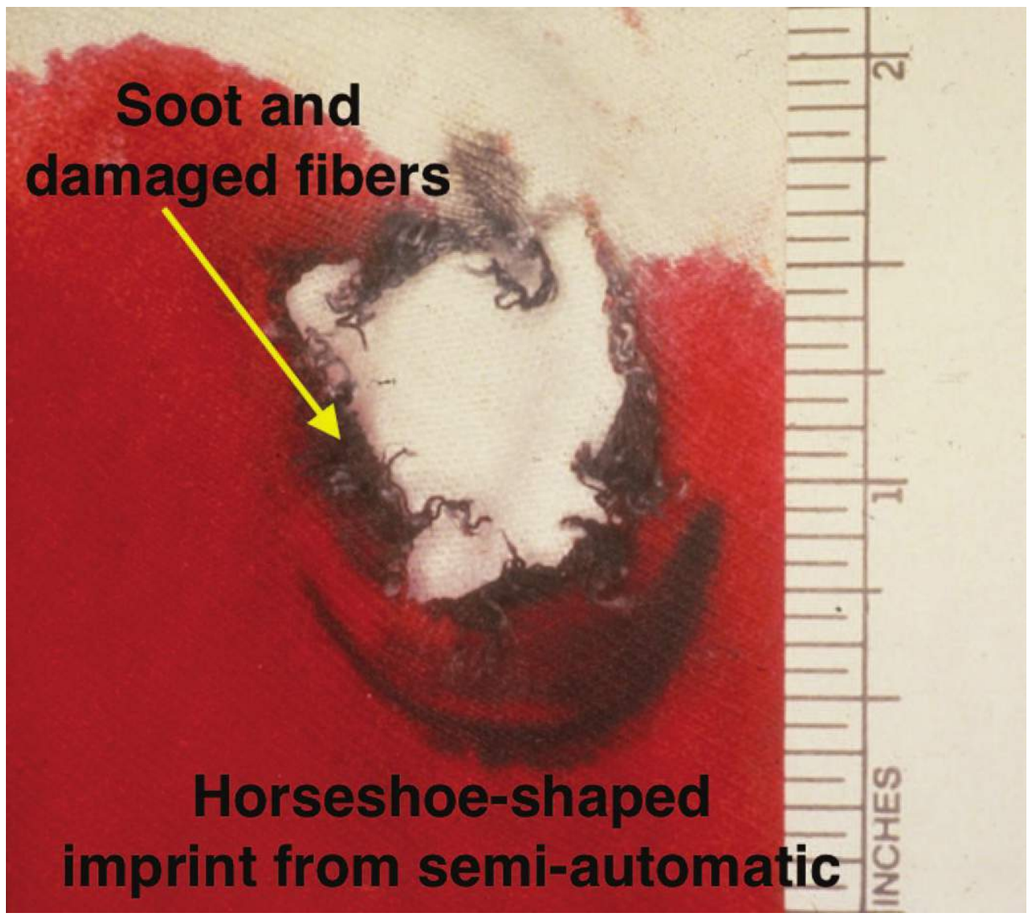


FIGURE 19.11 ■ **Muzzle Imprint, Soot, and Seared Fibers.** The clothing exhibits a "horseshoe-shaped" soot mark reflecting the outline of the frame of a semiautomatic handgun. Seared fibers are the result of flame and hot gases expelled from the barrel when it is in contact with clothing. (Photo contributor: William S. Smock, MD.)

Stellate tears are not pathognomonic for contact wounds. Tangential wounds, ricochet or tumbling bullets, and some exit wounds may also be stellate in appearance. These wounds are distinguished from tight-contact wounds by the absence of soot and powder within the wound. In some tight-contact wounds, expanding skin is forced back against the muzzle of the gun, causing a characteristic pattern contusion called a muzzle contusion (Fig. 19.9). An outline of the barrel can also be imprinted on the overlying clothing and is associated with contact wounds through clothing (Fig. 19.11). These patterns are helpful in determining the type of weapon (revolver or semiautomatic) used to inflict the injury and should be documented prior to wound debridement or surgery.

With a loose-contact wound, where the muzzle is angled or held loosely against the skin, soot and gunpowder residue will be present in and around the wound. The angle between the muzzle and skin will determine the soot pattern. A perpendicular, loose-contact or near-contact injury results in searing of the skin and deposition of the soot evenly around the wound. A tangential loose or near-contact injury produces an elongated searing pattern and deposit of soot around the wound.

"Bullet wipe" is a residue from soot, soft lead, or lubricant, which may leave a gray or black rim or streak on the skin or clothing overlying an entrance wound (Fig. 19.7). This discoloration may also be found around the abrasion collar but is usually more prominent on clothing.

Exit Wounds

Determining whether a wound is an entrance or an exit wound should be based on the physical characteristics and physical evidence associated with the wound and never upon the size of the wound. The size of the exit wounds are the result of a bullet pushing and stretching the skin from inside outward. The skin edges are generally everted, with sharp but irregular margins (Figs. 19.12-19.14). Abrasion collars, soot, searing, and tattooing are not associated with exit wounds. Soot can be seen at an atypical exit wound site if the entrance wound is close to the associated exit wound. Soot can be propelled through the wound from entrance to exit when the wound track is extremely short. If this is noted, the soot deposition will be more pronounced at the entrance and only faintly observed within the exit wound.

Exit wounds assume a variety of shapes and appearances and are not consistently larger than their corresponding entrance wounds. The size of an exit wound is determined primarily by the amount of energy transmitted to underlying tissue, bone for example, which is extruded from the wound. A bullet's size, shape, and attitude as it exits as well as postinjury tissue swelling will affect its size. A bullet's usual nose-first attitude can change upon entering the skin to a tumbling

and yawing one. A bullet with sufficient energy to exit the skin in a sideways attitude or one that has increased its surface area by mushrooming may produce an exit wound larger than its entrance wound. Energy transferred to bone, with resultant ballistic fracture, may also result in an exit wound larger than the entrance wound (Fig. 19.14). A "false abrasion collar" or "shored exit" wound may mimic an entrance wound. This occurs when the epithelium is pressed against a supporting surface such as a floor, wall, chair, firm mattress, or wallet (Figs. 19.15 and 19.16).

FIGURE 19.12 ■ **Exit Gunshot Wound.** A slit-like exit wound on the scalp from an intraoral gunshot. Exit wounds may take on a variety of appearances. (Photo contributor: William S. Smock, MD.)

FIGURE 19.13 ■ **Exit Gunshot Wound.** Stellate tears in an exit wound from a .22 caliber long rifle bullet that impacted the radius and ulna. The stellate configuration of an exit wound should not be confused with that of a contact wound. Exit wounds lack soot and seared skin. (Photo contributor: William S. Smock, MD.)

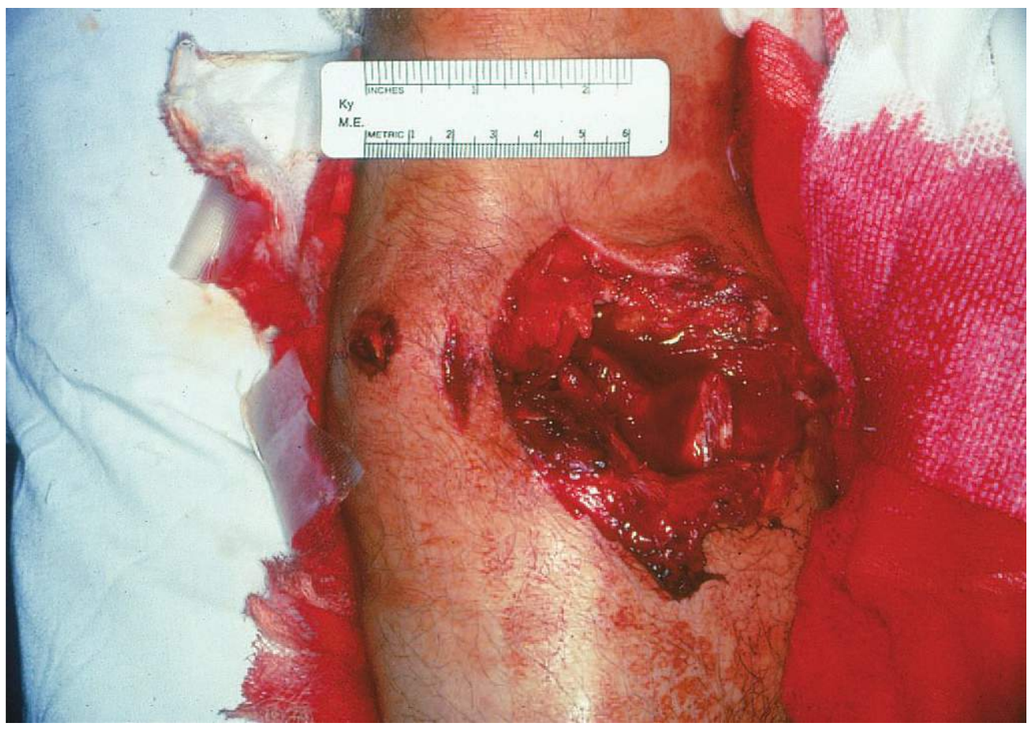


FIGURE 19.14 ■ **High-Velocity Gunshot Wound.** A perforating high-velocity gunshot wound to a lower extremity. The gaping exit wound resulted from the transfer of energy from the projectile to the tibia. The impact propelled multiple bony fragments through the skin. (Photo contributor: William S. Smock, MD.)

Graze Wounds

Graze wounds are considered atypical and result from tangential contact with a passing bullet. The direction of the bullet's path may be determined by careful wound examination. The bullet produces a trough and may cause the formation of skin tags on the lateral wound margins (Fig. 19.17). The base of these tags points toward the weapon and away from the direction of bullet travel.

Evidence Collection

Preservation of clothing, bullets, and cartridge cases is critical to the investigation of gun violence. Clothing should be place in separate paper bags and bullets in breathable containers. The microscopic marks on the exterior surface of a bullet can be used to identify the offending weapon. The use of booties on the ends of hemostats can prevent the loss of essential evidence and preserve these unique identifying microscopic marks from being altered or permanently destroyed (Fig. 19.18).

Pearls

1. Distant-range gunshot wounds are inflicted from a distance of greater than 4 feet and there is no tattooing, soot, or searing associated with the wound.
2. Intermediate-range gunshot wounds are inflicted at a distances up to 4 feet and characteristically are associated with tattooing from partially burned and unburned gunpowder impacting the skin.

FIGURE 19.15 ■ **Shored Gunshot Exit Wound.** A "shored exit" or "false abrasion collar" associated with a gunshot wound of exit. The false abrasion collar results when the skin is supported by a firm surface as the bullet exits. Shored exits occur when epithelium is pressed against a supporting surface (ie, floor, wall, chair, firm mattress, or, as in this case, wallet). (Photo contributor: William S. Smock, MD.)

FIGURE 19.16 ■ **Wallet Causing Shored Gunshot Exit Wound.** The wound seen in Fig. 19.15 occurred as a result of this wallet resisting the bullet's exit. (Photo contributor: William S. Smock, MD.)

FIGURE 19.17 ■ **Graze Gunshot Wound.** A deep graze wound from a handgun is seen. The dark wound margins are the result of drying artifact and should not be confused with the deposition of soot. (Photo contributor: Lawrence B. Stack, MD.)

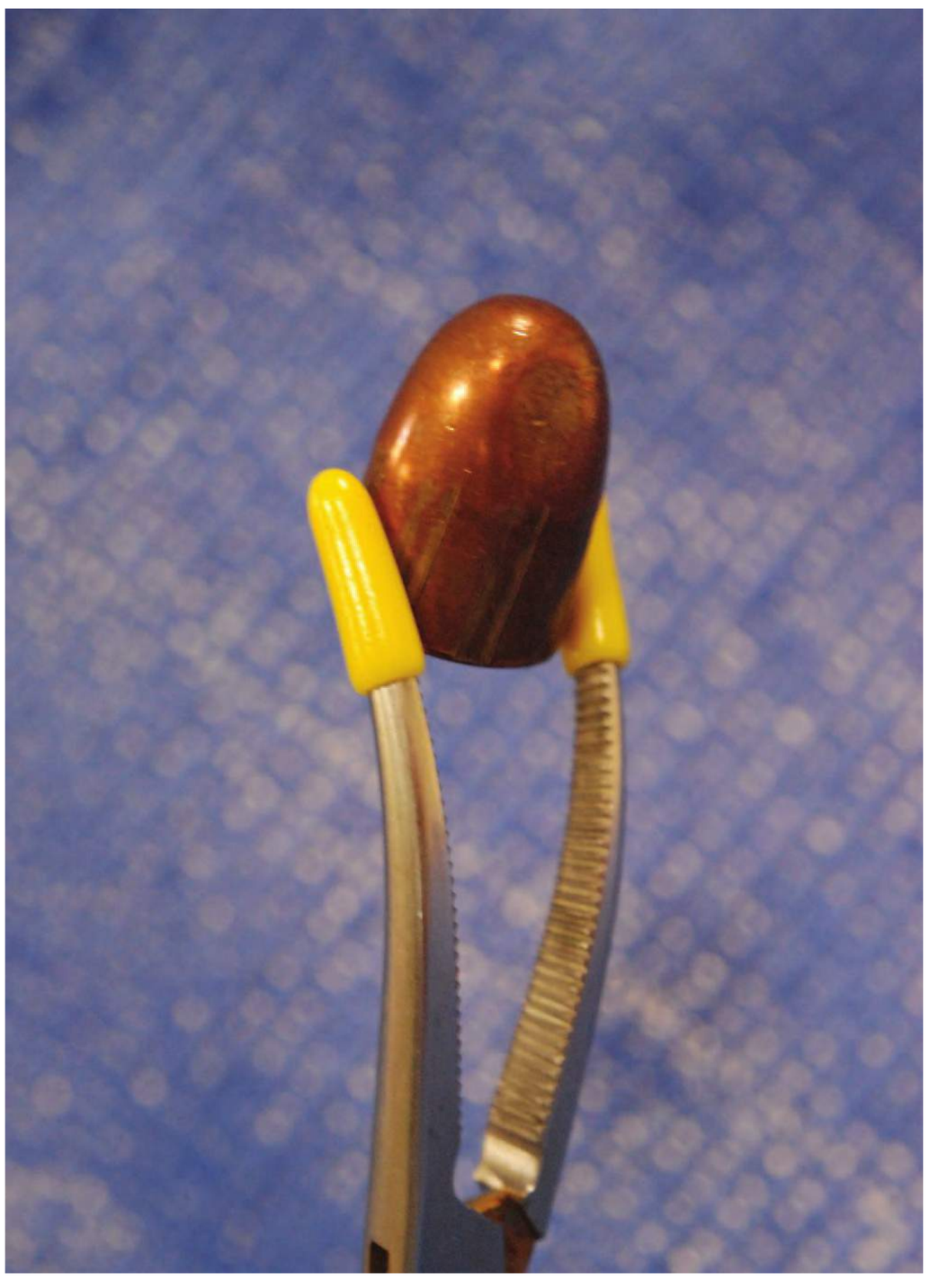


FIGURE 19.18 ■ **Suture Booties.** The use of booties on hemostats prevents the identifying microscopic marks on the bullet's surface from being damaged by handling. (Photo contributor: William S. Smock, MD.)

3. Near or close-contact gunshot wounds are defined as the maximum range at which soot is deposited on the wound or clothing and typically occur at a distance of 6 inches or less.
4. Contact gunshot wounds (barrel is in contact with the skin or clothing at time of discharge) vary in size but will include triangular tears, searing of the skin, and soot within or around the wound.
5. Abrasion collars, soot, searing, and tattooing are associated with entrance wounds.
6. Determination of whether a wound is an entrance or exit wound should **only** be based on the physical characteristics of the wound and clothing and not on the size of the wound.
7. The size of an exit wound is determined by: (1) energy transferred from the bullet to underlying tissue (bone pushed out), (2) bullet shape (mushroomed) and configuration, and (3) swelling of underlying tissue.
8. Emergency physicians should attempt to recognize, document, preserve, and collect short-lived evidence whenever the clinical situation allows.

Every "weapon" (hand, belt, hot iron, knife, electrical cord, baseball bat, tire iron) can leave a mark, design, or pattern stamped or imprinted upon or just below the level of the epithelium. The imprints of these weapons are called pattern injuries, which are considerably reproducible. These injuries can be categorized into three major classifications according to their source: sharp-force, blunt-force, and thermal-pattern injuries.

SHARP-FORCE-PATTERN INJURIES

Clinical Summary

There are two types of sharp-force injuries: incised and stabbed. The incised wound is longer than it is deep. The stab wound is defined as a puncture wound that is deeper than it is wide (Figs. 19.19-19.21). The wound margins of sharp-force injuries are clean and lack the abraded edges of lacerations from blunt forces. Forensic information can be gathered during the examination of a stab wound. Some characteristics of a knife blade, single- or double-edged, can be determined by visual inspection. Characteristics such as serrated versus sharp can be determined if the blade was drawn across the

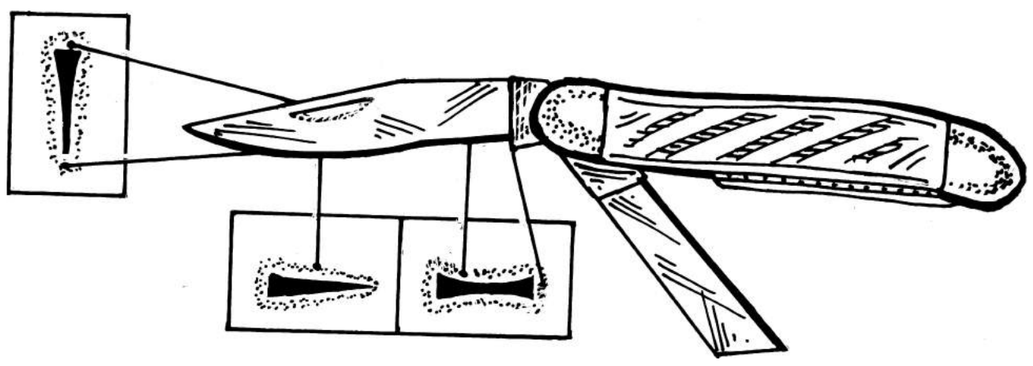


FIGURE 19.19 ■ **Stab Wound.** A stab wound from a single-edged knife blade will impart a sharp edge and a dull edge to the wound. If the blade penetrates to the proximal portion of the blade, a contusion may result from contact with the hilt of the knife.

FIGURE 19.20 ■ **Single-Edge Stab Wound with Hilt Mark.** A single-edged stab wound with a small hilt mark associated with the dull edge of the blade. (Photo contributor: William S. Smock, MD.)

FIGURE 19.21 ■ **Single-Edge Stab Wound.** A stab wound from a single-edged knife blade. The superior portion of the wound corresponds with the dull edge of the blade and the inferior with the sharp edge of the blade. (Photo contributor: William S. Smock, MD.)

skin during insertion or withdraw from the victim. Serrated blades do not always leave these characteristic marks.

When a patient presents with superficial and/or parallel incisions, the wounds must be considered intentionally self-inflicted until proven otherwise (Figs. 19.22 and 19.23). The emergency physician should obtain a more detailed history, including the number of "swipes" the alleged assailant made with the knife. Accidental, self-inflicted incised wounds on the palm or palmar aspect of the fingers are common on the hands of suspects in violent stabbings (Fig. 19.24). The suspects' hand slips down the handle and makes contact with the knife blade when the blade impacts a firm object, like bone (Fig. 19.25).

Pearls

1. Incised wounds have sharp wound margins and are longer than they are deep. Incisions result from contact with a sharp-edged implement: glass, knife blade, razor blade, or scalpel.
2. Stab wounds are puncture wounds that are deeper than they are long.
3. Knife-blade characteristics (single or dual edged, serrated or smooth) can frequently be determined by a visual inspection of the wound.
4. Superficial and/or parallel incisions should be assumed to be self-inflicted.

FIGURE 19.23 ■ **Self-Inflicted Incised Wounds.** Examination of the "victim's" chest reveals multiple superficial incisions. The patient initially claimed she was "assaulted" but later admitted that the wounds were self-inflicted. (Photo contributor: William S. Smock, MD.)

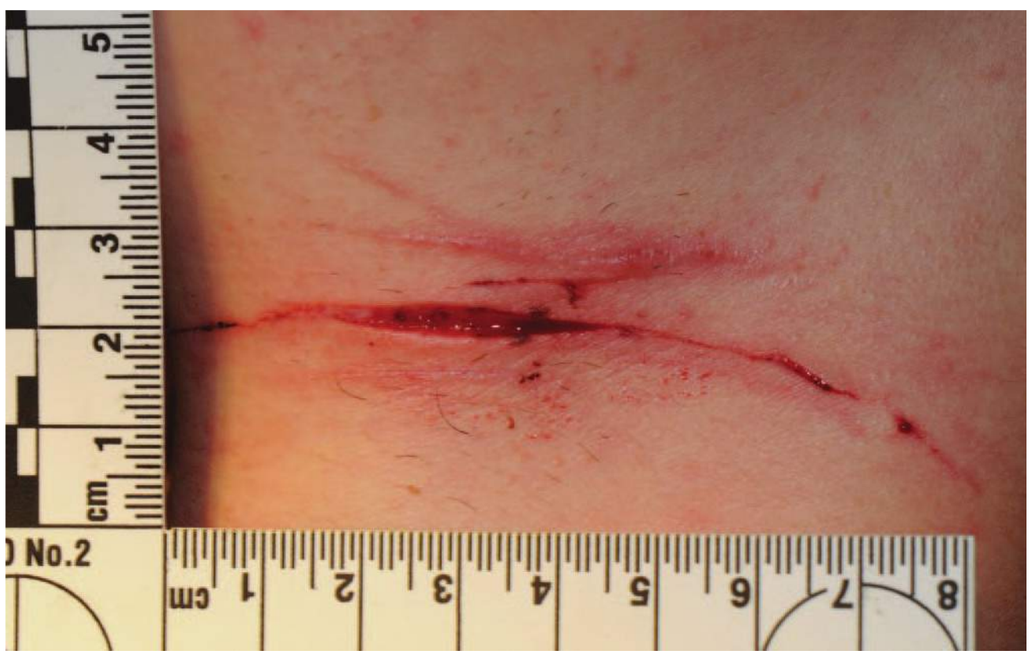


FIGURE 19.22 ■ **Self-Inflicted Incised Wounds.** Superficial and/or parallel incisions are pathognomonic for intentional self-inflicted wounds. This patient has acute and healed self-inflicted incisions in the same location on the anterior lateral aspect of her neck. (Photo contributor: William S. Smock, MD.)

FIGURE 19.24 ■ **Incised Wounds on the Palm.** Suspects in stabbings sometimes have incised wounds on their own palm or the palmar surfaces of their fingers. (Photo contributor: William S. Smock, MD.)

FIGURE 19.25 ■ **Hand Slippage on Knife.** When the knife blade suddenly stops (for example if the blade hits bone) the hand can slip down the handle and make contact with the blade incising wounds on the assailant's palm (Fig. 19.24). (Photo contributor: William S. Smock, MD.)

Clinical Summary

The most common blunt force injury is the contusion (Fig. 19.26). The pattern contusion is one that helps identify the causative weapon. A blow from a linear object leaves a contusion that is characterized by a set of parallel lines separated by an area of central clearing (Fig. 19.27). The blood underlying the striking object is forcibly displaced to the sides, which accounts for the pattern's appearance. Pattern injuries that an emergency physician should recognize include those caused by the hand (slap marks [Fig. 19.27], fingertip contusions [Fig. 19.28], grab marks, ligature marks [Fig. 19.30], fingernail abrasions), solid objects (baseball bat, tire iron, 2 by 4, belt, shoe, comb), and bite marks.

Other manifestations of blunt-force trauma to the skin are the abrasion and the laceration. A weapon with a unique shape or configuration may stamp a mirror image of itself on the skin (Fig. 19.29) and provide the emergency physician with additional information on the mechanism of the injury. The color of a contusion cannot be used to accurately date its age.

Pearls

1. A contusion is the most common blunt-force injury pattern in interpersonal violence.

FIGURE 19.26 ■ **Periorbital Contusion.** A contusion, without a discernable pattern, is the result of some type of blunt-force trauma. Victims of interpersonal violence frequently present with nonspecific, nonpattern contusions. A 24-hour-old cigarette burn is located on the victim's right upper lid. This victim was held against her will for 48 hours and repeatedly beaten about the face. (Photo contributor: William S. Smock, MD.)

FIGURE 19.27 ■ **Slap-Mark Pattern Contusions.** This victim of a "fall from the couch" has a pattern injury present on the right cheek. The pattern of central clearing surrounded by linear parallel contusions is pathognomonic for impact with a rounded linear object, in this case the extended fingers of a hand. (Photo contributor: William S. Smock, MD.)

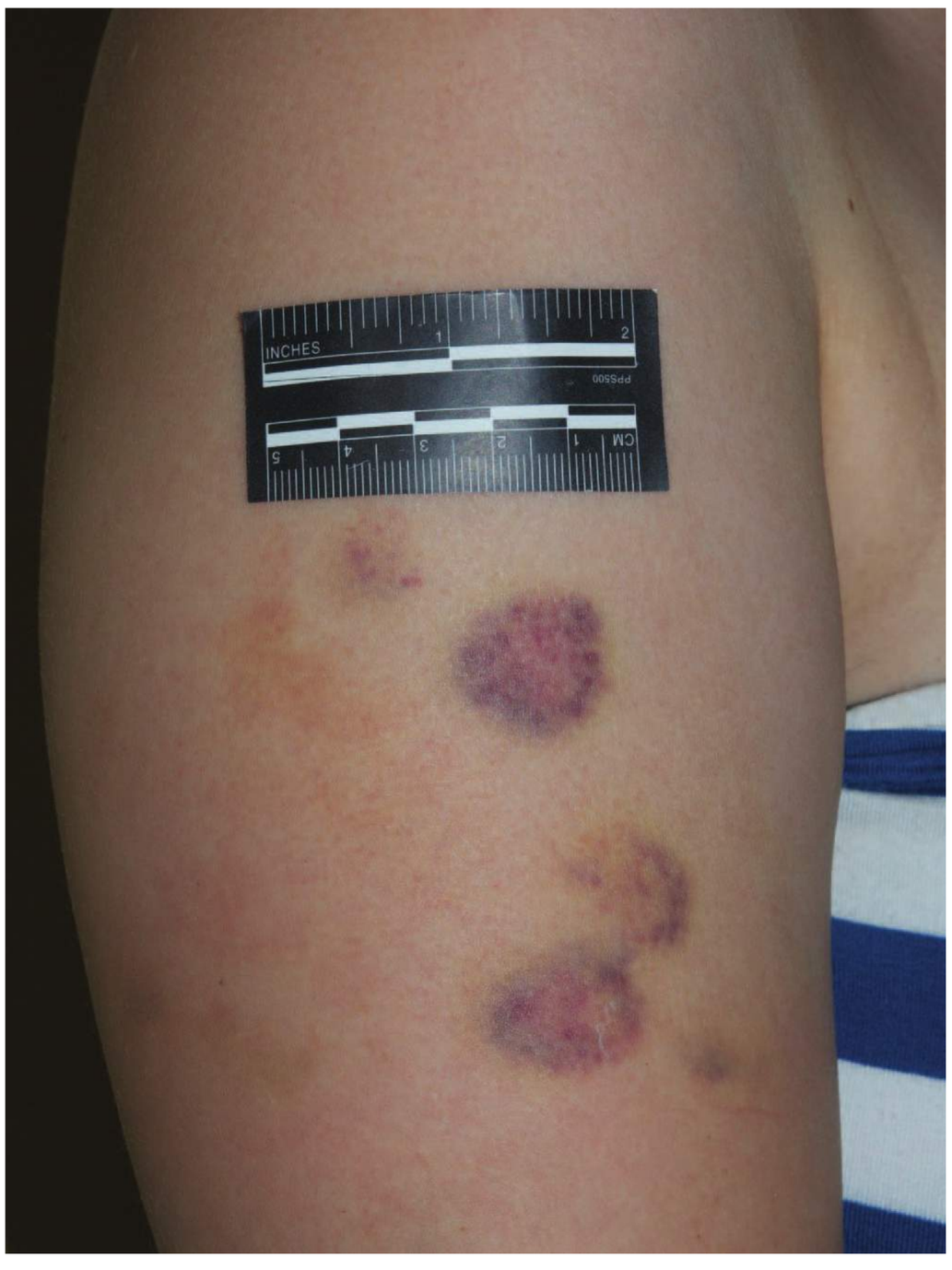


FIGURE 19.28 ■ **Fingertip Contusion Pattern.** This patient exhibits fingertip contusions. The emergency physician should never attempt to date the age of a contusion based upon its color. (Photo contributor: William S. Smock, MD.)

2. Blood underlying the force of the contusion is displaced to either side of the object, causing a pattern contusion in the shape of that object. This pattern is recognized by the central clearing surrounded by linear parallel contusions.
3. Emergency physicians must be able to recognize the pattern injuries caused by the hand (Fig. 19.27), solid objects, and bites.
4. Emergency physicians should not attempt to date the age of a contusion based upon its color or stage of resolution.

FIGURE 19.29 ■ **Tire Mark.** This tire mark, a mirror image of the tire tread, was the only evidence that connected this pedestrian with the vehicle involved in the hit-and-run incident. (Photo contributor: William S. Smock, MD.)

Clinical Summary

Manual strangulation is one of the most lethal forms of intimate partner violence and demonstrates the abuser's power and control over the victim. The brain will suffer an anoxic injury when the flow of oxygenated blood is stopped by occlusion of the carotid arteries. Strangulation victims report visual and auditory changes just prior to a loss of consciousness. The loss of bladder control indicates a deep anoxic insult to the brain of at least 15 seconds in duration. The loss of bowel control occurs with carotid artery occlusions of greater than 30 seconds. The jugular veins can be occluded with 4 psi, the carotids with 11 psi, and the trachea with 34 psi. Occlusion of venous return can cause capillary rupture from increased pressure and the development of petechial hemorrhage (Figs. 19.32-19.34). Petechial hemorrhages can develop in any vascularized tissue, including the brain. The most sensitive areas for petechial development include the conjunctiva, sclera, and intraoral mucosa. The use of an oral-pharyngeal scope (Fig. 19.35) often reveals vocal cord injury and the presence of petechial hemorrhage in the posterior pharynx. With simultaneous occlusion of both the arterial and venous systems, it is possible to induce fatal and nonfatal strangulation events without petechial hemorrhage and evidence of external trauma.

FIGURE 19.31 ■ **Strangulation Ligature Mark.** Ligature marks from the wires of her husband's "ear buds." The victim sustained neurological compromise from the compression of her carotids. (Photo contributor: William S. Smock, MD.)

FIGURE 19.32 ■ **Petechial Hemorrhage.** Extensive facial petechial was found above the area of neck compression in a nonfatal strangulation victim. (Photo contributor: William S. Smock, MD.)

FIGURE 19.30 ■ **Strangulation Ligature Belt Marks.** Ligature marks from a belt, with petechial hemorrhage above the marks, supported the mentally challenged patient's story of being "choked" by her caretaker. The majority of fatal strangulations do not exhibit external evidence of trauma. (Photo contributor: William S. Smock, MD.)

Pearls

1. Strangulation is associated with a 700% increased risk of interpersonal homicide.
2. Application of a ligature (Figs. 19.30-19.31) will leave a characteristic mark due to pressure applied over a small surface area.
3. Strangulation-induced petechial hemorrhage, from the rupture of venous vessels, may be found in the conjunctiva (Fig. 19.32), the soft palate, the posterior pharynx (Fig. 19.33), and above and below the area where pressure was applied (Fig. 19.34). Petechial hemorrhage is NOT required to prove strangulation. The majority of fatal strangulations exhibit no external evidence of trauma.
4. A victim of strangulation can be rendered unconscious in less than 8 seconds with bilateral occlusion of the carotid arteries. The carotids will be occluded with 11 pounds of pressure. Obtaining a history of neurological changes—loss of hearing, vision, consciousness, and bowel or bladder control—confirms neurologic injury and an anoxic insult to the brain.

FIGURE 19.33 ■ **Subconjunctival and Scleral Petechial Hemorrhage.** Increased venous pressure from the occlusion of the jugular veins during manual strangulation results in ruptured capillaries. (Photo contributor: William S. Smock, MD.)

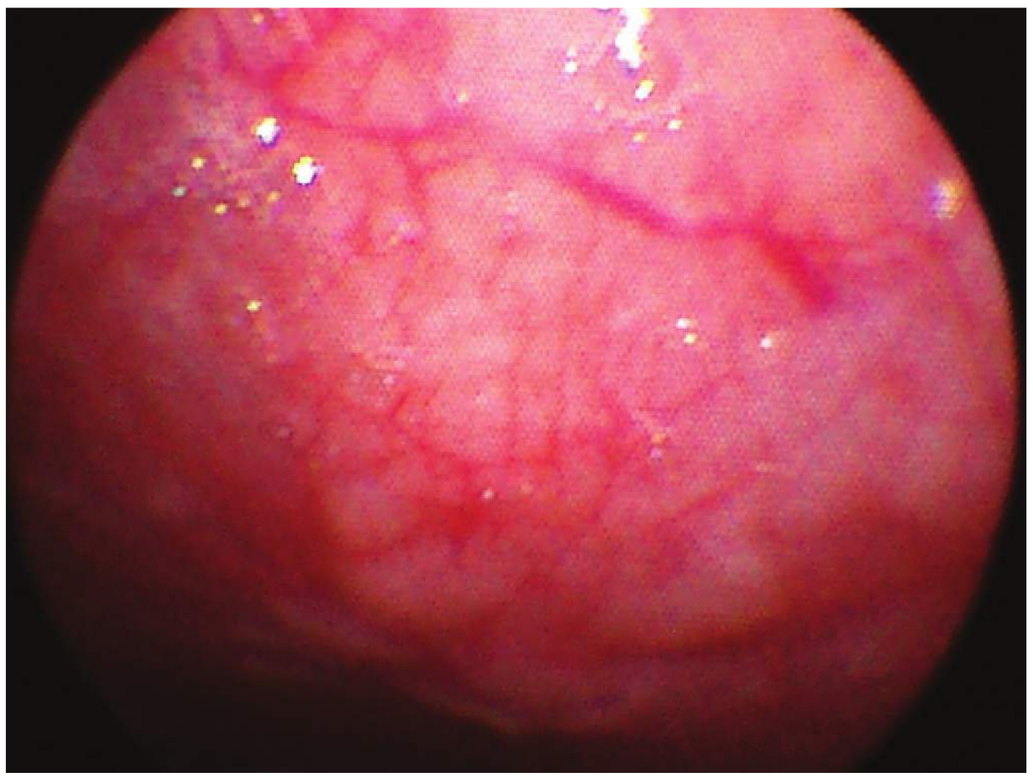


FIGURE 19.34 ■ **Petechial Hemorrhage in Posterior Pharynx.** Petechial hemorrhage in the posterior pharynx is a common finding in victims of strangulation. (Photo contributor: William S. Smock, MD.)

FIGURE 19.35 ■ **Oral-Laryngeal Scope.** An oral-laryngeal scope, equipped with a camera, is an excellent tool to document the extent of petechial hemorrhage in the pharynx and vocal cord injury in victims of strangulation. (Photo contributor: William S. Smock, MD.)

THERMAL-PATTERN INJURIES

Clinical Summary

Thermal pattern injuries are commonly seen in cases of abuse and assault especially when the victims are children and the elderly. A detailed history of the incident should include the position of the patient relative to the thermal source. This will help determine whether the injury was inflicted or accidental. Thermal injuries characterized by a distinct pattern that frequently present to emergency departments are those caused by cigarettes (Fig. 19.26), flatirons (Fig. 19.37), curling irons (Fig. 19.38), and scalding hot water (Fig 19.40). Water burns will be of two natures: immersion and splash. A sharp or clear line of demarcation between burned and unburned tissue characterizes an immersion or dipping burn. In contrast, splash burns are characterized by an irregular or undulating line or by isolated areas of thermal injury, usually round or oval in shape, caused by droplets of hot liquid. The severity of the scald injury depends upon two variables: the length of time that the skin was in contact with the offending substance and the temperature of the substance itself. Tap or faucet water causes full-thickness thermal damage in 1 second at 70°C (158°F), and 180 seconds at 48.9°C (120°F). Law-enforcement agencies should routinely measure the household's or institution's water temperature in any investigation involving a scald injury of a child, a developmentally delayed person, or an elderly one. The water temperature will give authorities an estimate of the duration of the insult and therefore the degree of malice of intention on the part of the assailant.

Pearls

1. A thermal-pattern injury is a common form of abuse seen in infants, institutionalized patients, and the elderly.
2. Emergency physicians must recognize thermal-pattern injuries of abuse.

FIGURE 19.37 ■ **Clothes-Iron Thermal Injury Pattern.** An iron was the weapon used to inflict this thermal injury. The areas of sparing are associated with its steam holes. (Photo contributor: William S. Smock, MD.)

FIGURE 19.36 ■ **Cigarette Thermal Injury Pattern.** A 24-hour-old cigarette burn is located on this victim's right upper lid. This victim was held against her will for 48 hours and repeatedly beaten about the face. (Photo contributor: William S. Smock, MD.)

FIGURE 19.38 ■ **Curling Iron Thermal Injury Pattern.** Curling irons can reach more than 200°F. This temperature can cause a full-thickness burn in less than 1 second. When this burn to the anterior surface of the leg was inflicted, someone's thumb was on the spring lever—evidence of intent (Fig. 19.39). (Photo contributor: William S. Smock, MD.)

FIGURE 19.39 ■ **Curling Iron.** The V-shaped configuration of the pattern injury (Fig. 19.38) indicated that the assailant's thumb was on the spring-loaded lever when the burn was inflicted. This was evidence of intent. The victim's aunt confessed to inflicting the burn. (Photo contributor: William S. Smock, MD.)

FIGURE 19.40 ■ **Immersion-Line Thermal Injury Pattern.** A characteristic "immersion line" is seen in a thermal-pattern injury. The line of demarcation is associated with the depth of the immersion. (Photo contributor: William S. Smock, MD.)

Chapter 20

HIV CONDITIONS

Stephen P. Raffanti
Anna K. Person

Kaposi Sarcoma. Multiple KS lesions surrounding the nipple of this patient with advanced HIV. (Photo contributor: Seth W. Wright, MD.)

The authors would like to acknowledge the special contributions of J. Michael Ballister, MD, Roderick Morrison, MD, Shane Cline, MD, and Michael Krentz, MD, for their excellent work on prior editions of this chapter.

PRIMARY HIV INFECTION

Clinical Summary

Establishing a definitive diagnosis in the earliest stage of HIV infection can have significant impact on the patient's outcome and may serve a public health function as well. Patients are highly infectious during acute seroconversion and may be unaware that they are infected. It is believed that a significant number of the new cases of HIV infection occurring in the United States are acquired from acutely infected individuals. Identifying acute seroconverters could offer the opportunity to discuss transmission reduction behavior. Clinical illness accompanies primary HIV infection in approximately two-thirds of patients. The usual time from HIV exposure to the development of symptoms is approximately 10 to 20 days, with average symptom duration of 1.5 to 2 weeks. The most common symptoms following seroconversion mimic a typical viral syndrome and may include mucocutaneous lesions and a generalized maculopapular rash located over the face, neck, and trunk. The rash is seen in over 50% of persons with symptomatic primary HIV infection. The lesions are typically small, well circumscribed, erythematous, nonpruritic, and nontender. Less frequently, patients may demonstrate neurologic signs and symptoms consistent with meningoencephalitis, myelopathy, and peripheral neuropathy. If obtained, laboratory studies may show lymphopenia and thrombocytopenia.

Management and Disposition

Although rapid HIV testing has become feasible in most emergency department (ED) settings, the diagnosis of acute infection cannot rely solely on rapid antibody-based tests. If the possibility of acute seroconversion is entertained, then a discussion should take place with the patient regarding level of risk behavior. If acute HIV is suspected, HIV-1 ribonucleic acid (RNA) quantitative polymerase chain reaction (PCR) is the appropriate diagnostic test. Patients who are acutely seroconverting should have extremely high levels of HIV RNA. Importantly, a negative HIV antibody test does not rule out HIV infection in these cases. During the "window period" of acute HIV infection, antibodies against the HIV virus have not yet formed and may not be detected for 8 to 10 weeks. If acute HIV is suspected or confirmed, patients should be educated about disease transmission and referred for prompt follow-up and further outpatient testing and evaluation.

Pearls

1. Maintain a high degree of clinical suspicion for acute primary HIV infection, especially when sexually active patients or patients who use intravenous drugs present with mononucleosis-like symptoms, unexplained rash, mucocutaneous ulcers, lymphadenopathy, or aseptic meningitis.
2. Obtain an HIV RNA quantitative PCR (and not just an HIV antibody test) if acute HIV is suspected.
3. Ensure proper follow-up for patients in whom the diagnosis of acute HIV infection is entertained, and counsel patients regarding potential risk of transmission.

FIGURE 20.1 ■ **Primary HIV Infection.** A maculopapular rash is seen in over half of persons with symptomatic acute HIV infection. This less typical papular/vesicular rash was present in a patient with primary HIV infection. (Photo contributor: Gregory K. Robbins, MD, MPH.)

IMMUNE RECONSTITUTION INFLAMMATORY SYNDROME (IRIS)

Clinical Summary

Effective antiretroviral therapy establishes virologic suppression and can allow the patient's immune system to rebuild to a near-normal competency. In some patients who develop a rapid immune response after a very advanced degree of immune suppression, the immune reconstitution inflammatory syndrome (IRIS) can develop. In these patients, it is thought that previously subclinical infections manifest with the development of a more robust immune status. IRIS is most often seen in patients who have recently started antiretroviral therapy (usually within 60 days of starting therapy) with an initial CD4 count below 100 cells/mm^3. Most common infections that can "trigger" IRIS include mycobacterium (tuberculosis [TB] and mycobacterium avium complex [MAC]), cytomegalovirus (CMV), cryptococcal disease, and histoplasmosis. Patients typically present with fever, malaise, lymphadenopathy, and symptoms associated with the active opportunistic infection.

Management and Disposition

The key for the ED physician is to identify those patients who have recently started antiretroviral therapy and may be presenting with IRIS-related findings. Appropriate evaluation and management depends on the underlying opportunistic infection. Patients will often require admission to the hospital and consultation with an infectious diseases specialist.

Pearl

1. HIV-infected patients who are currently taking antiretroviral therapy and present with a febrile illness should be questioned regarding the duration of antiretroviral therapy and pretreatment CD4 count. Patients with advanced disease who have recently started antiretroviral therapy may be presenting with signs and symptoms of IRIS-related disease.

FIGURE 20.2 ■ **IRIS.** This patient presented with fever and diffuse lymphadenopathy secondary to MAC infection. The patient had no manifestations of his disseminated disease until being started on antiretroviral therapy. (Photo contributor: Stephen P. Raffanti, MD.)

ORAL HAIRY LEUKOPLAKIA

Clinical Summary

Oral hairy leukoplakia (OHL) is a disease of the lingual squamous epithelium caused by the Epstein-Barr virus (EBV). OHL generally affects the lateral portion of the tongue, although the floor of the mouth, palate, or buccal mucosa may also be involved. The lesions are white corrugated plaques that, unlike Candida, cannot be scraped from the surface to which they adhere. Most often OHL is asymptomatic, although occasionally this condition can be painful. Diagnosis is usually clinical, though definitive diagnosis can be made by biopsy characteristically revealing acanthosis and parakeratosis.

FIGURE 20.3 ■ **Oral Hairy Leukoplakia.** Typical-appearing lesions on side of tongue in this patient with HIV. (Photo contributor: Robert Brandt, MD.)

Management and Disposition

Patients who are known to be HIV-seropositive can be educated about the disease and reassured. OHL is not considered to be a premalignant lesion. If the patient is not known to be infected with HIV, primary care provider referral and HIV screening, if available, should be offered.

FIGURE 20.4 ■ **Oral Hairy Leukoplakia.** Exudate does not scrap off the tongue in oral hairy leukoplakia, differentiating it clinically from oral thrush. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. Oral candidiasis can be distinguished from OHL by utilizing a swab in an attempt to remove the exudate characteristic of thrush and by observing pseudohyphal elements microscopically with candida. OHL cannot be scraped off and usually involves the lateral aspect of the tongue.
2. OHL is fairly specific for HIV infection as it is rarely observed in patients with other immunodeficiencies. If OHL is identified in a patient not known to be HIV infected, risk factors and HIV screening options should be discussed.

Clinical Summary

Oral candida infections are seen in over half of all HIV patients, with the severity of infection often correlating with the degree of immunosuppression. Oral candidiasis can occur at all stages of HIV disease. The usual causative agent is Candida albicans, but other Candida species have been isolated. Oral candidiasis, or "thrush," can be classified as pseudomembranous, angular, or erythematous. Pseudomembranous candidiasis can be diagnosed by identifying removable whitish plaques on the tongue, uvula, and buccal mucosa. Erythematous or atrophic candidiasis appears as smooth red patches along the soft and hard palate. Although isolated oral candidiasis is not an AIDS-defining illness (though esophageal candidiasis is), oral candidiasis is an indication for pneumocystis prophylaxis regardless of CD4+ cell count.

Vaginal candidiasis is common in HIV positive patients and can cause a severe whitish discharge with vulvar erythema.

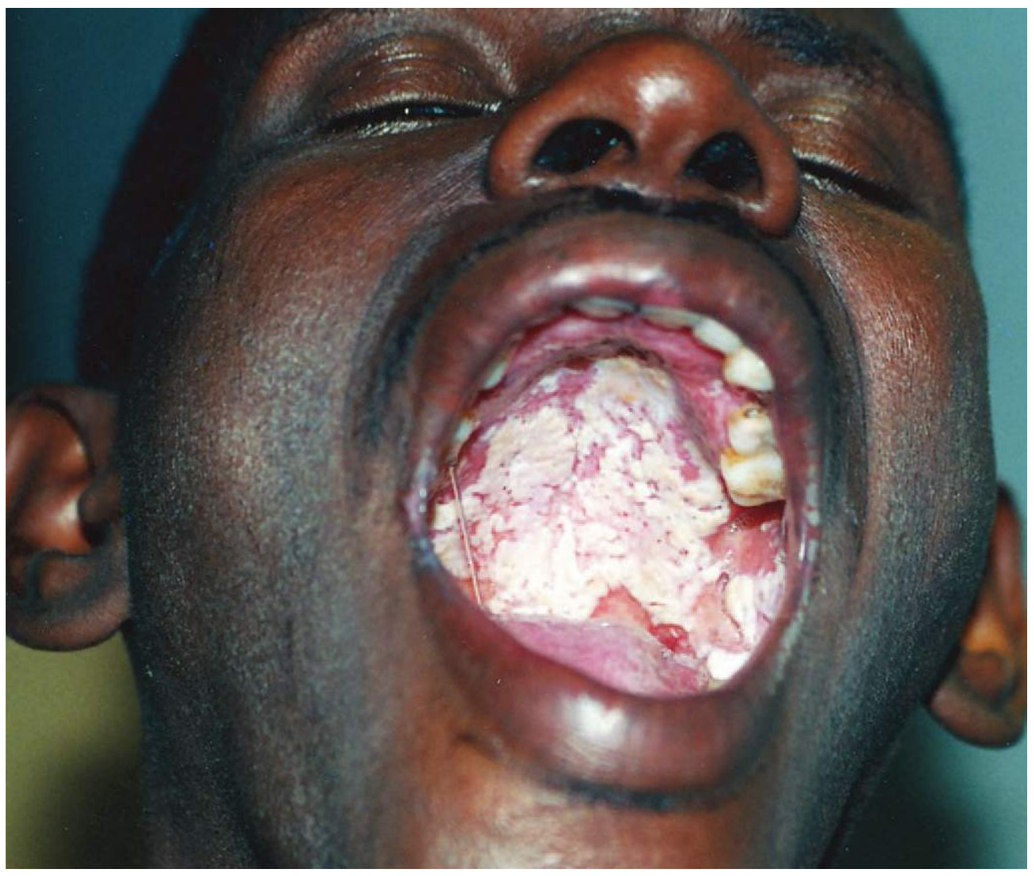


FIGURE 20.5 ■ **Oral Candidiasis.** Removable whitish plaques on the palate are seen in this HIV patient with pseudomembranous candidiasis. (Photo contributor: Thea James, MD.)

Management and Disposition

Patients presenting to the ED for dysphagia or odynophagia thought to be due to candidiasis should be evaluated for dehydration and may require inpatient admission. Most cases of minimally symptomatic oral candidiasis respond well to nystatin suspension or clotrimazole troches. More symptomatic oral or esophageal candidiasis usually responds to fluconazole. Rare cases of resistant candidiasis may require intravenous echinocandin therapy.

Pearls

1. Symptomatic oral and esophageal candidiasis is still a common ED presentation in HIV patients who are not receiving or are not compliant with effective antiretroviral therapy.
2. The diagnosis of oral candidiasis in a patient not already diagnosed with HIV infection should lead to a discussion of risk factors and should prompt HIV screening.
3. Mildly symptomatic oral, esophageal, or vaginal candidiasis usually responds to a one-time oral dose of fluconazole. Close clinical follow-up should be arranged.

FIGURE 20.6 ■ **Esophageal Candidiasis.** Endoscopy demonstrating esophageal candidiasis in this HIV patient. (Photo contributor: Edward C. Oldfield III, MD.)

FIGURE 20.7 ■ **Oral Thrush.** Typical plaque lesions are seen on the palate and uvula of this HIV patient. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Kaposi sarcoma (KS) is a low-grade vascular tumor associated with human herpesvirus 8 (HHV-8). Since the introduction of highly active antiretroviral therapy (HAART), the incidence of KS in HIV-infected persons has significantly declined. Skin involvement is characteristic but extracutaneous spread of KS is common, particularly to the oral cavity, gastrointestinal (GI) tract, and respiratory tract. The skin lesions appear most often on the lower extremities, face (especially the nose), oral mucosa, and genitalia. Most commonly, the lesions are papular, ranging in size from several millimeters to centimeters in diameter. Less commonly, the lesions may be plaque-like, especially on the soles of the feet, or exophytic and fungating with breakdown of overlying skin. Papular lesions may resemble lesions associated with bacillary angiomatosis or herpesvirus infections.

Pulmonary involvement can occur in AIDS-related KS. Affected persons may present with shortness of breath, fever, cough, hemoptysis, or chest pain or as an asymptomatic finding on chest x-ray. Diagnosis can be confirmed via bronchoscopy.

Management and Disposition

All HIV-infected patients with KS should receive antiretroviral therapy, which is generally effective in treating most cutaneous lesions. Systemic chemotherapy and newer immunomodulator therapy (interleukin-12, angiogenesis inhibitors) are reserved for more extensive disease including patients with visceral involvement.

Pearls

1. HIV patients who present with new papular lesions should be referred for dermatologic evaluation and biopsy.
2. A careful examination of the skin and oral cavity is appropriate in HIV-infected patients presenting with GI or respiratory complaints.
3. Half of patients with oral involvement have other GI tract lesions as well. Hemoccult testing of stool is a good preliminary screen.

FIGURE 20.8 ■ **Kaposi Sarcoma.** A single violaceous patch is seen on the face of an HIV-positive patient. (Photo contributor: George Turiansky, MD.)

FIGURE 20.9 ■ **Cutaneous Kaposi Sarcoma.** Characteristic non-raised violaceous Kaposi sarcoma lesions on the arm of this Caucasian patient with HIV. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 20.10 ■ **Kaposi Sarcoma.** Multiple black KS lesions, as typically seen in darker skinned patients. (Photo contributor: Seth W. Wright, MD.)

FIGURE 20.11 ■ **Kaposi Sarcoma.** Multiple KS lesions surrounding the nipple of this patient with advanced HIV. (Photo contributor: Seth W. Wright, MD.)

FIGURE 20.12 ■ **Oral Kaposi Sarcoma—Tongue.** Raised palpable erythematous Kaposi sarcoma on the tongue of this HIV patient. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Toxoplasma gondii is a widespread intracellular protozoan parasite with a definitive host stage in cats. Immunocompetent persons are usually asymptomatic, but immunocompromised persons (especially HIV-positive persons with <100 CD4 cells/μL) are susceptible to reactivation of latent disease with considerable morbidity and mortality.

Patients with central nervous system (CNS) toxoplasmosis most often present with symptoms consistent with intracranial mass lesions (headache, focal neurologic deficit, nausea/emesis, and/or seizure) or, less likely, encephalitis (fever, confusion, altered mental status). Contrasted computed tomography (CT) of the head typically reveals ring-enhancing lesions with a predilection for the basal ganglia. Magnetic resonance imaging (MRI) of the head is more sensitive than CT for identifying these lesions. Serological tests for T gondii antibodies (IgG) are usually positive. Other causes of mass lesions in the brain in HIV-infected individuals include CNS lymphoma, cryptococcoma, tuberculoma, and brain abscess.

Ocular toxoplasmosis is a less frequent complication of HIV disease. Patients typically present with eye pain and decreased visual acuity. Retinitis may be diagnosed by ophthalmoscopic evaluation revealing characteristic exudates and hemorrhage. Toxoplasmosis retinitis appears as raised yellow-white, cottony lesions in a nonvascular distribution, unlike the edematous perivascular exudates of CMV retinitis.

Management and Disposition

There are two combination regimens considered to be first choice for the treatment of toxoplasmosis. The most commonly used regimen is pyrimethamine and sulfadiazine for 6 weeks or until neurological findings have resolved; however, for those with sulfa allergies, pyrimethamine and clindamycin is an alternative. If the patient presents with seizures, loading doses of antiepileptics such as fosphenytoin or levatiracetam should be administered along with benzodiazepines. All patients with acute symptomatic CNS toxoplasmosis infection should be admitted for treatment.

In cases of suspected ocular toxoplasmosis, a complete eye examination including slit-lamp examination and measurements of intraocular pressure should be performed before consulting ophthalmology.

Pearls

1. HIV-infected patients presenting with focal neurologic findings or seizures should be evaluated for meningitis and space-occupying lesions in the brain. AIDS-specific differential includes toxoplasmic encephalitis, CNS lymphoma, and cryptococcal disease.
2. All HIV-infected patients should have a CNS space–occupying lesion excluded by imaging prior to lumbar puncture (LP).
3. Initial evaluation of the HIV-infected patient with possible CNS lesions should include CT or MRI imaging, LP, and triage management for bacterial meningitis. Further workup will require admission and infectious diseases consultation.

FIGURE 20.13 ■ Toxoplasma gondii **Infection.** Contrast head CT showing typical multiple ring-enhancing lesions seen in T gondii CNS infection. (Photo contributor: Edward C. Oldfield III, MD.)

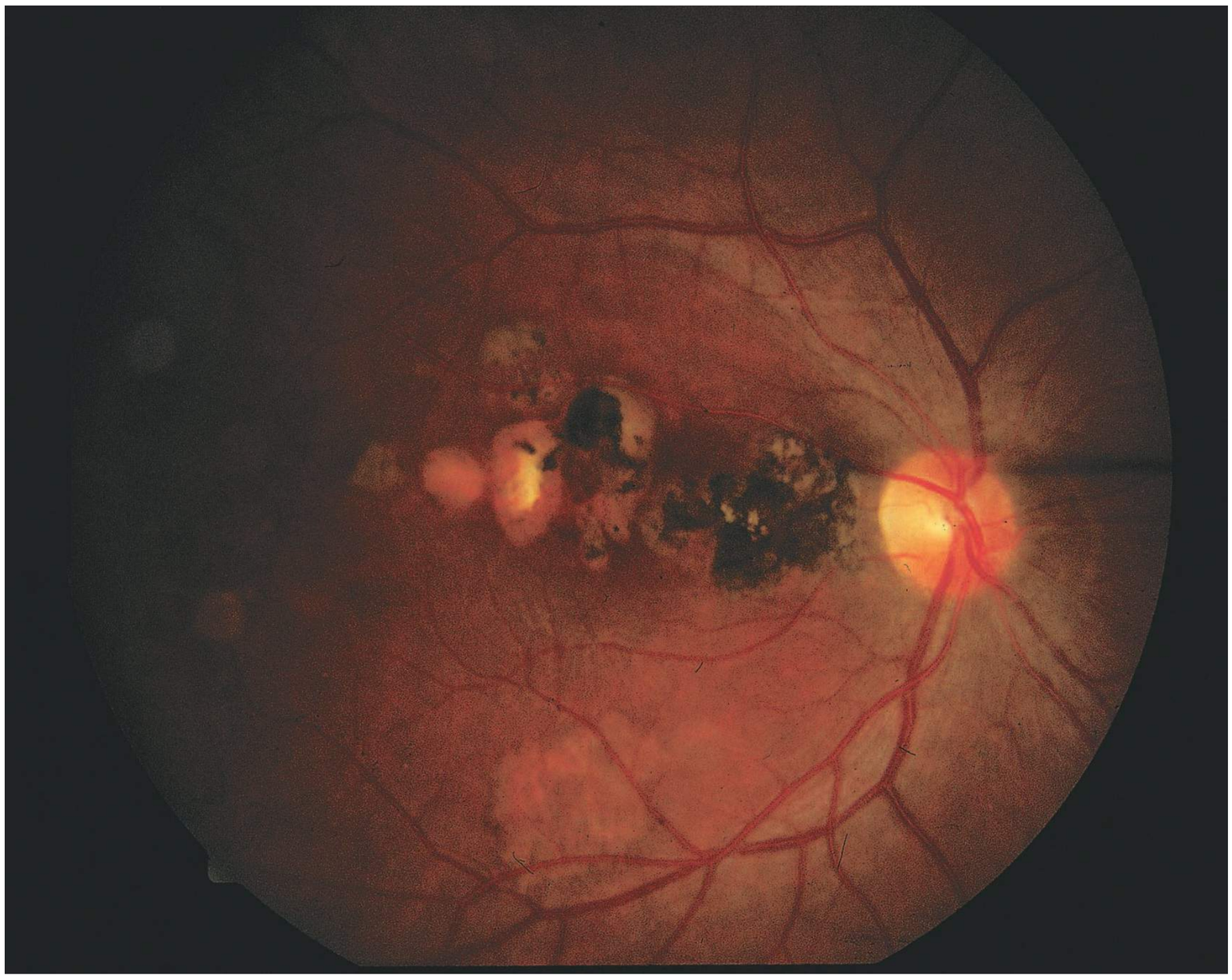


FIGURE 20.14 ■ **Toxoplasmosis Retinitis.** Ocular toxoplasmosis is a common complication of HIV disease. The lesion is a focal destructive chorioretinitis which leaves well-defined, heavily pigmented scars, especially in the macular area. (Photo contributor: Department of Ophthalmology, Naval Medical Center, San Diego, CA.)

Clinical Summary

Pneumocystis jirovecii (formerly carinii) pneumonia (PCP) is the most common opportunistic infection in HIV-infected patients. Clinical suspicion for PCP pneumonia in any HIV patient presenting with complaints of dyspnea and nonproductive cough should remain high. Presentations can be indolent, acute, or subacute, with associated symptoms including fever, fatigue, anorexia, weight loss, and chest pain. The complete blood count (CBC) is usually normal except for lymphopenia, while serum lactate dehydrogenase (LDH) is often elevated. Arterial blood gases (ABGs) most often reveal a respiratory alkalosis, Po_2 of 70 mm Hg or less, and an increased A-a gradient of 35 mm Hg or more. Radiographic chest findings are variable with diffuse interstitial alveolar infiltrates being common.

Management and Disposition

The initial treatment of PCP is often based on a presumptive diagnosis pending definitive results, usually a stained bronchial lavage sample obtained through bronchoscopy. It is essential that any HIV-infected patient who may have a CD4 count below 200 cells/mm^3 or CD4 cell percent less than 14 presenting with respiratory signs or symptoms be evaluated for PCP. Initial evaluation should include history of medications, especially adherence with prophylactic medications, clinical exam, chest x-ray, and room air ABG. Trimethoprim-sulfamethoxazole, orally or intravenously, is the first-line standard treatment. Clindamycin-primaquine, pentamidine, trimetrexate-leucovorin, atovaquone, and trimethoprim-dapsone are less effective alternatives. Treatment with corticosteroids should be initiated if the Po_2 is less than 70 or the A-a gradient is greater than 35. Any patient treated with steroids for presumptive PCP must have a definitive diagnosis made before discharge. Other pathogens that can cause pneumonia in HIV-infected patients may initially respond to steroid therapy.

Pearls

1. Include PCP in the differential diagnosis of any HIV patient who presents with a persistent fever or respiratory complaint.
2. Patients with significant dyspnea should have a room air ABG obtained. Oxygen saturation measures are not adequate to evaluate neither the oxygenation level nor the need for adjunct steroid therapy in a patient with suspected PCP.
3. The classic clinical presentation of PCP is a quiet patient with an increased respiratory rate, poor air movement, and a nonproductive cough on deep inspiration.
4. Over 30% of patients with PCP will have a normal chest x-ray. CT scanning of the chest is much more sensitive to reveal the classic interstitial ground glass pattern.

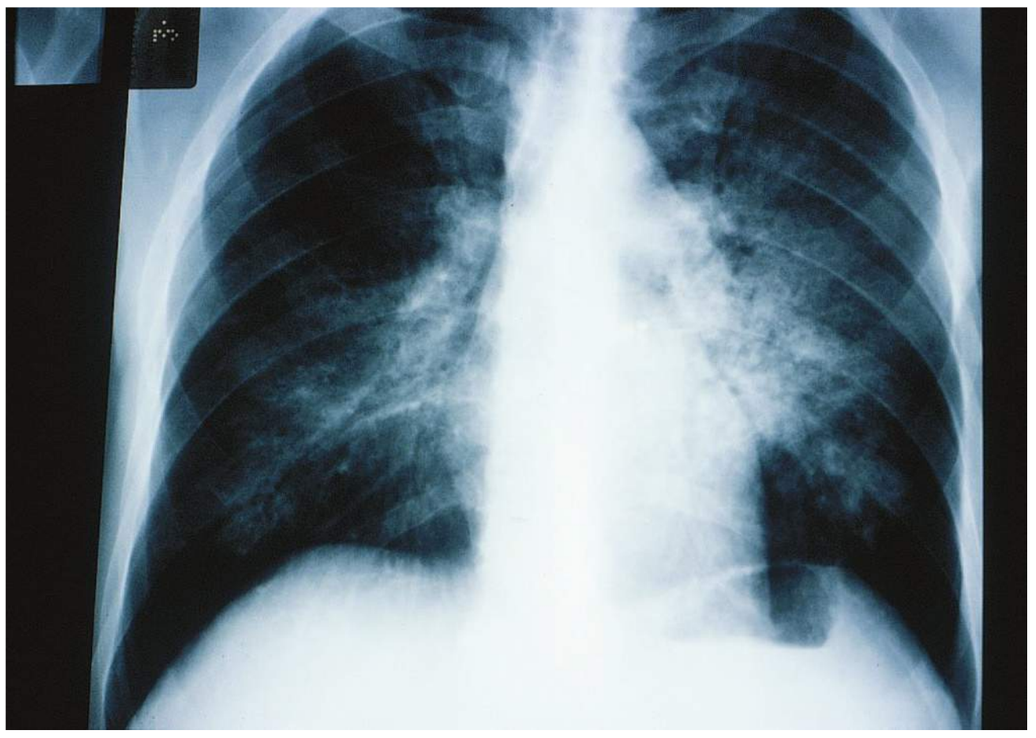


FIGURE 20.15 ■ Pneumocystis jirovecii **Pneumonia (PCP).** Chest radiograph showing diffuse interstitial alveolar infiltrates of PCP. (Photo contributor: Edward C. Oldfield III, MD.)

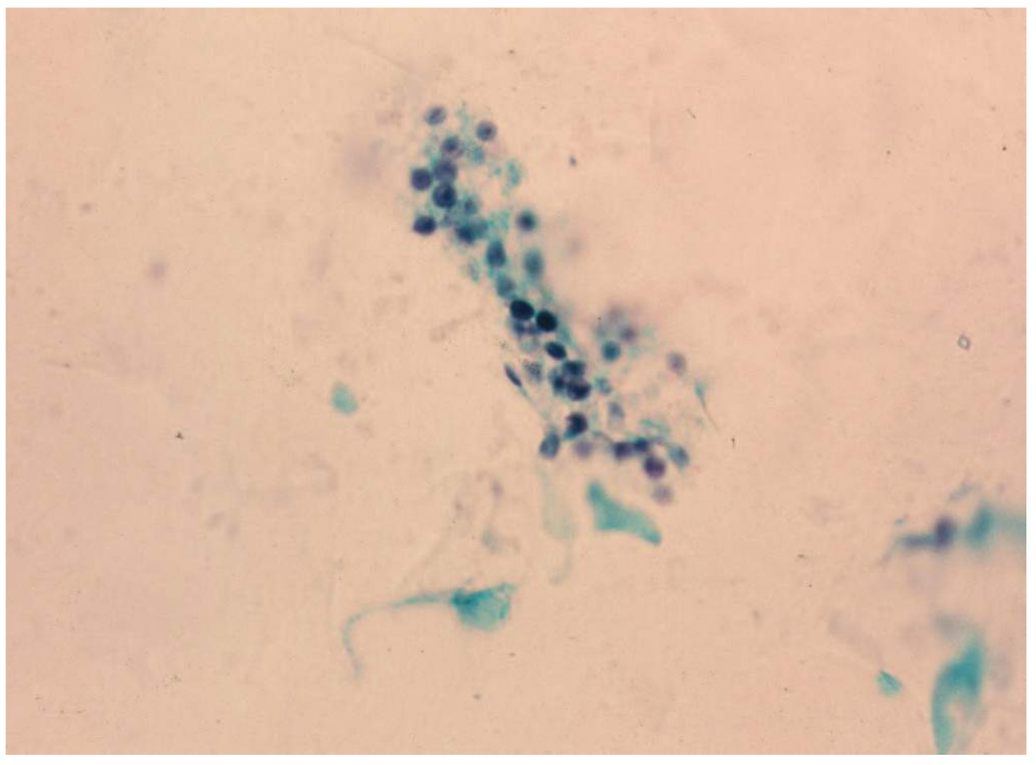


FIGURE 20.16 ■ **PCP on Silver Stain.** Pneumocystis jirovecii seen on methenamine-silver stain of a bronchoalveolar lavage sample from an HIV patient with PCP. (Photo contributor: Lawrence B. Stack, MD.)

CRYPTOCOCCAL INFECTIONS

Clinical Summary

Cryptococcus neoformans is the most common cause of meningitis in patients with AIDS. Cryptococcal meningoencephalitis typically manifests itself in patients whose CD4 cell counts are less than 50/μL. The onset tends to be insidious with fairly nonspecific symptoms such as fever, nausea, and headache. Symptoms may be present for several weeks. Seizures or focal neurological presentations are rare and neck stiffness and/or photophobia are usually absent. Diagnosis is usually made on examination of cerebrospinal fluid (CSF). Opening pressures may be quite elevated on LP, and CSF values usually reveal a normal CSF glucose concentration, a mildly elevated CSF protein concentration, and a CSF leukocyte count of less than 20/mL. India ink staining shows the organisms directly with an approximate sensitivity of 70%, while CSF cryptococcal latex antigen testing has a sensitivity approaching 90%. Fungal CSF cultures should also be sent. Cutaneous manifestations are seen in disseminated disease.

Management and Disposition

Treatment should begin for presumptive meningitis pending completion of CSF studies. Most often, the decision to treat for cryptococcal meningitis will be based on the results of CSF studies, along with consultation with infectious disease specialists. Cryptococcus can also cause pneumonia and skin lesions. Treatment regimens are usually based on amphotericin preparations followed by fluconazole.

Pearls

1. Perform the LP only after CT with contrast rules out space-occupying lesions, and do so with the patient in a lateral decubitus position so as to obtain an accurate opening pressure.
2. "False-positive" India ink stains can occur with other encapsulated organisms such as Klebsiella pneumoniae, Rhodotorula, Candida, and Proteus species.

FIGURE 20.17 ■ **Cryptococcal Infection.** Cryptococcal skin lesions in disseminated form. Note that the umbilicated centers give a similar appearance to that of molluscum contagiosum. (Photo contributor: Briana Hill, MD.)

FIGURE 20.18 ■ **Cryptococcal Infection.** Cutaneous cryptococcal lesions are often clinically difficult to distinguish from other cutaneous eruptions. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 20.19 ■ **Cryptococcal Infection.** This patient had extensive cutaneous involvement with disseminated cryptococcal infection. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

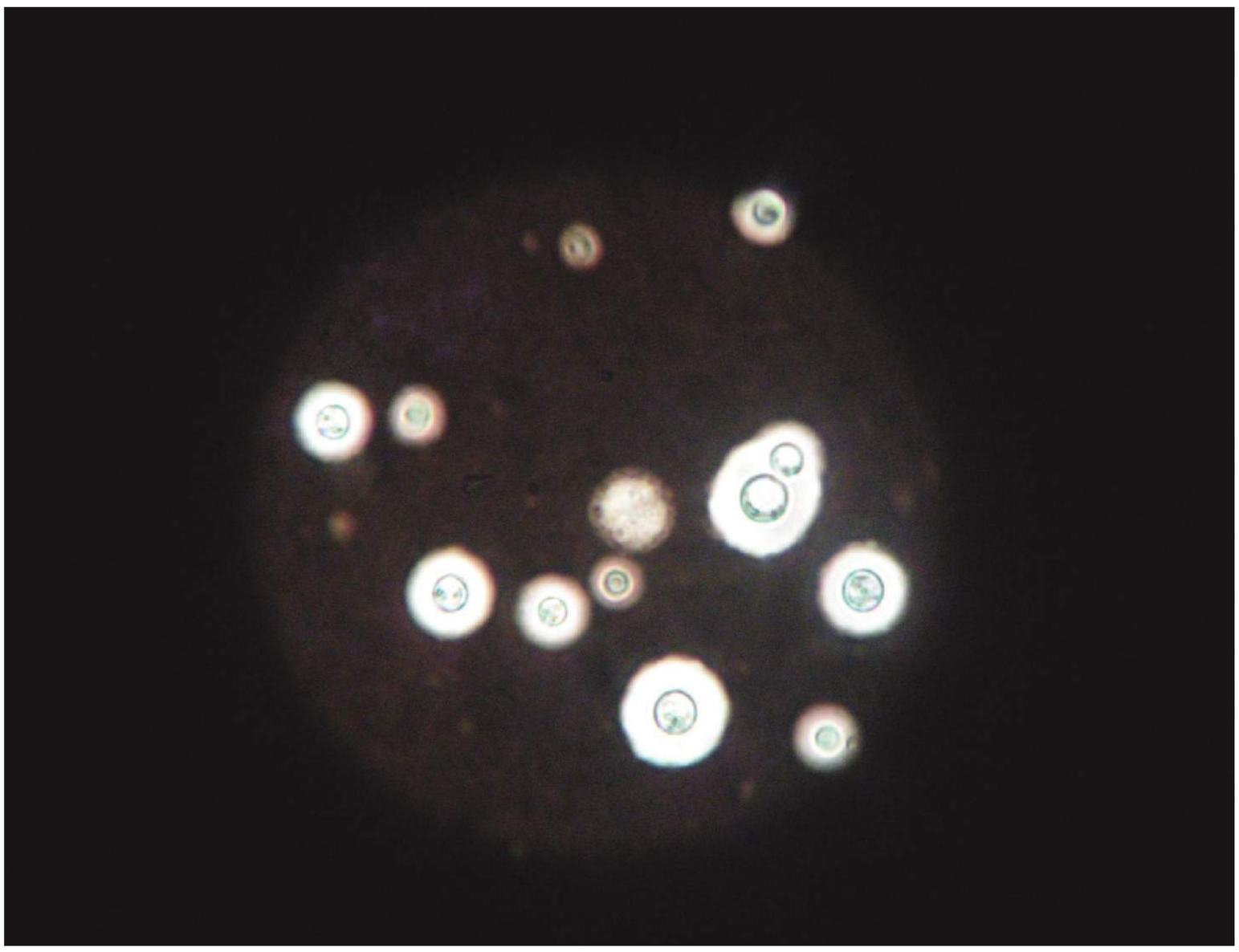


FIGURE 20.20 ■ **India Ink Preparation.** Cryptococcus neoformans seen as encapsulated yeast on India ink preparation of the CSF from an HIV patient with cryptococcal meningitis. (Photo contributor: Seth W. Wright, MD.)

HISTOPLAMOSIS

Clinical Summary

Disseminated histoplasmosis most commonly occurs in immunocompromised patients living in the Ohio and Mississippi River Valleys. HIV-infected patients usually have a CD4 count <200 cells/mm^3 and present with fever, weight loss, malaise, and pulmonary symptoms such as cough and dyspnea. Pulmonary symptoms predominate and in severe cases patients may present with acute respiratory distress syndrome. Patients may present with a diffuse papular rash in disseminated disease. Mediastinitis is a rare but serious complication of histoplasmosis. Urine and serum antigen testing carries a high sensitivity, but bronchoalveolar lavage may be needed to make the diagnosis. Serum LDH and alkaline phosphatase levels may be markedly elevated as well.

Management and Disposition

Disseminated histoplasmosis should be considered in any patient with moderate to advanced HIV infection and constitutional symptoms. Acutely ill patients with fever, weight loss, and functional decline should be admitted for evaluation treatment. Some patients with less severe disease may be treated with oral itraconazole but should be referred for evaluation and a definitive diagnosis.

Pearls

1. Consider disseminated histoplasmosis in any patient with moderate to advanced HIV infection and constitutional symptoms, rash, and appropriate geographic exposure.

FIGURE 20.21 ■ **Disseminated Histoplasmosis.** Skin lesions indicative of cutaneous involvement in an AIDS patient with disseminated histoplasmosis. (Photo contributor: Stephen P. Raffanti, MD.)

FIGURE 20.22 ■ **Pulmonary Histoplasmosis.** Chest x-ray demonstrating severe miliary histoplasmosis in a patient with AIDS. The patient presented with fever, cough, and respiratory distress. (Photo contributor: Jake Block, MD.)

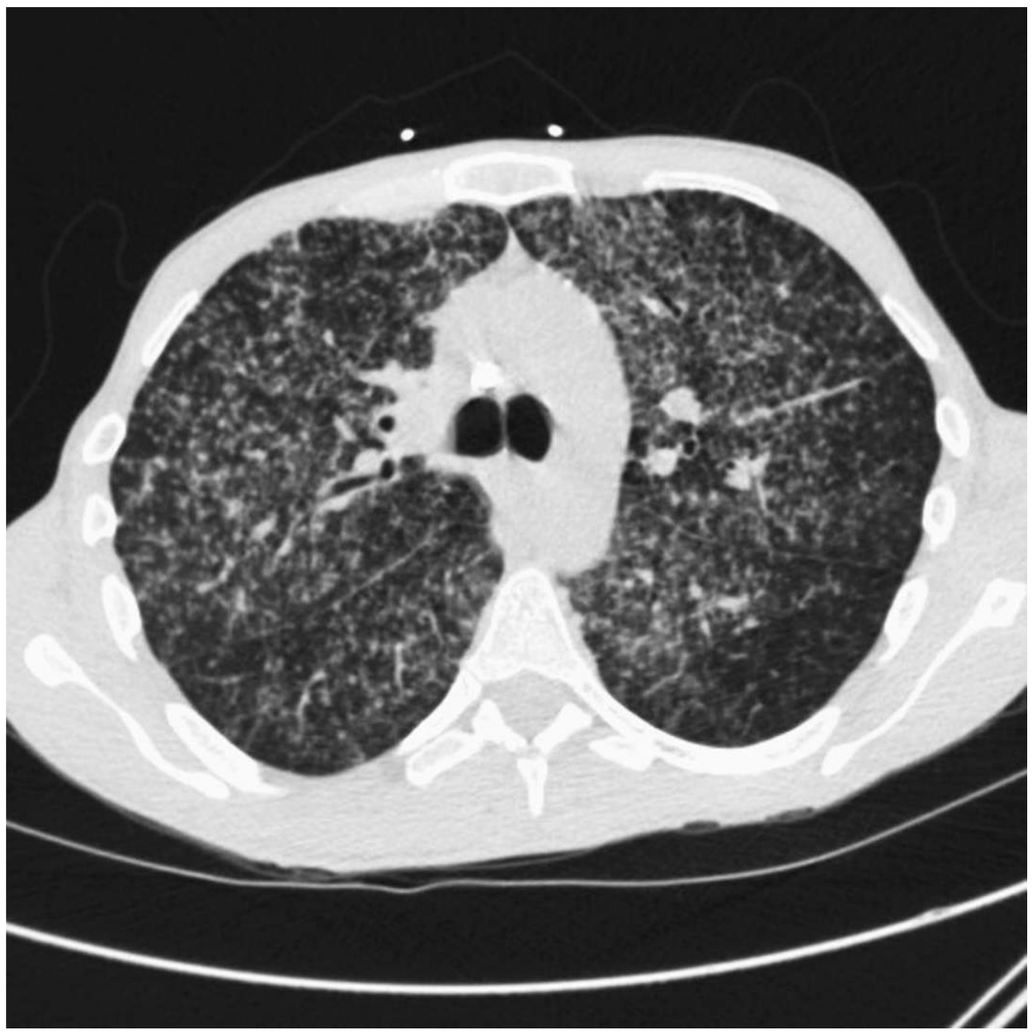


FIGURE 20.23 ■ **Pulmonary Histoplasmosis.** CT scan from the same patient in Fig. 20.22. (Photo contributor: Jake Block, MD.)

Clinical Summary

Cytomegalovirus (CMV) can be a cause of considerable morbidity in severely immunocompromised HIV-infected individuals. The major problems encountered are, in order of frequency, retinitis, colitis, esophageal ulceration, encephalitis, and pneumonitis.

CMV retinitis is the most common cause of blindness and eye disease in patients with AIDS. Patients typically present with loss of vision, floaters, scotoma, or visual field loss. Fundoscopic examination reveals exudates and hemorrhages, which follow the vasculature of the retina, giving it the typical "pizza pie" or "cottage cheese and ketchup" appearance.

CMV colitis is an uncommon but serious complication of HIV. The usual presenting features include generalized abdominal pain, diarrhea which may be bloody, and a low-grade fever. Loops of dilated large bowel may be seen on abdominal imaging, but definitive diagnosis is made with mucosal biopsy revealing characteristic "owls eye" inclusion bodies.

Management and Disposition

Treatment for CMV retinitis can be sight saving and should be started as soon as the diagnosis is considered. Emergent ophthalmologic consultation is needed. Treatment can be initiated with intravenous, intravitreal, or oral ganciclovir formulations. Relapse is common with CMV retinitis and if left untreated may progress to complete blindness.

Pearls

1. Ocular complaints in HIV patients require a comprehensive eye examination along with ophthalmology involvement if lesions are observed or complaints are significant.
2. Patients with CMV esophagitis often complain of odynophagia, but rarely complain of dysphagia, which may help clinically differentiate between CMV and candidal esophagitis.

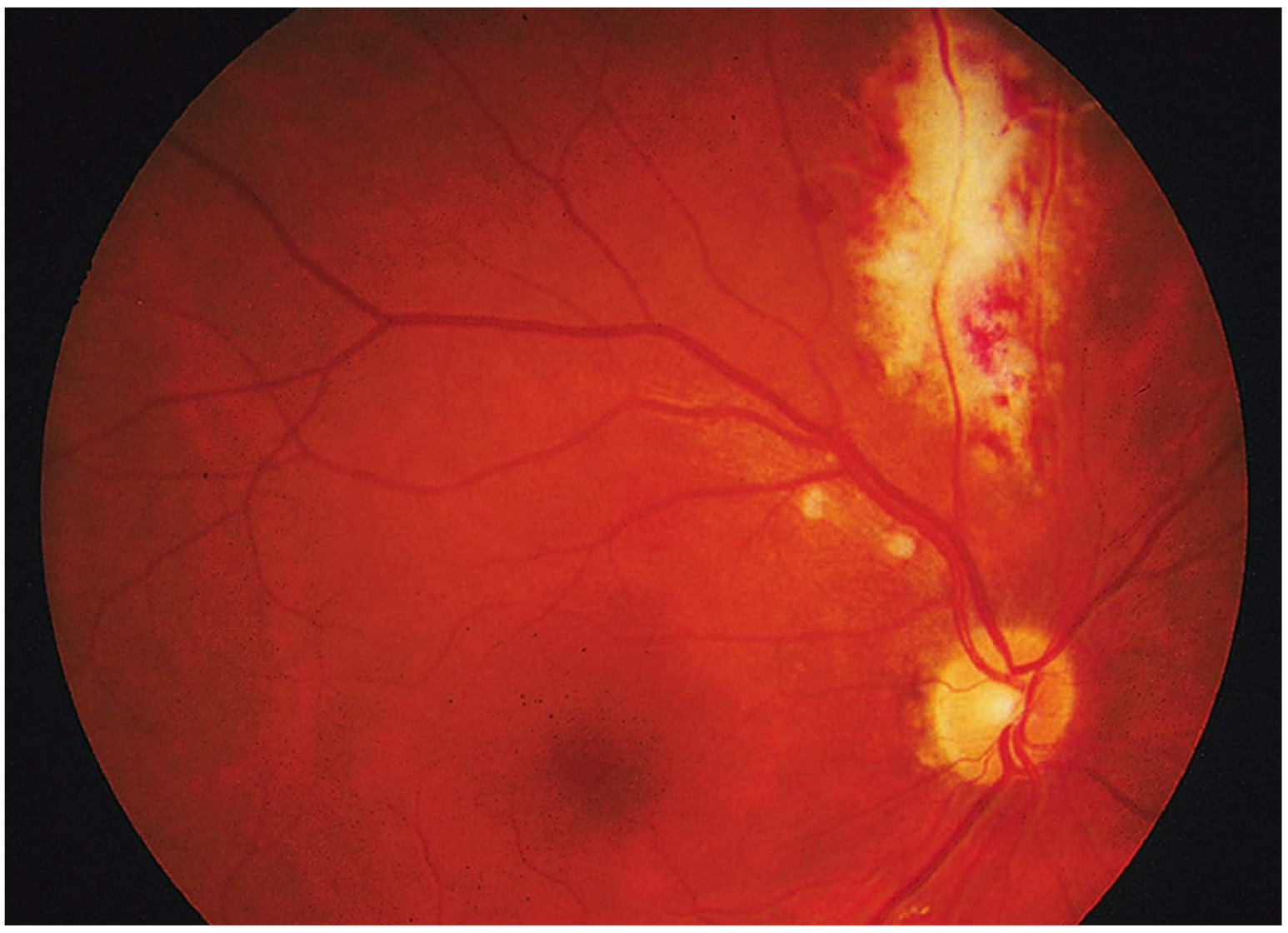


FIGURE 20.24 ■ **Cytomegalovirus Retinitis.** Funduscopic examination shows exudates and hemorrhages ("cottage cheese and ketchup" appearance) seen with CMV retinitis. (Photo contributor: Edward C. Oldfield III, MD.)

Clinical Summary

Varicella zoster virus (VZV) infection presents as shingles in HIV-infected patients at a greater frequency and severity than the general population. Despite the high incidence of shingles in this population, severe disseminated disease is rare.

The diagnosis of herpes zoster in the ED is often made clinically, by either recognizing the pain syndrome or visualizing the rash. Patients can present with a well-defined area of hyperesthesia for days prior to an outbreak of the characteristic lesions. Immunocompromised patients may demonstrate multidermatomal distribution or scattered vesicles at a distant site. Approximately 20% of patients will have systemic symptoms such as malaise, fever, headache, or fatigue. Culture, serologic testing, or PCR confirms the diagnosis. A Tzanck test can be obtained by scraping the base of a lesion in an attempt to demonstrate multinucleated giant cells.

Management and Disposition

Three antiviral drugs are used to treat herpes zoster infections: acyclovir, famciclovir, and valacyclovir. Patients with severe disseminated disease or ophthalmic zoster should receive intravenous acyclovir (10-12.5 mg/kg IV q 8 h for 10-14 days). Treatment of zoster ophthalmicus should include topical antibiotics and immediate ophthalmology consultation. Providing treatment of the disease within 72 hours of rash onset will result in a more rapid resolution of cutaneous lesions and decrease viral shedding, but will not change the incidence of postherpetic neuralgia. Immunocompromised patients should not be placed on glucocorticoids. Narcotics, capsaicin cream, and tricyclic antidepressants can be used for pain control.

Pearls

1. Worsening cases of herpes zoster, complicated herpes zoster, or ophthalmic zoster all require intravenous acyclovir and admission.
2. Herpes zoster encephalitis is rare, but can occur months after the cutaneous phase and can be difficult to diagnose. Common presenting symptoms include mental status changes, headache, fever, photophobia, vomiting, or even focal neurological deficits.
3. Patients presenting with localized hyperesthesia may have a zoster-related prodrome and treatment should be initiated if clinical suspicion is high.
4. The presence of vesicles on the tip of the nose (Hutchinson sign) indicates involvement of the nasociliary branch of cranial nerve V and is associated with a higher risk of ocular involvement.

FIGURE 20.25 ■ **HZV (Shingles).** A painful eruption of many tiny vesicles on an erythematous base in this patient with HIV. (Photo contributor: Jeffery Gibson, MD.)

FIGURE 20.26 ■ **HZV—Multiple Dermatomes.** Severe painful shingles (vesicles and bullae) spanning multiple cutaneous dermatomes in this HIV patient. (Photo contributor: John O'Mara, MD.)

FIGURE 20.27 ■ **Disseminated Herpes Zoster Infection.** Vesicles are seen over the entire face, representing disseminated HZV infection (multiple dermatomal distributions). (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

FIGURE 20.28 ■ **Ophthalmic Herpes Zoster.** This patient has vesicles in the ophthalmic division of cranial nerve V bilaterally. (Photo contributor: Daniel L. Savitt, MD.)

EOSINOPHILIC FOLLICULITIS

Clinical Summary

Eosinophilic folliculitis, commonly seen in advanced HIV patients, is a pruritic skin eruption usually involving the face, neck, trunk, and extremities manifesting in the form of pustules and papules. The skin lesions usually start as small pustular groups that later coalesce to create irregular erosions and plaques with central hyperpigmentation. The associated pruritus is often so intense that the lesions become excoriated. Most patients with this condition will demonstrate CD4 cell counts of less than 250/μL. The etiology of eosinophilic folliculitis is unknown, but is commonly thought of as an inflammatory process associated with immune dysfunction. It has become less common with the advent of HAART.

Management and Disposition

The diagnosis requires dermatology consultation and referral for biopsy. Antihistamines, potent topical steroids, itraconazole, topical permethrin, retinoids, metronidazole, dapsone, and ultraviolet B phototherapy have all shown variable levels of efficacy.

Pearls

1. The severe pruritus associated with this condition helps distinguish it from bacterial folliculitis. Symptomatic treatment for pruritus is necessary, and the disease generally improves with effective antiretroviral therapy.
2. Dermatological referral is warranted prior to initiating definitive therapy.
3. Patients with eosinophilic folliculitis may present with prurigo nodularis and lichen simplex chronicus as a result of severe itching and rubbing.
4. Secondary syphilis should be considered in all HIV-infected patients presenting with rash.

FIGURE 20.29 ■ **Eosinophilic Folliculitis.** The rash of eosinophilic folliculitis consists of small groups of pustules and vesicles, as seen on the face of this patient. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

FIGURE 20.30 ■ **Eosinophilic Folliculitis.** A magnified view showing pustules at the base of each hair follicle. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

Clinical Summary

Infection with herpes simplex virus (HSV) is extremely common in HIV-infected patients and may present with oral, genital, anal, esophageal, or ophthalmologic involvement. The hallmark for most clinical presentations of HSV outbreaks is an inflammatory cutaneous eruption, with or without vesicles and pain. HSV esophagitis is seen in immunocompromised HIV-infected patients and presents as dysphagia and odynophagia with or without oral lesions. Idiopathic apthous ulcerations in HIV-infected patients are indistinguishable from HSV lesions. Perirectal lesions are often erythematous, ulcerative, and extremely tender, with a predilection for the gluteal cleft. Perirectal HSV may also be associated with proctitis and anal fissures.

Ocular HSV may be demonstrated by observing dendritic lesions after fluorescein staining. HSV is associated with a syndrome of acute retinal necrosis characterized by pain, keratitis, and iritis that may lead to retinal detachment. The diagnosis is usually made clinically but can be confirmed by a Tzanck test, biopsy, or culture.

Management and Disposition

Multiple oral treatment regimens are available to treat HSV. Duration of treatment is usually increased for immunocompromised patients. Ocular HSV requires prompt ophthalmology consultation.

Pearls

1. Anticipate involvement of multiple sites in HIV patients; presentations of HSV may be atypical compared to HSV in immunocompetent individuals.
2. Suspect HSV in any HIV patient with a chronic painful ulcerative lesion.
3. Perirectal HSV outbreaks may be missed on a superficial history and physical examination. Ask if it is painful to wipe after stool and examine the perianal area. Do not assume hemorrhoids in HIV-infected patients at risk for HSV.

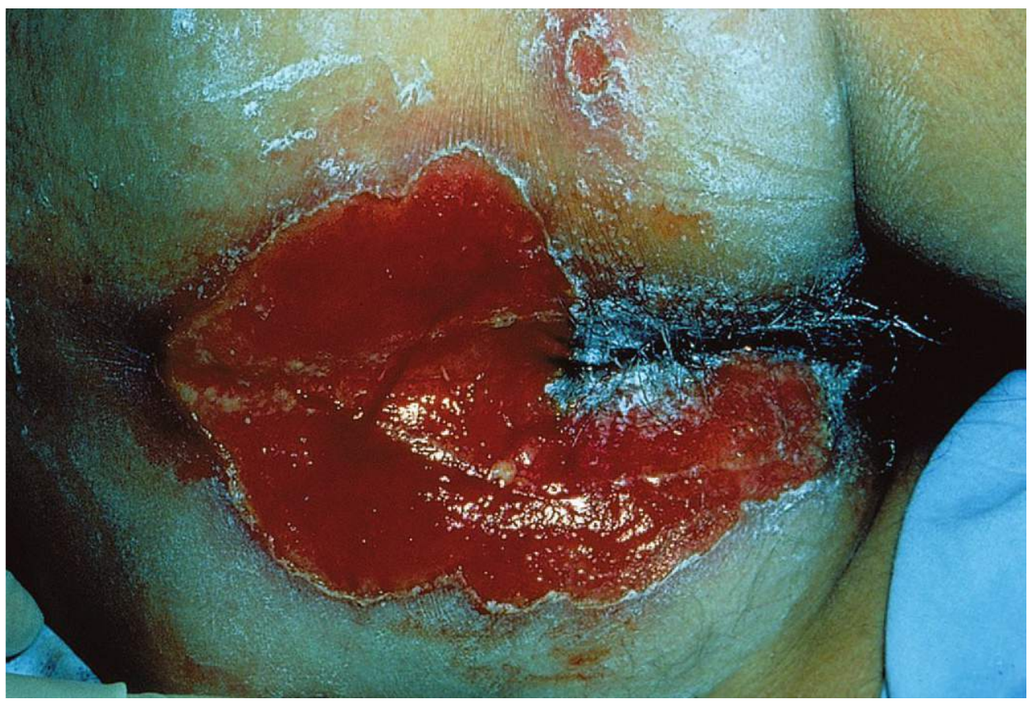


FIGURE 20.31 ■ **Herpes Simplex Virus in an HIV Patient.** Severe, recurrent perirectal HSV lesions in an HIV patient. (Photo contributor: Briana Hill, MD.)

FIGURE 20.32 ■ **Severe HSV on Genitalia.** Severe ulcerative HSV lesions on the penis and scrotum of an HIV patient. (Photo contributor: John O'Mara, MD.)

Clinical Summary

Human scabies, caused by the Sarcoptes scabiei mite, is one of the most common contagious dermatoses. In HIV patients, this organism can cause "crusted scabies," also known as Norwegian scabies, which denotes an overwhelming scabies infestation. In typical scabies, the mites cause extremely pruritic burrows, vesicles, and papules in a characteristic distribution involving the finger webs, sides of the hands and feet, breasts, waist, and groin. Pruritus is most intense at night. In contrast, crusted scabies typically affects the hands and the feet with asymptomatic crusting and does not cause significant pruritus. Transmission through infected linens or clothing is common in cases of Norwegian scabies. Risk factors include poor hygiene, crowding, and exposure to pets.

Most often the diagnosis of scabies is made clinically, with evidence of burrows and severe pruritus in a characteristic distribution. Definitive diagnosis is made from examination of shavings from the lesions. Placing mineral oil over a suspected lesion and then shaving it with a number 15 blade can demonstrate the mites, which are usually 0.3 to 0.4 mm in length.

Management and Disposition

Topical permethrin 5% cream has a low toxicity and is the treatment of choice for scabies. Patients are instructed to apply the cream from the neck down and leave it on for 8 to 14 hours before removal. One application is usually sufficient for treatment in nonimmunocompromised patients. The patient's clothes and bedding should be washed in hot water. Antihistamines should be prescribed to alleviate the pruritus. Often the pruritus persists after treatment as a hypersensitivity reaction to the mites and their feces and eggs. Any complicating secondary skin infection should be treated with appropriate systemic antibiotics. Localized infections can be treated with topical steroids.

For HIV patients with Norwegian scabies, permethrin should be applied to the face, scalp, behind the ears, and from the neck downward. Repeat treatments may be needed. For severe or refractory cases, oral ivermectin (200 mg/kg for one dose) can be tried with the exception of pregnant or lactating women.

FIGURE 20.33 ■ **Scabies.** Typical scabies rash showing unroofed papules secondary to scratching. Several small burrows are also seen. (Photo contributor: George Turiansky, MD.)

FIGURE 20.34 ■ **Norwegian Scabies.** Overwhelming scabies infestation causing "crusted scabies" in this patient with HIV. Hyperinfestations involve thousands to millions of mites. (Photo contributor: Francisco Bravo Puccio, MD, with permission from The Gorgas Course in Clinical Tropical Medicine, Universidad Peruana Cayetano Heredia, Lima, Peru.)

Pearls

1. Often secondary staphylococcal infections complicate the diagnosis, including impetigo, ecthyma, paronychia and furunculosis.
2. Oral antibiotics are often indicated for Norwegian scabies because of skin breakdown.
3. Close contacts of infected patients should be treated simultaneously. Inform patients that although the scabicide will kill the mites, itching may last for weeks. Patients often seek repeat treatments and inappropriately receive additional scabicides, which can cause contact dermatitis.

FIGURE 20.35 ■ **Norwegian Scabies.** Crusted lesions with hyperkeratotic scales on this patient's foot. Millions of mites were seen under microscopy in scrapings of the lesions. (Photo contributor: Larry Mellick, MD.)

MOLLUSCUM CONTAGIOSUM

Clinical Summary

Molluscum contagiosum is caused by a poxvirus that presents flesh-colored, dome-shaped lesions on affected individuals. Usually, molluscum contagiosum is an asymptomatic benign disease, but may become severe in HIV patients. Typical presentations consist of groups of 2 to 20 small, discrete, shiny lesions with central umbilication.

Management and Disposition

If the diagnosis is suspected in the ED, the patient should be reassured and referred to a dermatologist. Usually self-limited with spontaneous remission, treatment outside of genital lesions is for cosmetic reasons only.

Pearl

1. Clinically, it may be difficult to distinguish between cutaneous Cryptococcus and molluscum contagiosum. Dermatology or infectious disease consultation and biopsy may be required if the patient is clinically ill.

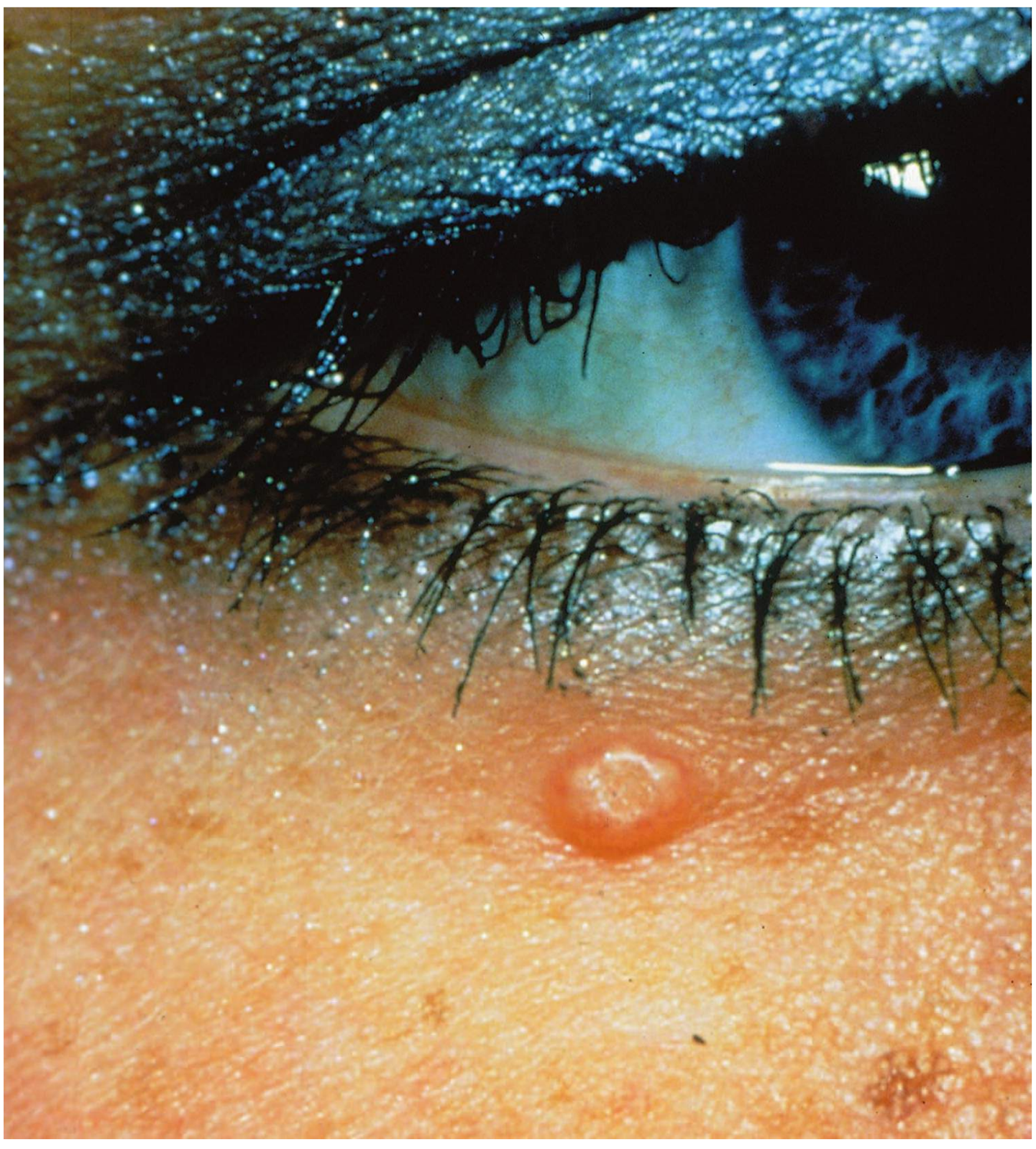


FIGURE 20.36 ■ **Molluscum Contagiosum.** Facial rash with ocular involvement is a common site of infection in HIV patients. Note the central umbilication. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

Clinical Summary

Dermatophyte infections are common superficial fungal infections involving the scalp (tinea capitis), body (tinea corporis), feet (tinea pedis), crural fold (tinea cruris), and nails (onychomycosis). Infection is very common in healthy individuals, but can be varied and atypical in patients with HIV. HIV patients also can demonstrate a much more severe and extensive form of disease. Three different dermatophytes are the usual source of infection: epidermophyton, trichophyton, and microsporum.

Tinea capitis is seen as an enlarging scaling patch on the scalp that may progress to a kerion. The initial lesion can be overlooked until alopecia is present. Untreated permanent scarring can occur. Tinea pedis is the most common dermatophyte infection seen in practice. Usually self-limited, intermittent, recurrent, and intensely pruritic, these lesions can often be secondarily infected due to mechanical irritation. Tinea corporis is the most common fungal infection seen in HIV patients. Usually, it begins as an intensely pruritic, oval scaling erythematous lesion that spreads centrifugally and can merge with other lesions. Tinea cruris is an infection involving the crural fold, much more common in men than women. It usually begins as a macular patch on the inner thigh, opposite the scrotal sac.

Management and Disposition

Tinea capitis responds to griseofulvin; however, other oral agents that can be effective include terbinafine, itraconazole, or fluconazole. Treatment usually is carried out for at least 3 weeks, with topical treatments ineffective. Tinea pedis can usually be treated with a topical antifungal cream for 4 weeks. Often patients have tried outpatient, over-the-counter antifungal medications without success. Extensive treatment can be initiated with griseofulvin, terbinafine, or itraconazole orally. Tinea corporis often responds to daily application of topical antifungal medications; however, systemic oral agents described above are also effective. Tinea cruris usually responds to topical antifungal medications.

Pearls

1. Confirming the diagnosis with a KOH preparation is important before initiating therapy, especially in diffuse disease. Often HIV patients suffer from a variety of skin infections that mimic dermatophyte lesions (eczema).
2. Athletes are especially at risk when close skin-to-skin contact occurs (wrestling, football). Oral treatment in these cases is preferred, with restriction in sports for 2 weeks or until lesions heal.
3. Some dermatophyte infections fluoresce under the Wood lamp examination.

FIGURE 20.37 ■ **Tinea Infection.** Tinea infection involving the scalp, neck, and upper back of this HIV-infected patient. (Photo contributor: Seth W. Wright, MD.)

FIGURE 20.38 ■ **Tinea Corporis.** Tinea infection involving the lower back and buttocks of the patient in Fig. 20.37. (Photo contributor: Seth W. Wright, MD.)

THROMBOCYTOPENIA

Clinical Summary

Thrombocytopenia occurs in 40% to 70% of all HIV patients. It can occur independently at all stages of HIV infection and may be encountered as the initial presentation of disease. HIV-associated anemia and granulocytopenia can occur concomitantly as course of the HIV infection worsens, with thrombocytopenia seen in 30% of patients with CD4 counts less than 200/μL. HIV patients with thrombocytopenia often present to the ED with bleeding (especially from the oral mucosa), ecchymosis, and petechiae. Secondary causes of thrombocytopenia are generally seen as the result of opportunistic infections, malignancy, or medications.

Management and Disposition

The emergency physician's efforts are initially focused on stabilization of the patient with two large intravenous lines, type and cross-match, and crystalloid infusion if significant bleeding has occurred. Because of the complexity of the differential diagnosis and potentially complicated treatment of HIV thrombocytopenia, an infectious disease specialist should be consulted early. In most cases, HIV patients with platelet count greater than 50,000 can be managed conservatively with spontaneous remission of approximately 20%. Zidovudine can increase platelet counts up to twofold in over 50% of patients. If the platelet count is less than 20,000, many infectious disease specialists recommend γ-globulin infusion and parenteral steroids. Other possible treatments include dapsone, danazol, interferon-α vincristine, anti-D immunoglobulin, splenic irradiation, and splenectomy. Even if the patient is to be managed conservatively, bone marrow analysis should be performed to rule out other causes of thrombocytopenia.

Pearls

1. Perform thorough skin and oral examinations in all patients with HIV looking for manifestations of thrombocytopenia.
2. Take a careful drug history and consider other infectious etiologies before assuming the thrombocytopenia is directly secondary to HIV infection.
3. Spontaneous bleeding is rare unless the platelet count is less than 10,000.

FIGURE 20.39 ■ **Thrombocytopenia.** Ecchymosis in an HIV patient with thrombocytopenia. (Photo contributor: Edward C. Oldfield III, MD.)

FIGURE 20.40 ■ **HIV-Associated Thrombocytopenia.** A large spontaneous subconjunctival hemorrhage is noted in this patient with HIV and severe thrombocytopenia. (Photo contributor: John O'Mara, MD.)

Clinical Summary

HIV patients have a 5 to 20 times higher rate of drug reactions than non-HIV patients. Up to 5% of ED visits by HIV patients are due to complications of therapy. Many of these manifest dermatologically, in order of decreasing frequency: (1) exanthems, (2) urticaria/angioedema, (3) fixed drug reactions, (4) erythema multiforme, and (5) photosensitivity reactions. The most common medications associated with rashes are antivirals, antibiotics, and antifungals.

Management and Disposition

The emergency physician may need to consult an infectious disease specialist, a pharmacist, or a dermatologist to help clarify the existence of a drug reaction. Clues besides recent initiation of a new drug are eosinophilia greater than 1000 or elevated liver function tests. Individual treatment varies depending on the situation. The offending agent should be discontinued. Antihistamines and steroids are indicated in certain situations.

Pearls

1. Ask about alternative and nonprescription medicines.
2. Half of all HIV patients will react adversely to sulfa drugs.
3. Beware of serious drug reactions such as Stevens-Johnson syndrome and toxic epidermal necrolysis (TEN).

FIGURE 20.42 ■ **Efavirenz Rash.** Drug exanthem typical of efavirenz. (Photo contributor: Stephen Burdette, MD.)

FIGURE 20.43 ■ **Efavirenz Rash.** Close-up image of drug exanthem caused by efavirenz in an HIV patient. (Photo contributor: Stephen Burdette, MD).

FIGURE 20.41 ■ **Drug Reaction.** Exanthematous drug reaction in an HIV patient. (Photo contributor: Kenneth Skahan, MD.)

FIGURE 20.44 ■ **Nevirapine Rash.** Drug exanthema caused by nevirapine in this HIV patient. (Photo contributor: Seth W. Wright, MD.)

ACUTE NECROTIZING ULCERATIVE GINGIVITIS

Clinical Summary

Acute necrotizing ulcerative gingivitis (ANUG), also known as Vincent angina or trench mouth, is commonly seen in HIV patients. It is a distinct and rapidly progressive ulceration typically starting at the tip of the interdental papilla, spreading along the gingival margins, eventually destroying the periodontal tissue. The triad associated with ANUG is oral pain, halitosis, and ulcerations. Other signs and symptoms include "metallic taste," "wooden teeth" sensation, tooth mobility, fever, adenopathy, and malnutrition. The cause of this aggressive, destructive process in HIV patients is a polymicrobial infection by oral anaerobes (Treponema, Selenomonas, Fusobacterium, Porphyromonas, Prevotella). ANUG represents a spectrum of disease from mild ulcerations to severe cellulitis and spread of the infection to the soft tissues, cheeks, lips, and bones.

Management and Disposition

ANUG is most frequently seen in four population groups: (1) HIV patients, (2) malnourished children, (3) young adults who are under a great deal of stress, and (4) polysubstance abusers. The first steps for the emergency physician are to eliminate other potentially more serious life-threatening infections and to address hydration status. Treatment includes (1) eliminating contributing factors (stress, poor nutrition, poor sleep, alcohol, and tobacco use), (2) chlorhexidine rinses twice a day, (3) surgical debridement by an oral surgeon if needed, and (4) oral penicillin and metronidazole.

Pearls

1. Beware of Ludwig angina (brawny submandibular induration and tongue elevation) associated with severe progression of ANUG as acute airway compromise is possible.
2. Noma (cancrum oris, gangrenous stomatitis), a rare disease of childhood associated with malnutrition, is characterized by an anaerobic destructive infectious process of the orofacial tissues that can clinically resemble ANUG.

FIGURE 20.45 ■ **Acute Necrotizing Ulcerative Gingivitis (ANUG).** ANUG (Vincent angina or "trench mouth") caused by spirochetal and fusiform bacteria in an HIV patient. Note the punched-out ulcerations of the interdental papillae, which are pathognomonic. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

Chapter 21

TROPICAL MEDICINE

Seth W. Wright
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Scan QR code or go to www.ematlas.com for video related to chapter

Uganda. (Photo contributor: Seth W. Wright, MD.)

The authors acknowledge Meg Jack for portions of this chapter written for the third edition.

FREE-LIVING AMEBA INFECTION

Clinical Summary

Free-living amebas, usually harmless protozoan residents of soil and water, can cause three distinct, occasionally devastating, human illnesses. Primary amebic meningoencephalitis (PAM) is a disease of the previously healthy and is caused by Naegleria fowleri. Granulomatous amebic encephalitis (GAE) is caused by Acanthamoeba species or Balamuthia mandrillaris, and occurs in both healthy and immunocompromised persons. In wealthier countries, contact lens users may suffer from chronic amebic keratitis, also caused by Acanthamoeba. While these diseases are found worldwide, they are more common in tropical and subtropical regions.

PAM is devastating, usually fatal, and found mostly in children or young adults with a history of recent freshwater exposure. The organism enters through the nose and penetrates the cribriform plate to the subarachnoid space and brain. The acute illness is indistinguishable from bacterial meningitis. Patients with GAE often present with an initial focus of infection in the skin or respiratory tract followed by neurologic changes reflective of extensive brain involvement.

Management and Disposition

The mainstay of management is consideration of these uncommon diseases. PAM is almost always fatal, but one survivor was successfully treated with amphotericin B, miconazole, and rifampin. Isolated cutaneous disease from Acanthamoeba and B mandrillaris can be cured, but brain involvement is fatal and often diagnosed at autopsy. Patients with suspected amebic keratitis should have immediate ophthalmologic referral.

Pearls

1. Lack of response to usual antimicrobials in a patient with severe meningitis symptoms should lead to the suspicion of PAM, particularly with recent freshwater exposure.
2. Global warming might increase the rate of N fowleri infection as the organism thrives in freshwater over 30°C.
3. Fatal cases of PAM have been reported following tap water nasal irrigation using a "neti pot" (a device used to clean nasal passages). Use of sterile or previously boiled water when irrigating eliminates the risk.
4. Consider Acanthamoeba infection in all contact lens wearers with a corneal infection. Early amebic keratitis can mimic the dendritic pattern of herpes simplex infection.
5. Immunocompromised patients with space-occupying lesions should have GAE on the differential.

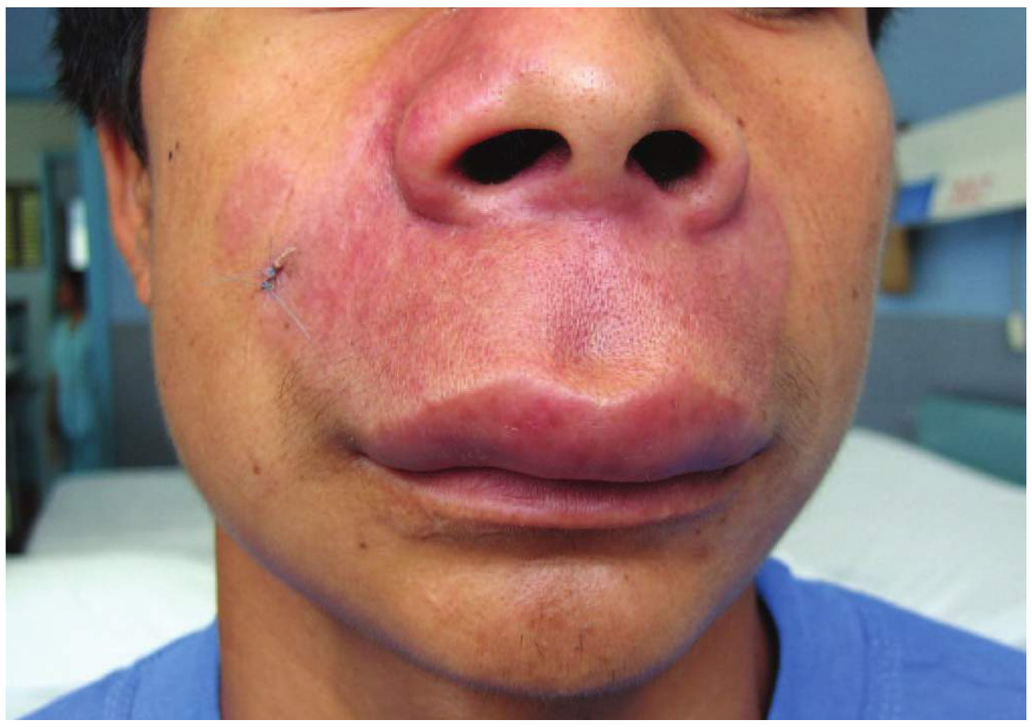

FIGURE 21.1 ■ **Ameba.** Twenty-one-year-old patient from South America with 1 year of symptoms from Balamuthia mandrillaris. The primary site often involves the mid-face and oral cavity. This patient did not have intracranial involvement. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

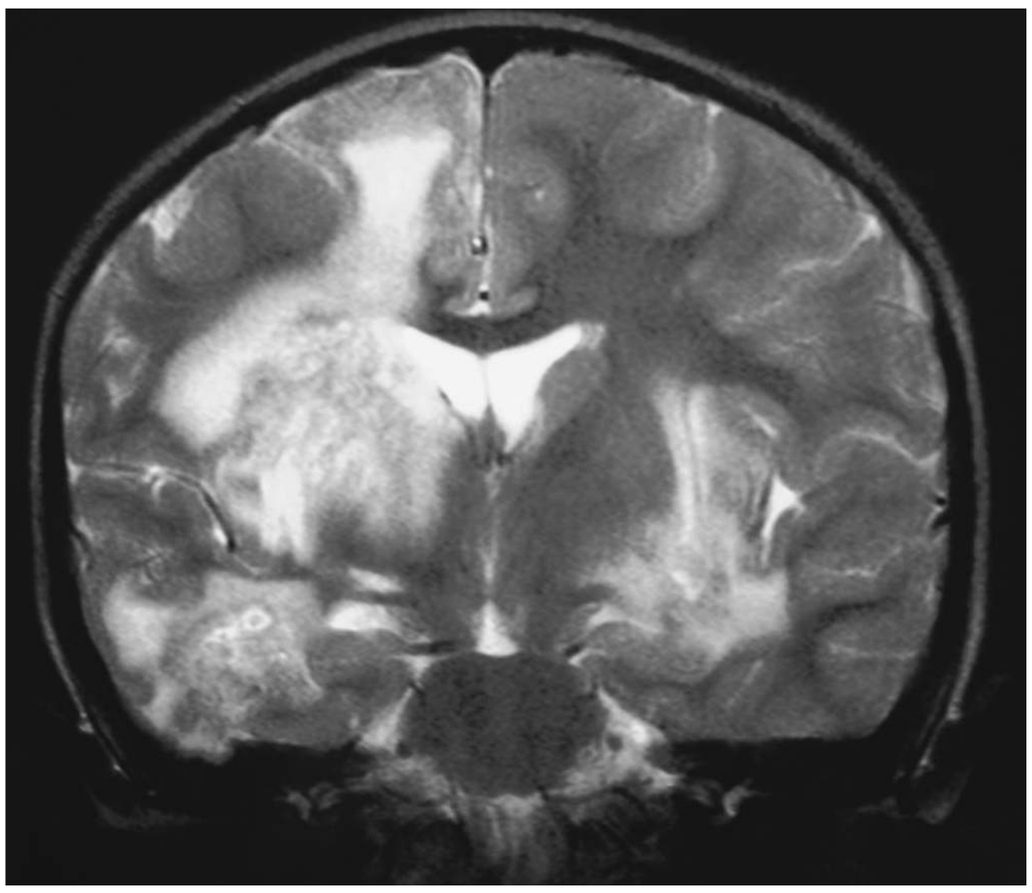

FIGURE 21.2 ■ **Ameba Imaging.** MRI showing extensive cerebral involvement in a fatal case of Balamuthia mandrillaris. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Clinical Summary

Anemia in the tropics is a common medical condition resulting from various nutritional deficiencies, infections, parasites, and inherited disorders. Its prevalence and causes differ significantly from developed countries, and is often multifactorial.

Overall, iron deficiency is the most common cause of anemia worldwide. Similar to deficiencies of folic acid and vitamin B_{12}, it results from diets rich in carbohydrates, but poor in meats, proteins, and vegetables. Most patients with AIDS in developing countries are anemic, with more severe cases seen in those coinfected with tuberculosis. Malaria causes hemolysis and hypersplenism, and should always be considered in anemic patients. Hookworm infection is another common cause of anemia, particularly in children and pregnant women. Other parasitic illnesses causing anemia include visceral leishmaniasis (kala-azar) and African trypanosomiasis (sleeping sickness). Hemoglobinopathies, including sickle cell anemia and thalassemia, are common in Africa, the Middle East, the Americas, and South Asia. Severe hemolytic anemia can occur in patients with G6PD deficiency.

Management and Disposition

Treatment depends on the suspected cause(s). Iron and folate supplementation can lead to a rapid and dramatic increase in red cell counts in patients with nutritional deficiencies. A low threshold for malaria diagnosis and treatment is important. Empiric treatment for hookworm is advisable. Red blood cell transfusion is rarely needed in stable patients, even those with extremely low counts.

Pearls

1. Administration of the antimalarial agent primaquine to patients with G6PD deficiency can lead to fatal hemolysis.
2. Thrombocytopenia in conjunction with anemia is highly suggestive of malaria in endemic areas.
3. Anemia in pregnancy should be screened for and aggressively treated as this improves both maternal and fetal outcomes.

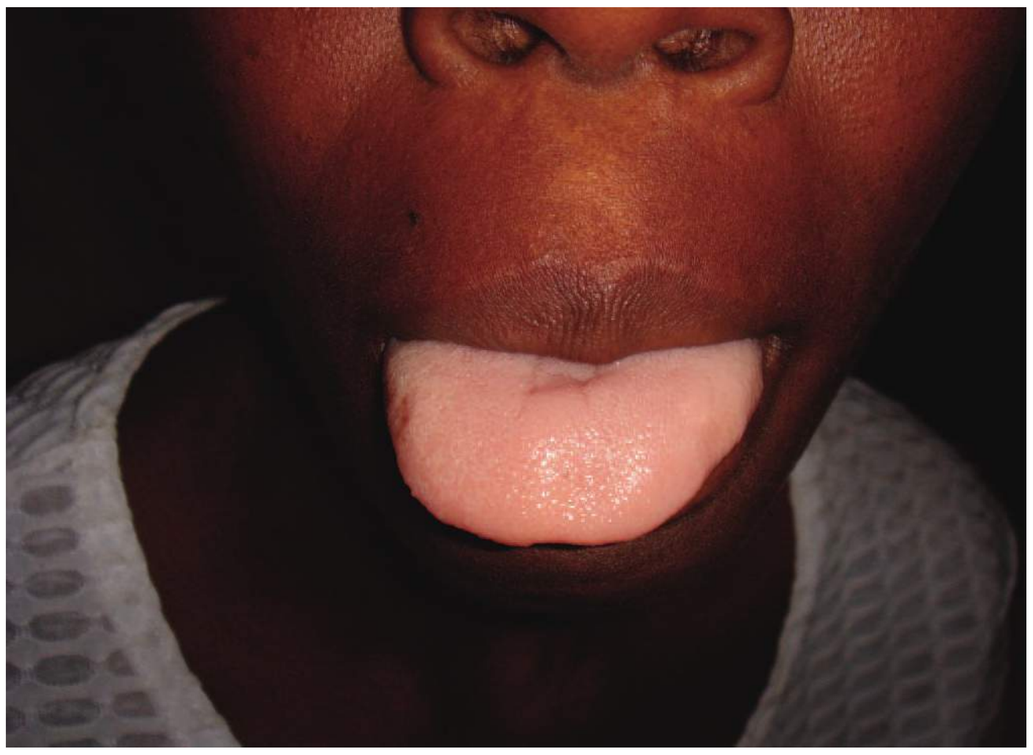

FIGURE 21.4 ■ **Anemia.** Pale tongue in a severely anemic Haitian woman. Pale mucus membranes, palms, and soles are often good indicators of anemia, particularly in dark-skinned individuals. (Photo contributor: Andreas Fischer, RN.)

FIGURE 21.3 ■ **Anemia.** Conjunctival pallor in a Haitian woman with complaints of weakness and a Hgb of 2.2. (Photo contributor: Andreas Fischer, RN.)

FIGURE 21.5 ■ **Anemia in AIDS.** Gums of Ugandan patient with end-stage AIDS and Hgb of 3.0. (Photo contributor: Seth W. Wright, MD.)

ANTHRAX

Clinical Summary

Anthrax, a worldwide zoonotic infection caused by Bacillus anthracis, was the first definitively recognized bacterial pathogen in human history. Most cases are sporadic, although occasional large outbreaks have been reported in Africa. Control of the disease in livestock has made endemic anthrax uncommon in developed countries, but it has taken on increased importance due to its use as a bioterrorism agent.

Cutaneous anthrax is the most common form, accounting for 95%. Infection occurs after spore exposure, with the initial manifestation of a small, painless, pruritic papule. The papule develops a large, central vesicle, followed by the classic necrotic ulcer with a black, depressed eschar. Local edema, regional lymphadenopathy, and systemic symptoms can occur. About 20% will progress to systemic bacteremia. Inhalational anthrax occurs after aerosolized spore exposure, and typically follows a biphasic course: (1) nonspecific upper respiratory infection–like symptoms and (2) then, in 1 to 4 days, fulminant bacteremia with fever, respiratory distress, hemorrhagic mediastinitis, and shock. Gastrointestinal (GI) anthrax is rare, affects the oropharynx or alimentary tract, and occurs after contaminated meat ingestion. It causes a nonspecific gastroenteritis, necrotic GI tract ulcers, and may progress to shock or death 2 to 5 days after symptom onset.

Management and Disposition

Uncomplicated endemic cutaneous anthrax is typically treated with oral or intramuscular penicillin. Doxycycline, erythromycin, and ciprofloxacin are alternatives. High-dose intravenous (IV) penicillin is used in cutaneous disease with systemic findings, and for inhalational or GI disease.

Patients with suspected bioterrorism-related anthrax require hospital admission with immediate public health notification. They should be decontaminated with removal (and sealed storage) of all clothing and cleansing with soap and water. The Centers for Disease Control and Prevention (CDC) guidelines recommend ciprofloxacin or doxycycline as first-line therapy. Inhalational and other severe forms are treated with additional multidrug antimicrobial therapy. Glucocorticoid therapy is controversial, but might be beneficial in meningitis or cutaneous disease with extensive head and neck edema.

Pearls

1. All forms can progress to hemorrhagic meningitis, which can mimic a traumatic tap on lumbar puncture.
2. Cutaneous and inhalational anthrax have been reported from use of imported animal hide drums.
3. Injection anthrax is rare and recently found among heroin users in Great Britain and Germany. Symptoms include abscess, cellulitis, necrotizing fasciitis, and sepsis.
4. The length of antibiotic therapy is usually 7 to 10 days, but postexposure prophylaxis involves 60 days, including vaccine administration.
5. The classic pathologic finding of inhalational anthrax is hemorrhagic mediastinitis, demonstrated as a widened mediastinum on chest radiography.

FIGURE 21.6 ■ **Anthrax.** A black eschar with a central hemorrhagic ulceration on the thumb associated with massive edema on the hand. (Used with permission from Wolff K, Johnson RA, Suurmond D. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 5th ed. New York, NY: McGraw-Hill; 2005: p. 631.)

FIGURE 21.7 ■ **Anthrax Progression.** (**A**) Initial facial cutaneous anthrax with early lesion development and massive facial edema. (**B**) Interval development of septicemia. The lesion is developing a more typical dark coloration. (**C**) The patient following resolution of sepsis. (Photo contributor: Seth W. Wright, MD.)

ASCARIASIS

Clinical Summary

Ascaris lumbricoides is the most common human intestinal roundworm infection. The parasites are found worldwide and are highly endemic where sanitation and hygiene are poor; they commonly cause infection in regions where human feces are used as fertilizer. Adult worms live in the small intestine and produce enormous numbers of eggs, which are excreted in the feces. Passed eggs require at least several weeks in warm, moist soil before embryonating into an infective egg. Ingested eggs hatch in the jejunum, migrate through the intestinal wall into the bloodstream, and are transported to the lungs. Larval worms burrow through the alveolar walls, ascend up through the trachea, and are swallowed back into the small intestine where they develop into adults.

Most infections are asymptomatic. Patients may present for care if they pass a worm in their stool. A heavy worm burden may lead to abdominal pain, pancreatic/biliary disease, or intestinal obstruction. Failure to thrive and decreased cognitive development are seen in heavily infected children. Migrating worms may cause biliary obstruction, appendicitis, or liver abscesses.

Diagnosis is from identification of a passed worm or examination of stool for eggs.

Management and Disposition

Outpatient treatment with mebendazole or albendazole is usually effective but only on worms in the adult intestinal stage. Thus, it is recommended that a stool examination be done at 2 to 3 months and retreatment initiated if positive for eggs.

Pearls

1. Marked peripheral eosinophilia can be seen during the migratory stage.
2. While in the lungs, migrating worms can cause an eosinophilic pneumonitis with asthma-like symptoms, or "Löffler syndrome."
3. Ascaris worms occasionally migrate from the anus, mouth, or nose following antihelmintic treatment.
4. Ascaris is the most common cause of acute abdominal surgical emergencies in some highly endemic countries.

FIGURE 21.8 ■ **Ascariasis.** Worms spontaneously extruding from the anus of a boy with a high fever. Worms often migrate in patients with fever. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.9 ■ **Ascariasis.** Adult ascaris worm after passage from the anus. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.10 ■ **Ascariasis.** Distended abdomen of a Haitian girl with a large ascaris worm burden. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

American trypanosomiasis, or Chagas disease, is a zoonotic protozoal infection caused by Trypanosoma cruzi. Chagas is spread by triatomine insects with a large variety of mammals serving as natural reservoirs. Disease is limited to the western hemisphere and is most common in areas of substandard housing as the vector insects proliferate in adobe, unfinished brick walls, and in thatched roofs. Chagas is considered the most important protozoal disease in the Americas, surpassing malaria in morbidity and mortality. It is estimated that 10 million people are infected, including 300,000 immigrants within the United States.

Chagas is usually acquired during childhood and persists for life. Infection occurs when the insect defecates while taking a blood meal and the infectious stool is rubbed into the bite wound or a mucus membrane (often the conjunctiva). Acutely, infection is often asymptomatic but can present with a skin lesion at the infection site (chagoma), fever, adenopathy, myocarditis, and hepatosplenomegaly. The organism can be isolated during the indeterminate phase (years or even lifespan). Chronically, about 30% develop a cardiomyopathy with progressive biventricular heart failure. Severe arrhythmias, including complete heart block, are common. Approximately 10% of patients will develop digestive complications due to loss of lumen wall neurons, including megaesophagus and megacolon.

During acute infection, trypanosomes are seen on the blood smear and polymerase chain reaction (PCR) is useful for diagnosis, but serology is typically negative. Serological assays are used to diagnose chronic disease. Blood culture and xenodiagnosis can also be used, but lack sensitivity.

Management and Disposition

Patients presenting with acute symptoms are treated with benznidazole or nifurtimox. Patients with chronic disease should be worked up with an electrocardiogram (ECG) and echocardiography, and referred as indicated. Antitrypanosomal treatment is warranted in the indeterminate phase of chronic infection and in those with early stage cardiomyopathies.

Pearls

1. Chagas is the most common cause of cardiomyopathy in many Latin American countries. The disease should also be considered in patients with complete heart block or other arrhythmias.
2. Chagas is surprisingly common in wild and domestic mammals in the Southern and Southwest United States. The prevalence in Tennessee dogs is 6.4%, and is higher in wild animals such as raccoons, skunks, and rodents.
3. Human infection in the United States is rare since the vector insects are uncommon inside modern housing, but occasional cases have been reported in those without a history of travel.
4. Universal screening of blood for Chagas is now routine in the United States.

FIGURE 21.11 ■ **Chagas Disease.** A child with unilateral periorbital swelling from local inflammation at the site of a Chagas organism inoculation (Romana's sign). (Photo contributor: Jorge Kleisinger, MD, and Ana Rosa de Benedetto).

Clinical Summary

Cholera is a severe diarrheal disease caused by Vibrio cholerae, a gram-negative bacterium, and is spread via the fecal-oral route. It is associated with poor hygiene and overcrowding, as well as the resultant contamination of food and water. Endemic disease is present in many areas of the world with occasional epidemics. Massive outbreaks in previously disease-free areas can occur following disasters or breakdowns in public health measures, such as seen in Haiti following the 2010 earthquake.

Cholera is characterized by massive, watery, gray, and painless diarrhea. The stool resembles "rice water" without blood or pus. Patients may have associated vomiting. Renal failure, hypotension, and circulatory collapse can occur within hours of diarrhea onset. Mortality rates as high as 20% to 50% among severe cases can be seen if adequate rehydration is not available. Death rates of less than 2% are seen with good case management.

FIGURE 21.12 ■ **Cholera.** Severely dehydrated child during an outbreak in Uganda. Lethargy and sunken eyes are typical findings. IV fluids are typically reserved for severe cases. The child is on a typical cholera cot. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.13 ■ **Dehydration in Cholera.** "Tenting" of the skin of a dehydrated cholera patient. The presence of tenting implies moderate to severe dehydration. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

The mainstay of treatment is hydration. Oral rehydration solutions are adequate for mild and moderate dehydration. Patients with severe dehydration are treated with IV lactated Ringer solution and oral rehydration. Per World Health Organization (WHO) guidelines, adult patients may require 10 to 15 L in the first 24 hours. Monitoring of electrolytes and renal function is ideal, but often not available in areas struck by cholera epidemics. Hypokalemia is common. Antibiotics are not required for recovery as the illness is self-limiting, but doxycycline may reduce the volume of diarrhea and shorten the duration of illness.

Pearls

1. Cholera, plague, and yellow fever are the three diseases internationally notifiable to WHO.
2. John Snow's removal of the Broad Street water pump handle during the 1854 cholera epidemic in London is considered to be the beginning of modern field epidemiology.
3. Rapid cholera test kits are available and are essential for early confirmation of disease in the first suspected cases.
4. An oral cholera vaccine is available, but only confers short-term immunity and is not available in the United States.

FIGURE 21.14 ■ **Rice Water Stool.** Typical "rice water" stool from cholera patient. Patients with cholera often lose a liter or more of watery stool an hour. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.15 ■ **Cholera Cot.** Patient on cholera cot. Use of the cholera cot allows for monitoring of fluid output and allows for easier waste control in patients too weak to leave the bed. (Photo contributor: Seth W. Wright, MD.)

CUTANEOUS LARVA MIGRANS

Clinical Summary

Cutaneous larva migrans (CLM), also known as "creeping eruption," is the most common dermatologic problem following a trip to the tropics. CLM is a worldwide, parasitic infection commonly seen in warm, tropical environments; it is uncommon in developed countries due to shoe wearing habits and routine deworming of pets.

CLM is most commonly caused by dog and cat hookworms, Ancylostoma caninum and Ancylostoma braziliense; humans are an accidental host. The infected animal passes eggs in its feces where they hatch, molt, and feed on soil bacteria. Upon contact with a human host, the larval worm penetrates the skin and attempts dermal migration. The worm remains under the skin since it lacks collagenase and cannot penetrate deeper layers. The larvae are unable to complete their life cycle, and, if left untreated, are trapped in the epidermis and will die in 2 to 8 weeks.

CLM is commonly seen on the lower extremities of travelers who walk barefoot on beaches with contaminated sandy soil. Symptoms are manifested by an erythematous tract with a distinctive serpiginous dermal pattern. The tract is markedly pruritic and may feel like a thread on palpation.

Management and Disposition

Treatment of CLM consists of mebendazole, albendazole, ivermectin, or topical application of thiabendazole. Antipruritics may also help.

Pearls

1. The lesions advance a few millimeters to several centimeters daily as the larva migrates.
2. Excoriation and secondary infections frequently occur. This can make the distinctive wandering rash more difficult to visualize.
3. Biopsy is usually not helpful for diagnosis since the organism lies 1 to 2 cm away from the leading edge of the eruption.

FIGURE 21.16 ■ **Cutaneous Larva Migrans.** A serpiginous, linear, raise, tunnel-like erythematous lesion outlining the path of migration in the larva. Upon palpation, it feels like a thread within the superficial layers of the skin. (Photo contributor: Janet Rohde.)

FIGURE 21.17 ■ **Cutaneous Larva Migrans.** CLM with secondary infection on the foot of a toddler from Zambia. (Photo contributor: Meg Jack, MD.)

Clinical Summary

The larval form of the pork tapeworm Taenia solium causes cysticercosis and affects 50 million people worldwide. Disease in developed countries is usually due to immigration or foreign travel. Taeniasis (intestinal tapeworm) is acquired from ingesting cysts from undercooked pork. Eggs passed in the stool from these carriers are highly infectious and may survive in the environment for months. Humans acquire cysticercosis from the ingestion of these eggs/larva, usually through unhygienic food preparation. Once the eggs are consumed, they hatch, penetrate the bowel wall, and travel to the subcutaneous tissue, skeletal muscle, and brain, though they may involve any organ. Two distinct types of diseases exist: neurocysticercosis (which can be parenchymal or extraparenchymal) and extraneural cysticercosis. Symptoms are dependent on the affected organ system; significant morbidity is associated with ocular, cardiac, and neurologic involvement. The larval worm will eventually die and leave calcified lesions.

Intestinal taeniasis is diagnosed through egg identification on stool exam or by passage of an intact worm or worm segment. Computed tomography (CT) and magnetic resonance imaging (MRI) have facilitated recognition of neurocysticercosis with visualization of a contrast-enhancing ring lesion. Diagnosis may also be made by biopsy, serum, or cerebrospinal fluid (CSF) antibody testing. Neurocysticercosis should be strongly considered in endemic regions or in immigrants with new onset seizures.

Management and Disposition

Seizures are treated with standard medications. Calcified lesions do not require specific anticysticercal therapy. Viable cysts can be treated with albendazole or praziquantel, but should be done with expert consultation. Inpatient management is advisable if viable cysts are to be treated as inflammatory reactions may occur. Corticosteroids are recommended in this situation. Surgical intervention may be indicated for obstructing neurologic lesions or intraocular lesions.

Pearls

1. Infection is most common in rural areas of developing countries where pigs are allowed to roam freely and ingest infected human feces. This completes the cycle and propagates infection.
2. Cysticercosis and taeniasis are rare in predominantly Muslim countries where eating pork is uncommon; however, cysticercosis is still possible due to ingestion of contaminated nonpork food products.

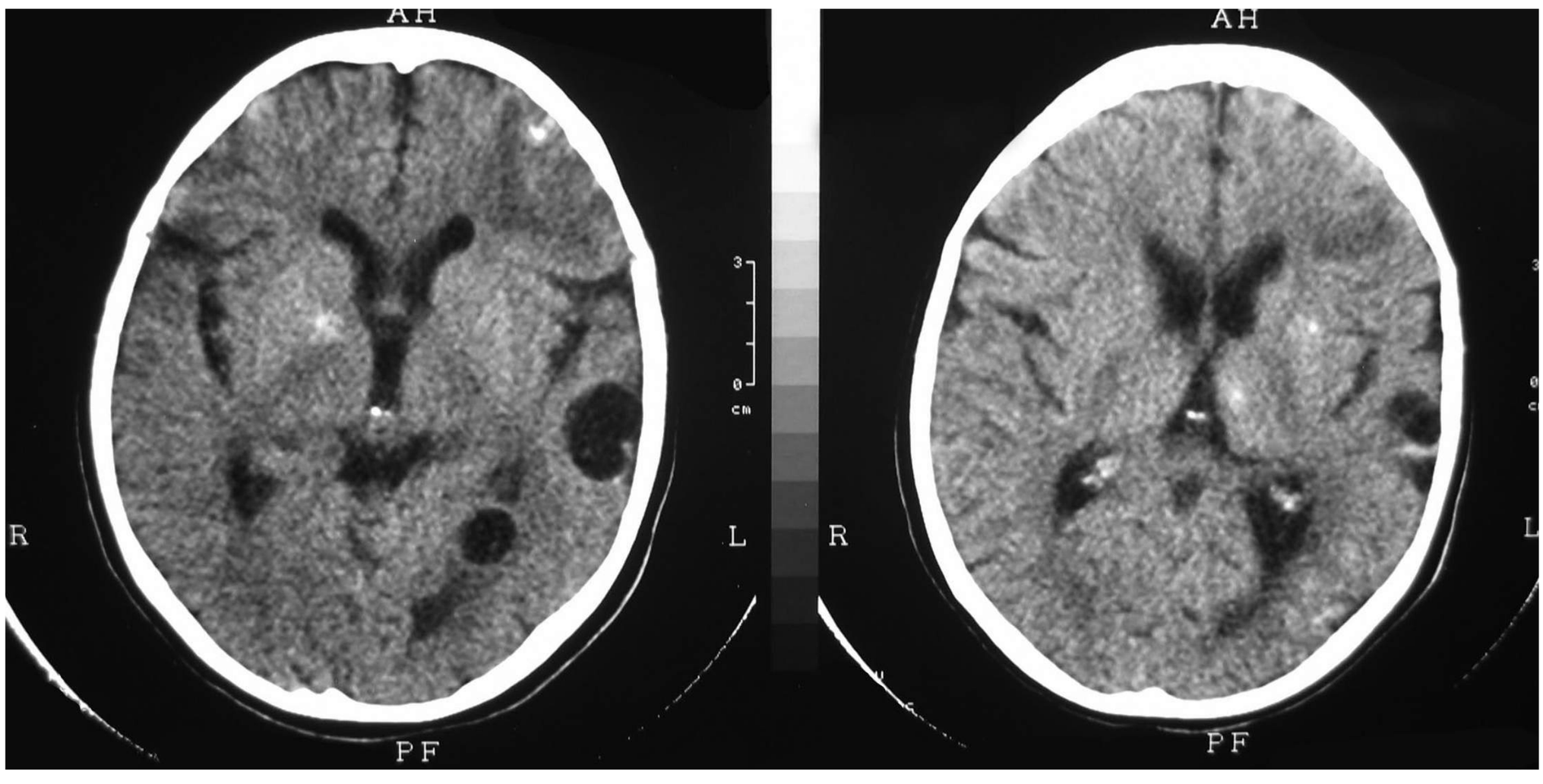

FIGURE 21.18 ■ **Neurocysticercosis.** Noncontrast head CT showing new (cystic) and old (calcified) lesions of neurocysticercosis. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

3. The adult tapeworm can attain a length of 20 feet or more, and can live up to 20 years in the intestine.
4. Most people with an intestinal pork tapeworm do not have cysticercosis.
5. The only pathognomonic CT or MRI finding of neurocysticercosis is a 2- to 4-mm bright nodule within the cyst ("cyst with dot sign") that represents a solid larval tapeworm.

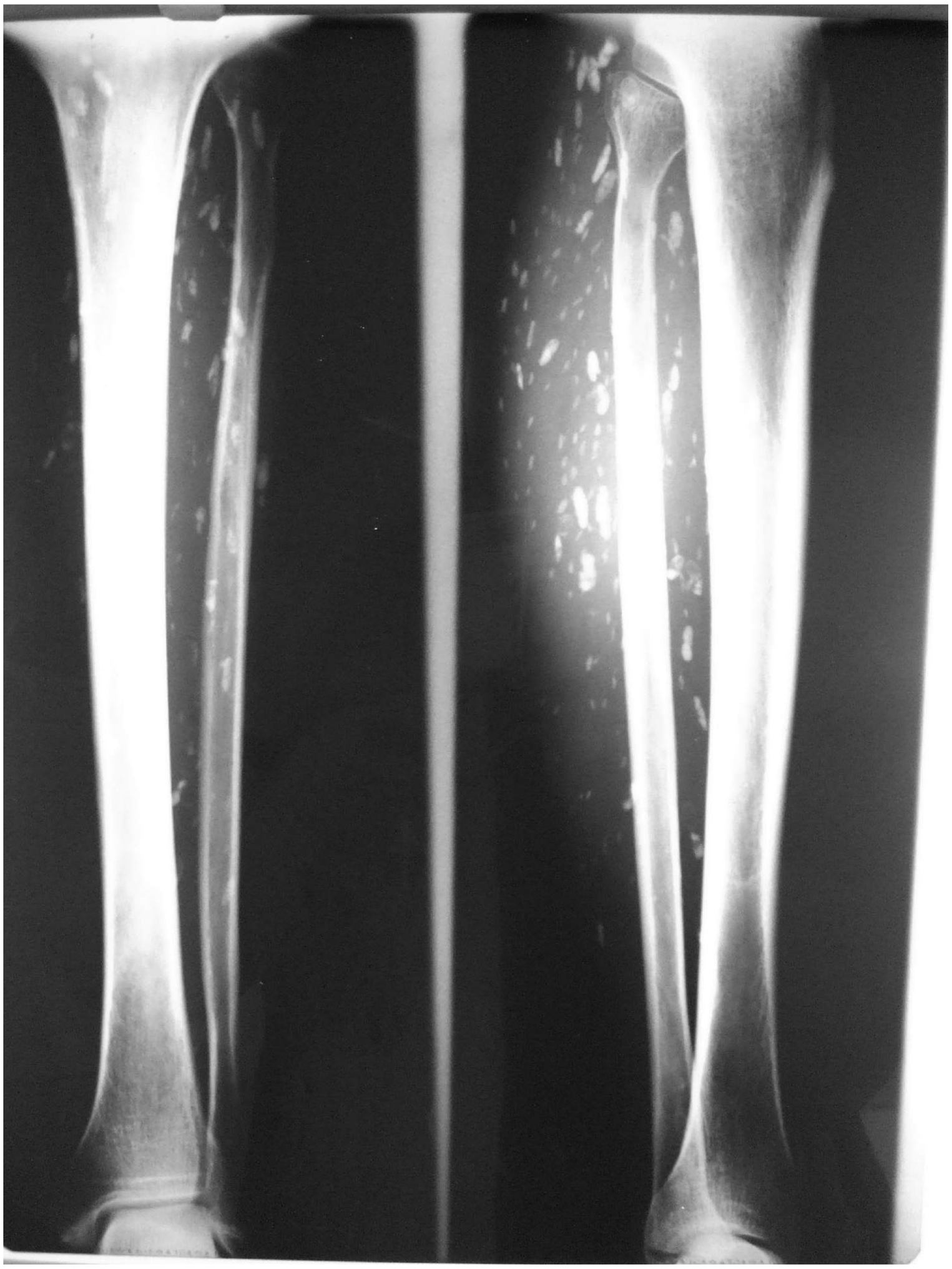

FIGURE 21.19 ■ **Cysticercosis Cysts.** Multiple calcified soft tissue cysts noted as an incidental finding on a lower leg radiograph in a patient with disseminated cysticercosis. The "cigar-shaped" calcifications are highly suggestive of cysticercosis. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Clinical Summary

Dengue fever, also known as "breakbone fever," is a mosquito-borne viral disease. A flavivirus, dengue is a rapidly emerging illness due to reintroduction of Aedes species mosquitoes into areas of previous eradication. It is now distributed throughout tropical and subtropical regions, and widespread epidemics have occurred in the Caribbean and Southeast Asia, among other world regions. Dengue is one of the most common causes of fever in returning travelers.

The incubation period is usually 3 to 7 days, with symptom resolution within 10 days. Infection ranges from subclinical symptoms to a hemorrhagic state with possible shock. Most commonly, it is characterized by fever, severe myalgias, retro-orbital pain, and headache. A rash, seen in approximately half, is usually maculopapular, but may be mottled, flushed, or petechial. The recently revised classification of dengue severity consists of dengue without warning signs, dengue with warning signs, and severe dengue based upon the degree of plasma leakage, spontaneous bleeding, organ impairment, and shock.

There are four serotypes; exposure to one serotype provides lifelong immunity to that type. Previous infection with one serotype may predispose an individual to a more severe infection with another serotype. Thus, severe dengue is more common in indigenous individuals than the previously unexposed traveler. The diagnosis is primarily clinical. The presence of thrombocytopenia, leukopenia, and hemoconcentration is suggestive, and confirmatory tests with PCR or immunoglobulin M antibodies via enzyme-linked immunosorbent assay (ELISA) are available.

Management and Disposition

Supportive therapy is the only treatment available, with a focus on fluid repletion. Patients with mild illness are treated as outpatients. Admission of those with severe illness, hemorrhagic manifestations, shock, or an uncertain diagnosis is warranted.

Pearls

1. Consider dengue fever in recently returned travelers with fever, headache, and myalgias, particularly travelers from the Caribbean or Southeast Asia.
2. The fever tends to be high and may suddenly resolve and then return, also known as a "saddle back" fever pattern.

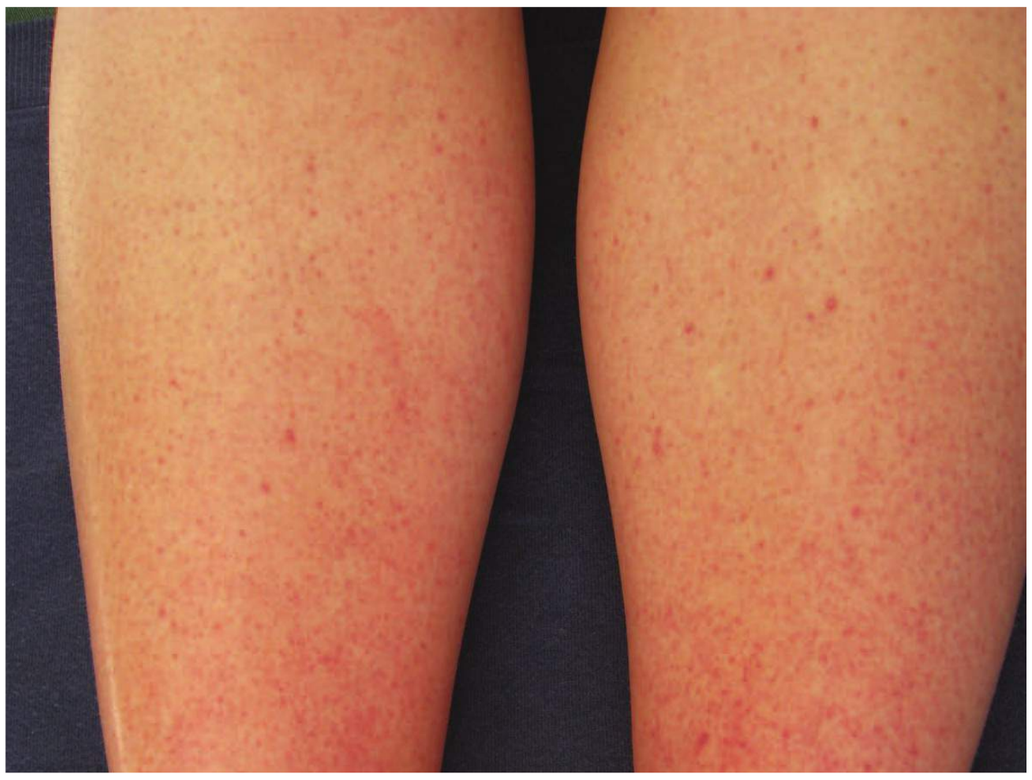

FIGURE 21.20 ■ **Dengue Rash.** Maculopapular rash from dengue on legs of an American medical worker during an outbreak in Guyana. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.21 ■ **Dengue Petechiae.** Measurement of petechiae following a tourniquet test in a patient with dengue. Twenty or more petechiae in the template area are suggestive of dengue diagnosis. (Photo contributor: WHO/TDR.)

3. A positive "tourniquet test" is suggestive of dengue. A blood pressure cuff is applied and inflated to a point between the systolic and diastolic pressures for 5 minutes. The test is positive if there are more than 20 petechiae per square inch.
4. Occasional cases of dengue occur in South Texas and an outbreak recently occurred in Key West, Florida.

FIGURE 21.22 ■ **Dengue Rash.** A hemorrhagic bullae on the arm of a patient with dengue hemorrhagic fever. (Photo contributor: WHO/TDR.)

Clinical Summary

Dracunculiasis is a debilitating roundworm infection caused by Dracunculus medinensis, commonly known as the Guinea worm. Dracunculiasis was historically widespread throughout equatorial Africa, the Middle East, and South Asia but the disease is now isolated to limited areas of Africa due to intensive eradication efforts. Dracunculiasis has a complex life cycle beginning when infected water fleas, or cyclops, are ingested while drinking from unclean water sources. Following ingestion, the larval worm penetrates through the stomach and intestine and migrates to the subcutaneous tissue, where it matures and reproduces. A painful wound develops, usually on the lower extremity, when the female emerges to release larvae. Immersion in water relieves the pain, allowing the worm to release larvae and complete the life cycle.

Emergence of the worm leads to a painful wound that is often accompanied by fever, nausea, vomiting, edema, intense pruritus, ulceration, and eosinophilia, all of which can last for months. The wounds often become secondarily infected, leading to significant disability. Drinking boiled or filtered water prevents dracunculiasis.

Management and Disposition

The worm is carefully extracted, usually around a stick or rolled gauze, in order to avoid retained worm products and the resultant inflammatory reaction. This process can take days to weeks. Daily immersion in water, with proper disposal to avoid larval spread, helps facilitate worm removal. Surgical excision is possible.

Pearls

1. The Guinea worm is the largest tissue parasite of humans. While very thin (2 mm in diameter), a full-grown worm is about a meter in length.
2. Dracunculiasis is rarely fatal, but the wound can cause significant morbidity and economic loss in areas with high infection rates.
3. It is predicted that dracunculiasis will be the first parasitic disease to be fully eradicated, largely through patient education and use of filtered water.

FIGURE 21.23 ■ **Dracunculiasis.** Gradual extraction of a Guinea worm using rolled gauze in Ghana. (Photo contributor: The Carter Center/Louise Gubb.)

ELEPHANTIASIS

Clinical Summary

Elephantiasis affects more than 120 million people worldwide with over 40 million severely disfigured. It is not a specific disease, but rather a syndrome caused by chronic obstruction of lymphatics. The most common cause is lymphatic filariasis, which is caused by the thread-like worms Wuchereria bancrofti, Brugia malayi, and Brugia timori. The infection is transmitted to humans by mosquitoes. Adult worms lodge in the lymphatics, disrupting the fluid balance between tissues and blood vessels, causing lymphedema of the extremities, breast, or genitourinary (GU) system. The infection is generally acquired in childhood, although clinical manifestations may take years to develop. Adult worms live 4 to 6 years, producing millions of blood circulating microfilariae. Acute symptoms of lymphadenopathy and dermal inflammation may precede and later accompany chronic swelling. With persistent infection and inflammation, the skin develops a hyperkeratotic, pebbly, or warty appearance that may become ulcerated and darkened. Bacterial and fungal superinfections contribute to morbidity.

Mosquito nets and insect repellants are the main means of prevention of lymphatic filariasis. Parasites may be detected microscopically in the blood, but the nocturnal periodicity makes identification challenging. Eosinophilia is common, but is nonspecific. Availability of a rapid card test that identifies circulating antigens has overcome this problem and is available in some endemic areas.

Management and Disposition

Treatment of lymphatic filariasis depends upon the presence or absence of other filarial organisms and includes various combinations of albendazole, ivermectin, and diethylcarbamazine. Cleansing of the affected areas and topical antibiotics aid in thwarting secondary disease. Local massage and elevation of the extremity improve lymphatic flow.

Pearls

1. Elephantiasis bears a heavy social burden due to physical limitations, disfigurement, sexual disability, and social stigmatization. Affected patients may be shunned by their families, are often unwed, and unable to work.

FIGURE 21.24 ■ **Elephantiasis.** Unilateral lymphatic filariasis. Note diffuse edema with early evidence of chronic skin changes. (Photo contributor: Larry Stack, MD.)

FIGURE 21.25 ■ **Elephantiasis.** Late-stage bilateral elephantiasis with chronic nodular skin changes. (Photo contributor: Seth W. Wright, MD.)

2. Testicular hydrocele is the most common manifestation of chronic W bancrofti infection for males in endemic areas.
3. Symptomatic filariasis is occasionally seen in travelers, even in those with only short-term stays in endemic regions.
4. W bancrofti occasionally causes an acute asthma-like condition known as tropical pulmonary eosinophilia.
5. Podoconiosis is a noninfectious cause of elephantiasis that occurs in people who walk barefoot in areas with large amounts of volcanic ash. Kaposi sarcoma is another emerging cause of nonfilarial elephantiasis in sub-Saharan Africa.

FIGURE 21.27 ■ **Elephantiasis.** Nonfilarial elephantiasis due to extensive Kaposi sarcoma in a 26-year-old Ugandan woman with AIDS. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.26 ■ **Elephantiasis.** Large hydrocele from lymphatic filariasis in a man from Haiti. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.28 ■ **Elephantiasis.** Bilateral disfigurement from lymphatic filariasis in an elderly Haitian man. (Photo contributor: WHO/TDR.)

EPIDEMIC MENINGITIS

Clinical Summary

Meningococcal disease in developed countries usually consists of sporadic cases and small outbreaks. In contrast, massive epidemics of serogroup A or C meningococcal meningitis occur in tropical countries, most notably in sub-Saharan Africa. These seasonal outbreaks tend to occur during the dry season along a wide swath of equatorial Africa known as the "meningitis belt."

The clinical presentation of epidemic meningitis is the same as in developed countries. Patients will typically present with fever, headache, nausea/vomiting, photophobia, and neck stiffness. Coma and death typically ensue if not treated. Diagnosis is with a spinal tap, although patients in resource-limited countries are often treated based on clinical grounds during a known outbreak. Mortality rates of less than 10% are obtainable. Large-scale vaccination programs are effective in decreasing spread of the disease within the affected areas and in adjacent population centers.

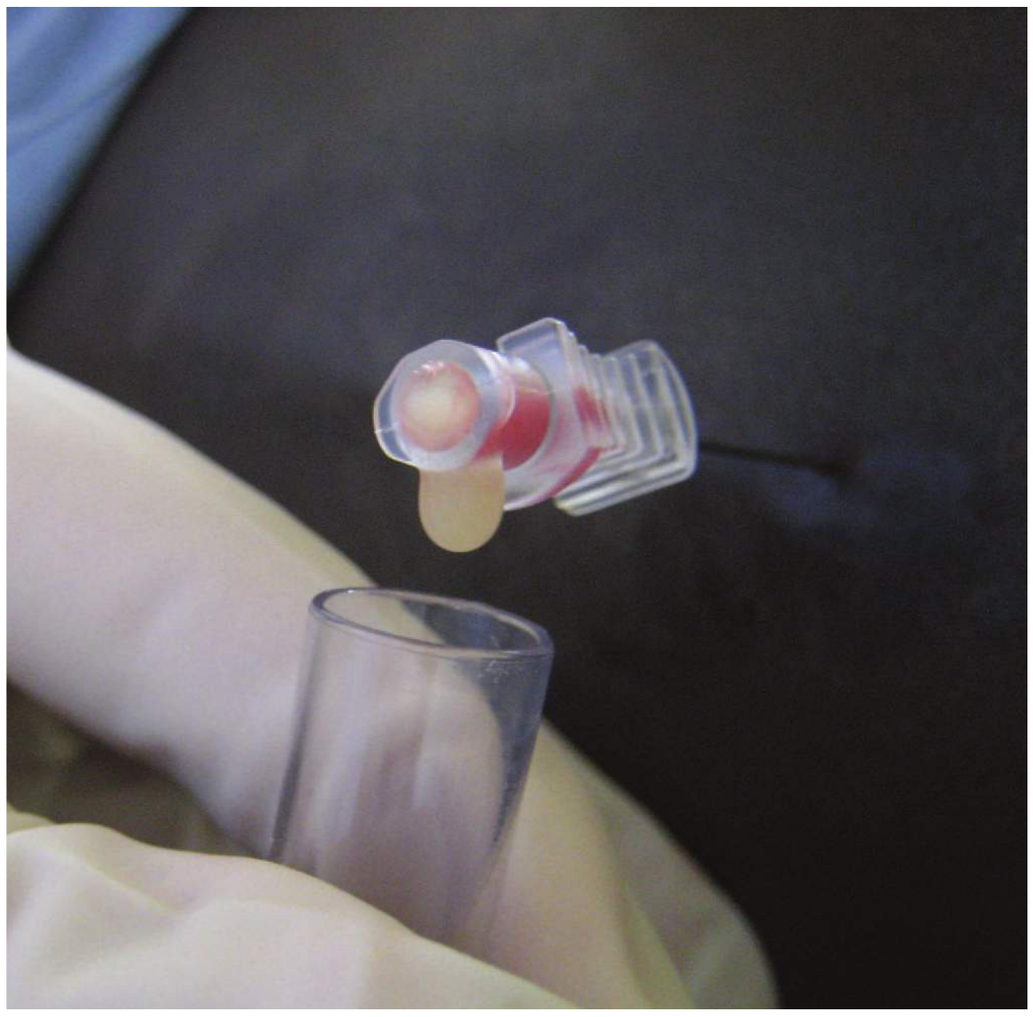

FIGURE 21.29 ■ **Meningitis Spinal Fluid.** CSF obtained from a 33-year-old Ugandan woman treated during an epidemic of serogroup A meningococcal meningitis. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

The mainstays of treatment are antibiotics and supportive care. Penicillins, cephalosporins, and ampicillin are still the treatments of choice in many settings, and are usually efficacious.

Pearls

1. Single-dose oily chloramphenicol injections have been used with success and allowed for easier management of large numbers in limited-resource settings. A second dose is given in 24 hours if there has been no improvement.
2. Massive outbreaks of meningitis have occurred during the Hajj, a pilgrimage to Mecca in Saudi Arabia. They are the only country requiring proof of vaccination before entry.
3. An often-used critical threshold to define an epidemic in Africa is 15 cases per 100,000 population per week. This would be equal to over 1400 cases per week in the Chicago metropolitan area.
4. Both serogroup A and C are covered by the meningococcal vaccine recommended for travelers by WHO and CDC.

FIGURE 21.30 ■ **Meningitis in a Child with a Very Rigid Neck.** Cerebral malaria should also be in the initial differential diagnosis. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 7-14.)

Clinical Summary

Endemic goiter is one of a spectrum of iodine deficiency disorders and is characterized by thyroid gland enlargement. It can occur in any location where environmental iodine is limited, but is rarely seen in developed countries as dietary iodine supplementation is routine. Goiters are often seen in inland and mountainous areas where iodine is leached from the soil, and access to iodine-rich foods (i.e., fish) is limited. Iodine deficiency disorders are common in widespread areas of Africa, Asia, and South America.

Patients with inadequate iodine in their diet have decreased T3 and T4 production, and resultant increased pituitary thyroid-stimulating hormone (TSH) secretion. The thyroid becomes hyperplastic and enlarged; however, patients are typically euthyroid.

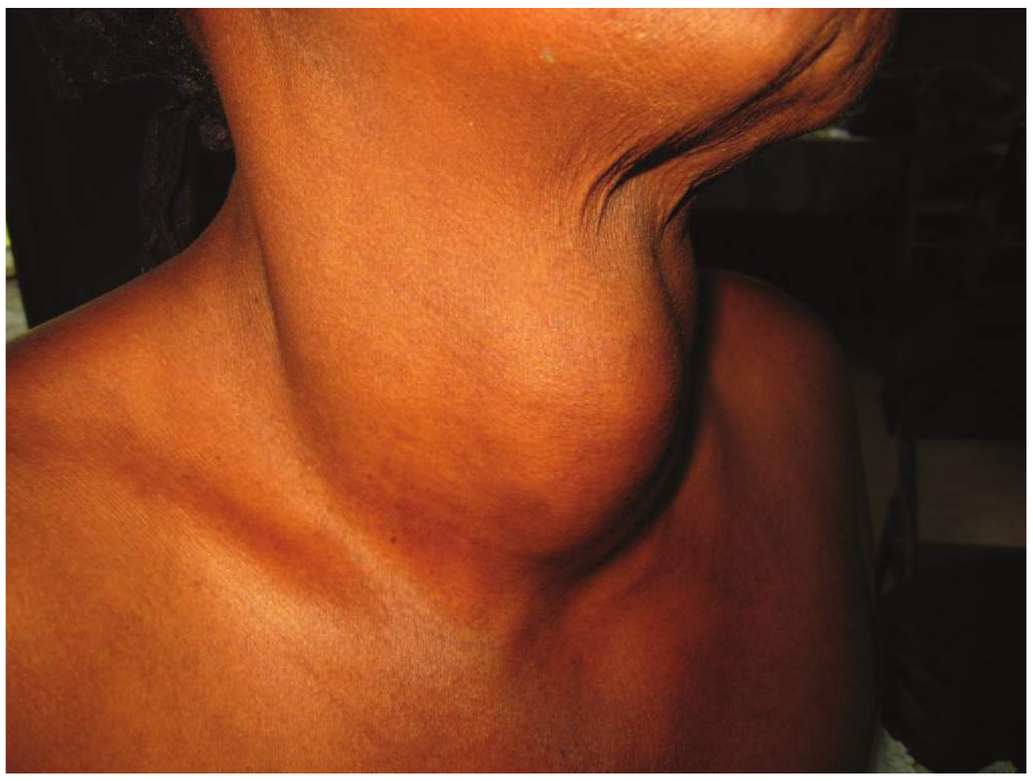

FIGURE 21.31 ■ **Goiter.** Easily visualized and palpable goiter. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

Prevention with dietary supplemental iodine is the mainstay of goiter management at the community level. Individual patients with goiter can be treated with potassium iodide solution or Lugol iodine, with iodized salt as a permanent solution. Surgical treatment can be indicated for massive goiter, particularly in the setting of tracheal or esophageal compression.

Pearls

1. Goiters in developed countries are most likely to be caused by defects in thyroid hormone production, resulting in an increase in TSH.
2. Iodine deficiency is the most common cause of preventable mental retardation (endemic cretinism) in the world.
3. Some dietary staple items, such as cabbage, cassava, lima beans, and sweet potatoes, have a goitrogenic factor which may be superimposed upon primary iodine deficiency.
4. Malignancy is not a common complication despite the massive size of many goiters, although it may interfere with detection.
5. In children, the goiter is usually diffuse; in adults, however, it is more commonly nodular since individual thyroid nodules proliferate at different rates.

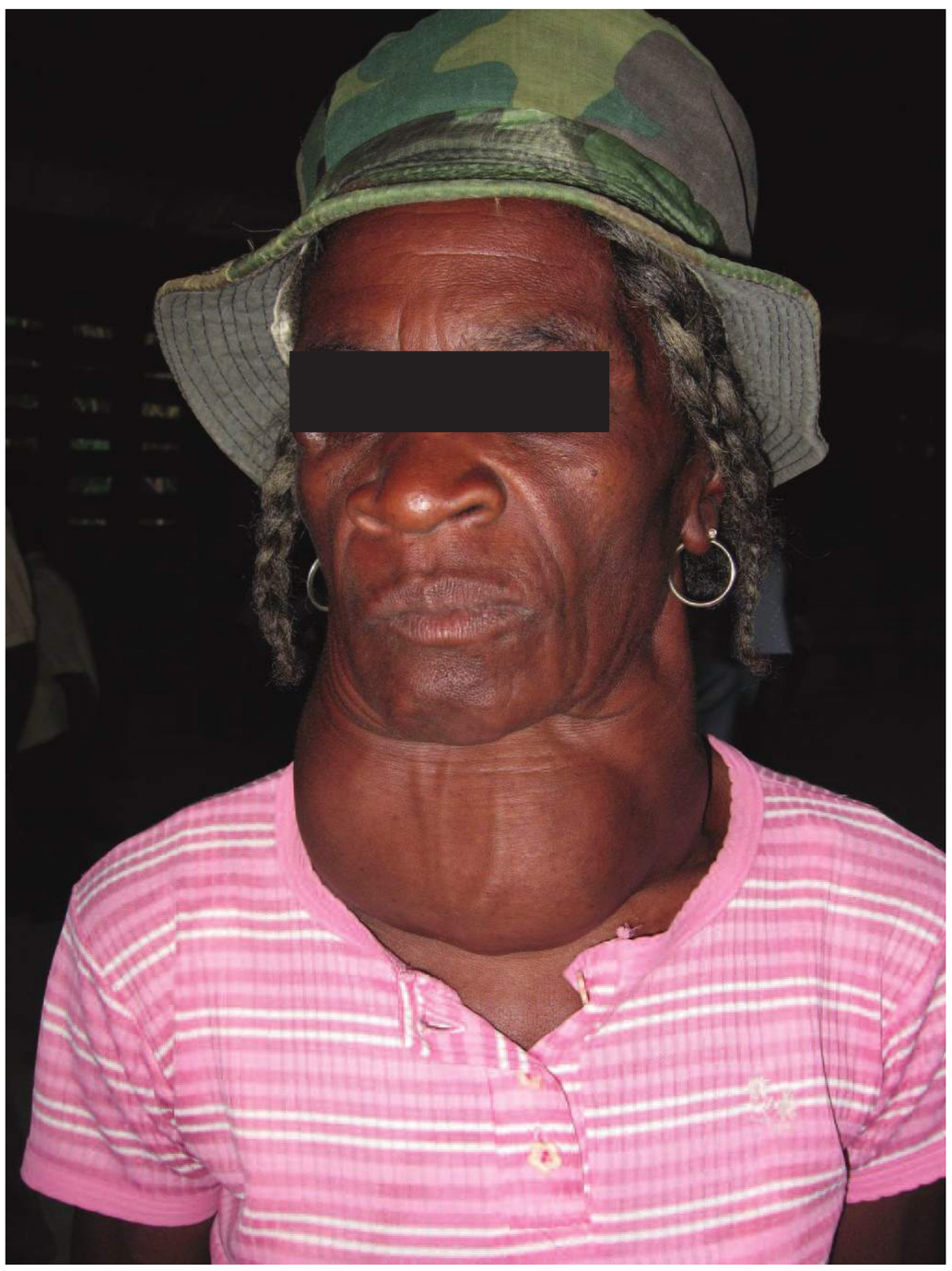

FIGURE 21.32 ■ **Goiter.** Patient from mountainous inland region of Haiti with massive goiter. This island nation has a high rate of goiter due to the complex interaction of poverty, political instability, deforestation, and erosion of soil. (Photo contributor: Ian D. Jones, MD.)

HYDATID CYST

Clinical Summary

Hydatid cysts are formed by the larval stage of Echinococcus granulosus tapeworms. The life cycle is maintained between canines and various farm animals (usually sheep or cattle). Adult worms inhabit the canine small intestine and eggs are passed in the stool. Farm animals become infected when they feed upon stool-contaminated material. The life cycle is completed when dogs or other canines ingest larval cysts formed in infected farm animals. Humans are infected from inadvertent ingestion of canine fecal material and are considered accidental hosts.

Hydatid cysts most commonly affect the liver and the lungs. Most patients who are infected have no symptoms until the cysts rupture, or are large enough to produce mass effect. Diagnosis is centered upon ultrasonography and serologic testing. Changes in farming practices have led to a marked decline in most industrialized countries, but they remain common in many developing areas. Most cases in the United States are immigrants from endemic regions.

Management and Disposition

Hydatid cysts are often an incidental finding when x-rays, CT scans, or ultrasounds are done for other purposes. These patients can be referred for follow-up. Patients with symptomatic cysts may need admission. Depending on cyst size and location, treatment may include percutaneous aspiration, surgical resection, or pharmacotherapy. Care must be taken with surgery to avoid spillage of cysts, which may lead to anaphylaxis.

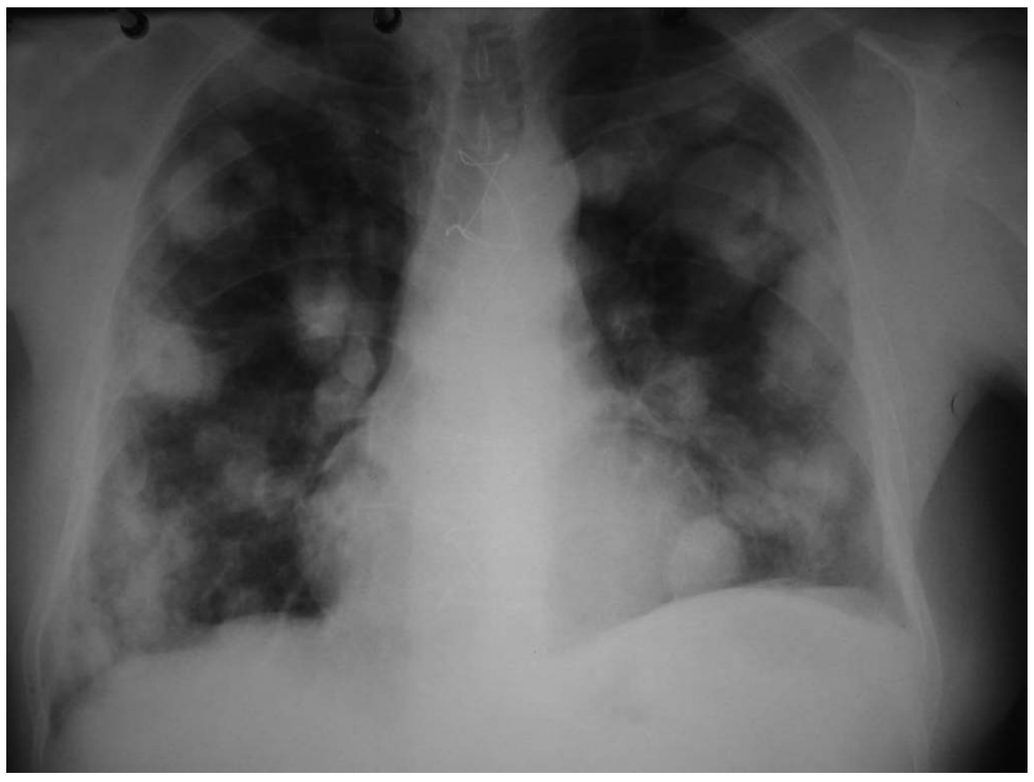

FIGURE 21.33 ■ **Hydatid Cyst Radiograph.** Chest x-ray showing multiple hydatid cysts. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Pearls

1. Liver cysts may produce obstructive jaundice, abdominal pain, or cholangitis.
2. Cysts are slow growing with an estimated average growth of 1 to 1.5 cm per year.
3. A chronic cough, pleuritic chest pain, hemoptysis, and dyspnea may be seen with lung involvement.
4. Though cerebral involvement is uncommon, it is seen more often in children.

FIGURE 21.34 ■ **Hydatid Cyst CT.** Scan demonstrating large hydatid cyst in the liver. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

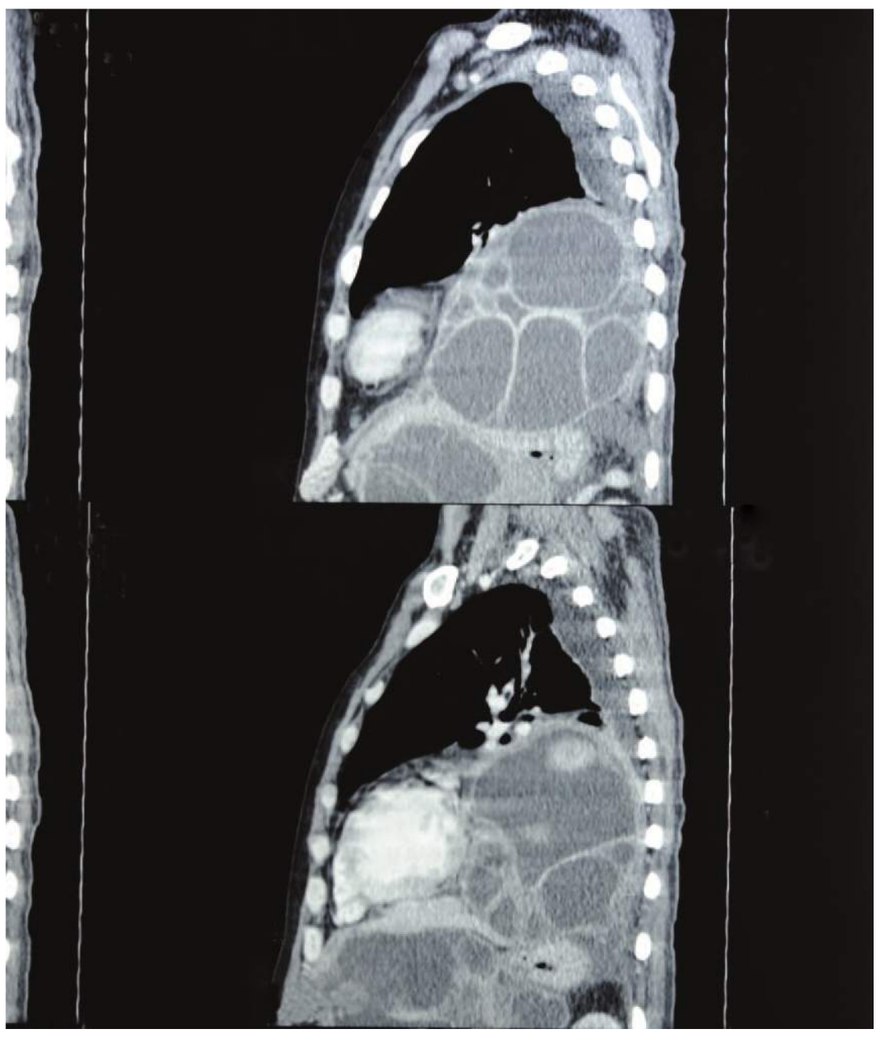

FIGURE 21.35 ■ **Hydatid Cyst CT.** Multiple echinococcal cysts are seen in the chest and liver of this middle-aged Latin American man with chronic cough, chest pain, and hemoptysis. (Photo contributor: Larry Stack, MD.)

Clinical Summary

Leeches are parasitic blood sucking aquatic or land-based invertebrates. Aquatic leeches are found worldwide, have relatively weak jaws, and usually attach to thinner tissues such as the pharynx or vagina. Land leeches are primarily found in tropical and subtropical areas of Asia, the South Pacific, and South America, typically have powerful jaws, can attach to any body surface, are fast and painless feeders, and often detach from the host without being noticed.

Leeches have been historically used for bloodletting, their saliva utilized to derive modern anticoagulants, and are considered currently for elimination of venous congestion after microvascular surgery. They are not known to directly transmit infectious diseases, but bacterial infection can follow bites. Leeches are prodigious feeders, so large infestations can cause significant anemia. Bites from detached land leeches can also bleed for prolonged periods due to injected anticoagulants. Allergic and anaphylactic reactions may occur. Aquatic leeches have potential to cause complications, particularly when attached within the esophagus or airway.

Management and Disposition

Treatment of detached leech bites consists of local wound care, hemostasis if needed, tetanus prophylaxis, and antibiotics if secondarily infected. Attached leeches are usually detected while in the field. They can be detached by carefully using a fingernail to break the suction under the sucker. Many other modalities (perhaps associated with increased risk of infection or other injury) have been suggested, including use of saline solution, vinegar, or a lighted match.

Pearls

1. The presence of a small y-shaped lesion on the lower extremity is suggestive of a detached land leech bite.
2. Aeromonas infections have been reported following use of medicinal leech therapy. Ciprofloxacin may aid in prevention.
3. Hemostasis with QuikClot gauze has been reported to be effective in patients with prolonged bleeding.
4. Leeches have been used for bloodletting in ancient medicine through the 19th century; it was believed the withdrawal of blood would help correct imbalances in four humors of the body or, later, treat a wide range of ailments.
5. Direct thrombin inhibitors, bivalirudin and lepirudin, are derived from the salivary gland of leeches.

FIGURE 21.36 ■ **Leech.** Nonengorged leech attached to thigh of patient in remote area of Tasmania. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.37 ■ **Leech.** Engorged leech attached to toe of a backpacker in Papua New Guinea. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.38 ■ **Leech.** Leech bite wound on the leg seen after detachment of a land leech. Note the sharp wound edges and surrounding ecchymosis. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Leishmaniasis is a protozoan, zoonotic parasite spread by sand fly bites. It is endemic in over 80 countries and has an incubation period of 2 to 6 months, though it may range from days to years, with relapse possible. Infection occurs when flagellated forms of more than 20 infective species of Leishmania are injected into the skin, taken up by macrophages, and multiply. Often, the bite goes unnoticed.

The primary cutaneous lesion begins as an enlarging papule, develops a scaly or ulcerative lesion with indurated edges and a central crater, and usually heals spontaneously in 6 to 12 months, but may progress to more diffuse syndromes. Mucocutaneous involvement or "espundia" is mostly seen in Latin America following initial cutaneous infection with Leishmania braziliensis. Nasal congestion and epistaxis may progress to septum perforation and nasal bridge collapse, causing a "tapir nose" deformity. Visceral involvement, known as "kala-azar," involves the spleen and liver. It is associated with fever, anemia, cachexia, splenomegaly, and may progress to hemorrhagic symptoms, secondary infections, and GI involvement. Visceral leishmaniasis is generally fatal without treatment.

Coinfection with advanced HIV causes synergistic immunologic disturbances due to the lack of a critical CD+ T-cell response.

The characteristic parasites can be identified from a smear or biopsy of the lesion identifying parasite amastigotes (Leishman-Donovan bodies). Culture and PCR are also diagnostic options.

Management and Disposition

No specific emergency treatment exists for leishmaniasis. Treatment of cutaneous or mucosal disease is with pentavalent antimony, miltefosine, pentamidine, or amphotericin B after referral to an experienced clinician. Protective clothing, bed nets, and insect repellent are the most effective ways of avoiding transmission as the flies are usually active from dusk to dawn. Visceral leishmaniasis should be managed by a tropical medicine or infectious disease specialist as the treatment is difficult and often toxic.

FIGURE 21.39 ■ **Cutaneous Leishmaniasis.** Lesions in a girl from the highlands of Peru. Most leishmania lesions are on exposed body areas. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 21.40 ■ **Cutaneous Leishmaniasis.** Lesion involving the face of an HIV-infected patient. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Pearls

1. More than 90% of visceral leishmaniasis cases are in India, Bangladesh, Nepal, Sudan, and Brazil, while more than 90% of cutaneous forms are in Afghanistan, Algeria, Brazil, Iran, Iraq, Peru, Saudi Arabia, and Syria.
2. Leishmaniasis is common in the Middle East and many cases have been identified in troops deployed to that region.
3. Leishmaniasis should be considered in immigrants from Latin America with chronic skin or mucosal lesions.
4. With visceral involvement, the primary cutaneous lesion will usually have resolved before clinical symptoms of kala-azar have developed.
5. After treatment of the visceral form, depigmented or nodular cutaneous lesions that are often confused with leprosy may occur.

FIGURE 21.41 ■ **Mucocutaneous Leishmaniasis.** Mucous membrane involvement (espundia) and "tapir-nose" deformity from Leishmania braziliensis. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 21.42 ■ **Mucosal Leishmaniasis.** Oral mucosal leishmaniasis caused by L braziliensis. Mucosal involvement can occur years after spontaneous resolution of a nontreated cutaneous lesion. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Clinical Summary

Leprosy (Hansen disease) is caused by Mycobacterium leprae and presents with anesthetic skin lesions and peripheral nerve complications. This chronic disease is common in many parts of the developing world, including areas of South America, South Asia, and Africa. Illness severity is inversely proportional to the patient's ability to produce a cell-mediated response and creates a range from localized (tuberculoid leprosy) to disseminated disease (lepromatous leprosy), with many having borderline cases falling between the two extremes.

Localized disease is limited to one to three skin lesions, typically sharply demarcated, flat, hypopigmented, anesthetic plaques with elevated margins. This is often accompanied by easily discernible peripheral nerve thickening. The most severe forms are characterized by poorly demarcated erythematous macules, papules, or nodules. The peripheral nerves have less palpable findings, but more diffuse nerve involvement is seen in a stocking glove pattern. This results in loss of bone length and insensate extremities, often leading to repetitive trauma, infection, and loss of digits. With diffuse infiltration of the face, the characteristic "leonine facies" is exhibited.

Diagnosis is primarily clinical along with staining of a slit skin smear or biopsy for acid-fast bacilli. PCR can aid in the diagnosis.

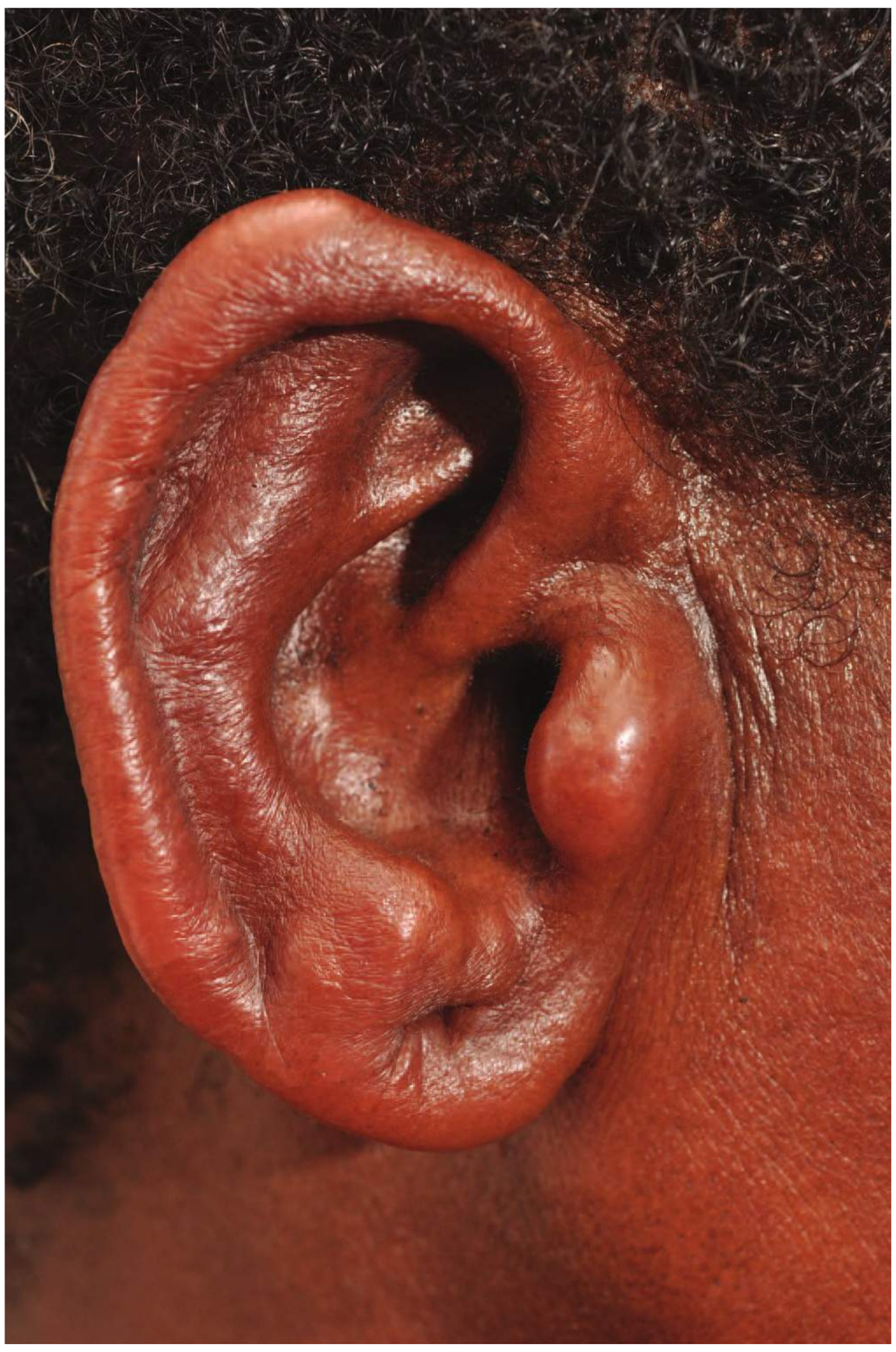

FIGURE 21.43 ■ **Leprosy.** Typical thickened skin on the external ear of a patient with lepromatous leprosy. (Photo contributor: Larry Stack, MD.)

Management and Disposition

There is no specific emergency therapy for leprosy. Patients should be referred to the national treatment program or a clinician experienced with leprosy management. Multidrug therapy is curative using varying combinations of dapsone, rifampin, and clofazimine. The class of disease dictates type and length of treatment. An ophthalmic exam is essential due to a high propensity for corneal abrasions and ulcers.

Pearls

1. Leprosy acquired in the southern United States is highly associated with armadillo handling.

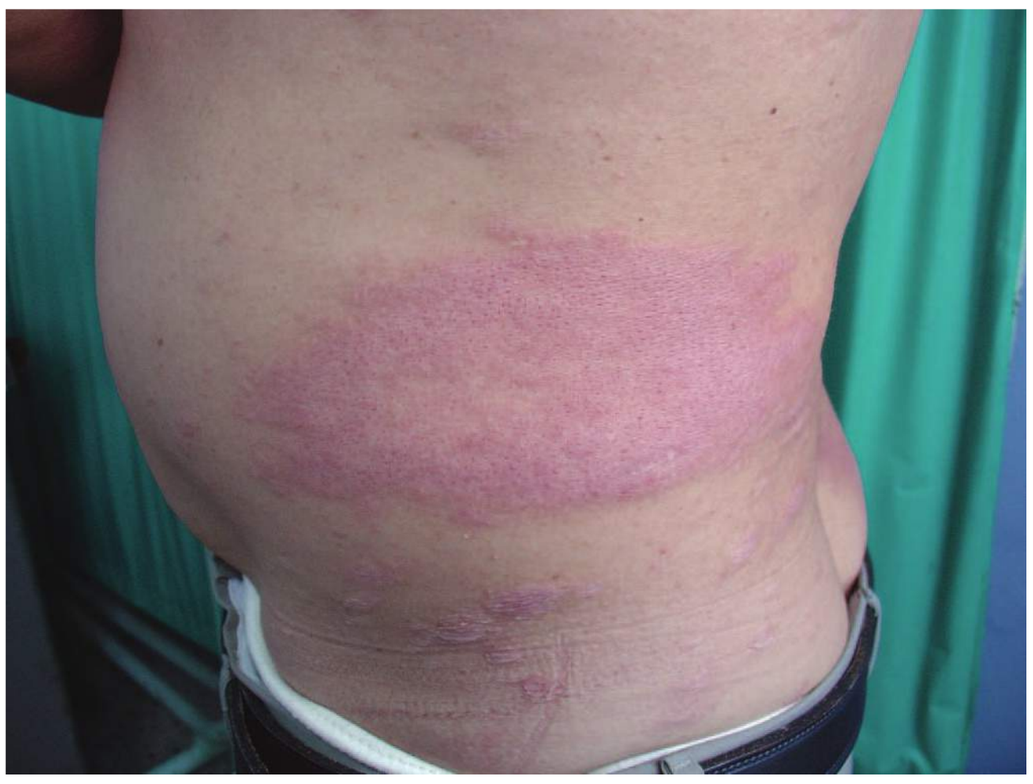

FIGURE 21.44 ■ **Leprosy Skin Lesions.** Anesthetic skin lesions in a Peruvian patient with borderline leprosy. Borderline disease is not as localized as tuberculoid leprosy and not as widespread as lepromatous disease. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

2. The presence of an anesthetic skin lesion should suggest the diagnosis of leprosy.
3. Multidrug therapy has been made available by WHO free of charge to all patients worldwide and provides a highly effective cure for all types.
4. The simpler WHO classification categorizes leprosy into localized (paucibacillary) or disseminated (multibacillary) disease.

FIGURE 21.45 ■ **Leprosy.** Thickened skin due to bacillary infiltration. Later stage may require digit amputation. (Photo contributor: David Effron, MD.)

FIGURE 21.46 ■ **Leprosy.** Chronic foot changes with ulceration and shortening of the toes. Extremity damage in leprosy results from loss of sensation, repeated trauma, neurotrophic atrophy, and direct bacillary deposition. (Photo contributor: Seth W. Wright, MD.)

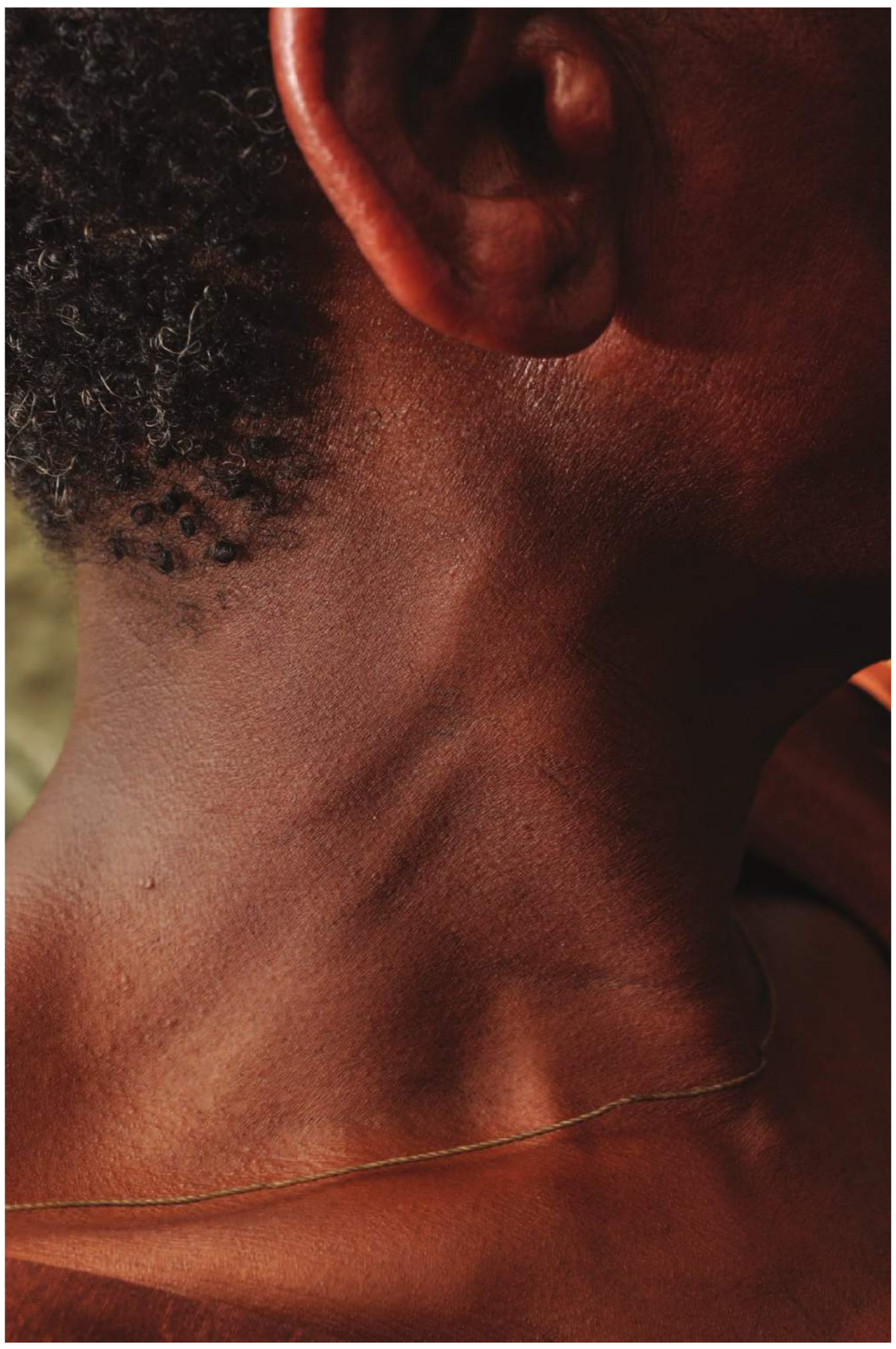

FIGURE 21.47 ■ **Leprosy.** Enlargement of the greater auricular and transverse cervical nerves of a patient with leprosy. Enlarged peripheral nerves are a cardinal manifestation. (Photo contributor: Larry Stack, MD.)

Clinical Summary

Leptospirosis is a zoonotic bacterial disease acquired by exposure to water contaminated by the urine of infected animals. Rodents are the usual reservoir, although pigs, dogs, and cattle can harbor the organism. It is most commonly attained by indirect skin or mucosal contact through occupational work, such as sugarcane or rice farming, or through recreational activities such as rafting, swimming, and hiking. Slaughterhouse workers can acquire leptospirosis through contact with infected body fluids. Leptospirosis is most common in tropical regions, especially during the rainy season.

The incubation period is usually 7 to 14 days; more than 90% of cases are asymptomatic, or mild and self-limited. Typical symptoms include the abrupt onset of high fever, chills, headache, severe myalgias, conjunctival suffusion (dilatation of vessels without inflammation or exudate), cough, jaundice, abdominal pain, rash, or diarrhea. Aseptic meningitis is common. Dermatologic findings are nonspecific and include macular, papular, and petechial rashes. Approximately 10% of patients develop the potentially life-threatening icteric form known as Weil syndrome. The initial symptoms are indistinguishable from the milder form until several days after the onset when complications such as renal failure, liver failure, rhabdomyolysis, pulmonary hemorrhage, or cardiopulmonary failure develop.

Laboratory findings in leptospirosis are nonspecific. An elevated creatine kinase is common. Markedly elevated bilirubin values are seen in Weil syndrome. Culture of the organism is possible, but diagnosis is usually serologic. Rapid test kits are available in some countries.

Management and Disposition

Empiric treatment is often indicated. Outpatient management with doxycycline, penicillin, amoxicillin, or a macrolide can be considered for mild illness. Severe disease is treated with IV penicillin or ceftriaxone. Supportive care, including optimal fluid management, is essential to help prevent or ameliorate renal dysfunction. Severely ill patients often need ventilatory support in an intensive care unit and dialysis.

Pearls

1. About half of the cases reported in the United States are from Hawaii, with increased outbreaks during periods of heavy rainfall and flooding.
2. Consider leptospirosis in an acutely febrile adventure traveler with recent freshwater exposure.
3. The presence of conjunctival suffusions, with or without jaundice, is highly suggestive of leptospirosis.
4. A Jarisch-Herxheimer reaction may occur after treatment with antibiotics.
5. Weekly doxycycline is effective prophylaxis for short-term travelers to highly endemic areas.

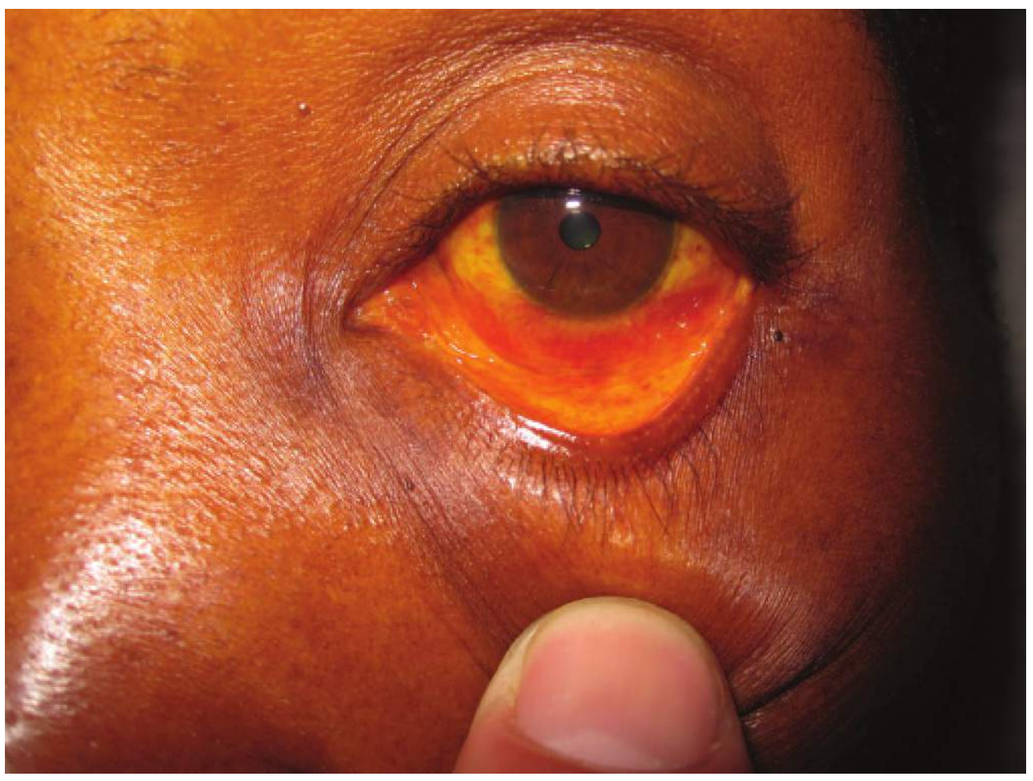

FIGURE 21.48 ■ **Leptospirosis.** Jaundice and conjunctival suffusions in a Haitian patient with laboratory-confirmed leptospirosis. The patient also had renal failure and myocarditis, both commonly seen in Weil syndrome. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Malaria infects up to 300 million people annually with over 1 million deaths; it is the most deadly vector-borne disease in the world. This parasitic disease is transmitted by the night-biting female Anopheles mosquito and is caused by four protozoa of the genus Plasmodium (Plasmodium falciparum, Plasmodium malariae, Plasmodium ovale, and Plasmodium vivax), with P falciparum causing the most morbidity and mortality. It is most common in tropical areas, particularly sub-Saharan Africa and Southeast Asia, with specific species predominating in each area.

The parasites undergo a hepatic cycle before entering circulating red blood cells and replicating. Lysis of the cell then occurs, releasing toxic by-products and further parasites into the blood, thereby causing cyclical clinical manifestations. Symptoms include fever, rigors, headache, myalgias, and malaise. The classic cyclical fevers do not always occur. P falciparum infection can cause massive hemolysis due to overwhelming parasitemia. Parasitized erythrocytes lose flexibility leading to microcirculatory obstruction, hypoxia of vital organs, and splenomegaly. Symptoms from P ovale and P vivax may be delayed for many months due to hepatic dormancy. Pregnancy is a risk factor for severe P falciparum infection and can result in maternal anemia, prematurity, and increased infant mortality.

Diagnosis is established by identification of the parasites on thick and thin smears. Rapid antigen kits are available in some regions. Bed nets, insecticides, and protective clothing are effective adjuncts for prevention. Chemoprophylaxis with an appropriate agent is highly recommended for travelers to endemic regions.

Management and Disposition

Treatment depends upon geographic location, suspected species, and illness severity. Close adherence to CDC, WHO, or national guidelines is highly recommended for both prophylaxis and treatment. Oral treatment of uncomplicated malaria is feasible with one of several regimens. Quinidine is the only approved treatment for severe malaria in the United States, although artesunate is available on an emergency basis from the CDC when quinidine is not tolerated or available. Artemisinin combination therapy for severe disease is now

FIGURE 21.49 ■ **Malaria Jaundice.** Jaundice due to acute hemolysis in a Haitian child with documented P falciparum infection. (Photo contributor: Seth W. Wright, MD.)

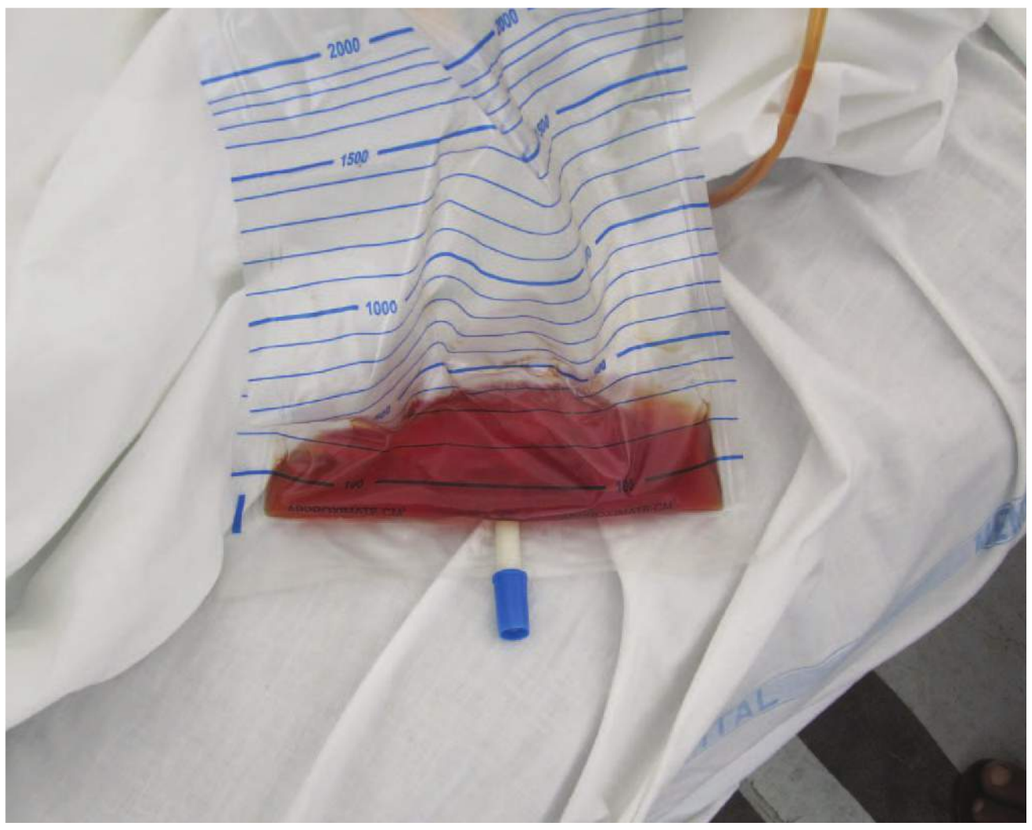

FIGURE 21.50 ■ **Malaria.** Hemoglobinuria following hemolysis in the patient from Fig. 21.49. The patient was severely anemic with a hemoglobin of 3.0 mg/dL. (Photo contributor: Seth W. Wright, MD.)

available in many endemic countries, although IV quinine remains a common treatment. Patients in developing countries are often treated as outpatients. With rare exception, returning travelers with suspected malaria should be admitted for management and treatment. Admission is always warranted for patients with suspected or confirmed P falciparum, symptoms of cerebral malaria, children, pregnant women, or immunocompromised individuals.

Pearls

1. Malaria should be strongly considered in any patient exhibiting fever following recent travel to the tropics. It is the single most common cause of fever in this population.
2. An emerging species, Plasmodium knowlesi, causes severe disease, but is currently limited in distribution to Southeast Asia.
3. The average incubation period for P falciparum is about 13 days; average incubation periods are longer for the other species. Incubation periods for all species can be variable.
4. Parasitemia fluctuates over time with the highest incidence during episodes of fever. Failure to identify the parasites on initial smears is not an indication to withhold therapy when the diagnosis is likely.
5. Residents of highly endemic areas will often have partial immunity to malaria due to repeated exposure. Immigrants visiting their native country are at high risk for severe infection due to loss of immunity.

FIGURE 21.51 ■ **Malaria.** Massive hyperreactive malarial splenomegaly in a surgical patient. This condition can occur with chronic infection, but malaria smears are often negative. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 21.52 ■ **Malaria.** A toddler with cerebral malaria. Abnormal motor posturing including decorticate, decerebrate, and opisthotonic posturing are all common in this condition. (Photo contributor: Seth W. Wright, MD.)

MUMPS

Clinical Summary

Mumps is a highly contagious viral disease best known as a cause of parotitis. Vaccination has markedly reduced the rate of disease in the developed world, but many resource-limited countries do not include mumps in their vaccine programs. It remains common worldwide; occasional large outbreaks occur even in countries with well-developed vaccine programs. Mumps is most common in children and spread by respiratory droplets, direct contact, or fomites. Subclinical infection is common in younger children. Disease in adolescents and adults is associated with a higher rate of serious complications.

A nonspecific illness with malaise, myalgias, fever, and headache follows an incubation period of about 2 to 3 weeks. Unilateral or bilateral parotid swelling is the hallmark, although orchitis is seen in about one-third of postpubertal males and pancreatitis in about 4% of patients. Diagnosis is usually clinical, particularly in developing countries. A high amylase from a salivary or pancreatic source might be present; viral isolation, PCR, and serology are available for definitive diagnosis.

Management and Disposition

Mumps is a self-limited, usually mild, disease. Treatment is supportive and consists of antipyretics, fluids, and analgesics. Patients with orchitis might benefit from scrotal support. Patients should be advised to stay home from school or work for 5 days after symptom onset.

Pearls

1. Natural infection confers life-long protection, although rare recurrent cases have been reported.
2. Aseptic meningitis with CSF pleocytosis occurs in more than one-half of patients with mumps, although most are minimally symptomatic. Mumps encephalitis also occurs, but is extremely rare.
3. Mumps should be considered in the differential diagnosis of aseptic meningitis, even in the absence of parotitis.
4. Mumps is one of the leading causes of acquired deafness in developing countries.
5. Ovarian and breast inflammation are occasionally seen in females, although it is not as common as orchitis among males. Sterility is rarely seen with either condition.

FIGURE 21.53 ■ **Mumps.** Parotid swelling in a Haitian girl with mumps. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.54 ■ **Mumps.** Note the classic submandibular and preauricular enlargement of the parotid gland. (Photo contributor: Centers for Disease Control and Prevention.)

Clinical Summary

Mycetoma is a localized, chronic, granulomatous infection of subcutaneous tissue with possible extension to underlying bone. Two classifications include eumycetoma, caused by filamentous fungi, and actinomycetoma, caused by bacteria of actinomycetes species. The organism is inoculated into subcutaneous tissue following minor trauma, most commonly the lower extremity and hand, though it may arise anywhere on the body. Both types are similar clinically; eumycetoma, however, causes more morbidity. Initially, a painless subcutaneous swelling is seen with induration, numerous suppurative nodules, and chronically draining sinus tracts. Remote abscesses may rarely be seen due to hematogenous extension. Actinomycetoma occurs more frequently (60%) and has a much better outcome. Mycetomas are rarely fatal, but may cause significant dysfunction and disfigurement.

Expulsion of "grains" containing aggregates of the organisms is common. The presence of black grains is diagnostic of a fungal origin, while pale grains could be either fungal or from an actinomycetes species. Further identification of the causative organism may be done by means of various stains, culture, or by serology.

Management and Disposition

There is no specific emergency management except recognition and referral. Surgical resection of large lesions may reduce organism load, but relapse rates can be as high as 50%. Amputation is common. Eumycetoma is treated with 1 to 2 years of antifungal agents, while actinomycetoma responds to various combinations of trimethoprim-sulfamethoxazole, dapsone, streptomycin, and amikacin.

Pearls

1. Mycetoma is usually seen in young male adults living in rural areas of Africa, Mexico, South America, or India who work as farmers or laborers. Walking or working barefoot is a risk factor for lower extremity disease.
2. Patients may complain of a deep itching sensation rather than pain. If pain is present, it may indicate secondary infection or bone involvement.
3. Sweating of the affected area is commonly seen.
4. A similar condition, botryomycosis, is caused by a chronic Staphylococcus infection with sinus formation.
5. Mycetoma was first described in the Madura district of India and is often referred to as "Madura foot."

FIGURE 21.55 ■ **Mycetoma.** A eumycetoma with typical chronic sinus drainages in the foot and ankle of a 33-year-old agricultural worker in Peru. Treatment was with itraconazole. (Photo contributors: Rob Griedanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 21.56 ■ **Mycetoma.** A large actinomycetoma of the upper leg with extensive active and healed sinus tracts. (Photo contributors: Stuart Skinner, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

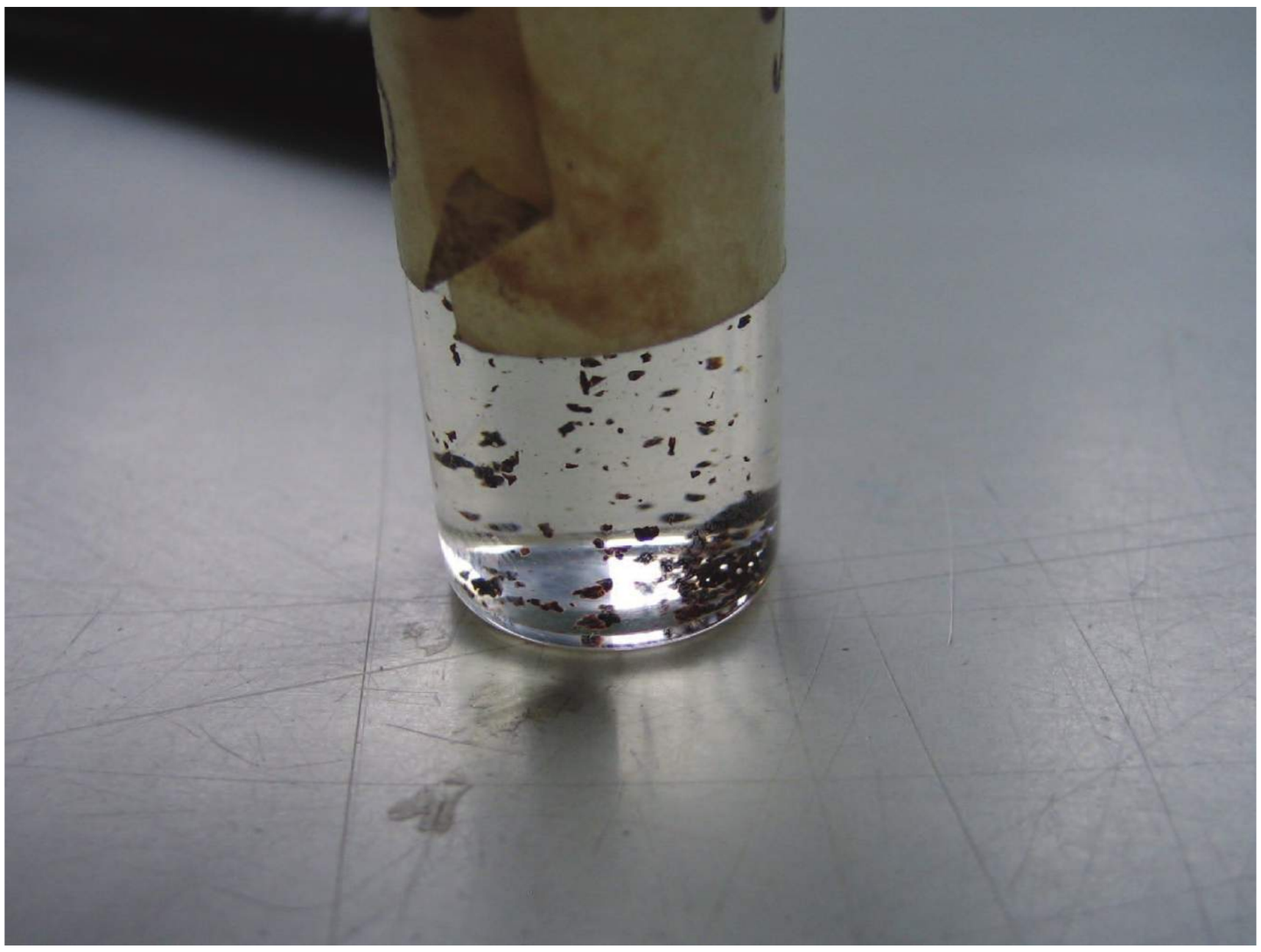

FIGURE 21.57 ■ **Mycetoma Granules.** Sinuses discharge characteristic dark granules (sclerotia) from a eumycetoma. The granules represent microcolonies of the organism. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 21.58 ■ **Mycetoma.** Mycetoma of the foot from Nocardia sp.: typical location with multiple draining sites. (Reproduced with permission from Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ, Wolff K. Fitzpatrick's Dermatology in General Medicine. 8th ed. McGraw-Hill; 2012: Fig. 185-6, p. 2249.)

Clinical Summary

Myiasis is the invasion of living human or animal tissue by maggots, the larvae of flies. Infection is most commonly subcutaneous, but may be seen in wounds and body cavities. The human botfly (Dermatobia hominis) is a major cause of furuncular myiasis in the New World; mosquitoes courier eggs to the host. A papule develops as the larvae feed, followed by a pruritic furuncle, often with drainage from a central punctum. After maturation, the larva emerges and falls to the ground, where it pupates in the soil and evolves into an adult fly.

The tumbu fly, found in Africa, also causes furuncular myiasis and is spread from eggs deposited in soil, sand, or clothes. Screwworm species are found in the Old and New Worlds. These worms are notorious for direct deposition of eggs after flying into the nasal cavity, leading to nasal cavity myiasis. Feeding maggots can cause extensive tissue damage. Wound myiasis, caused by numerous fly species, is seen in open sores and gangrenous tissues. Wearing protective clothing, utilizing insect repellant, and covering wounds are preventative measures.

Management and Disposition

Lidocaine injection at the lesion base, or application of suffocating occlusive substances such as Vaseline, may cause the larva to surface and facilitate removal in furuncular myiasis. Incisional extraction is challenging due to the larva's tapered shape and many rows of spines and hooks that it uses to grip tissue. Wound myiasis is treated with thorough debridement. Secondary infection is the only indication for antibiotics; it is uncommon due to bacteriostatic activity in the gut of the larvae, but may occur by leaving portions of them after removal.

Pearls

1. Patients with lesions may have the sensation of something moving under the skin.
2. Myiasis is self-limiting and usually not harmful, but it does cause psychological distress to the host, especially when the larvae surface. Though rare, deaths from meningitis have occurred after tissue penetration from infection in the eye, nose, or ear canal.
3. Screwworms are major pests of livestock. A sterile male release program has eliminated the screwworm from the United States.
4. Some feel that fly maggots are a useful method of wound debridement. Packets of fly maggots have been commercially developed for this purpose.

FIGURE 21.59 ■ **Myiasis.** Preserved specimens of D hominis. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 21.60 ■ **Wound Myiasis.** Wound myiasis in an elderly patient following dressing removal. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.61 ■ **Myiasis.** Wound on the arm of a woman who had visited the Amazon jungle. A D hominis larva was extracted from the wound. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 21.62 ■ **Tumbu Fly Lesions.** Multiple lesions from tumbu fly infestation on the skin of a 14-month-old American boy living in Botswana. The child had been wearing clothes dried on infected ground. (Photo contributor: Anonymous.)

Clinical Summary

Mycobacteria other than tuberculosis and leprosy cause disorders primarily seen in tropical countries. As opposed to Mycobacterium tuberculosis and M leprae, they are usually environmental saprophytes, not obligate human/animal pathogens. Human exposure may be via inhalation, contaminated water, or preexisting wound contamination. Overall, nontuberculosis mycobacteria can cause four different clinical syndromes: pulmonary disease, regional lymphadenitis, disseminated disease in immunocompromised patients, and skin/soft tissue infections.

Buruli ulcer is the most important disorder caused by infection with Mycobacterium ulcerans. They are seen throughout the tropics, but are particularly prevalent in Africa. The ulcer is slow-growing with extensive necrosis and deep undermining of wound edges. Serious complications can occur, including osteomyelitis, contractures, and scarring.

A variety of other cutaneous forms of mycobacterial diseases exist. Fish tank granuloma (from Mycobacterium marinum) is seen following handling of tropical fish tanks and occasionally in fishermen. Posttraumatic abscesses are caused by the rapidly growing mycobacteria, Mycobacterium fortuitum and Mycobacterium chelonae.

Disseminated disease from Mycobacterium avium complex is a late-stage opportunistic infection in HIV/AIDS, but is occasionally seen in immunocompetent individuals.

Management and Disposition

Providers should consider these illnesses in the differential diagnosis when patients present with typical skin lesions and in severely immunocompromised AIDS patients. Buruli ulcer is typically treated for 8 weeks with rifampin and either streptomycin or clarithromycin. Surgical excision with grafting may be needed. Other infections are treated with macrolides or other antibiotics.

Pearls

1. Buruli ulcer is the third most common mycobacterial disease worldwide, after tuberculosis and leprosy.
2. Consider M marinum infection on patients with a hand lesion following tropical fish tank manipulation.
3. Fast-growing mycobacteria such as M chelonae cause "cold abscesses," often seen after nonsterile injections that are given for cosmetic purposes. A recent outbreak of this organism was also seen following tattooing.
4. Some postulate that Crohn disease and sarcoidosis may be caused by a yet unidentified mycobacteria.
5. Mycobacterium haemophilum is considered the second most common cause of cervical lymphadenitis in children.

FIGURE 21.64 ■ **Cold Abscess.** A cold abscess following a cosmetic injection for weight loss. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 21.63 ■ **Buruli Ulcer.** Typical shallow base and deep undermining of the edges in a patient from Benin. (Photo contributor: World Health Organization.)

FIGURE 21.65 ■ **Cold Abscesses.** A patient with multiple recurrent cold abscesses from M chelonae following nonsterile weight loss injections in Peru. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

PROTEIN-ENERGY MALNUTRITION—KWASHIORKOR AND MARASMUS

Clinical Summary

Protein-energy malnutrition (PEM) applies to a group of disorders including kwashiorkor and marasmus. These are characterized by an imbalance between the body's supply and demand of energy and nutrients. Kwashiorkor means "the sickness of weaning" as it is often seen following weaning after birth. It usually occurs between the ages of 1 and 4 with a deficiency of dietary protein in the presence of normal to high carbohydrate intake. The etiology is complex, but there is decreased synthesis of proteins resulting in hypoalbuminemia. Kwashiorkor is an acute illness manifested by edema secondary to fluid and sodium retention. Patients have peripheral edema, anasarca, moon facies, apathy, and a protuberant abdomen secondary to hepatomegaly. The skin can become hyperkeratotic and may split open in pressure-prone areas.

Marasmus is often seen under the age of 1 and is associated with inadequate intake of both protein and calories. The body's own energy stores are utilized, resulting in emaciation. These individuals do not have edema, but rather a loss of subcutaneous fat, muscle wasting, and wrinkled loose skin. Marasmic-kwashiorkor refers to the combination of both forms simultaneously. Laboratory studies are useful, but often are not available. Patients with PEM may be left with permanent neurological and physical deficits due to lack of calories, vitamins, and essential amino acids.

Management and Disposition

These conditions are rarely seen or treated in developed countries, so acute treatment is often in settings with limited resources. These children often have other acute illnesses and are also at greater risk for infection, specifically pneumonia, gastroenteritis, and sepsis. IV fluids are limited to those with shock as they are prone to congestive heart failure; WHO oral rehydration salt solution is usually used for the first 6 to 10 hours. Low-protein milk formula is then started with a goal

FIGURE 21.66 ■ **Kwashiorkor.** A Zambian child with typical light hair, moon facies, peripheral edema, and dry skin of kwashiorkor. (Photo contributor: Meg Jack, MD.)

FIGURE 21.67 ■ **Marasmus.** An African infant with severe marasmus due to poor feeding following maternal death. The infant is severely underweight with loose skin and little subcutaneous fat. (Photo contributor: Meg Jack, MD.)

FIGURE 21.68 ■ **Kwashiorkor.** A Ugandan boy with edema typical of acute kwashiorkor. The "enamel paint peeling" rash is occasionally seen. The patient died several days after the photo was taken. (Photo contributor: Seth W. Wright, MD.)

of 100 kcal/kg/day. A normal diet is gradually started over a few weeks. Inpatient admission for evaluation, intervention, and arrangement of long-term care is advised when possible.

Pearls

1. PEM is one of the leading causes of death among children <5 years of age worldwide.
2. Depigmentation of dark hair causes it to turn reddish, and curly hair may become straight and brittle. Intermittent periods of proper nutrition may lead to alternating bands of light and dark hair known as the "flag sign."
3. Kwashiorkor is an acute illness, often of only several days duration. It is commonly precipitated by acute infections.
4. The mid upper arm circumference (MUAC) is often used for rapid assessment of nutritional status in developing countries.
5. Newer nutritional products, such as "Plumpy'nut," have been developed and allow for outpatient management.

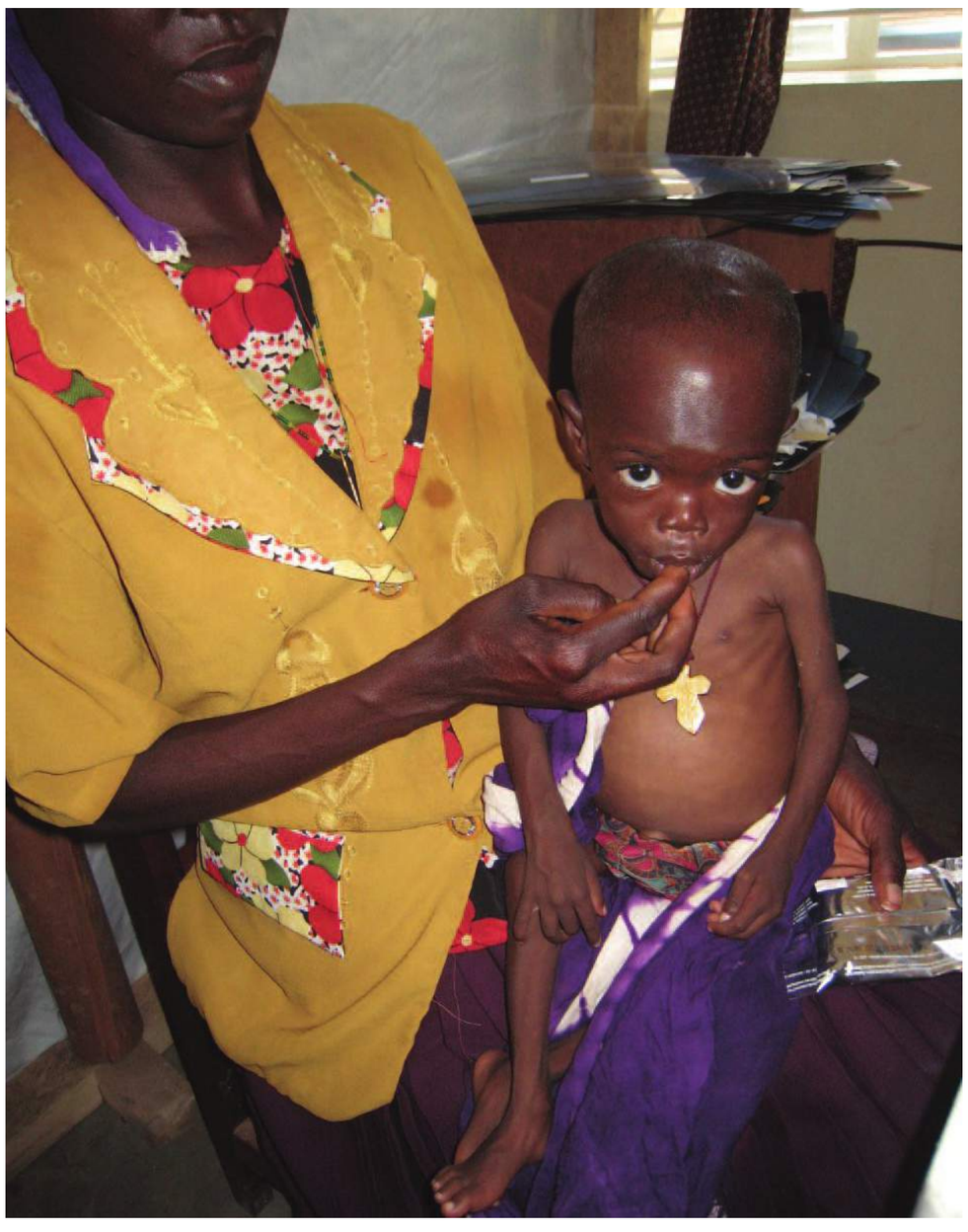

FIGURE 21.69 ■ **Marasmus.** An HIV-positive Ugandan girl recovering from marasmus. She is eating Plumpy'nut, a commonly used ready-to-use therapeutic food. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.70 ■ **Marasmus.** An African boy with marasmus. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Rabies is a viral zoonotic disease typically acquired through the bite of an infected animal. Transmission via aerosolized virus has been documented in bat-infested caves and in laboratory workers. Human-to-human transmission has only been documented in transplant recipients. Infection can be prevented by preexposure or postexposure vaccination.

Rabies has the dubious distinction of having the highest case fatality rate of any infectious disease. It is a severe progressive encephalitis and presents in one of two forms. In 80% of cases, patients will develop the encephalitic (furious) form, with typical symptoms of fever, agitation, laryngeal spasms, severe pain on swallowing, hydrophobia, confusion, and a host of other neurologic signs and symptoms. Patients with the paralytic (dumb) form (20%) present with an ascending paralysis similar to Guillain-Barré syndrome. Patients with either form often have preceding paresthesias and pain at the exposure site, as well as a nonspecific prodromal phase lasting up to 1 week. Autonomic instability is seen in all patients prior to death. The diagnosis of rabies in developing countries is primarily clinical. Fluorescent antibody testing, biopsy, and PCR are done in areas where available.

Management and Disposition

Management of clinical rabies is traditionally palliative as the outcome has been uniformly fatal. Vaccination after development of clinical rabies is futile. Treatment in developing countries is usually limited to sedation with benzodiazepines and other comfort measures. Survival of several patients using the "Milwaukee protocol" has been reported. This uses an induced coma (midazolam and ketamine) with continuous electroencephalography (EEG) monitoring, antiviral agents (amantadine), and aggressive diagnosis and treatment of cerebral vasospasm. This regimen is considered experimental in the United States and is not practical in most developing countries.

Pearls

1. Insect-eating bats cause the majority of US rabies cases, but most patients are unable to recall a bite. Dog bites are the most common cause worldwide.
2. The shortest incubation periods are associated with extensive bites and bite wounds to the face and scalp.
3. Rabies vaccine and immunoglobulin are crucial in avoiding rabies following an exposure. They are, however, contraindicated after the onset of clinical rabies as they might lead to an alteration of the natural immune response.
4. Human-to-human transmission is extremely rare or nonexistent. Nevertheless, postexposure prophylaxis of close contacts is recommended as the virus is present in saliva.
5. A report from Peru has suggested the possibility of subclinical rabies disease in Amazon residents.

FIGURE 21.71 ■ **Rabies.** A 20-year-old Ugandan patient with furious rabies on the fourth day of clinical illness after a dog bite 2 months earlier. The neck is visibly swollen due to subcutaneous emphysema. The patient developed spontaneous pneumomediastinum due to forceful vomiting during a period of severe laryngeal spasms. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.72 ■ **Rabies.** Same patient on the sixth day, now unable to swallow or control secretions due to laryngeal pain and spasms. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Over 200 million people worldwide are infected with schistosomiasis, with approximately 200,000 annual deaths. Transmission has been documented in over 75 tropical countries; it is considered the second most devastating parasitic disease, behind malaria. Humans become infected by contact with blood flukes in contaminated freshwater, usually through bathing, swimming, fishing, or agricultural work. The infectious larval form of the worm, known as cercariae, penetrates the skin, develops into an adult, and deposits eggs causing a host immune response and tissue damage.

Acute symptoms are most often seen in nonimmune individuals such as travelers. Cercarial dermatitis, or swimmer's itch, is a pruritic papular rash discovered immediately following exposure. Katayama fever may occur 2 to 8 weeks after exposure from systemic hypersensitivity and resembles serum sickness: fever, cough, chills, myalgias/arthralgias, diarrhea, lymphadenopathy, and hepatosplenomegaly. Resolution usually occurs after a few weeks, but patients can progress to coma or death.

Chronic symptoms are related to parasite load and involved organs. Intestinal symptoms (caused by Schistosoma mansoni) include abdominal pain, poor appetite, bloody diarrhea, with possible hepatosplenomegaly, periportal fibrosis, ascites, and portal hypertension. Urogenital disease (caused by Schistosoma haematobium) typically causes hematuria and dysuria, as well as genital lesions and vaginal bleeding in women. It can progress to fibrosis and calcification of the bladder and ureters, leading to hydroureter/nephrosis, infection, glomerular disease, and bladder cancer. Schistosomiasis is also associated with neurologic and pulmonary complications, including transverse myelitis, seizures, and pulmonary hypertension. Diagnosis is made through egg detection via stool or urine microscopy. Anemia and eosinophilia can be found in up to two-thirds of patients. Serology is useful for diagnosis in travelers.

Management and Disposition

Cercarial dermatitis usually resolves within 1 week; topical or systemic glucocorticoids may be considered. Therapy for Katayama fever is supportive, although glucocorticoids might be a useful adjunct. Praziquantel is effective against all forms of chronic disease but is not active on immature worms. Thus, patients with acute Katayama fever require delayed treatment in order to eliminate matured worms. Central nervous system (CNS) disease is treated with a combination of praziquantel and steroids.

Pearls

1. Hematuria is a common presenting symptom in areas endemic for S mansoni. These patients are often treated empirically with praziquantel.
2. Schistosomiasis can impact other infections, leading to more severe disease in patients with HBV, HCV, HIV, and malaria.
3. Prevention includes avoiding high-risk waterways, wearing protective boots, and vigorous toweling/drying following contact/exposure.
4. Swimmer's itch can occur as an initial manifestation of human schistosomal infection but is most often caused by avian schistosomes and is not otherwise infectious.

FIGURE 21.73 ■ **Schistosomiasis.** Bladder thickening and calcification in a patient with S haematobium infection. (Courtesy of the J.D. MacLean Centre for Tropical Diseases at McGill University.)

FIGURE 21.74 ■ **Schistosome Cercarial Dermatitis.** Rash due to avian schistosomes in a man following freshwater exposure in a Guyana river. Cercarial dermatitis usually follows freshwater exposure, but occasionally is seen with salt water. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.75 ■ **Schistosome Cercarial Dermatitis.** A highly pruritic papulovesicular eruption on the knee acquired after wading through a slow-flowing creek. (Reproduced with permission from Wolff K, Johnson RA, Saavedra AP (eds). Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. New York, NY: McGraw-Hill; 2013: Fig. 28-26.)

Clinical Summary

The infective spores of Clostridium tetani are widely distributed in soil and resistant to heat and disinfectants. Disease occurs when toxin forms after organism growth in wounds; it is characterized by acute onset of skeletal muscle rigidity and convulsive spasm.

The toxin affects inhibitory GABA and glycine receptors, leading to unopposed contraction and spasm of skeletal muscle. Initial symptoms involve the facial musculature producing trismus (lockjaw) and risus sardonicus (sneering grin). As larger muscles are involved, one may see opisthotonos, arm flexion and abduction, fist clenching against the thorax, abdominal rigidity, and lower extremity extension. Other symptoms include laryngeal spasm resulting in asphyxia, seizures, hyperthermia, hypertension, diaphoresis, and tachycardia. Reflex spasms may be triggered by minimal external stimuli. Fractures, dislocations, and rhabdomyolysis may occur due to forceful sustained muscle contractions.

Management and Disposition

Diagnosis is primarily clinical. Treatment includes airway protection, metronidazole or penicillin, active immunization with tetanus vaccine, tetanus immune globulin, benzodiazepines, and supportive therapy. Wounds should be cleansed and debrided to eliminate further toxin production. Tetanus immune globulin facilitates removal of unbound tetanus toxin, but does not affect toxin attached to nerve endings. Recovery of nerve function requires sprouting of new terminals and formation of new synapses which may take months to occur.

Pearls

1. There are four clinical forms of tetanus: local, cephalic, neonatal, and generalized disease (most common).
2. Neonatal tetanus occurs in infants born without passive immunity 3 to 21 days after birth. Inoculation usually occurs through infection of the umbilical stump and is common in developing countries.
3. Magnesium sulfate decreases spasms and cardiovascular instability.
4. In developed countries, tetanus is primarily a disease of the elderly, diabetics, and inadequately vaccinated immigrants.
5. Tetanus disease does not always lead to immunity.

FIGURE 21.76 ■ **Tetanus.** Fatal generalized tetanus in an 8-year-old Haitian child resulting from an infected puncture wound. Severe opisthotonic posturing and rigid jaw clenching are evident. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.77 ■ **Tetanus.** Abdominal wall contractions in a 12-year-old Ugandan girl with tetanus. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.78 ■ **Tetanus.** Severe facial tetany. The distorted grin and raised eyebrows is known as risus sardonicus. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.79 ■ **Neonatal Tetanus.** Neonatal tetanus in a 11-day-old infant. Opisthotonic posturing, muscular rigidity, and inability to swallow are seen. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Trachoma is the leading cause of infectious blindness in the world. It is endemic in areas of Africa, Asia, Latin America, the Middle East, and aboriginal communities in Australia. It is a chronic follicular conjunctivitis caused by Chlamydia trachomatis. Trachoma is prevalent in populations with limited access to adequate sanitation and clean water; it is spread through ocular and respiratory secretions as well as flies.

The distinct active phase produces mild itching, irritation, and eye discharge, as well as conjunctival inflammation, particularly of the superior tarsal plate. This may progress to marked photophobia, blurred vision, and eye pain. The scarring, or cicatricial, phase occurs after repeated or severe infection; chronic inflammation causes the upper lid to shorten (entropion), with subsequent eyelash inversion (trichiasis). Trichiasis may cause painful corneal abrasions and subsequent corneal edema, ulceration, scarring, opacities, and, ultimately, blindness. Lacrimal gland involvement leads to dryness and increased irritation. Trachoma usually affects both eyes. Diagnosis is largely clinical, but may be confirmed by culture. Community-based efforts on hygiene education and behavior modification can decrease the incidence.

Management and Disposition

For acute and subacute infections, the most effective treatment is a single dose of azithromycin; tetracycline ophthalmic ointment is an alternative. Eyelid surgery to correct trichiasis and entropion may prevent blindness.

Pearls

1. Trachoma was once endemic to North America and Europe, but has disappeared with improved sanitation and living conditions.
2. It is extremely contagious and may be spread through direct contact with eye, nose, or throat secretions.
3. Young children are particularly susceptible, but the disease progresses slowly and the more painful symptoms may not emerge until adulthood.
4. Adult women are at much greater risk due to their close contact with small children who are the main reservoir of infection.
5. The WHO is attempting to eradicate trachoma by 2020 through their public health program known as SAFE. This multifaceted approach involves surgery for advanced disease, antibiotics, facial cleanliness, and environmental/sanitation improvements.

FIGURE 21.80 ■ **Trachoma.** Extensive inflammatory response with trichiasis. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.81 ■ **Trachoma.** Chronic corneal scarring with excessive tearing in an African patient. (Photo contributor: Meg Jack, MD.)

Clinical Summary

According to WHO, traditional medicine is the sum total of the knowledge, skills, and practices based on the theories, beliefs, and experiences indigenous to different cultures, whether explicable or not, used in the maintenance of health as well as in the prevention, diagnosis, improvement, or treatment of physical and mental illness. Such practices are common worldwide and, while frequently used in developed countries, they are even more prevalent in the tropics. As many as 80% of people in some countries rely upon traditional medical practices as their primary form of health care. Poverty, lack of education, decreased access to standard medical care, and mistrust of Western medical concepts all play a role.

Traditional medicine practices are as varied as the societies of the world. Local practices for acute and chronic illnesses might include prayer, meditation, diets, fasting, massage, exercise, herbal remedies, acupuncture, skin scraping, and scarification. Underlying principles of traditional therapies vary greatly; in some societies, they are related to the balance or homeostasis between negative (bad, dark, devil, etc) and positive (good, light, angels, etc) forces. Ayurvedic medicine is practiced throughout South Asia and roughly translates to "the science of life." This common practice seeks to promote spiritual harmony based upon the theory that health exists

FIGURE 21.83 ■ **Evil Eye Make-Up.** Make-up on a girl in Bangladesh used to ward off the "evil eye," a sickness transmitted by someone who is envious, jealous, or covetous. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.82 ■ **Coin Rubbing.** Typical coin-rubbing marks on the back of a Southeast Asian immigrant with a minor illness. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.84 ■ **Traditional Healing Practice.** Razor marks placed by a traditional healer in a Zambian patient with fever and cough. A chest x-ray showed an infiltrate corresponding to the razor marks. (Photo contributor: Seth W. Wright, MD.)

when there is a balance between body, mind, and spirit. Other concepts, such as witchcraft or the "evil eye," are prominent in dozens of countries. The evil eye, or ocularis sinister, is thought to be the cause of a curse, spell, misfortune, or disease, and various amulets, decorations, or procedures may be used to ward off the unwanted effects.

Pearls

1. More than 70% of Americans using traditional therapies will not inform their physician of their use.
2. The majority of pediatric fevers from malaria are treated with herbal medicines at home in many African countries.
3. Coin rubbing (cao gio) is a common treatment for minor illnesses in Southeast Asia and is frequently seen in US immigrants. This should not be mistaken for child abuse.
4. Many common medications, including important antimalarial agents, are derived from traditional herbal remedies.
5. Ayurvedic and Unani systems have a recognized place in national health programs in India, and have hundreds of thousands of registered practitioners.

FIGURE 21.85 ■ **Fire Cupping.** Fire cupping is used in traditional Chinese medicine for a variety of ailments including musculoskeletal pain and various respiratory, digestive, and gynecologic diseases. The distinctive temporary cupping marks develop as a result of vacuum formation within the cups as the heated air cools. (Photo contributor: Allison Bollinger, MD.)

FIGURE 21.86 ■ **Traditional Healing Practice.** Use of plant material for healing of an otherwise untreated open ankle fracture following the 2010 Haiti earthquake. (Photo contributor: Seth W. Wright, MD.)

TROPICAL SPOROTRICHOSIS

Clinical Summary

Sporotrichosis is a subcutaneous infection caused by the fungus Sporothrix schenckii and is acquired from moss, decaying vegetation, hay, and soil. It is usually seen in those whose vocation brings them into contact with the environment. The two primary manifestations involve the cutaneous and lymphocutaneous systems; however, osteoarticular, pulmonary, and disseminated forms (primarily in immunocompromised patients) may be seen from direct inoculation or through hematologic seeding.

The extremities are usually involved with the initial lesion at the site of injury from a thorn, barb, pine needle, or wire. After approximately 1 to 10 weeks, a localized red, purple, or pink papule develops, often resembling an insect bite. The papule evolves into one or more nodules that form painless chronic ulcers with a nonpurulent, clear discharge. In the lymphocutaneous form, the nodules will progress proximally along lymphatic tracts and blood vessels. Many strains do not grow at temperatures above 35°C, decreasing their ability to spread and commonly resulting in a localized lesion. It is not communicable from person to person though it may be acquired through exposure to infected animals, with cats being the most infectious.

Wearing gloves and long sleeves while working in the outdoors and avoidance of skin contact with sphagnum moss are the mainstays of prevention. Confirmatory diagnosis is made with biopsy or culture.

Management and Disposition

Treatment with oral itraconazole for 3 to 6 months, or alternatively potassium iodide or terbinafine, is effective with complete recovery in cutaneous and lymphocutaneous forms. Variable response to treatment, often initially with amphotericin B, is seen in patients with severe disease or systemic involvement.

Pearls

1. This diagnosis should be considered when a cutaneous lesion is found on a patient involved with landscaping, rose gardening, Christmas tree farming, berry picking, baling of hay, and in veterinarians.
2. Lesions typically are noted on the distal upper extremity and the patient may have already failed multiple treatments with antibacterial agents.
3. While first described in the southern and central United States, it is most common in Mexico, Central/South America, Japan, Australia, and Africa. In Peru, the incidence is approximately 1 in 1000 people.
4. Sporotrichosis is easily confused with leishmaniasis, and the two illnesses coexist in many locations. Lymphatic spread is more characteristic of sporotrichosis, but possible with either. Other diseases or causes on the differential include nocardia, tularemia, and mycobacteria.

FIGURE 21.87 ■ **Sporotrichosis.** Typical sporotrichoid spread up the arm from an inoculation of the hand. Note the ulcers that resemble pyoderma gangrenosum in this Panamanian child. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 174-13.)

FIGURE 21.88 ■ **Sporotrichosis.** Typical lymphatic spread of sporotrichosis along the lateral aspect of the foot in a 30-year-old resident of the Peruvian Amazon. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Clinical Summary

Tuberculosis (TB) is a chronic bacterial infection spread from human to human through respiratory droplets containing M tuberculosis, an acid-fast bacillus. Upon inhalation, the organisms are transported to regional lymph nodes where the immune system forms granulomas or "tubercles." Most people undergo complete healing following exposure with only a positive PPD. However, it may lie dormant as latent disease until the immune system is suppressed, leading to organism release. TB occurs worldwide, but is more common in impoverished nations.

The organisms are dependent on high oxygen content and are typically found in the upper lobe or superior segment of the lower lobes of the lungs. Though it is primarily a respiratory illness, 15% will exhibit extrapulmonary manifestations involving the adrenal glands, long bones, vertebrae, GI tract, GU tract, skin, lymph nodes, meninges, pericardium, or peritoneum. Patients with HIV infection have a much higher overall prevalence and rate of extrapulmonary disease. Common symptoms of active TB are fever, night sweats, malaise, weight loss, cough, hemoptysis, and pleuritic chest pain. Diagnosis is made by the PPD and chest x-ray, but is confirmed through AFB smears and sputum culture. The mainstay of diagnosis in resource-limited countries is the AFB smear.

Management and Disposition

Typical first-line chemotherapy consists of isoniazid, rifampin, pyrazinamide, and ethambutol for 2 months, followed by isoniazid and rifampin for 4 months. Patients in developed countries are usually admitted with respiratory isolation. Outpatient therapy is common in developing countries. After 2 weeks of treatment, the patient is usually no longer contagious. Poor long-term adherence is a major contributing factor to the development of multidrug-resistant strains. Directly observed therapy (DOT) where patients are observed taking the medication may be required to ensure compliance and is routine in most countries.

Pearls

1. Any immunocompromised person with pulmonary symptoms should be placed in respiratory isolation until TB is excluded.
2. "Scrofula" refers to cervical lymph node involvement of TB, while vertebral involvement is called "Pott disease."
3. Isoniazid combined with rifapentine for treatment of latent TB, given weekly for 12 weeks, is now an alternative to the 9-month regimen of daily isoniazid.
4. TB is the most important opportunistic infection in HIV patients in sub-Saharan Africa. The HIV epidemic has led to a massive increase in the incidence of TB in many countries.

FIGURE 21.89 ■ **Tuberculosis Adenopathy.** Bilateral cervical adenopathy (scrofula) in a Peruvian child with documented TB. Pediatric cervical adenopathy in highly endemic areas is strongly suggestive, and the most common form, of extrapulmonary TB. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 21.90 ■ **Tuberculosis Scarring.** Chronic discharging sinuses with extensive scar formation in a Peruvian man with extrapulmonary tuberculosis. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 21.91 ■ **Tuberculosis Cold Abscess.** Cold abscess on the back of a Haitian woman with untreated Pott disease. (Photo contributors: Seth W. Wright, MD.)

FIGURE 21.92 ■ **Tuberculosis Pott Disease.** Severe spinal deformity ("gibbus") in an 8-year-old Ugandan boy with Pott disease. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.93 ■ **Abdominal Tuberculosis.** Severe wasting and ascites in a Ugandan boy with abdominal tuberculosis. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.94 ■ **Clubbing.** Clubbing of the fingers in a Ugandan man with longstanding pulmonary tuberculosis. (Photo contributor: Seth W. Wright, MD.)

FIGURE 21.95 ■ **Pulmonary Tuberculosis.** Severe wasting in a Sudanese soldier with smear positive pulmonary tuberculosis. His initial weight was 38 kg with a BMI of 11. His weight increased to 52 kg and BMI to 15 by the end of the 2-month intensive phase of treatment. (Photo contributor: Seth W. Wright, MD.)

TUNGIASIS

Clinical Summary

Tungiasis is an ectoparasitic condition caused by the cutaneous penetration of a Tunga penetrans flea. These tiny fleas are commonly found in sandy beaches and areas with warm, dry soil. Risk factors for infection include household dirt floors, lack of closed footwear, and the presence of farm animals. In some poverty-stricken regions, the prevalence of tungiasis approaches 50%. Tungiasis is also common in returned travelers, usually following beach vacations.

The female flea burrows into the skin of a host, feeds on blood, matures, lays eggs, and eventually dies in the cutaneous tissue. The rear of the flea remains visible as a black spot and serves for respiration and excretion of feces. A pronounced inflammatory response is seen leading to characteristic lesions. The vast majority of lesions are seen on the feet with the area at the rim of the toenails being the preferred site.

Early stage lesions demonstrate erythema surrounding a 0.5- to 2.0-mm black dot. A yellowish-white halo then develops around the dot with increasing erythema, pain, and pruritus. Later stage lesions develop a brownish coloration with crusting and desquamation. Secondary bacterial infection is common. Tungiasis is usually a clinical diagnosis; a small central dot representing the flea is an important clue. A biopsy will show the flea, but is not required for diagnosis.

FIGURE 21.96 ■ **Tungiasis.** Acute tungiasis lesion from an adult flea seen on the foot of a 30-year-old American aid worker in the Democratic Republic of Congo. Lesions are more obvious in patients who do not habitually walk barefoot. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

No oral drug has been proven to be effective against an embedded flea. Occlusive ointments will kill the flea, but does not aid in removal. The flea can be removed with excision or gently with a needle. Untreated, the flea will eventually die after 4 to 6 weeks with subsequent sloughing of the flea and eventual resolution of the inflammatory lesion. Antibiotics are indicated for secondary infection.

Pearls

1. Tungiasis is known by numerous local names including sand fleas, jiggers, bicho de pie, nigua, pigue, suthi, and chigoe.
2. The causative flea is native to the West Indies and was first reported in crewman who sailed with Christopher Columbus. They are now widely found throughout Latin America, Africa, and Asia.
3. The initial stage of penetration is manifested by a stinging sensation followed by itching that is surprisingly described as being pleasant.
4. Numerous cases of tetanus and gangrene have been reported in patients with secondarily infected T penetrans lesions.

FIGURE 21.97 ■ **Tungiasis.** Chronic lesions in a Kenyan man. Crusted scales are common in those with chronic infections and thicker skin. (Photo contributor: Larry Stack, MD.)

Chapter 22

AIRWAY

Steven J. White
Richard M. Levitan
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Lawrence B. Stack

Glottic Opening. Standard direct laryngoscopy reveals the glottic opening in this cadaver. A Mac 4 blade is used like a Miller blade in this instance as the epiglottis lies anterior to the blade tip. (Photo contributor: Lawrence B. Stack, MD)

MAXIM: Endotracheal intubation is not always the best initial intervention for respiratory failure.

Clinical Summary

Some patients in respiratory distress may benefit from other interventions, short of intubation. Patients with flash pulmonary edema may have dramatic improvement with intravenous nitroglycerin, intravenous furosemide, and BiPAP (Bi-Level Positive Airway Pressure) ventilation (see Fig. 22.1). Patients with airway narrowing (edema, neoplasm, stricture, foreign body) can have significant decreased work of breathing by decreasing airway resistance to inspired gas using administration of helium-oxygen (HELIOX) mixture. HELIOX, usually as a 78%: 22% helium: oxygen mixture, is much less dense than either air or 100% oxygen by virtue of helium replacing nitrogen or oxygen, respectively. This lowers resistance to laminar flow by as much as 20% to 25% and the effects are immediate.

Pearl

1. Alternative ventilatory adjuncts include HELIOX, CPAP, BiPAP, and Vapotherm (see Fig. 22.2). These adjuncts may prevent the need for intubation in selected patients.

FIGURE 22.1 ■ **Bi-Level Positive Airway Pressure.** This patient with COPD rapidly improved with the application of BiPAP. (Photo contributor: Steven J. White, MD.)

FIGURE 22.2 ■ **Vapotherm.** Application of Vapotherm (high-flow warmed oxygen) rapidly improved this toddler with pneumonia and reactive airway disease. (Photo contributor: Lawrence B. Stack, MD.)

MAXIM: The most important initial airway intervention may be to ask for help.

Clinical Summary

Clinical scenarios where asking for help include:

1. Laryngeal injury/tracheal disruption, for whom a nonendoscopic intubation attempt can result in tracheal disruption and fatally lost airway (see Fig. 22.3).
2. Recent neck surgery, with pending loss of airway from an expanding hematoma; definitive and life-saving intervention in this case is to open up the recent incision and evacuate the hematoma.
3. Suspected epiglottitis, where an immediate operative tracheostomy or cricothyroidotomy may be required if intubation fails due to epiglottic edema.
4. Severe angioedema with tongue, oropharynx swelling where endotracheal or nasotracheal intubation may be obstructed (see Fig. 22.4).

Pearls

1. Emergency physicians are emergency airway experts, but know your limitations.
2. Do not let your ego get in the way of proper patient airway management and care.

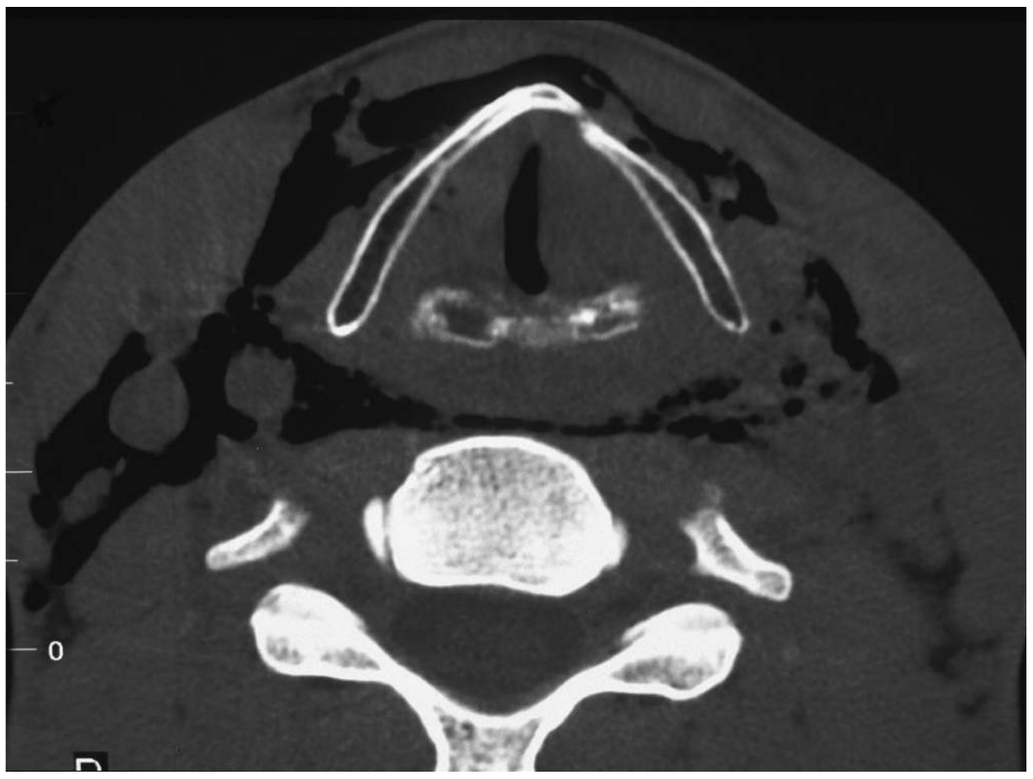


FIGURE 22.3 ■ **Laryngeal Fracture.** This 17-year-old male was kicked in the neck by a bull at a rodeo causing a laryngeal fracture. (Photo contributor: Rudy Kink, MD.)

FIGURE 22.4 ■ **Angioedema.** This patient with ACE-inhibitor angioedema and respiratory failure required awake fiber optic nasotracheal intubation by anesthesia. (Photo contributor: David Effron, MD.)

MAXIM: Most patients with airway/respiratory problems should be positioned for their comfort, not ours.

Clinical Summary

If mentating normally and physically able, a patient with airway difficulty will assume a position which optimizes their airway patency and gas exchange, usually sitting up and leaning forward. Such patients include those with incomplete airway obstruction, flash pulmonary edema, and massive airway bleeding from oropharyngeal trauma. Unfortunately, during preparation for intubation, such patients often are placed supine prematurely, increasing the patient's respiratory distress and anxiety, increasing the likelihood of spontaneous emesis and aspiration, and decreasing his ability to handle oropharyngeal bleeding or secretions. In these clinical situations, we should rethink the desire to immediately place a patient supine for endotracheal intubation. Intubation can be accomplished with the patient sitting up, by either of the following.

1. Altering the intubation technique (see Fig. 22.5).
2. Altering the intubator position relative to the patient (see Fig. 22.6).

Pearls

1. Keep patients in optimal position for spontaneous ventilation until they are sedated just prior to intubation; then, place them supine.
2. Consider intubating a patient sitting upright if you feel the supine position will compromise their ability to be ventilated.
3. Titrating ketamine in small doses (10 mg every 1-2 minutes) may facilitate awake intubation in a patient sitting upright.

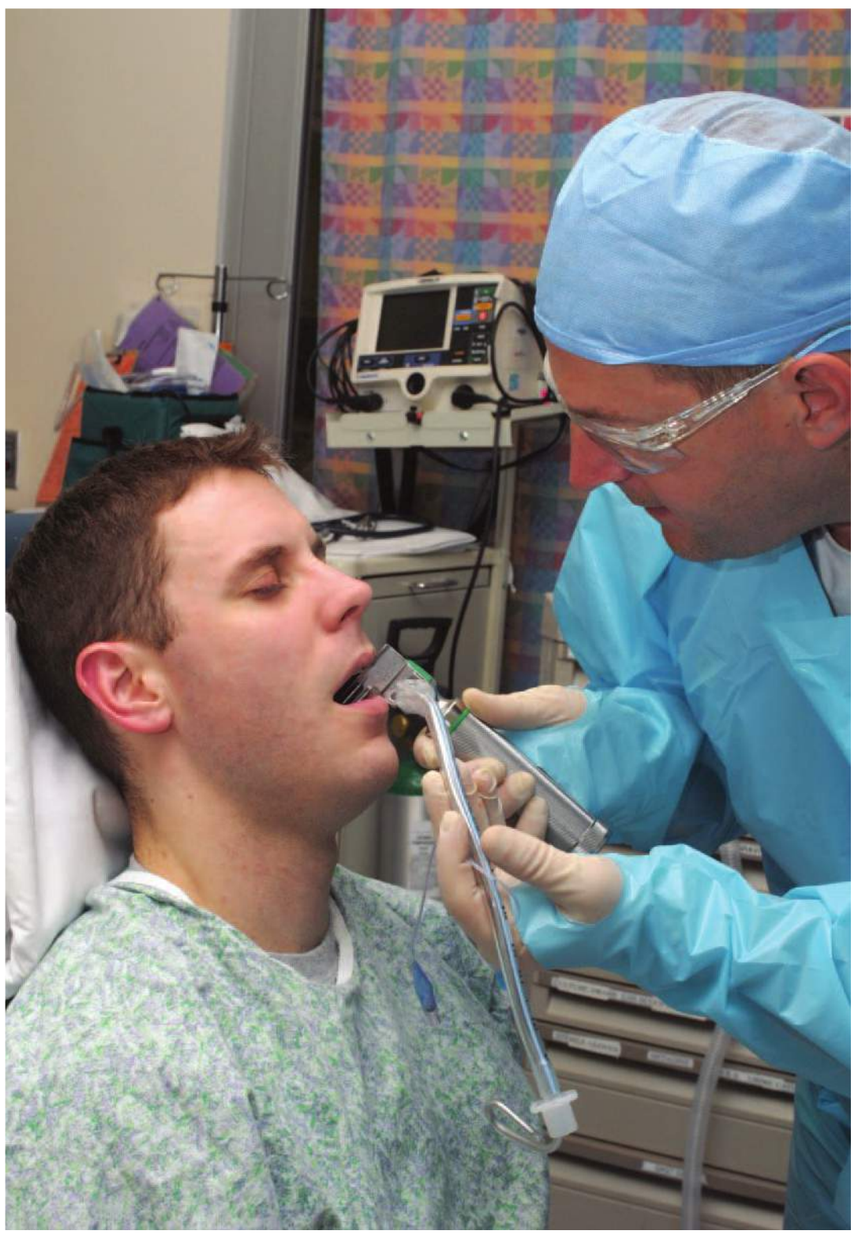


FIGURE 22.5 ■ **Intubation Facing the Sitting Patient or "Tomahawk Technique."** Holding the laryngoscope in the right hand and displacing the mandible forward and passage of the tube with the left hand is an alternative position to supine intubation. (Photo contributor: Lawrence B. Stack, MD.)

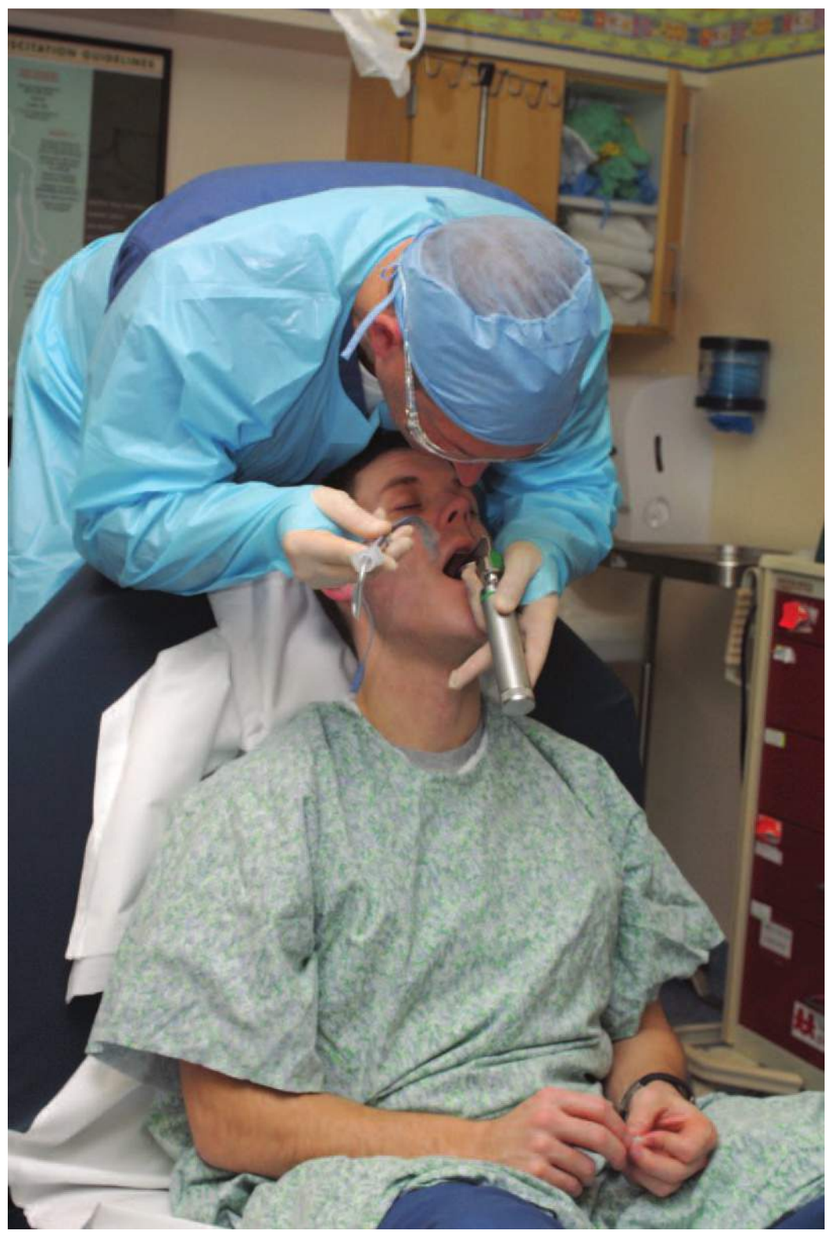


FIGURE 22.6 ■ **Intubation above the Sitting Patient.** Standing above a patient and intubating in the conventional manner is an alternative airway management position in a patient who may be difficult to ventilate in the supine position. An assistant may be necessary to hold the patient upright if sedated and/or paralyzed. (Photo contributor: Lawrence B. Stack, MD.)

MAXIM: Think before you paralyze.

Clinical Summary

Before committing to rapid-sequence induction (RSI) for direct laryngoscopy, consider the following:

1. Are any planned medications contraindicated?
2. Can rescue ventilation be achieved?
3. Is direct laryngoscopy possible? (See Fig. 22.7.)
4. What are my secondary and tertiary backup plans in the event of primary plan failure?
5. Are my equipment and personnel ready for RSI?

Pearls

1. A good backup plan to direct laryngoscopy should have at least one alternative intubation technique, one alternative ventilation technique, and one surgical airway technique.
2. Prepared equipment, correct patient position, proper drug dosing, having a backup plan, effective communication, and good technique will promote first-pass intubation (see Figs. 22.8 and 22.9).

FIGURE 22.8 ■ **Difficult Intubation.** This morbidly obese man with beard and no neck was successfully intubated after the third attempt. Ventilation was extremely difficult after paralysis. (Photo contributor: Lawrence E. Heiskell, MD.)

FIGURE 22.7 ■ **Wired Mandible.** This 33-year-old male with recent mandibular wire fixation of his mandibular fracture. A nasal or neck approach would be the only options for an emergency airway in this patient if no wire cutters were available. (Photo contributor: David Effron, MD.)

FIGURE 22.9 ■ **Difficult Intubation.** Anticipation of difficulty ventilating this patient if paralyzed prompted planning to optimize intubation success, which required three attempts. (Photo contributor: Lawrence E. Heiskell, MD.)

MAXIM: Patients will not die if they are not intubated; they will die if their lungs are not ventilated and their blood is not oxygenated.

Clinical Summary

The goal for airway management in any patient must be to maintain adequate ventilation and oxygenation. This does not necessarily mean intubation. Correct bag-valve-mask (BVM) ventilation/oxygenation technique is an underrated skill that will buy you time in the patient with a difficult airway. Proper steps for optimal two-person BVM ventilation include:

1. Positioning—ear-to-sternal notch alignment (when clinical scenario permits). Neck slightly flexed, head slightly extended (see Fig. 22.10).
2. Jaw thrust—displace mandible anteriorly with pressure from long, ring, and small fingers on mandible, not on soft tissues.
3. Mask compression—thumb and index fingers should apply firm pressure to face and nasal bridge (see Figs. 22.11 and 22.12).
4. Oral/nasal airways—may help maintain airway patency during BVM ventilation (see Fig. 22.13).
5. Use 7 mg/kg tidal volume, over 1 to 2 seconds at 12 breaths/min.

FIGURE 22.10 ■ **Optimal Ear-to-Sternal Notch Alignment.** Optimal position for ventilation and laryngoscopy occurs when the external auditory canal and the sternal notch are aligned in the horizontal plane (**B**) and the face is parallel to the ceiling (**C**). This position optimizes airway patency and ventilation mechanics. (Photo contributor: Lawrence B. Stack, MD.)

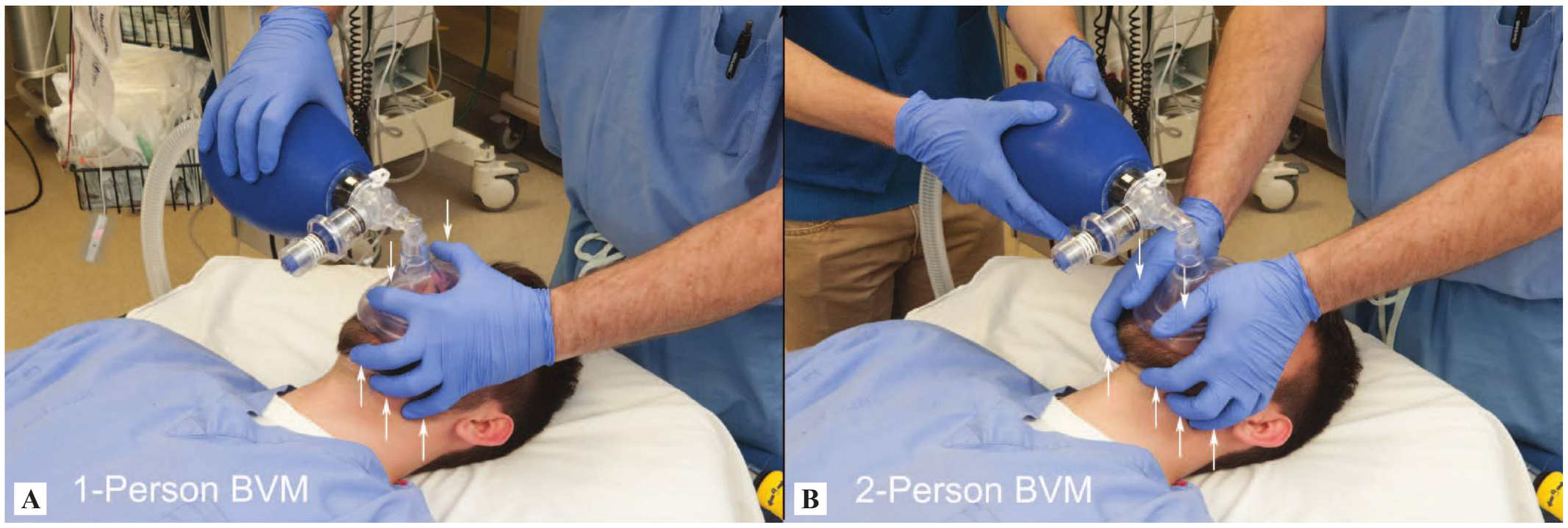


FIGURE 22.11 ■ **Bag-Valve-Mask Ventilation.** Correct positioning and forces during bag-valve-mask ventilation are demonstrated for the one- and two-person (thumbs) techniques. Upward force on the mandible, not soft tissue, is key to effective jaw-thrust technique. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Be an expert at BMV ventilation.
2. Ear-to-sternal notch positioning is most beneficial in obese patients and those with obstructive sleep apnea.
3. Keeping dentures in place facilitates BVM, while removing them facilitates orotracheal intubation.
4. Midface and mandibular disfiguration from whatever cause will interfere with optimal BVM ventilation.
5. Consider LMA, King LT, or Air-Q if unable to intubate or obtain adequate seal for BVM ventilation.

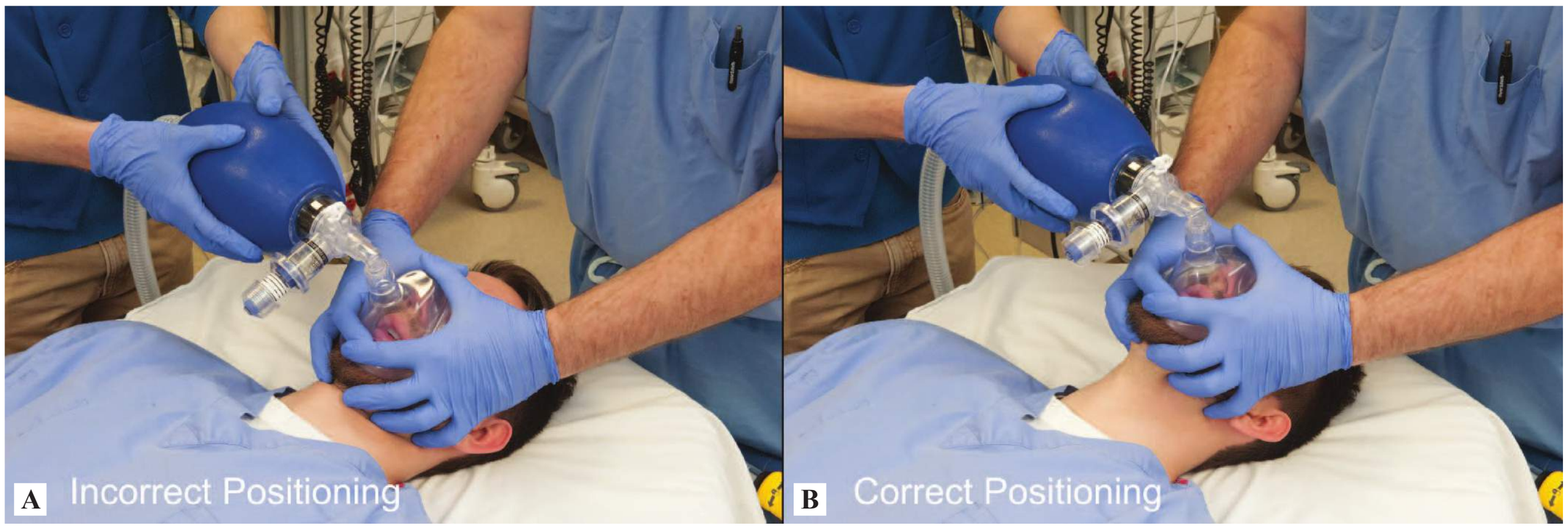


FIGURE 22.12 ■ **Bag-Valve-Mask Ventilation.** Incorrect (**A**) and correct (**B**) positioning and forces during bag-valve-mask ventilation are demonstrated for the two-person techniques. Lack of jaw thrust during BVM will allow the tongue to block air passage. (Photo contributor: Lawrence B. Stack, MD.)

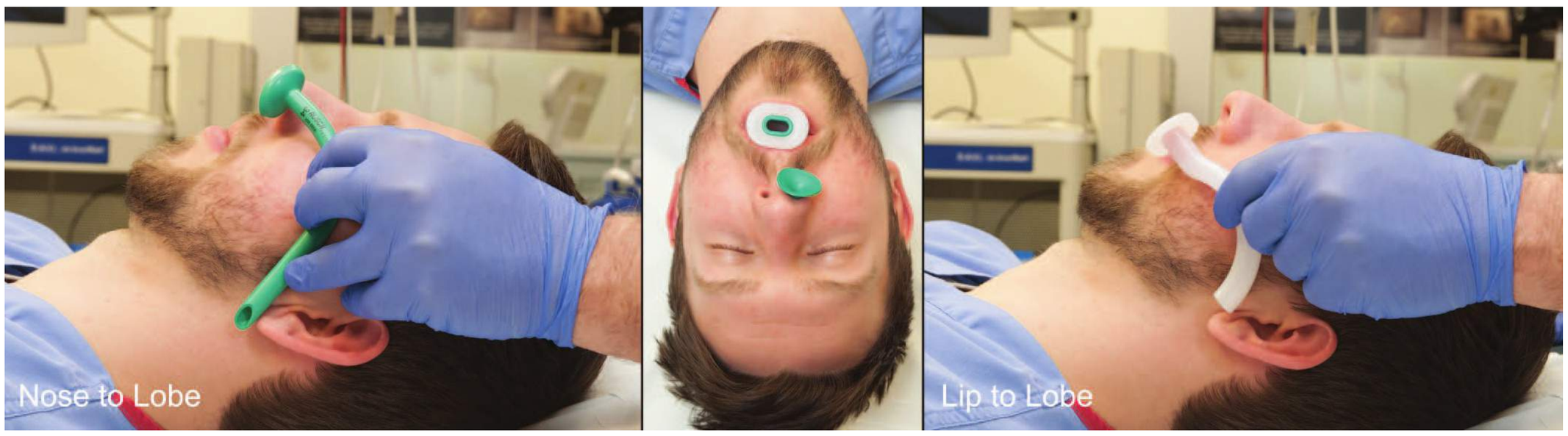


FIGURE 22.13 ■ **Nasal and Oral Airways.** Appropriately sized and placed nasal and oral airways maximize upper airway patency during bag-valve-mask ventilation. (Photo contributor: Lawrence B. Stack, MD.)

Patient Positioning

The optimal position to maximize laryngoscopic visualization of the larynx is:

1. The head extended.
2. The neck flexed.
3. The base of the ear is aligned with the sternal notch (see Fig. 22.10).
4. The facial plane is horizontal, parallel to the ceiling (see Fig. 22.10).
5. The facial plane should be positioned between the laryngoscopist's xiphoid process and umbilicus (see Fig. 22.14). This position most closely replicates in a supine posture which the patient would assume sitting up. For very large individuals or those with significant morbid obesity, this may require creation of a textile ramp of blankets, sheets, or towels to raise the head and shoulders to proper elevation and alignment.

Preoxygenation and Passive Apneic Oxygenation

Preoxygenation, when possible, should be used to create an oxygen surplus in the blood and tissue which permits a period of apnea to occur without arterial oxygen desaturation. This process hopefully begins in the prehospital setting with the placement of a nonrebreather mask with high-flow oxygen on the patient. This should continue in the ED in the event intubation is required. In addition, a nasal cannula on high flow (15 L/min) should also be placed when the patient arrives in the ED if there is a high likelihood of intubation. High-flow oxygen delivered by nasal cannula provides passive apneic oxygenation during the apneic phase of endotracheal intubation.

Technique

Stylet Shape

A stylet should be used with all oral intubations in which a laryngoscope is employed. The laryngoscopist will typically

FIGURE 22.14 ■ **Correct Intubating Height.** Optimal height for intubation is typically when the plane of the patient's face is between the intubator's umbilicus and xiphoid process. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.15 ■ **Preoxygenation and Passive Oxygenation.** In preparation for intubation, simultaneously placing an NRBM (preoxygenation before induction) and nasal cannula (passive oxygenation after paralysis) at 15 L/min will help prolong tissue oxygenation during the apneic phase of intubation. (Photo contributor: Lawrence B. Stack, MD.)

fashion an inexpensive stylet, essentially a malleable wire, into a shape of their preference, to permit control of the distal endotracheal tube (ETT) tip. One should take care not to place too much bend or curvature to the stylet, because such a configuration can actually impair the glottic view and control of the ETT tip. Optimal stylet configuration has been described as "straight to the cuff" and then a gentle anterior bend of 30 degrees.

Even with a correct bend on the stylet, it may be difficult to advance the styletted ETT into the trachea. The tip of the left-sided beveled tube can impact and catch on the anterior tracheal cartilages, preventing advancement. This can often be remedied with a generous clockwise or rightward rotation of tube and stylet, which acts to rotate the bevel anteriorly and depress the ETT tip.

Optimizing Biomechanics

Holding the laryngoscope handle and blade where the proximal end of the blade is in the palm of the hand creates a natural extension of the forearm and provides fine control of the blade tip. The left thumb should run parallel to the axis of the handle. Effort mechanics are more efficient with this grip. Placing the elbow close to the body requires less effort and more

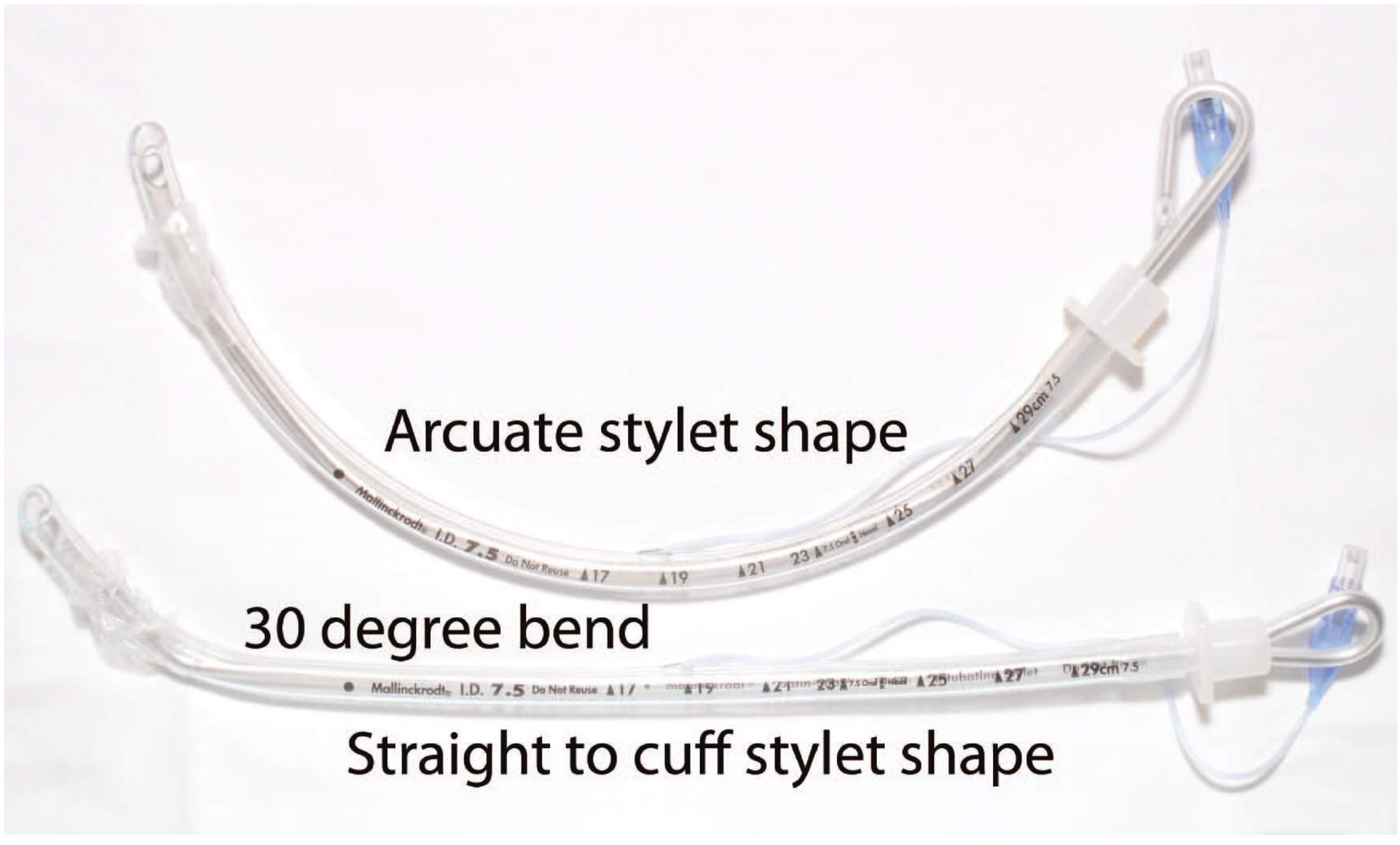


FIGURE 22.16 ■ **Stylet Shape.** Straight to cuff with a 30-degree bend is the optimal stylet shape as this offers the most ETT tip control and view of the glottic opening. (Photo contributor: Lawrence B. Stack, MD.)

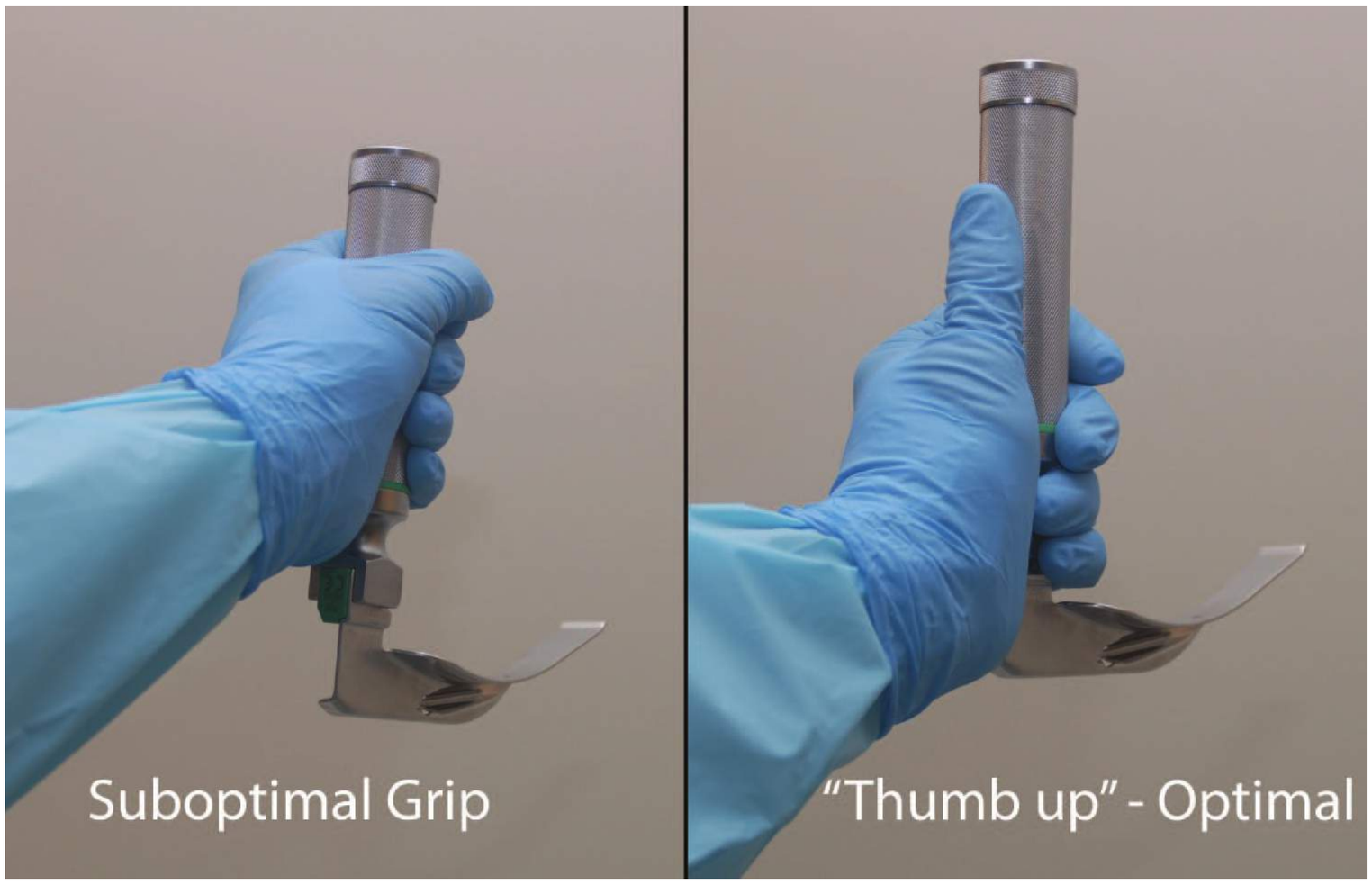


FIGURE 22.17 ■ **Laryngoscope Grip.** The laryngoscope should be gripped as low as possible and the thumb extended. This makes a natural extension of the forearm. (Photo contributor: Lawrence B. Stack, MD.)

mechanical control than if the elbow abducted from the body. A memory aid for these concepts are "Thumb up-Wing in."

Mouth Opening Techniques

The scissor technique places the laryngoscopist's right thumb on the occlusive surface of the patient's lateral mandibular teeth or gum and long or index finger on the maxillary teeth or gum. A scissoring motion opens the mouth allowing blade insertion. The fingers should be removed once the blade is adequately inserted. An alternative technique is to place the laryngoscopist's right thumb on the nasal bridge and long finger between the mental protuberance and alveolar margin. The thumb and long finger are moved away from each other, causing the mouth to open.

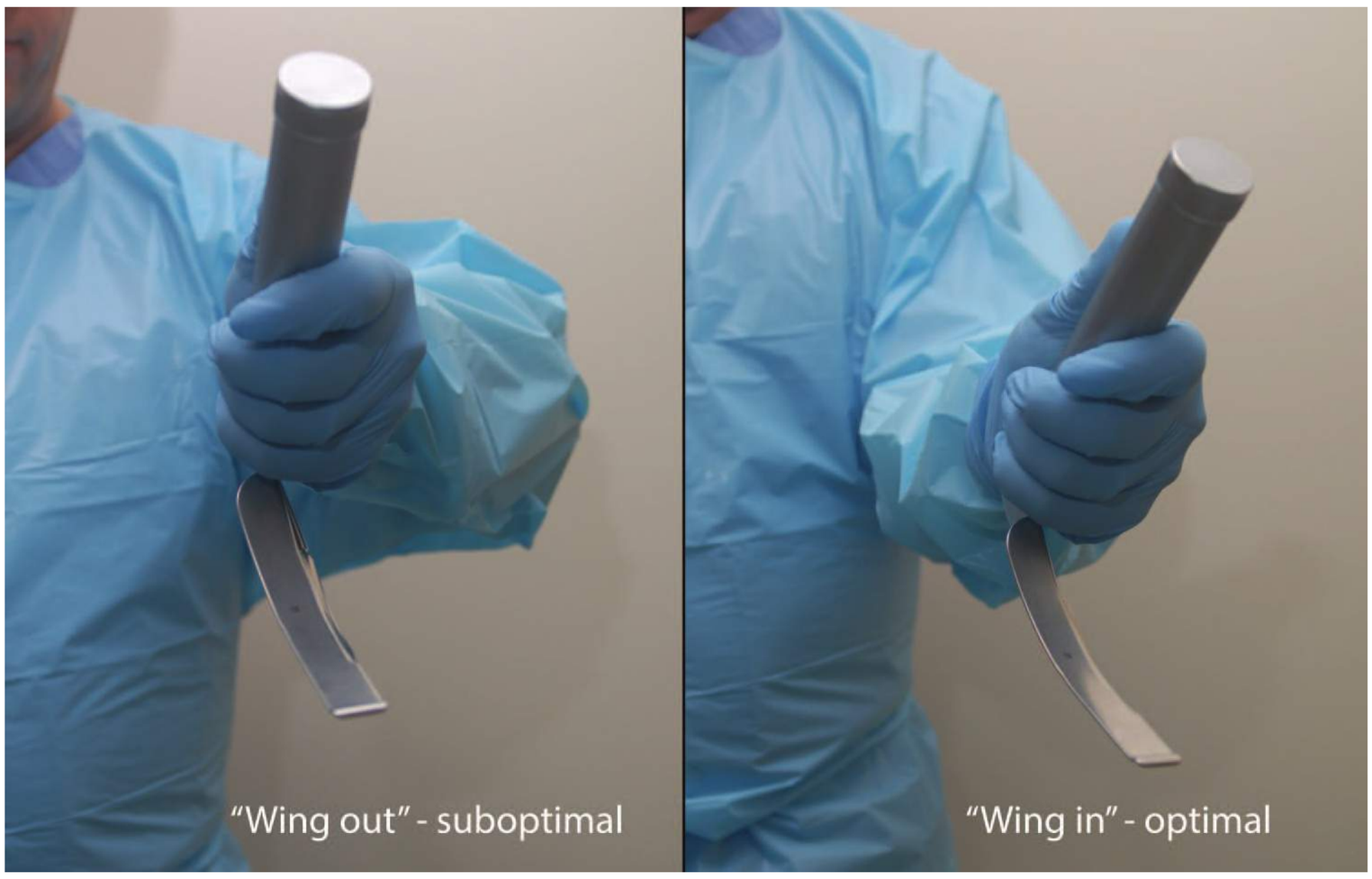


FIGURE 22.18 ■ **Laryngoscope Biomechanics.** Keeping the elbow close to the body requires less effort and more control during laryngoscopy. (Photo contributor: Lawrence B. Stack, MD.)

Blade Insertion

The blade is initially inserted just to the right of midline with the handle pointed toward the patient's feet. As the tip of the blade advances downward along the tongue base and into vallecula, the tongue is swept slightly to the left to increase the amount of workspace. When fully inserted into the vallecula, the angle of the handle is now approximately 40 degrees from horizontal with lifting forces directed upward and forward.

Optimal Epiglottoscopy

The key to using the laryngoscope to optimally visualize the glottic opening is to first visualize and control the epiglottis, a relatively fixed anterior structure. Pulmonary secretions, blood, and/or vomitus that pools in the posterior pharynx may obscure the posterior laryngeal structures, dependent with gravity. The epiglottis itself may be camouflaged in this pool of muck (see Fig. 22.20). One should carefully insert the laryngoscope blade with a goal of adequately visualizing and controlling the epiglottis. Failure to do so risks inserting the laryngoscope blade too deeply, and often results in displacing the larynx anteriorly to expose the esophagus, which, as a consequence of anterior-ward tension on the laryngoscope blade, may then look like a glottic opening begging for a tube. By carefully controlling the epiglottis, the intubating clinician will be able to locate important airway landmarks. Be careful to displace the tongue to the left side as you insert the blade. If the bulk of the tongue wraps around the blade, it can both impair your view and impede insertion of the ETT.

Progressive visualization of laryngeal structures is predictable:

1. Uvula and posterior pharynx (see Fig. 22.21A)
2. Epiglottis (see Fig. 22.21B)
3. Posterior arytenoids cartilages and interarytenoid notch (see Fig. 22.21C)
4. Glottic opening (see Fig. 22.21D)
5. Vocal cords (see Fig. 22.21E)
6. Esophagus (see Fig. 22.21F)

Bimanual Laryngoscopy

Even with well-performed laryngoscopy, adequate visualization of the laryngeal structures may be difficult. Bimanual laryngoscopy (external laryngeal manipulation by the operator), where the laryngoscopist performs laryngoscopy while simultaneously manipulating the external larynx, facilitates optimal visualization. This eliminates any delay or miscommunication between an assistant and laryngoscopist. Once optimal position is found, an assistant can maintain that position. Alternatively, an assistant's hand, placed on the laryngeal structures, guided by the laryngoscopist's hand can maintain optimal position after the laryngoscopist removes his hand from the assistant's hand.

Lip Commissure Retraction

The lip commissure is the junction of the upper and lower lips. Lateral retraction of the patient's right lip commissure by an assistant facilitates visualization of oral structures and insertion of the ETT into the oral cavity. This creates an optimal workspace for tube delivery. After glottis visualization is achieved, an assistant places their right index finger in the patient's right commissure and thumb against the maxilla. The index finger retracts the commissure laterally while applying an opposing force with the thumb to avoid excessive movement of the head. A memory aid of this concept is "pull out commissure, push on maxilla," or "POC-POM." Retraction of the lip commissure aids in keeping the ETT from

FIGURE 22.19 ■ **Mouth Opening Techniques.** The traditional "scissor" technique (**A**) and the "clean hand" technique (**B**) for opening the mouth during endotracheal intubation. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.20 ■ **Epiglottic Camouflage.** Vomitus, blood, and pulmonary secretions may pool in the posterior pharynx and obscure the gravity-dependent epiglottis. Elevating anterior laryngeal structures will expose the epiglottis. (Photo contributor: Lawrence B. Stack, MD.)

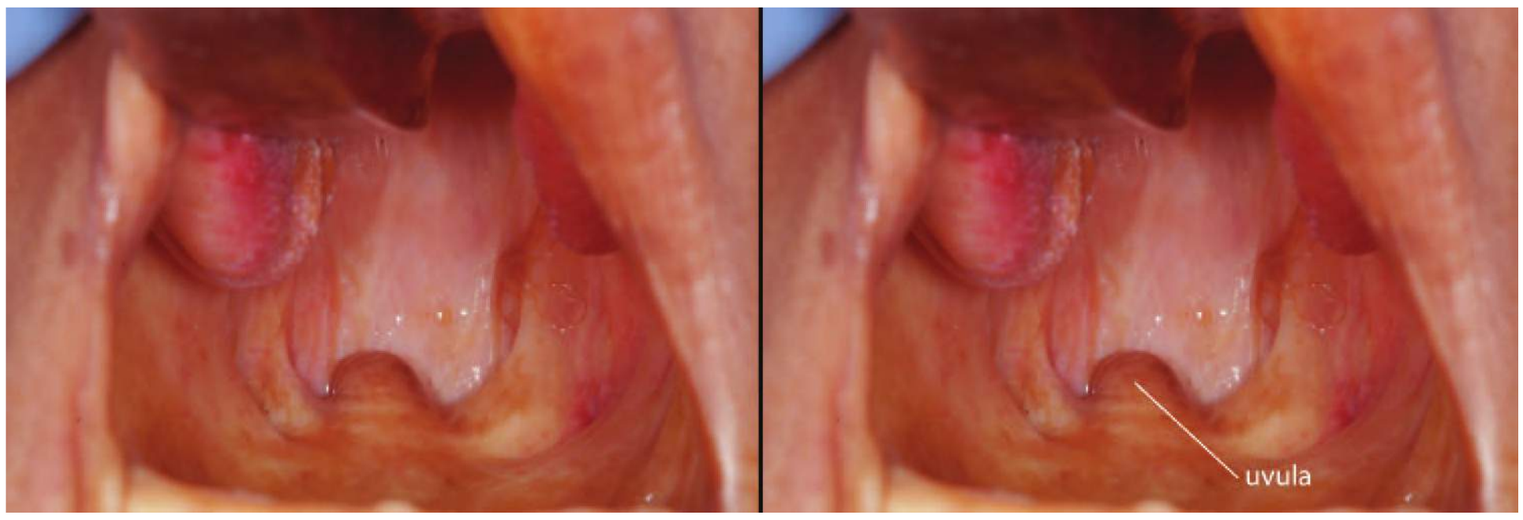


FIGURE 22.21A ■ **Progressive Visualization—Uvula.** The uvula and posterior pharynx are the first structures visualized during correct laryngoscopic technique. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.21B ■ **Progressive Visualization—Epiglottis.** The key to first-pass intubation is finding and identifying the epiglottis laryngoscopy. Following epiglottis will take one to the glottic opening. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.21C ■ **Progressive Visualization—Interarytenoid Notch.** The interarytenoid notch is a vertical cleft between the posterior cartilages. Above the notch lies the glottic opening. Below the notch is the esophagus. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.21D ■ **Progressive Visualization—Glottic Opening.** The posterior glottic opening is seen before the vocal cords are visualized. (Photo contributor: Lawrence B. Stack, MD.)

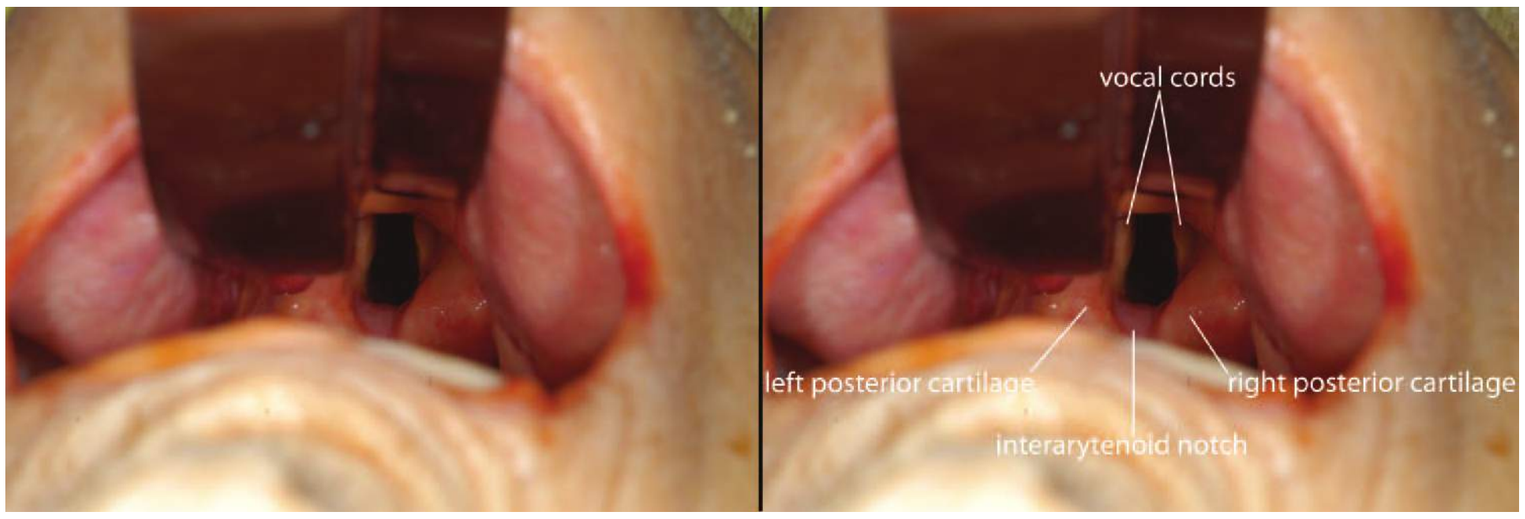


FIGURE 22.21E ■ **Progressive Visualization During Laryngoscopy—Vocal Cords.** The vocal cords have a distinct white appearance. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.21F ■ **Progressive Visualization During Laryngoscopy—Esophagus.** The esophagus lies directly below the interarytenoid notch. (Photo contributor: Lawrence B. Stack, MD.)

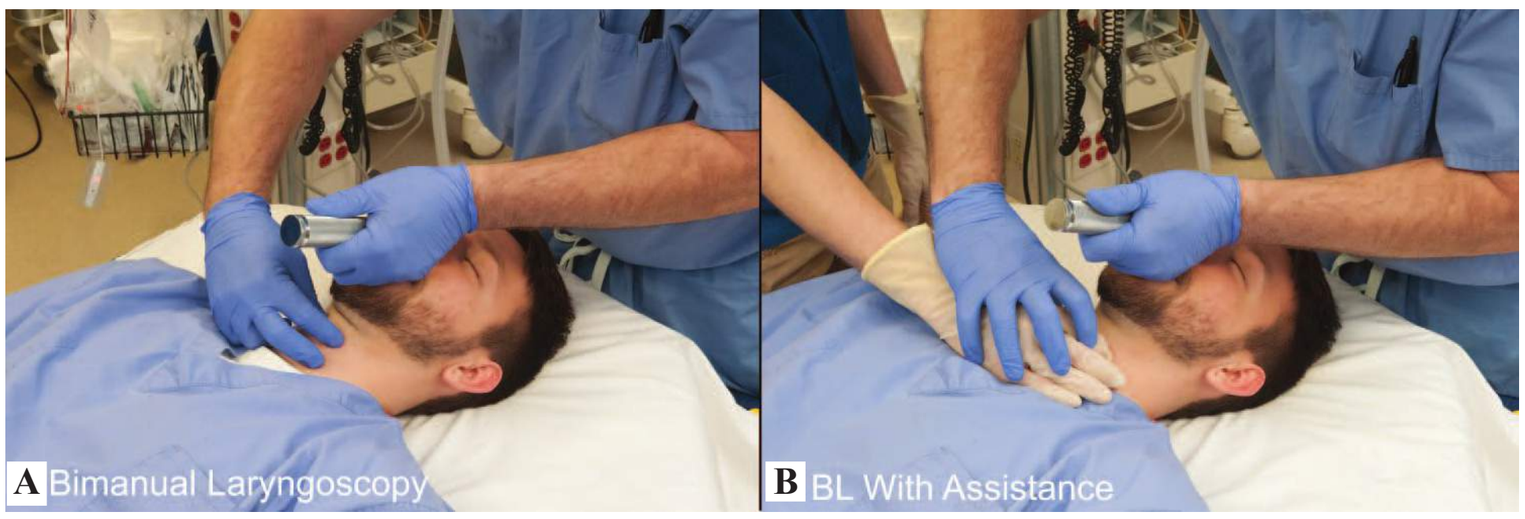


FIGURE 22.22 ■ **Bimanual Laryngoscopy.** Laryngoscopist uses right hand to manipulate laryngeal structures for optimal visualization during laryngoscopy. Once optimal position is identified, an assistant maintains it during intubation. (Photo contributor: Lawrence B. Stack, MD.)

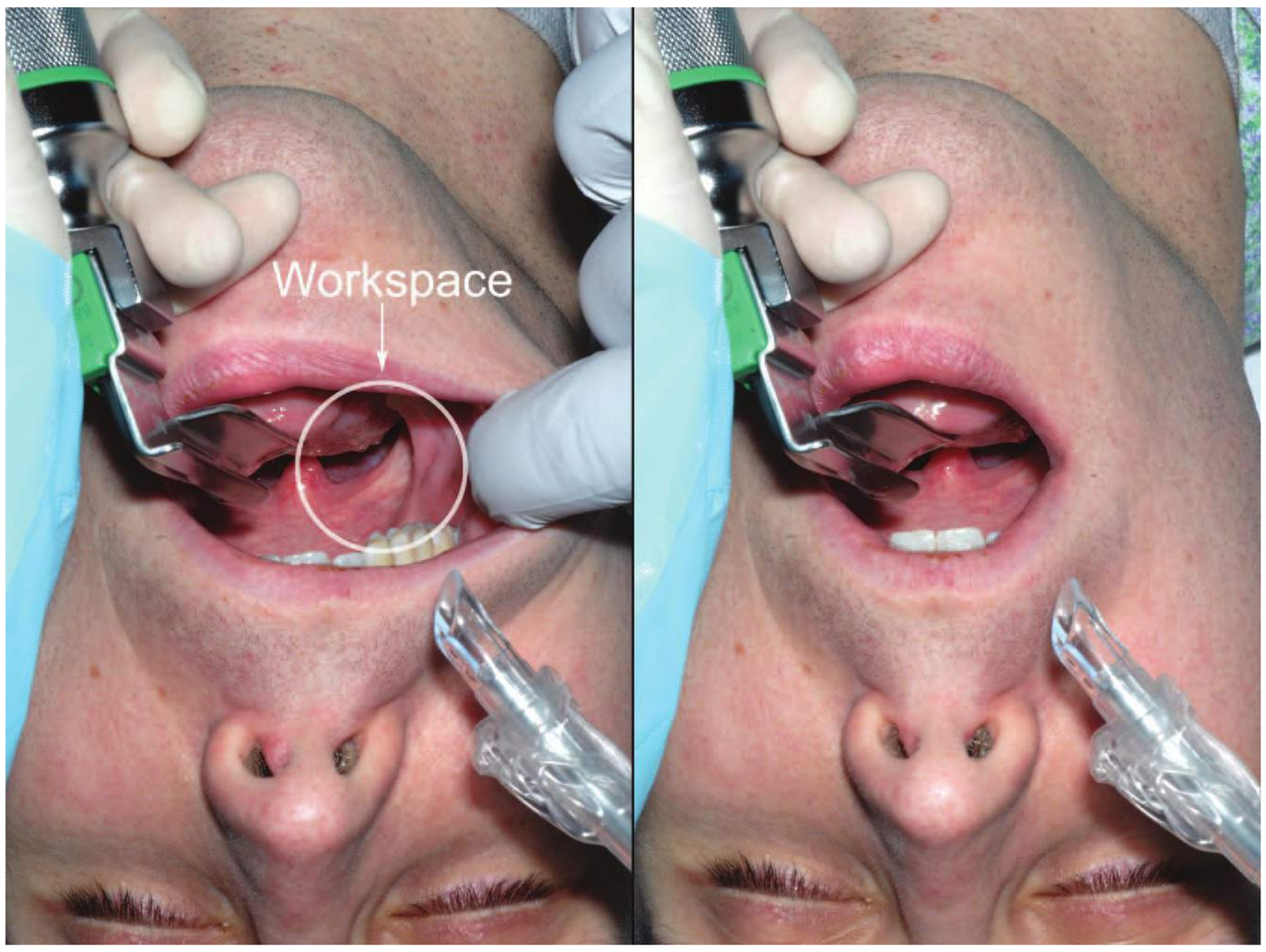


FIGURE 22.23 ■ **Commissure Retraction.** An assistant retracts the right commissure to create a generous "workspace" to visualize and deliver the endotracheal tube. (Photo contributor: Lawrence B. Stack, MD.)

blocking the view of the glottic opening during advancement of the ETT. Using the retracted lip commissure as a fulcrum or "toggle" gives optimal control of the ETT tip, especially when the stylet is in the "straight to cuff" shape. Retraction may also facilitate intubation by allowing clockwise rotation of the tube if the ETT tip becomes hung up on the proximal tracheal cartilages. A memory aid of this concept is "right on rings" or rotate the tube to the right or clockwise if it hangs up on the tracheal rings.

Endotracheal Tube Handling

The thumb, index, and long fingers provide the fine dexterity for the hand, and should be used to control the tube during endotracheal intubation. The ETT should be held like a "throwing dart" between the thumb, index, and long fingers midway along the tube for precise control during the procedure. A trained assistant should hand the tube to the laryngoscopist in this manner.

Endotracheal Tube Delivery

Obtain the best glottic view and have an assistant provide right lip commissure retraction. Introduce the ETT at the 3-o'clock position of the mouth. Advance the tube posteriorly in a manner that will not obstruct the direct view of the "target." Advance the tube through the glottis. The last thing you should see is

FIGURE 22.24 ■ **Pull on Commissure-Push on Maxilla.** When pulling on the commissure with the index finger, simultaneous pushing on the maxilla with the thumb will minimize head movement during intubation. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.25 ■ **Correct ETT Grip.** Using the thumb, index, and long fingers to grip the mid-endotracheal tube like a "throwing dart" will provide the most precise control during tube deliver. (Photo contributor: Lawrence B. Stack, MD.)

the tube going through cords. If resistance is met, it is most likely due to the bevel being caught on a tracheal ring. Turn the tube to the right or clockwise and attempt to advance the tube. Advance the tube to 22 cm at the lip, inflate the cuff, and confirm placement. Secure the tube in place (see Table 22.1).

Pearls

1. The key to first-pass intubation success is identification and control of the epiglottis during direct laryngoscopy.
2. The glottic opening lies between the epiglottis and interarytenoid notch. Identification and passing the tube between these structures will improve first-pass success.
3. While most intubations are performed without difficulty, if a difficult airway is anticipated and there is time to prepare, optimize the patient's ear-to sternal notch position.
4. Approximately 250 mL/min of oxygen will move from the alveoli into the blood during apnea, which may extend the period of adequate arterial oxygenation during intubation.

FIGURE 22.26 ■ **Incorrect ETT Grip.** These ETT grips will provide a powerful force on the tube but will lack precise handling of the tube, critical to tube delivery. (Photo contributor: Lawrence B. Stack, MD.)

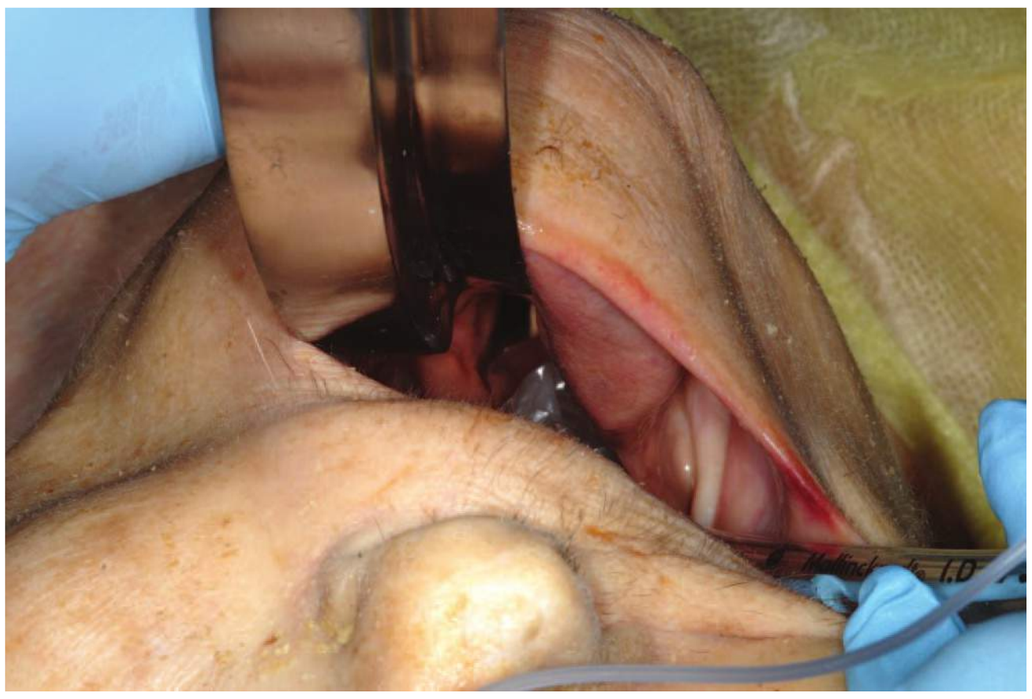


FIGURE 22.27 ■ **Tube Delivery.** Obtain the best glottic view. Introduce the ETT at the 3-o'clock position of the mouth. Advance the tube posteriorly in a manner not to obstruct the view of the "target." Advance the tube through the glottis. (Photo contributor: Lawrence B. Stack, MD.)

TABLE 22.1 ■ ENDOTRACHEAL TUBE (ETT) DELIVERY STEPS

- Obtain best possible glottic view
- Introduce ETT at 3-o'clock position of the mouth
- Advance ETT posteriorly to avoid obstruction of "target" view
- Advance ETT through glottis (last thing to see is ETT through cords)
- If resistance is met (bevel against rings), rotate tube clockwise, then advance
- Inflate cuff
- Confirm tube placement (ETCO2, breath sounds, O_2 saturation monitor)
- Secure tube

Technique

The Eschmann stylet, also known as a "gum elastic bougie" or simply "bougie," is a 60-cm long, flexible introducer, which is designed to assist intubation of the anterior larynx, especially those with an "epiglottis-only" view. The tip of the bougie has an anterior fixed flexion with an angle of 40 degrees to facilitate entering an anterior glottic opening. Using a laryngoscope, one obtains the best view possible of the glottic opening. In the case of limited glottic view, the bougie is inserted such that the tip is introduced just under the epiglottis and probes for the glottic opening. The intubator should feel a tactile "pop" as the bougie enters the trachea and he or she may also have a tactile sensation of "speed bumps" as the bougie is advanced and tracks across the tracheal cartilaginous rings. However, the more sensitive indicator of tracheal bougie position is the resistance encountered as the tip abuts against the carina, at approximately 27 to 30 cm. Should the bougie be inserted into the esophagus, no such endpoint is encountered.

While continuing to hold anterior traction with the laryngoscope, the laryngoscopist should direct an assistant to thread the ETT over the bougie again while maintaining anterior traction; the intubator should then advance the ETT over the bougie to the appropriate insertion depth. Resistance to tube advancement may indicate "arytenoid arrest" and can be remedied by rotating the tube counterclockwise 90 degrees followed by attempts to advance the ETT tip past the arytenoid cartilages.

A one-handed technique can be employed in which the ETT is preloaded onto the bougie, which is then curled in the right hand with approximately 20 cm of bougie protruding through the distal end. The laryngoscopist performs laryngoscopy as before, maintains anterior traction on the mandible, and inserts the bougie through the glottic opening as above. He or she then uncoils the ETT/bougie and advances the pair until resistance of the carina is met, confirming intratracheal position. The ETT is then advanced to the proper depth and the bougie is removed.

Use of the bougie does not have to be limited to the anticipated difficult airway. In fact, to gain proficiency, we recommend that practitioners use it as their routine stylet, as it has come to be used in the UK. The practitioner will then be better prepared for the unanticipated anterior larynx/limited view airway.

FIGURE 22.28 ■ **Bougie Tip Shape.** The bougie tip has a 40-degree bend to help facilitate entering the glottic opening when the glottis is not adequately visualized. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.29 ■ **Epiglottis-Only View.** In this view, despite optimal laryngoscopy, the epiglottis may be all that is seen. The bougie may be a reasonable alternative in this situation (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.30 ■ **Passing the Bougie.** The two-person technique for using the bougie. Standard DL is preformed and the bougie is used like a stylet, but hugging the underside of the epiglottis. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.31 ■ **Bougie under the Epiglottis.** Here the bougie is hugging the underside of the epiglottis. Tracheal rings can be felt by "tactile speed bumps," confirming correct placement. (Photo contributor: Lawrence B. Stack, MD.)

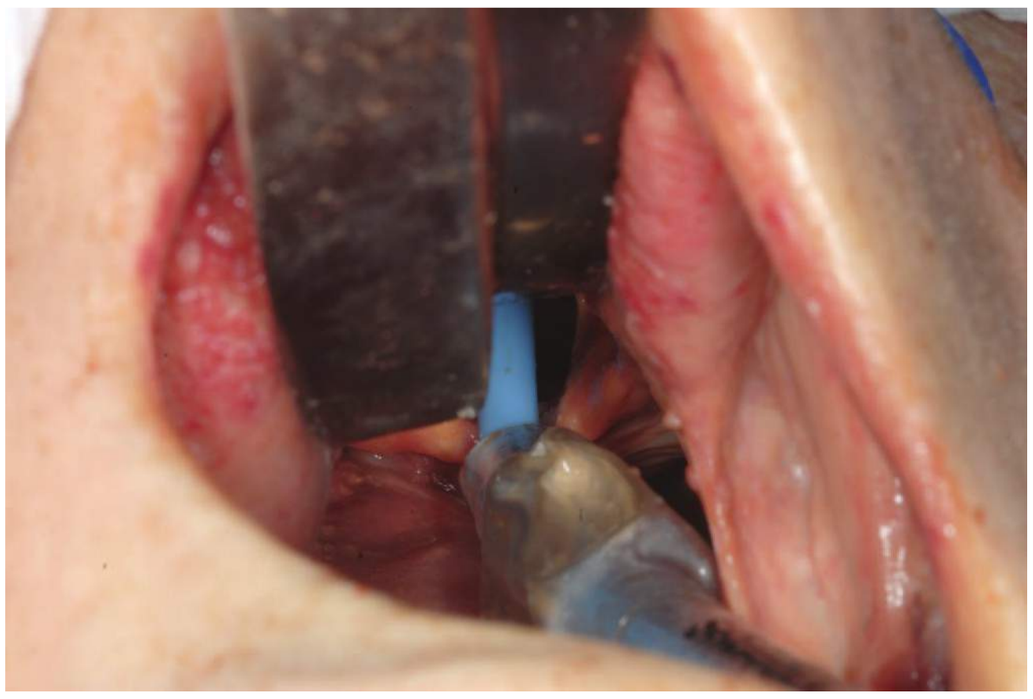


FIGURE 22.33 ■ **Tube Advancement Stall.** Stalling of tube advancement while using the bougie is most likely due to catching the right arytenoids cartilage with the bevel of the ETT. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.32 ■ **Tube over the Bougie.** An assistant places the bougie over the tube while direct laryngoscopy is maintained. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.34 ■ **Arytenoid "Escape."** Turning the tube counterclockwise, as shown here, will release the bevel of the ETT from the arytenoid cartilage and allow the tube to advance. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.35 ■ **One-Handed Bougie Configurations.** Holding the bougie in one of these configurations will allow one person to pass the bougie. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. The bougie is an excellent adjunct in the limited view or "epiglottis view" airway.
2. Bougie-assisted intubations is best deployed as a two-person technique.
3. Stall of ETT advancement when using the bougie is most frequently due to catching the bevel on the right arytenoid cartilage and can be relieved by rotating the tube counterclockwise.

FIGURE 22.36 ■ **One-Handed Bougie Technique.** Passing the bougie using the one-handed technique. See text for details. (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The Glidescope is a video laryngoscope composed of an integrated blade/handle which houses a blade tip–mounted camera and light source, connected by cable to a video LCD display. The Glidescope affords an excellent view of the laryngeal structures with little anteriorly directed force. Use of proprietary stylet, conformed to angle of Glidescope blade, is recommended. A more portable version, suitable for out-of-hospital use, is marketed as the Glidescope Ranger.

Technique

The blade is inserted into the mouth, along the center of the tongue, and advanced to visualize the epiglottis and laryngeal structures. Little-to-no anterior force is required, and in contrast to standard laryngoscope, levering the blade is recommended to enhance the view. Once the tube is passed through the cords, the proprietary stylet is withdrawn 5 cm to allow further passage of the tube. The steep angle of Glidescope blade and stylet causes the tube to abut the anterior tracheal rings and arrest tube delivery in most patients. Withdrawing the stylet 5 cm and rotating the tube clockwise will allow complete tube delivery to an appropriate depth.

FIGURE 22.37 ■ **Glidescope.** The Glidescope video LDC display on a stand with blade (inset) and proprietary stylet (inset). (Photo contributor: Lawrence B. Stack, MD.)

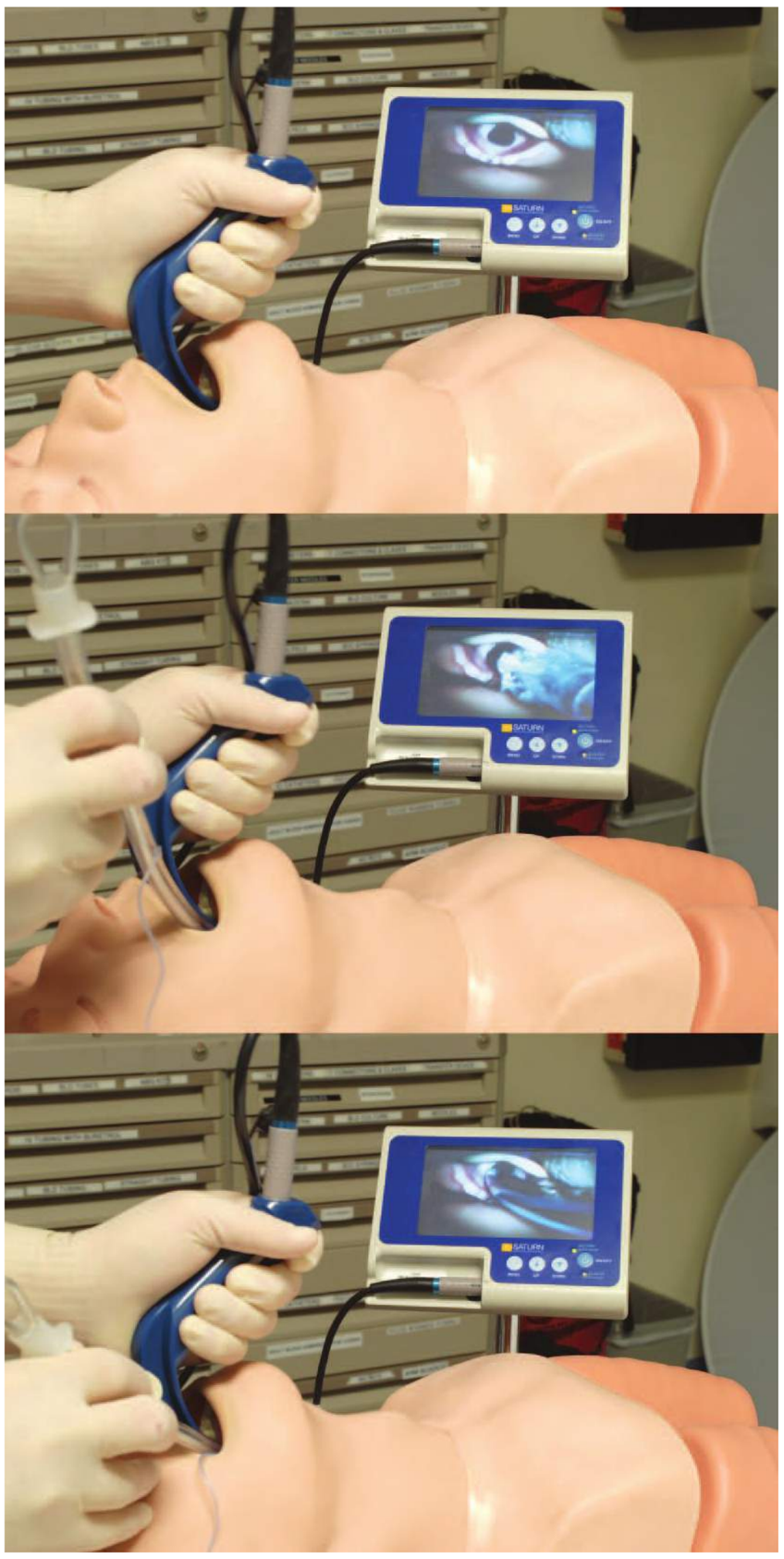


FIGURE 22.38 ■ **Glidescope in Use.** The Glidescope in action demonstrating progressive images of passage of an ETT. (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The Storz C-MAC video laryngoscope system consists of stainless steel, reusable blades, which house a CMOS camera chip and high-output LED light, an interchangeable powered coupling, video screen, and mobile tower. Available blades are similar in size and shape to the Mac 2, Mac 3, Mac 4, Miller 0, Miller 1, and a D-blade designed for patients with difficult airway anatomy. The near-standard shape of the blade allows direct laryngoscopy for glottic visualization as well as video laryngoscopy.

Technique

The blade connected to the power coupling should be handled like a standard laryngoscope blade. It should be inserted just to the right of midline of the tongue with the handle pointing toward the feet. As the tip of the blade advances downward along the tongue base and into vallecula, the tongue is swept slightly to the left to increase the amount of workspace. When fully inserted into the vallecula, the angle of the handle is now approximately 40 degrees from horizontal with lifting forces directed upward and forward. At anytime, the laryngoscopist can switch from a standard direct laryngoscopy to a video laryngoscopy; however, once changed to video laryngoscopy, it is best not to switch back and forth from video laryngoscopy to direct laryngoscopy.

FIGURE 22.39 ■ **Storz C-MAC Tower.** Storz video laryngoscope system including tower and adult blades. (Photo contributor: Lawrence B. Stack, MD)

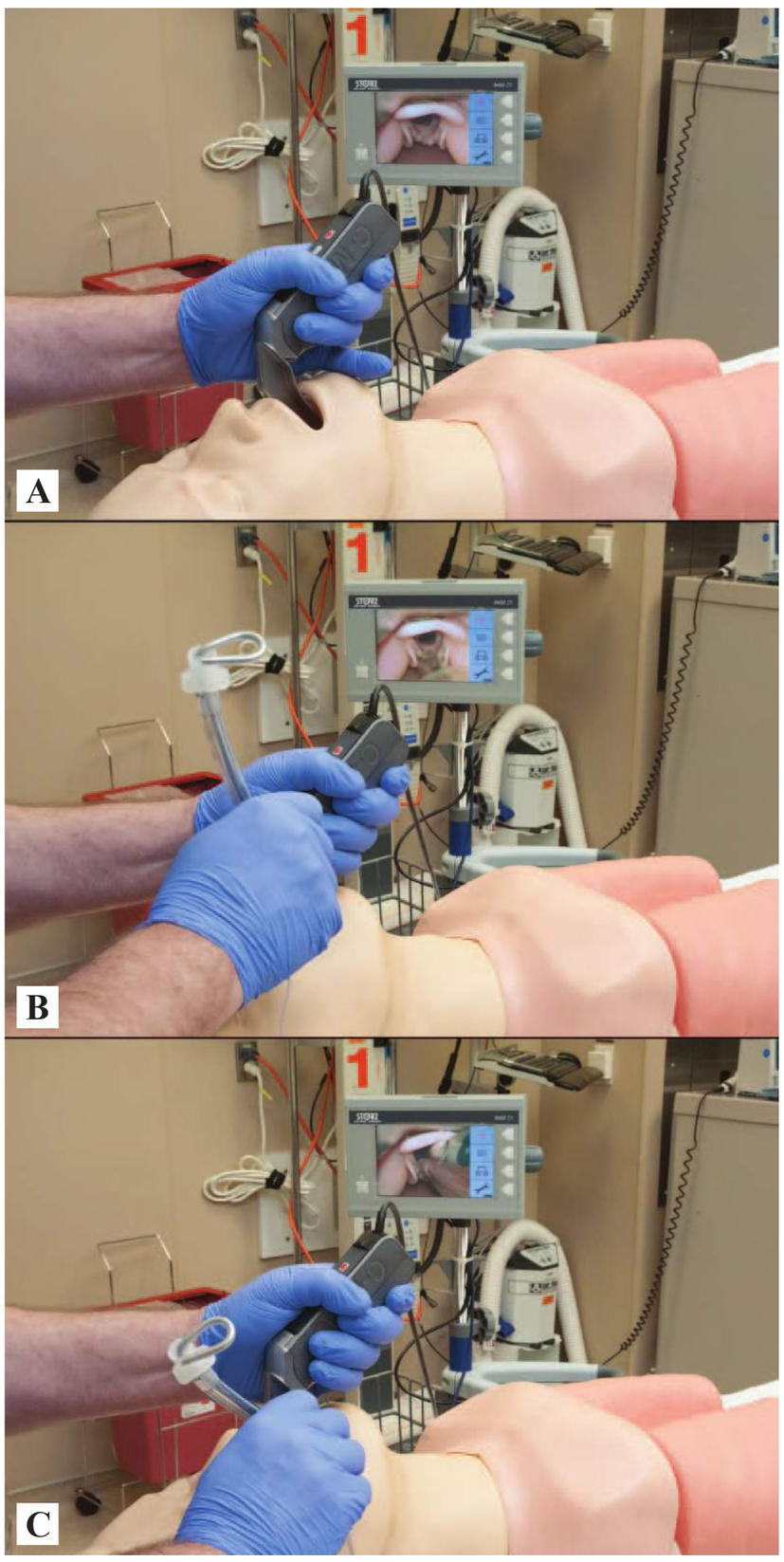


FIGURE 22.40 ■ **Storz C-MAC in Use.** The Storz C-MAC in action demonstrating progressive images of passage of an ETT. (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The McGrath MAC enhanced direct laryngoscope is a combination of direct and video laryngoscope in a compact, battery-powered, device that uses disposable standard curved Macintosh blades and has an adjustable LED screen. It can be used in direct laryngoscopy and video laryngoscopic situations.

Technique

The disposable plastic Macintosh blade is placed onto the McGrath MAC EDL and the device is powered on. The blade is inserted just to the right of midline of the tongue with the handle pointing toward the feet. As the tip of the blade advances downward along the tongue base and into vallecula, the tongue is swept slightly to the left to increase the amount of workspace. When fully inserted into the vallecula, the angle of the handle is now approximately 40 degrees from horizontal with lifting forces directed upward and forward. At anytime, the laryngoscopist can switch from a standard direct laryngoscopy to a video laryngoscopy; however, once changed to video laryngoscopy, it is best not to switch back and forth from video laryngoscopy to direct laryngoscopy.

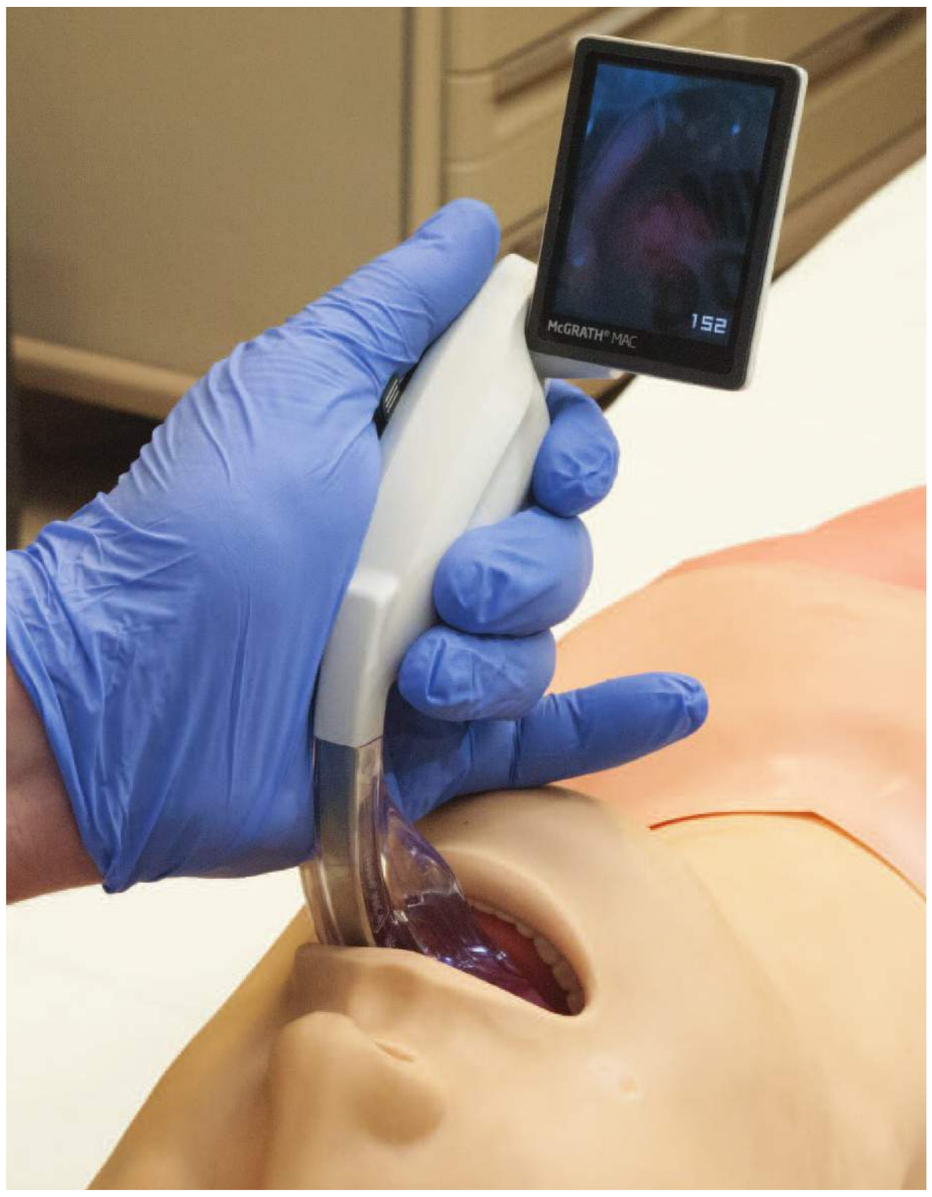


FIGURE 22.42 ■ **McGrath MAC EDL.** Cord visualization using the McGrath MAC which can also be used as a direct laryngoscope. (Photo contributor: Lawrence B. Stack, MD.)

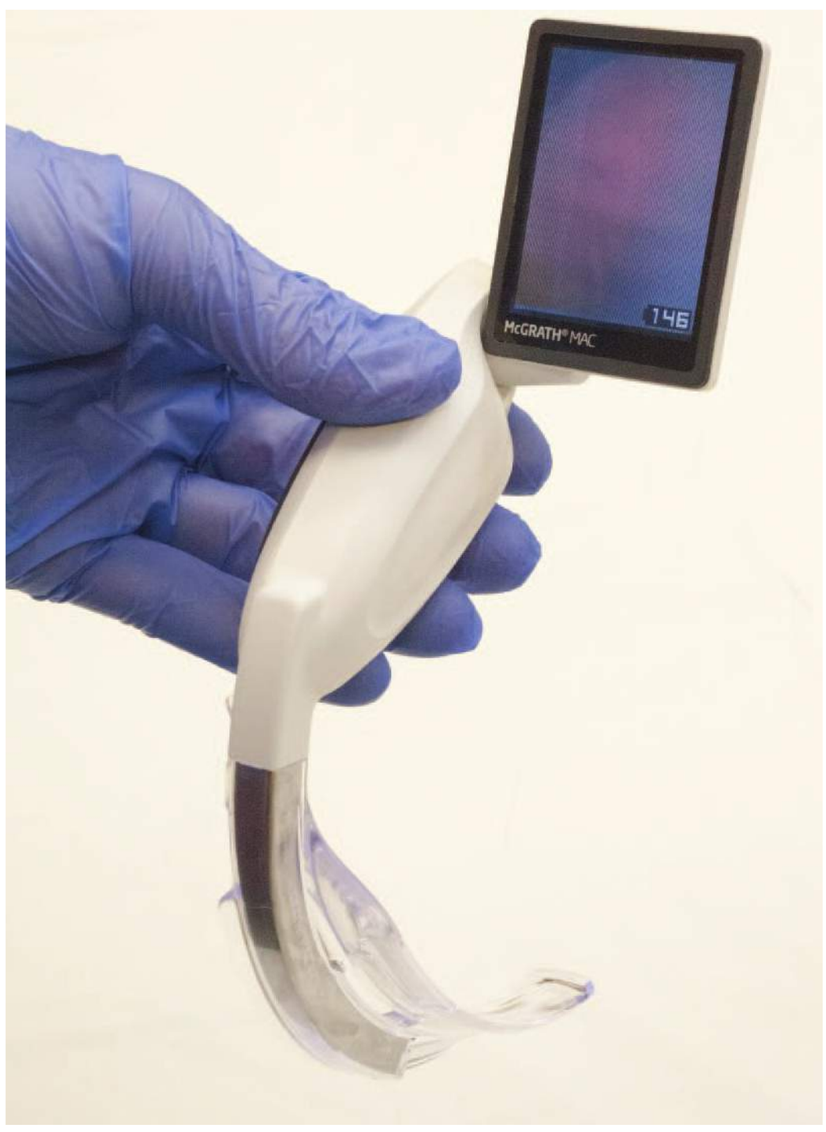


FIGURE 22.41 ■ **McGrath MAC Enhanced Direct Laryngoscope.** Compact video laryngoscope system shown here with a MAC 3 disposable blade. (Photo contributor: Lawrence B. Stack, MD.)

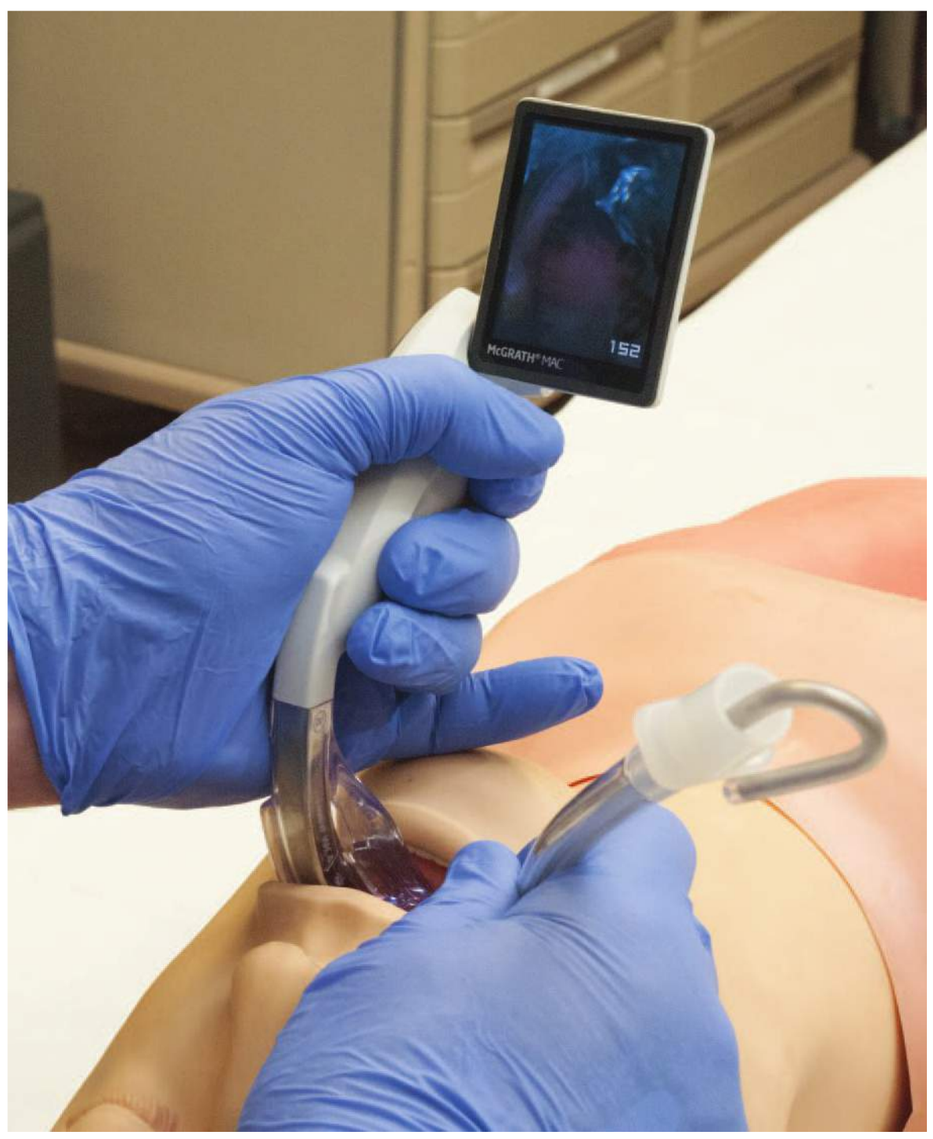


FIGURE 22.43 ■ **McGrath MAC EDL.** Vocal cord visualization and ETT delivery are demonstrated here. (Photo contributor: Lawrence B. Stack, MD.)

The laryngeal mask airway (LMA) was originally designed to facilitate ventilation during anesthesia for short operating room procedures. It has been shown to provide improved ventilation in cardiac arrest and failed airway cases and has a shallow learning curve, promoting its use by relative novices.

Equipment

The LMA consists of a short curved tube connected to a small mask with inflatable cuff. The shape promotes blind insertion with an endpoint detected as resistance as the leading edge of the cuff just enters and obstructs and the esophageal inlet. The inflated cuff then seals around the laryngeal inlet. The distal "mask" incorporates a small grate to prevent prolapse of the epiglottis within the mask. The LMA has several configurations including a disposable model, the LMA "Unique" (Figs. 22.44 and 22.45). LMAs are available in three adult sizes, #3 (30-50 kg), #4 (50-70 kg), and #5 (>70 kg). They are

FIGURE 22.45 ■ **LMA Unique.** The laryngeal surface of the LMA Unique with inflated cuff. Note the "grate" which prevents epiglottic prolapse into the mask. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.44 ■ **LMA Unique.** Adult sizes 3, 4, and 5 and pediatric size 2.5 are seen here. (Photo contributor: Lawrence B. Stack, MD.)

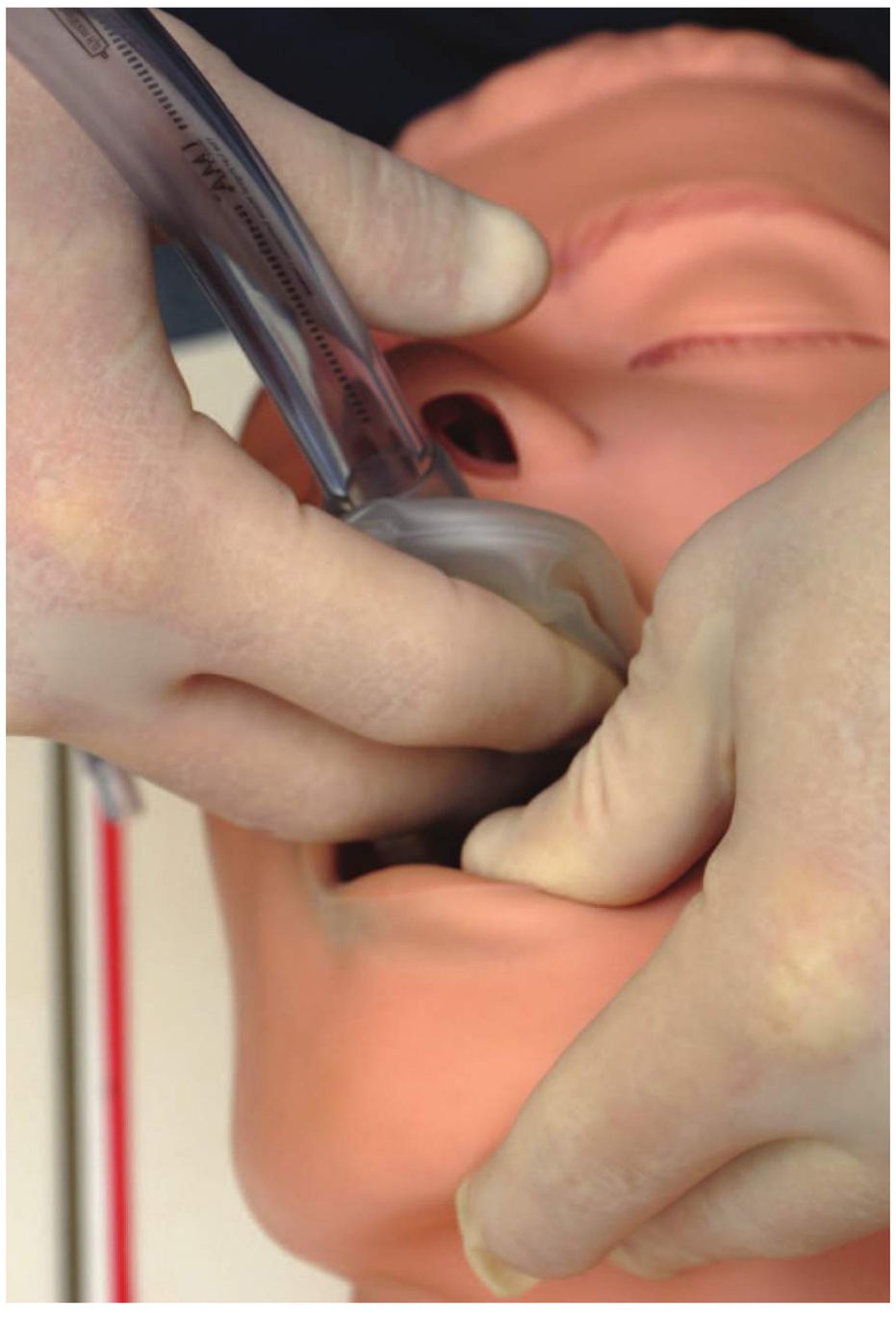


FIGURE 22.46 ■ **LMA Insertion.** LMA with deflated cuff is held against the hard palate during insertion by the index and third fingers. (Photo contributor: Lawrence B. Stack, MD.)

also available in pediatric sizes from size 0 to 2.5 for infants through toddlers.

Technique

The LMA is inserted into the mouth and held against the hard palate while being advanced into the hypopharynx. The LMA is advanced until resistance is met, and the cuff is then inflated with 20 to 40 mL of air to effect a seal. The mask should be lubricated prior to insertion.

Because the LMA aligns with the glottic opening, it is possible to intubate the trachea through the LMA with minimal interruption of ventilation. The distal grate prevents anything larger than a 6.0 ID ETT through a #3 and #4, and a 7.0 ID ETT through a #5. In addition to passing a small ETT directly, it is also possible to pass a flexible fiberoptic scope or a rigid fiberoptic scope (Levitan or Shikani) through the LMA under direct visualization. One must be sure to have an ETT, sized to pass through the LMA, loaded onto the stylet prior to passage. It is recommended to rotate the ETT counterclockwise 90 degrees to orient the bevel posteriorly, rotating clockwise as the tube is passed into the glottis.

FIGURE 22.47 ■ **Correct LMA Placement.** The LMA is correctly seated when resistance is met. Slight adjustments may be needed once seated. (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The tracheal lightwand is a device which consists of a bright light bulb on the end of a wire. The best current embodiment is the TrachLight (Laerdal), which incorporates (1) a very bright distal light encased in a tube-like flaccid stylet, (2) a removable stiffening wire to provide rigidity and allow the stylet to conform to the required bending angle, (3) a handle which comprises a water-proof battery housing, on-off magnetic reed switch, and a track which provides for easy length adjustment of the stylet for different size ETTs. There is cam-like locking device to firmly secure the tube to the stylet. The stylet is marked in centimeter markings to align with those on the ETT, ensuring that the distal bulb is always at the proper position at the tip of the ETT.

Technique

The intubation technique relies on the unique appearance and location of the transilluminated glow of the skin overlying the anterior neck to provide feedback to the intubator about the ETT tip position and the subsequent stylet movements needed for tracheal placement and advancement. For intubation, the stylet is bent at the distal 5 cm, conveniently labeled with "BEND HERE." The bend angle is critical and depends upon the head and neck position of the patient. For a patient who is in a neutral head/neck position, such as a patient in a halo device, the stylet should be configured to about an 80 degree acute angle. If the patient is hyperflexed, a more acute angle is required, approximately 60 degrees. Finally, for patients who are hyperextended, an obtuse angle of 110 or 120 degrees would be appropriate.

The technique can be performed with the intubator facing the patient, or with the intubator at the head of the bed behind the patient. The lightwand should be held in the dominant hand with a pencil grip at the ETT hub/handle junction. With his nondominant hand, the intubator should grasp the tongue with gauze, gently distracting forward. This acts to bring the epiglottis anteriorly. The lightwand/ETT is introduced into the center of the mouth and advanced to the

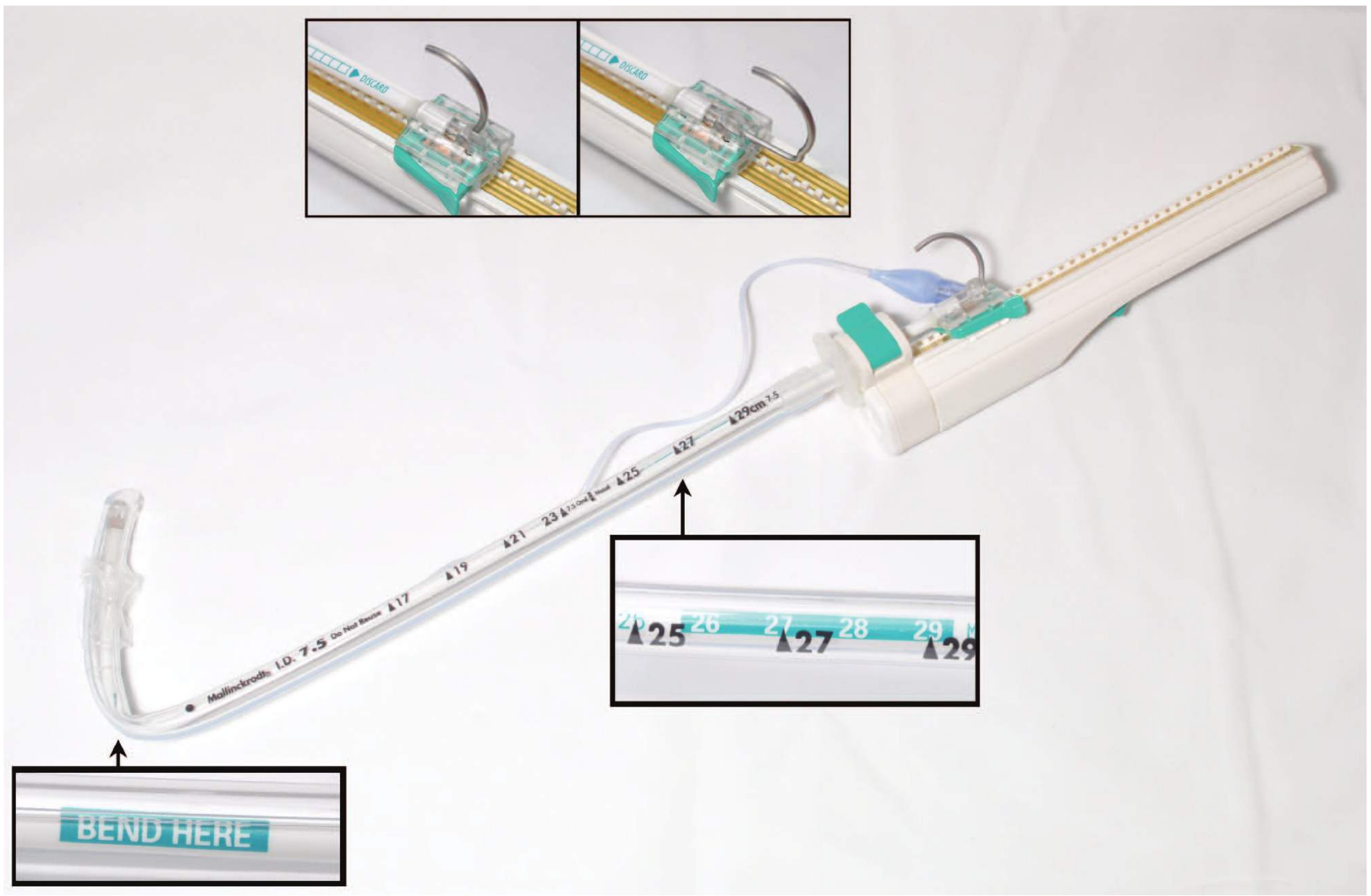


FIGURE 22.48 ■ **TrachLight by Laerdal.** The correctly assembled TrachLight demonstrates the correct alignment of the centimeter markings (inset) on the tube, the correct location of the bend (inset), and the correct engagement of the wire portion of the stylet (inset). (Photo contributor: Lawrence B. Stack, MD.)

hypopharynx while maintaining anterior pressure, holding the elbow high and the arm abducted, to keep the stylet bulb anterior. Advancement stops when the intubator recognizes an orange glow on the anterior neck tissues or when tube advancement meets resistance. The appearance and position of this glow determines the next maneuver.

If transillumination positional feedback indicates that the tube tip is in one of the pyriform fossae, the stylet/ETT is withdrawn slightly and rotated toward the midline and reinserted. This process is continued repeatedly in an iterative manner until the correct midline glow indicating tracheal position is encountered. At that point, the stiffening wire is withdrawn several centimeters, resulting in a caudad displacement of the glow as the intratracheal stylet "relaxes." The tube and stylet is then advanced until the glow just disappears below the sternal notch. The tube is now confirmed to be intratracheal and the tube tip is confirmed to be reliably in the mid-trachea, with no need for confirmatory chest radiograph.

Failure to modify the position of the transilluminated glow usually indicates that the stiffening wire has displaced from the locking notch that keeps the wire from rotating in place.

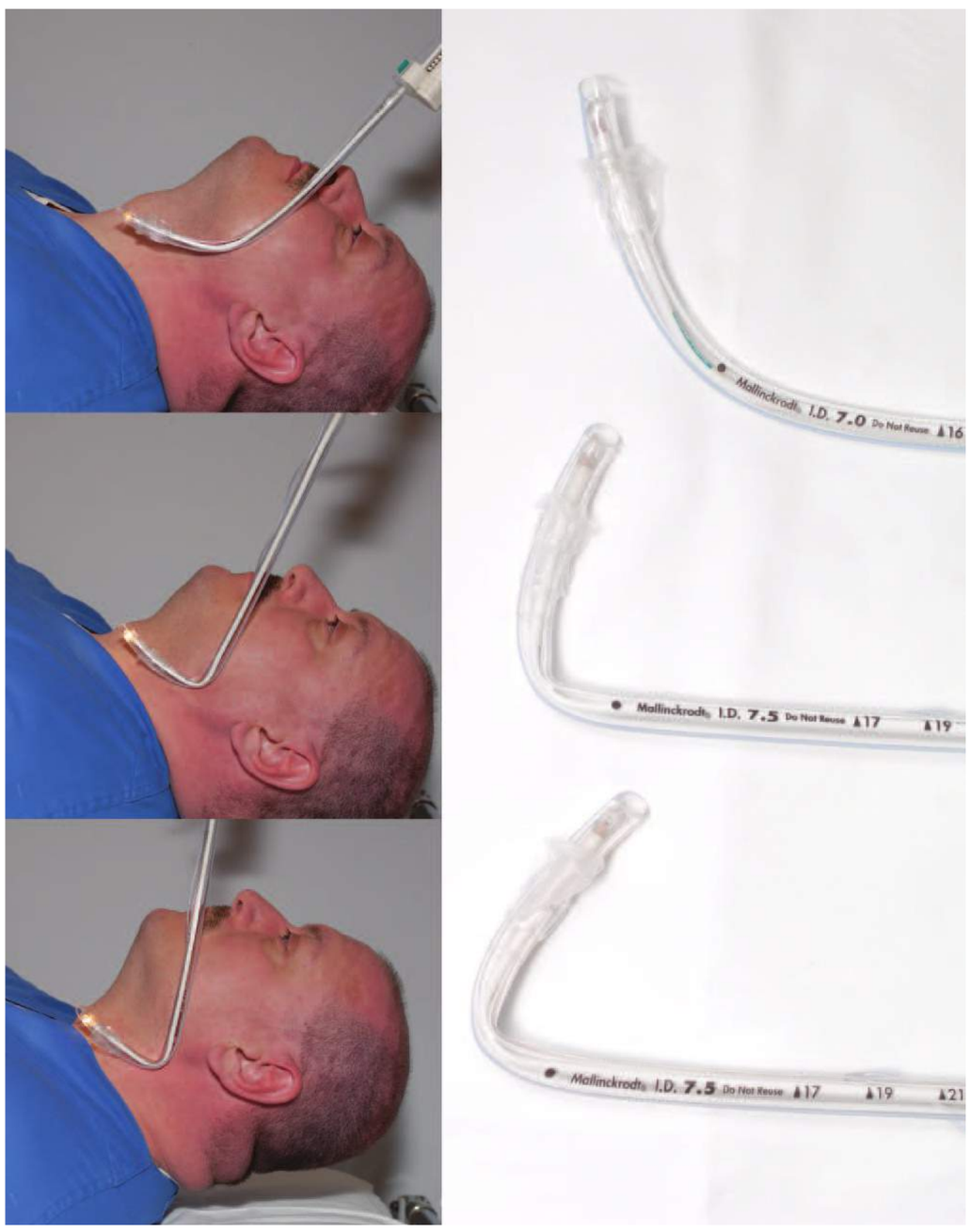


FIGURE 22.49 ■ **TrachLight Bend Angle.** The head/neck position and suggested corresponding bend angle of the TrachLight. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.50 ■ **TrachLight Positioning.** The correct TrachLight position is demonstrated here. The device is placed in the center of the mouth while the left hand is lifting the tongue anteriorly. (Photo contributor: Lawrence B. Stack, MD.)

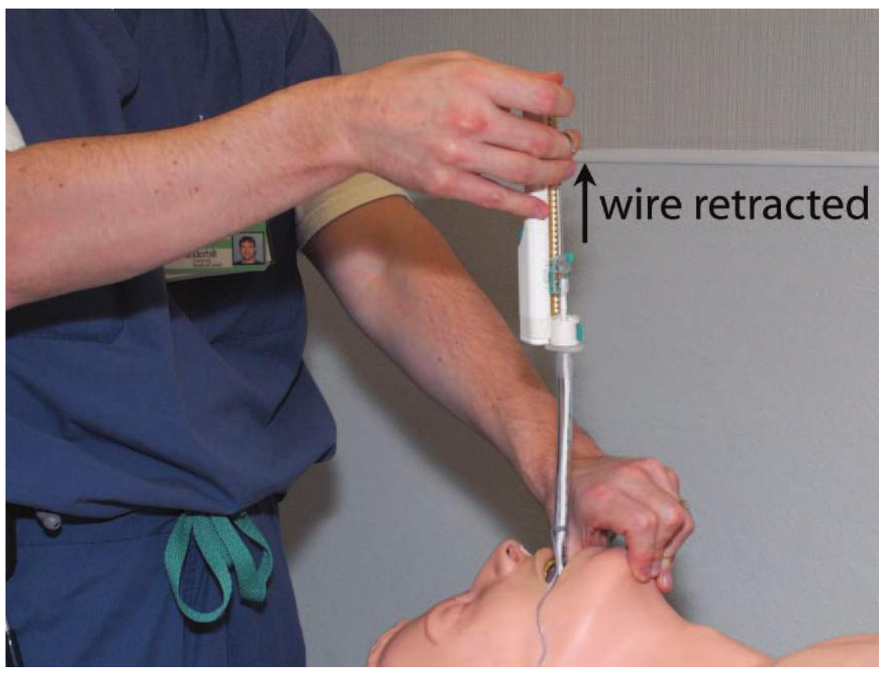


FIGURE 22.51 ■ **TrachLight Technique.** Once the tracheal position is encountered, the stiffening wire is retracted several centimeters before advancing ETT into the trachea. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.52 ■ **TrachLight Technique.** Once the tracheal position is encountered, the stiffening wire is retracted several centimeters before advancing ETT into the trachea. (Photo contributor: Lawrence B. Stack, MD.)

TABLE 22.2 ■ TRACHLIGHT MANEUVER DECISIONS

Glow appearance	Position of glow	Anatomic position of stylet tip	Next maneuver
Bright orange, well localized	High neck/submental	Vallecula	Remove stylet, decrease acuity of bend angle
Dull orange glow	Right/left of neck midline	Right/left pyriform fossa	Partially withdraw stylet, rotate slightly to midline, re-advance, keeping tip anterior
No glow or very dim	If present, thin line at lateral neck may be bilateral	Esophagus	Remove stylet, increase acuity of bend angle
Bright midline glow	Thyroid prominence	Glottic opening	Withdraw stiffening wire by 3 cm, advance stylet/ETT until glow just disappears beyond sternal notch

(Photo contributor: Lawrence B. Stack, MD.)

DIGITAL (TACTILE) INTUBATION

Digital intubation relies on tactile definition of intubation landmarks, primarily the epiglottis but often the posterior cartilages and interarytenoid notch.

Technique

Digital intubation technique is performed with the intubator facing the patient from the side. With the gloved nondominant hand, the intubator should insert the index and third fingers into the patient's mouth. He should next "walk" the fingers down the tongue to progressively displace the tongue anteriorly as the fingers are advanced to the epiglottis. The epiglottis will have a feel similar to the earlobe. As the third finger encounters the epiglottis, the finger traps and holds the epiglottis anteriorly. The ETT, with stylet configured to an open "C," is introduced by the dominant hand into the corner of the mouth and advanced to the palmer side of the intraoral index finger. The intraoral index finger is then used to guide the ETT tip to the third finger, where the ETT is slipped between the epiglottis and third finger. The third finger is used to keep the ETT anterior against the laryngeal surface of the epiglottis while the ETT is advanced by the dominant hand and assisted by the intraoral index finger. It is often possible to palpate the arytenoid cartilages/interarytenoid notch, in which case one need only ensure that the ETT tip remains anterior to those cartilages and is prevented from drifting posteriorly to the esophageal inlet. The posterior cartilages project cephalad, feeling like snake fangs. Correct ETT position can be confirmed by palpating the arytenoid cartilages posterior to the ETT.

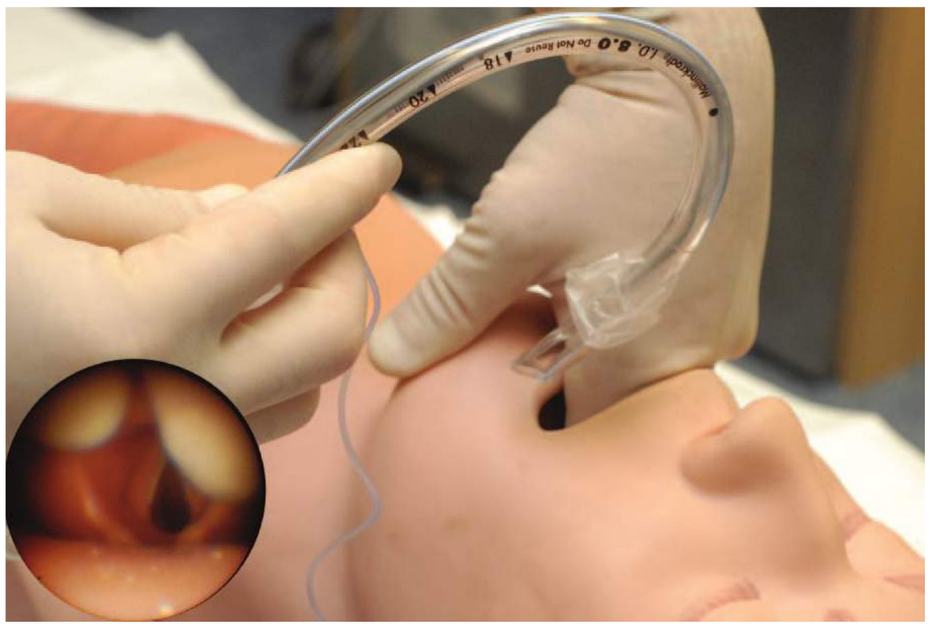


FIGURE 22.56 ■ **Digital Intubation Technique.** Index and third finger inserted into the mouth with "C" shaped stylet held in left hand. Third finger holds epiglottis anteriorly (inset). (Photo contributor: Lawrence B. Stack, MD.)

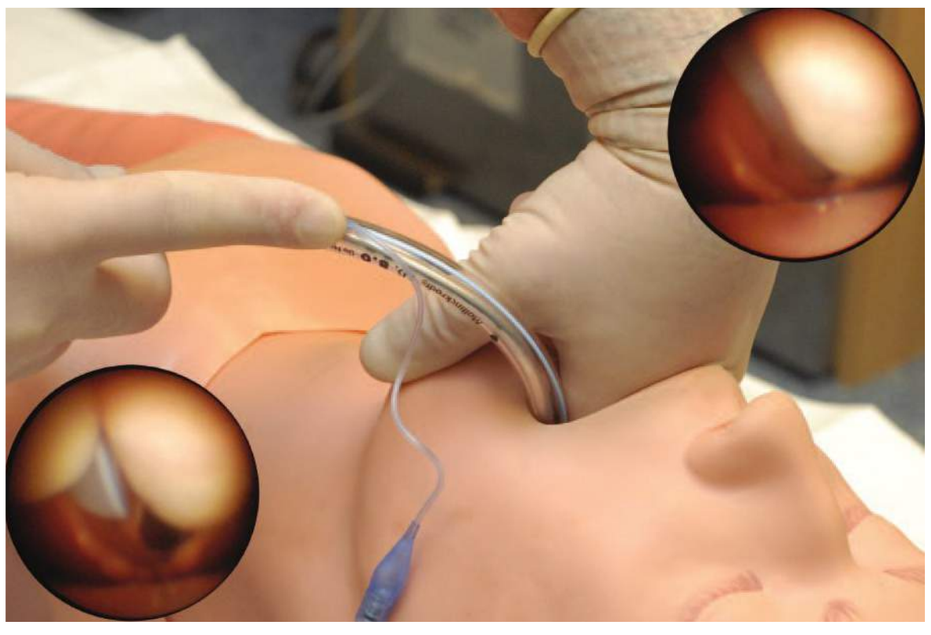


FIGURE 22.57 ■ **Digital Intubation—ET Tube Passage.** Intraoral index finger guides ETT to third finger (inset) which directs the tube into the glottic opening (inset). (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Digital intubation may be the best airway delivery option in austere conditions or in patient with copious airway secretions or blood.
2. Can only be used in a paralyzed, comatose, or dead patient.

Equipment

The reusable ILA is similar in concept to the classic LMA but has a structural features that provide an easier and more stable insertion with less likelihood of tube kink. In addition, it has a unique "keyhole" like airway outlet which helps to direct an ETT to the midline at the proper glottic entry angle. Because it has a larger lumen, the ILA can accommodate standard ETT up to 8.5 mm ID. The ILA comes in five sizes—1.0 (<5 kg), 1.5 (10-20kg), 2.4 (20-50kg), 3.5 (50-70kg), and 4.5 (70-100kg), accommodating maximum ETT sizes of 4.5, 5.5, 6.5, 7.5, and 8.5, respectively.

FIGURE 22.60 ■ **Intubating Laryngeal Airway Insertion.** The ILA is inserted by displacing the tongue and jaw anteriorly and is advanced down the curvature of the oropharynx and hypopharynx. (Photo contributor: Lawrence B. Stack, MD.)

Technique

The mandible is displaced anteriorly with a jaw lift maneuver, bringing the tongue and epiglottis forward. The ILA is

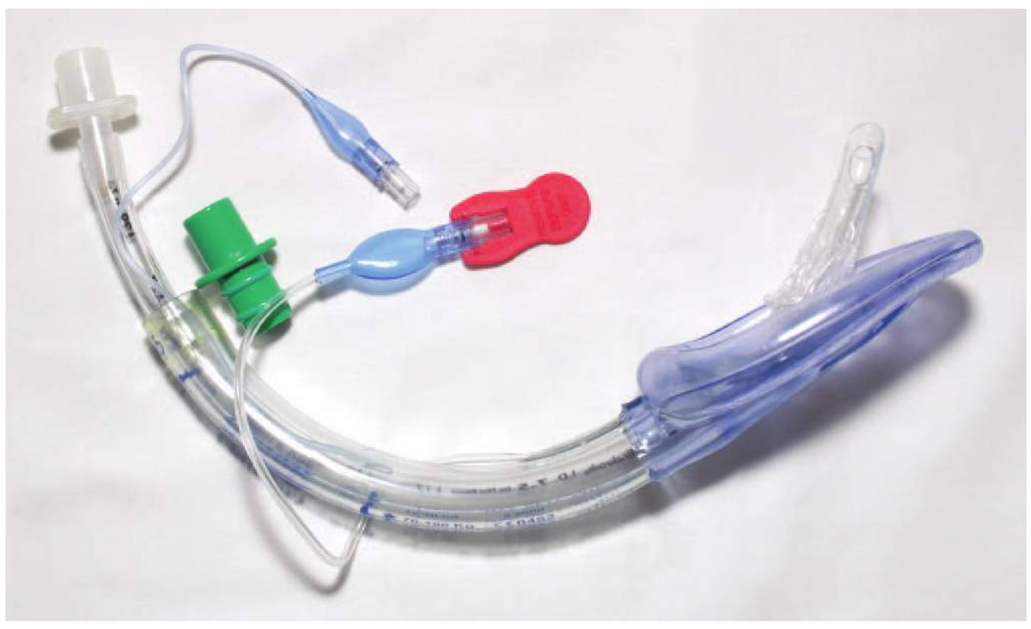


FIGURE 22.58 ■ **Intubating Laryngeal Airway**. Intubating LMA demonstrating an ETT through the device lumen. (Photo contributor: Lawrence B. Stack, MD.)

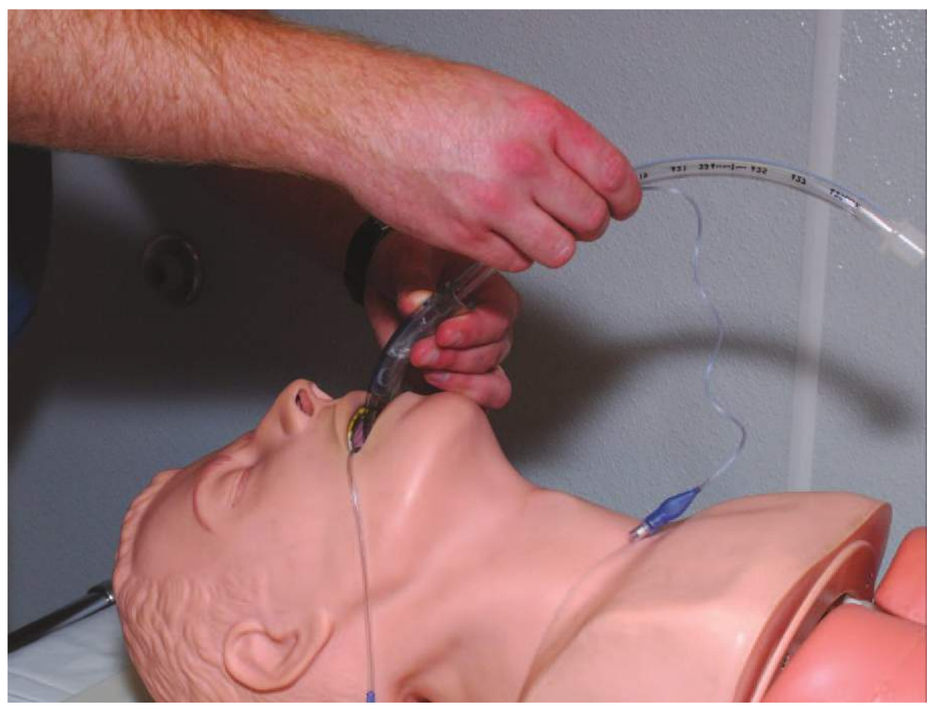


FIGURE 22.61 ■ **ETT Insertion Through the Intubating Laryngeal Airway.** An ETT is advanced through the ILA. (Photo contributor: Lawrence B. Stack, MD.)

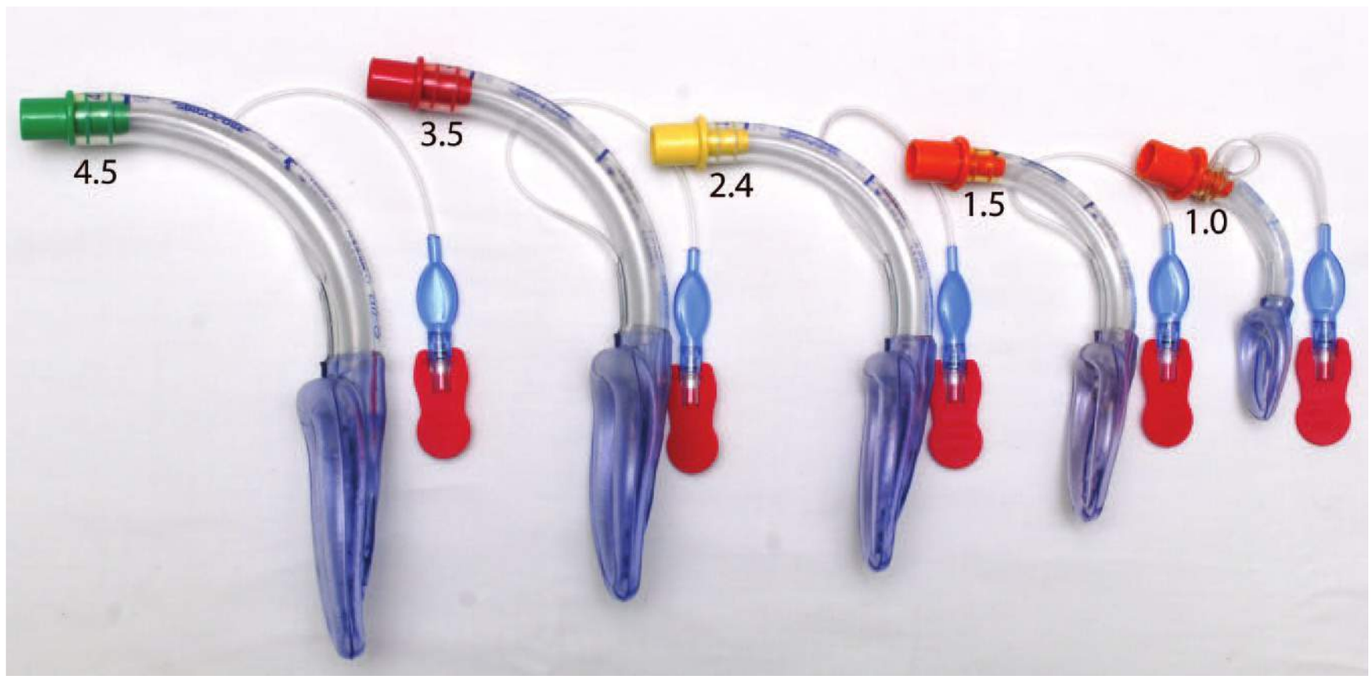


FIGURE 22.59 ■ **Intubating Laryngeal Airway Sizes.** ILA in five different sizes. (Photo contributor: Lawrence B. Stack, MD.)

then inserted into the mouth, advancing with a downward and inward force to follow the curve of the oropharynx/hypopharynx. The insertion endpoint is firm resistance to further insertion. The cuff is then inflated with approximately 20 mL of air or until the pilot balloon is firm. Ventilation can proceed by attaching a ventilating bag to the proximal port.

Intubation can be accomplished through the ILA in a well-sedated/chemically paralyzed patient after first removing the proximal 15-mm adaptor port. A well-lubricated standard ETT can then be inserted through the ILA. This procedure can also be aided by using a lightwand, opitcal sytlet, or fiberoptic intubating bronchoscope, or bougie, through the ETT to facilitate optimal alignment for intubation.

Once the ETT is inserted, the ILA can be easily removed by inserting the disposable tube stabilizer/stylet.

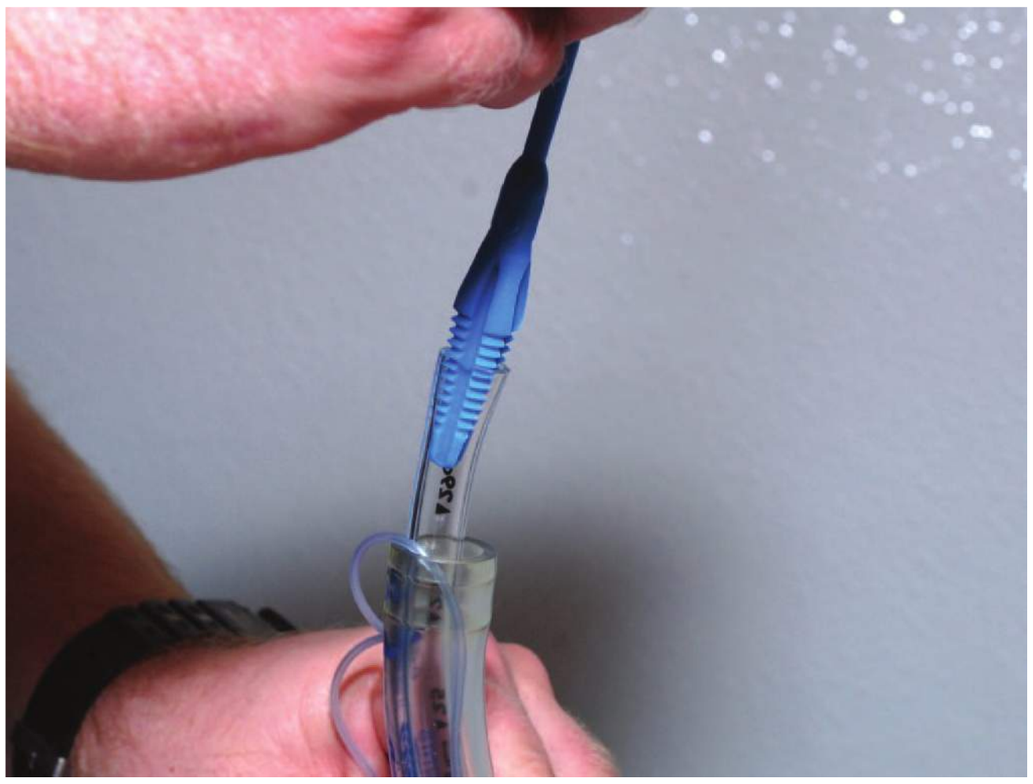


FIGURE 22.62 ■ **Intubating Laryngeal Airway Removal.** Removal of the ILA while keeping the ETT in place is accomplished by using the disposable tube stabilizer (blue). (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The King laryngeal tube (King LT) consists of an airway tube with two cuffs. The distal cuff seals the esophagus and the proximal seals the oropharynx and nasopharynx when inflated. Between the cuffs are multiple ventilation channels. Inflation of the cuffs occurs through one inflation line. A separate gastric drainage channel is present in the disposable model (King LTS-D).

Technique

A lubricated King LTS-D is inserted behind the tongue while preforming a jaw lift, placing the patient in a sniffing position. The tube is advanced until resistance is met or until the connector base is aligned with the teeth or gums. The cuffs are inflated with 60 to 90 mL of air, depending on the size of the King LTS-D. Some "giggling" may be required to properly seat the tube.

Pearls

1. Most unsuccessful King LT placement attempts are due to failure to keep the tube in midline during insertion.
2. Anticipate vomiting when the King LT is removed.

FIGURE 22.63 ■ **King LTS-D Airway.** The King LTS-D is shown with the distal esophageal balloon and the proximal hypopharynx balloon inflated. (Photo contributor: Lawrence B. Stack, MD.)

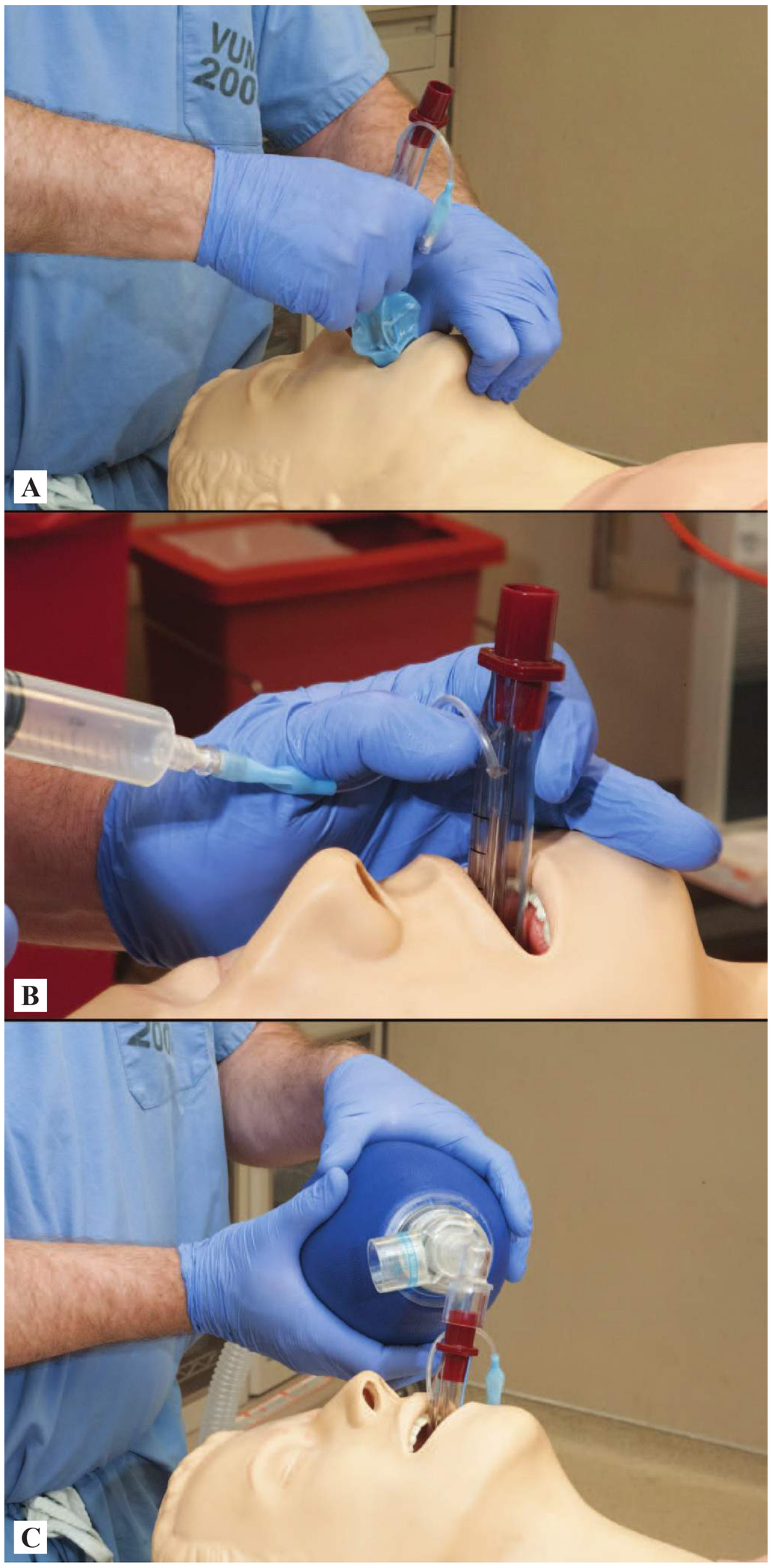


FIGURE 22.64 ■ **King LTS-D Airway Technique.** The King LTS-D is passed down the anterior surface of the tongue while the tongue and mandible are pulled upward. The tube is advanced until resistance is met or the base of the connector is at the level of the lip or gum. The balloon is inflated with 45 to 90 mL of air depending on the size of the tube. (Photo contributor: Lawrence B. Stack, MD.)

Patient Preparation

The nares should be inspected for patency and the side deemed most hospitable to tube passage should be anesthetized and vasoconstricted, Additionally, it is helpful to confirm nasal patency by inserting a well-lubricated nasal trumpet. Lidocaine jelly can be used as the lubricant to provide additional topical anesthesia. We favor the use of disposable atomizers to instill 2 mL of a mixture of tetracaine 1%/phenylephrine 0.5%, which yields excellent mucosal anesthesia and vasoconstriction. Favorable intubation conditions can be achieved in about 5 minutes.

In preparation for nasal intubation, the ETT can be conformed into a circle by inserting the tip into the 15-mm proximal adaptor, and soaked in warm water to soften the tube for nasal passage. Success can also be enhanced by the use of a special ETT, called an Endotrol control tip ETT. This tube incorporates a length of nylon fishing line within a channel in the anterior wall of the ETT. Pulling on the proximal loop "trigger" causes anterior deflection of the tube tip.

FIGURE 22.65 ■ **Verification of Patency.** A well-lubricated nasopharyngeal airway will verify patency of the nasal passage prior to NTT insertion. (Photo contributor: Lawrence B. Stack, MD.)

Technique

Once the selected naris is suitably anesthetized, the intubator faces the patient, who can be sitting up or supine, and inserts the tube into the naris, parallel to the nasal floor/perpendicular to the facial plane (not along the nasal axis). To minimize trauma to the nasal turbinates, the bevel of the tube should face the turbinates. This implies that if the left naris is being intubated, the tube can be inserted along the normal curve of the nose-nasopharynx; however, if the right naris is being intubated, the tube should be inserted with the curve of the tube in a cephalad direction until the tube is advanced to the nasopharynx, at which point the tube is rotated to the correct anatomic configuration. As the tube is slowly advanced to the hypopharynx, the intubator listens closely for transmitted breath sounds, rotating the tube right or left to maintain maximal transmission. It takes a more-severe-than-expected rotation externally to generate a small rotation of the supraglottic tube tip. The tube is then rapidly advanced unless resistance is encountered, as would be expected with pyriform sinus tube tip position. Correct intratracheal placement should elicit cough, a reflex deep inspiration, and a loss of phonation. The intubator may also be sprayed with coughed secretions. If breath sound transmission decreases with advancement, the tube is withdrawn and re-advanced, preferably with more

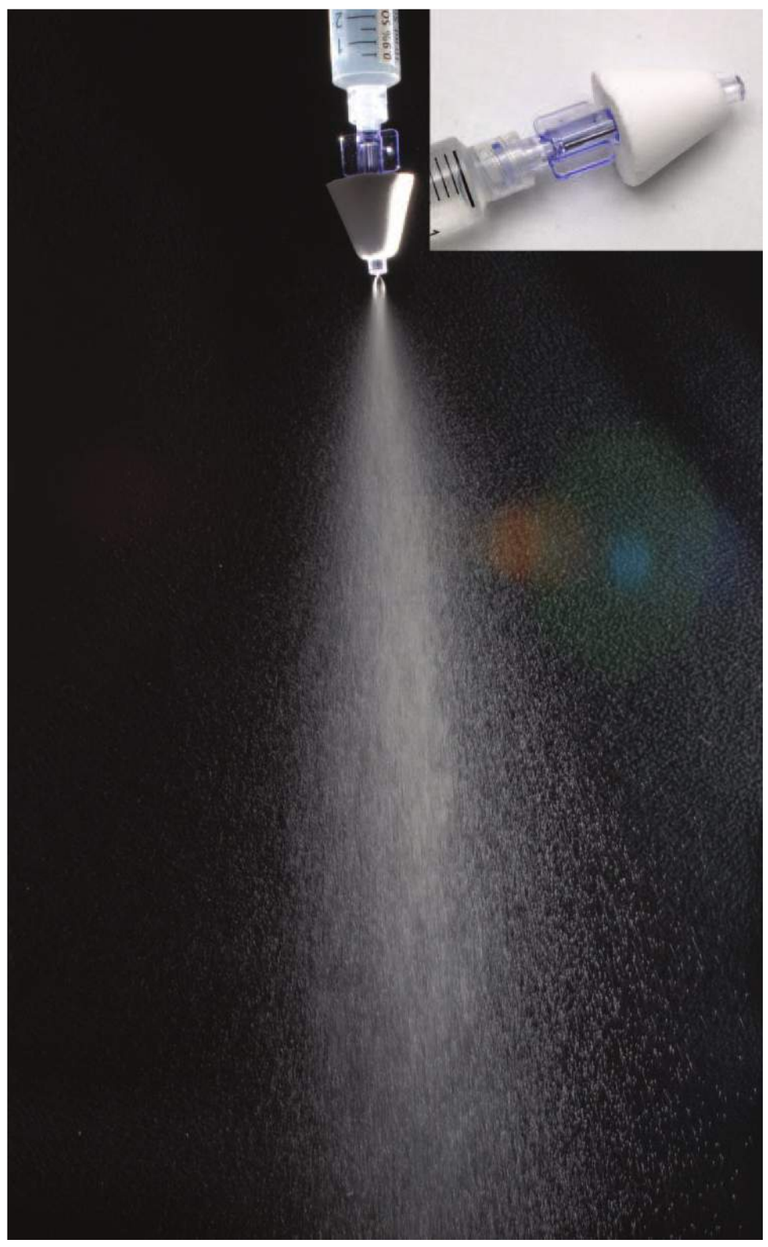


FIGURE 22.66 ■ **Atomizer.** A disposable atomizer used to deploy topical vasoconstrictors and analgesics prior to NTT insertion. (Photo contributor: Lawrence B. Stack, MD.)

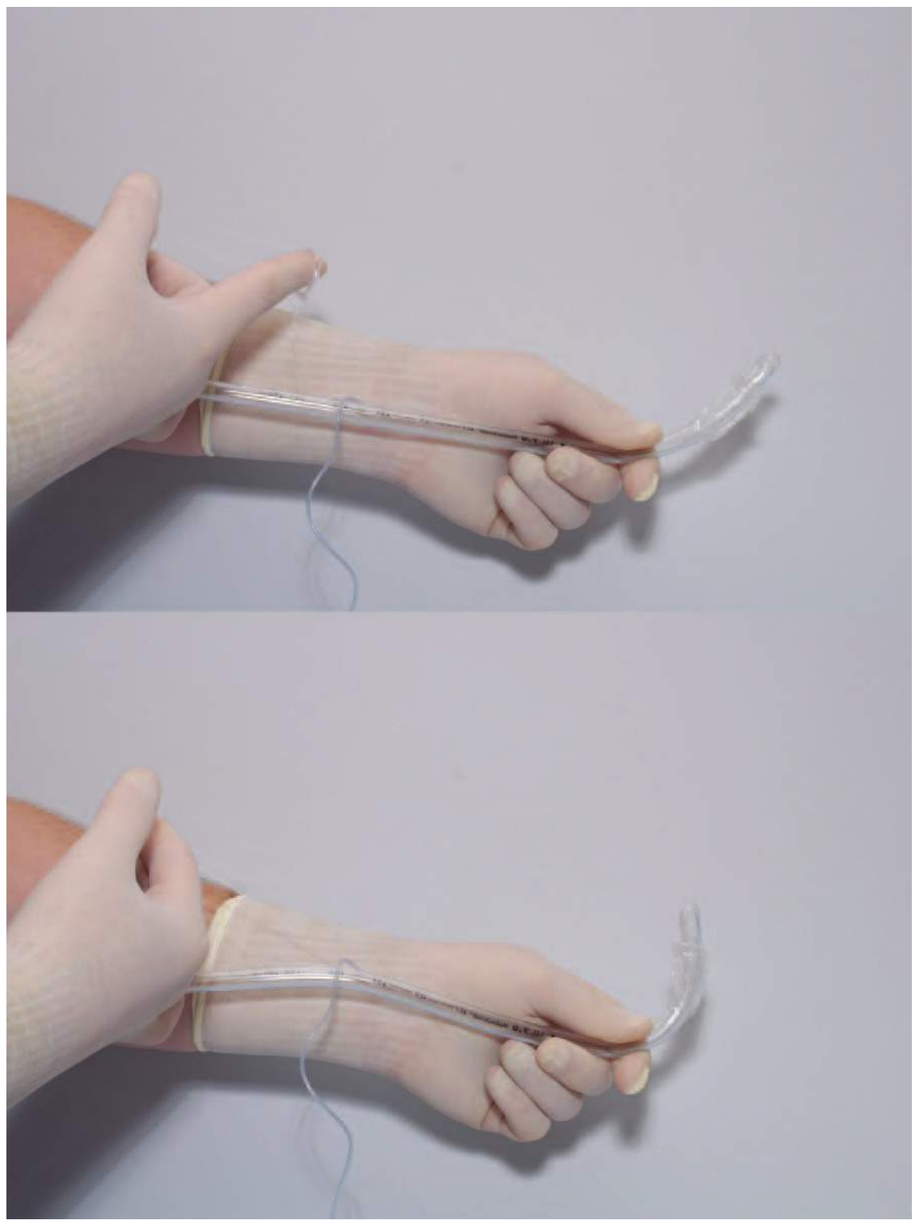


FIGURE 22.67 ■ **Endotrol ETT.** Demonstration of the ETT tip control using the Endotrol trigger. (Photo contributor: Lawrence B. Stack, MD.)

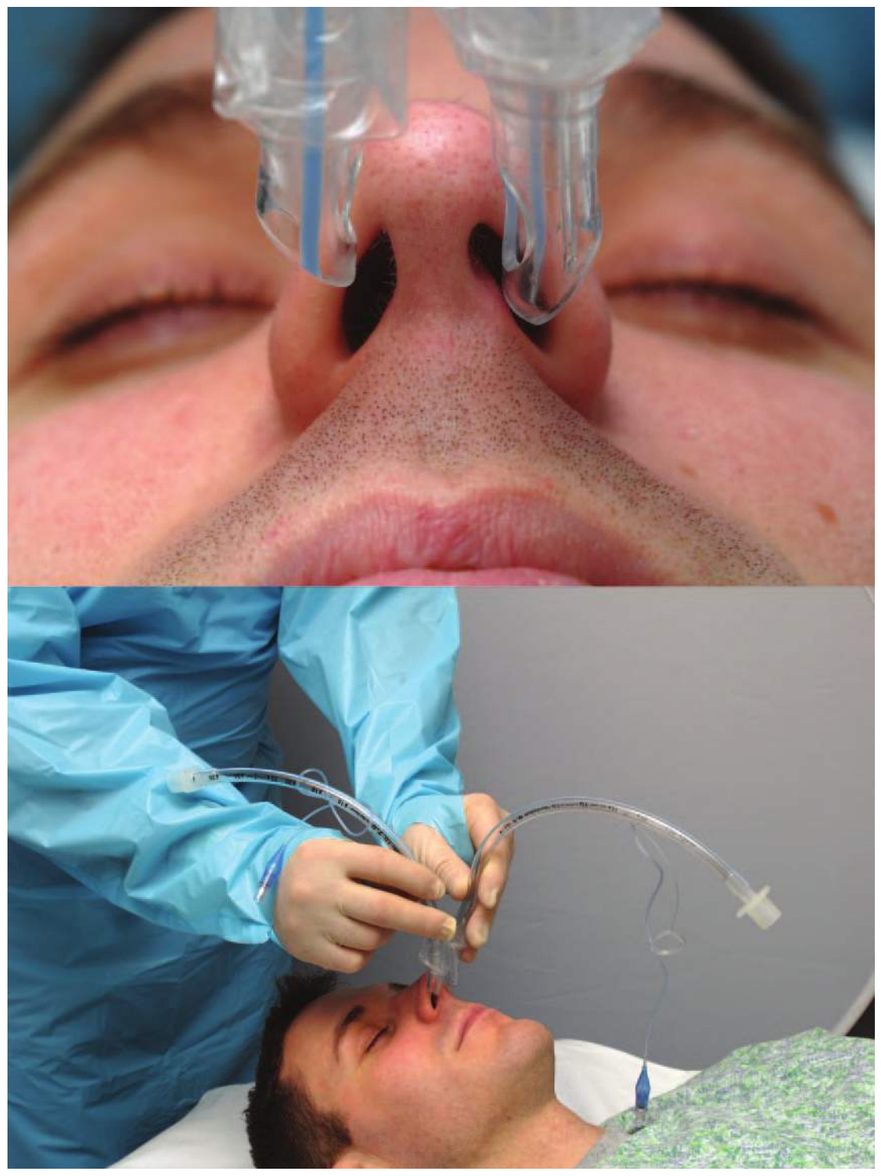


FIGURE 22.68 ■ **Bevel Placement for NT Intubation.** When inserted into the nose, the bevel of the tube should face the turbinates to minimize the trauma to these structures. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.69 ■ **Maneuvering for Maximized Breath Sounds.** Once the ETT is in close proximity to the glottic opening, the intubator must rotate the tube left or right till maximum air movement is heard, then advance the tube. (Photo contributor: Lawrence B. Stack, MD.)

anterior displacement of the tip if the Endotrol tube is used. If such a tube is not available, anterior displacement can be produced by partially inflating the ETT cuff.

The audible feedback to determine tube tip position can be enhanced by the attachment of the Beck Airway Airflow Monitor (BAAM), which creates an amplified sound with air movement, much like the sound of a tea kettle. Alternatively, one can remove the diaphragm/bell from a stethoscope and insert the distal end of the stethoscope tubing into the proximal end of the ETT. In addition to amplifying transmitted sounds, this trick also can spare the intubator from a sputum ear wash. Positional feedback can also be enhanced by use of a quantitative waveform capnometer/capnograph.

Pearls

1. Blind nasotracheal intubation offers a viable option in the event that oral intubation is rendered impossible in the short term, such as a patient who has a wired jaw or large subglossal hematoma.
2. The blind nasotracheal intubation technique requires maintenance of spontaneous respirations because the intubator relies on audible variation in transmitted breath sounds via the ETT.
3. The TrachLight lightwand can be used for blind nasal tracheal (NT) intubation, with the stiffening wire removed. Tube tip position is determined identically to that with orotracheal transillumination, with identical repositioning maneuvers, except that rotational maneuvers are likely to be more extreme as noted above.

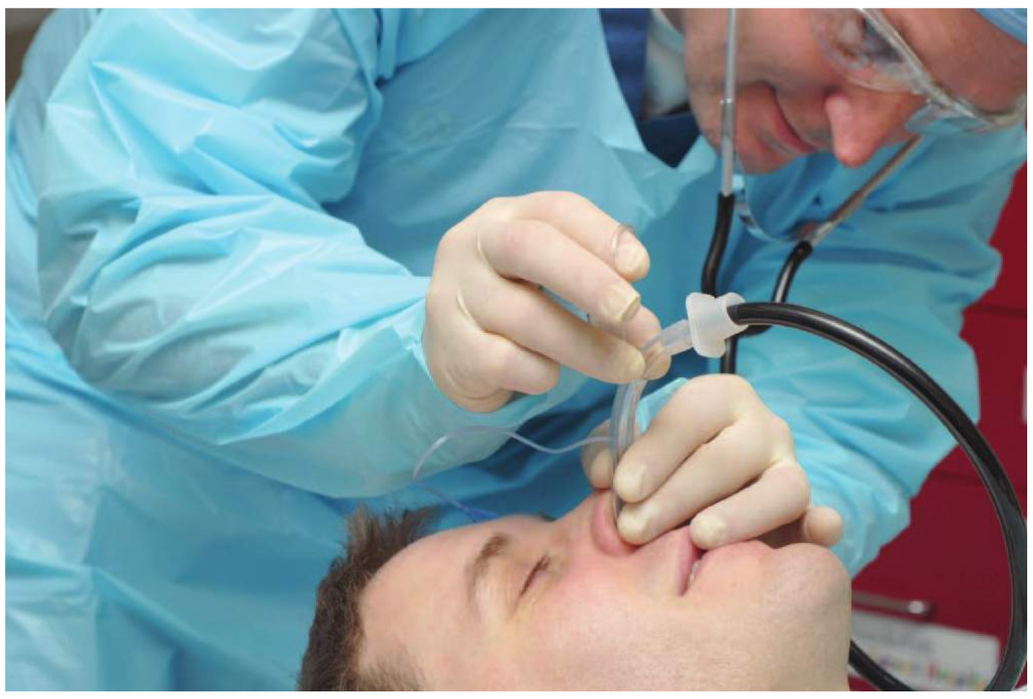


FIGURE 22.71 ■ **Stethoscope Airflow Monitor.** Removing the diaphragm/bell from a stethoscope and placing it in the end of the ETT will enhance identify the location of maximum air movement. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.70 ■ **Beck Airway Airflow Monitor (BAAM).** The BAAM makes an amplified sound to help identify location of maximized air movement. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.72 ■ **TrachLight Augmentation of NT Intubation.** Remove the stiffening wire, and place the stylet into the ETT and use conventional landmarks to guide maneuvering decisions for proper tube placement. (Photo contributor: Lawrence B. Stack, MD.)

There are two methods of nasal flexible fiberoptic technique: (1) scope-first and (2) tube-first.

Patient Preparation

For both methods, the nose is prepared as described above under blind nasotracheal intubation.

Technique: Scope-First

The largest possible ETT, preferably 7.0 mm ID, is inserted over the endoscope to the most proximal position of the endoscope. The fiberoptic endoscope is then inserted through the selected naris. Under direct visual guidance, the endoscope is advanced along the floor of the nose, navigating under the inferior turbinate. The endoscope tip is manipulated by a combination of trigger deflection (up/down) and scope rotation (left/right) to keep the lumen in the center of the optical field. As the tube is advanced to the posterior nasopharynx, the scope tip is deflected downward to reveal the epiglottis and laryngeal inlet. The scope is advanced further, with the operator maneuvering the scope tip to keep the glottic opening in the center of the optical field. With the endoscope tip just above the cords, 2 mL of 1% lidocaine should be injected through the injection port of the fiberscope to provide vocal cord anesthesia prior to tube passage. The endoscope advancement continues until the endoscope tip is approximately 3 to 5 cm above the carina. The tube is then gently advanced over the fiberoptic sheath into the naris and to the trachea. If resistance is encountered at 14 to 16 cm, the tube may be

FIGURE 22.73 ■ **Scope-First Method.** The ETT is placed at the proximal extreme. The endoscope is then guided to the glottic opening and the tube is then advanced over the endoscope. (Photo contributor: Lawrence B. Stack, MD.)

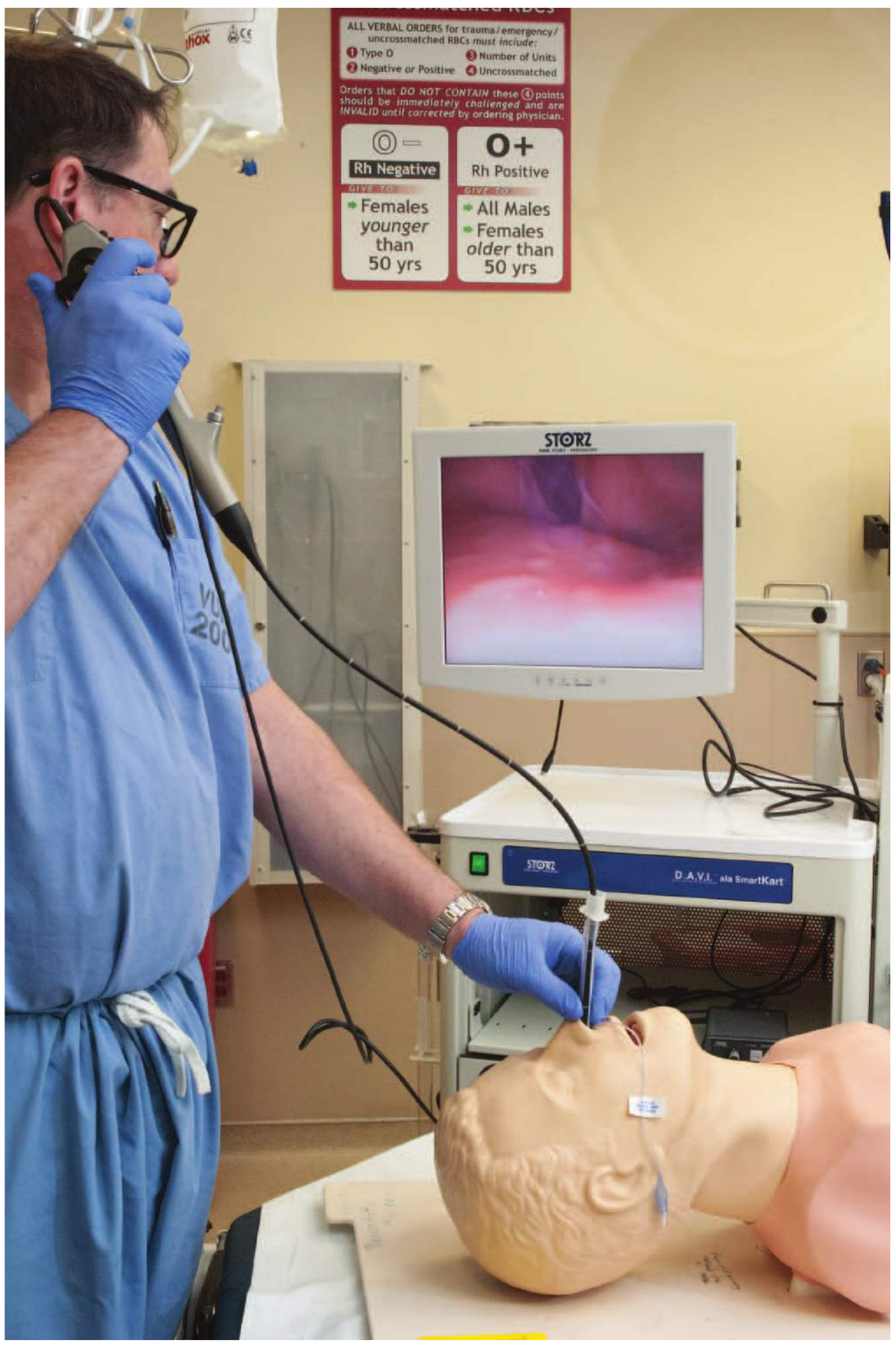


FIGURE 22.74 ■ **Tube-First Method.** The ETT is advanced to the hypopharynx. The endoscope is then advanced through the tube and maneuvered to the glottic opening. The tube is then advanced over the endoscope. (Photo contributor: Lawrence B. Stack, MD.)

impacting upon the right arytenoid. As discussed previously, this can be corrected by a severe counterclockwise rotation. The tube tip should be verified to be the correct distance from the carina.

If, during endoscope navigation through the nose, the view becomes blurry because of adherent secretions, the tip can be cleared by gently deflecting the tip into "pink" mucosa. If one becomes lost or disoriented during scope advancement or if there is no obvious lumen through which to advance, the endoscope should be withdrawn slightly and the tip maneuvered to provide a recognizable view. The endoscope should not be advanced if a lumen is not readily apparent.

Technique: Tube-First

To avoid some of the technique difficulty inherent with nasal navigation, the ETT can be passed through the naris and advanced into the hypopharynx first, before scope insertion. The endoscopist then inserts the intubating fiberscope through the ETT, and advances it to and through the glottic opening to the appropriate distance above the carina. Tube advancement proceeds as described above. The tube-first method may also facilitate endoscopic intubation in the event of epistaxis.

CRICOTHYROTOMY (SELDINGER TECHNIQUE)

Equipment

The Melker kit manufactured by Cook Medical includes all the equipment necessary: catheter, thin-walled needle, Seldinger wire, cuffed tracheostomy tube 5.0 mm ID, and tapered introducer.

Technique

1. Identify the thyroid cartilage and the cricothyroid membrane directly caudally.
2. Prep the neck with suitable antiseptic; anesthetize the skin overlying the cricothyroid membrane with 1% lidocaine.
3. Isolate the trachea between the thumb and third finger of the sterilely gloved nondominant hand, using the index finger to palpate the cricothyroid membrane landmark. With a syringe attached, preferably containing sterile water or remaining lidocaine, insert the catheter through the cricothyroid membrane at a 30-degree angle to the horizontal, aiming toward the feet. Once through the skin, begin aspirating as the catheter is advanced. Air and bubbles entering the syringe confirm entry into the trachea.
4. Carefully slide the catheter off of the needle and into the trachea.
5. Reattach syringe and reconfirm tracheal position.
6. Thread Seldinger wire through catheter into trachea; lack of resistance to wire passage confirms correct position.
7. Remove catheter and, with the included scalpel, make a small incision at the wire-skin entry site.
8. Insert tracheostomy tube with dilator/introducer over wire into trachea. Expect significant resistance. Inability to pass dilator may indicate an inadequate skin incision
9. Withdraw dilator/introducer and wire as one unit, inflate cuff and initiate ventilation.
10. Secure tracheostomy tube with tracheostomy ties

FIGURE 22.76 ■ **Identify Landmarks.** Palpate thyroid cartilage and cricothyroid cartilage caudally. Angle needle 30 degrees caudad to enter the trachea. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.75 ■ **Melker Cricothyrotomy Kit.** Components of the kit produced by Cook Inc. (www.cookmedical.com). (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.77 ■ **Bubbles during Aspiration.** Aspiration of bubbles during needle advancement confirms tracheal placement. (Photo contributor: Lawrence B. Stack, MD.)

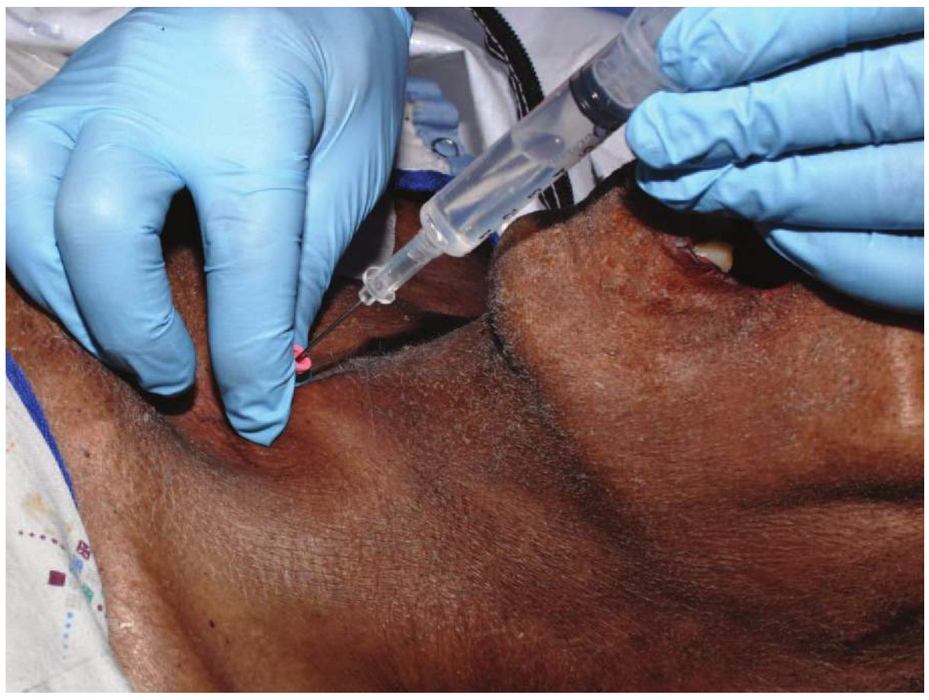


FIGURE 22.78 ■ **Slide Catheter Into Trachea.** Carefully slide the catheter off the needle into the trachea. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.79 ■ **Confirm Tracheal Placement.** Again, aspirate bubbles from the catheter to confirm tracheal placement. (Photo contributor: Lawrence B. Stack, MD.)

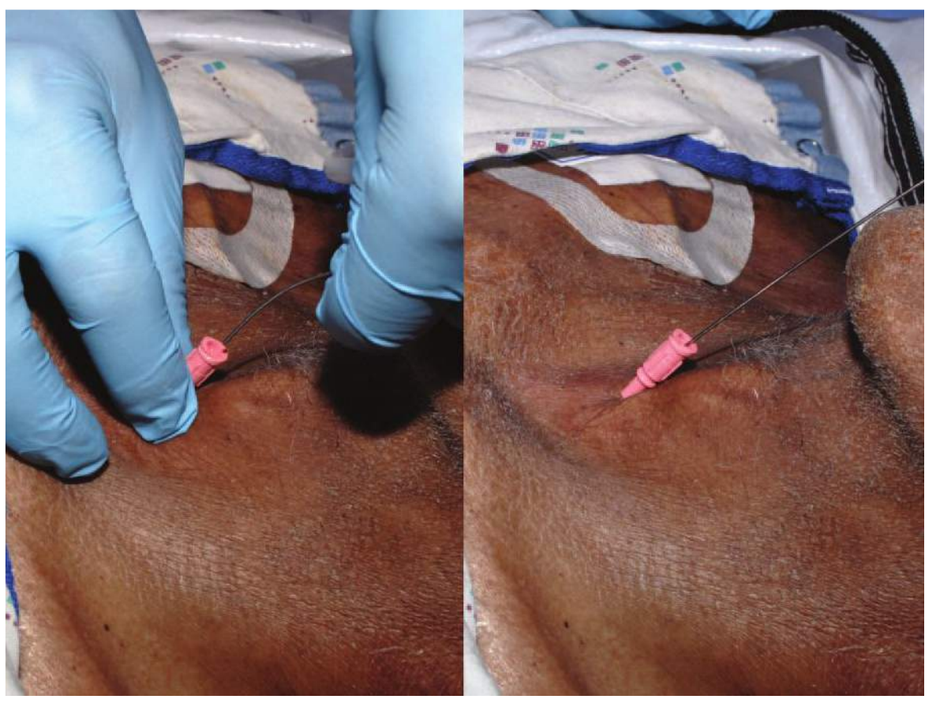


FIGURE 22.80 ■ **Thread Wire.** Lack of resistance during threading of Seldinger wire confirms tracheal placement. (Photo contributor: Lawrence B. Stack, MD.)

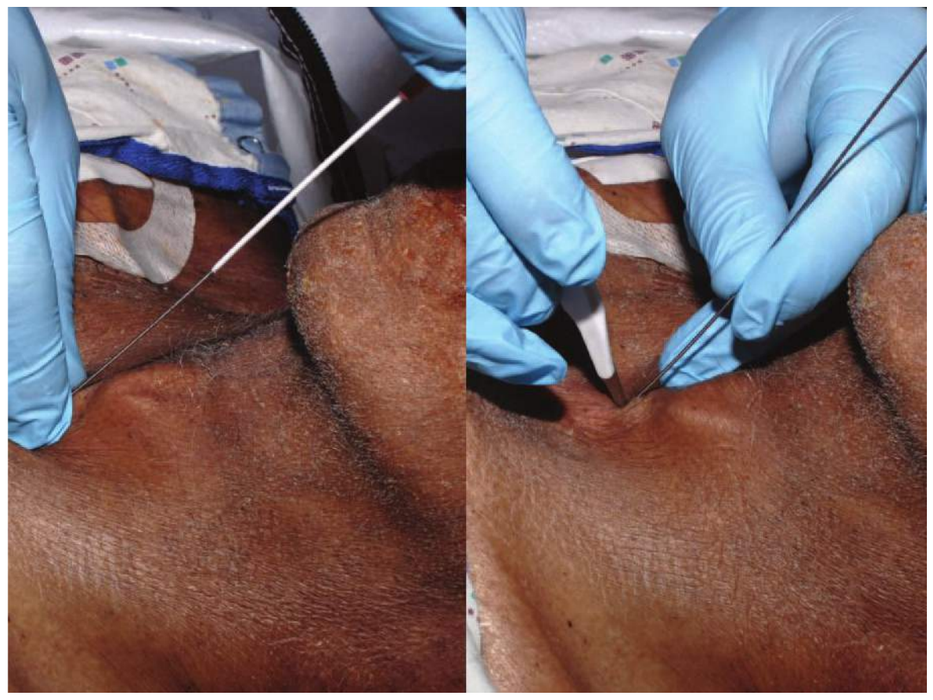


FIGURE 22.81 ■ **Incision at Wire Entry Site.** Remove catheter and make incision at the wire entry site. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.82 ■ **Insert Tracheostomy Tube/Introducer.** Insert both devices over the wire and into the trachea. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.83 ■ **Remove Wire and Introducer.** Remove these devices together as a single unit. (Photo contributor: Lawrence B. Stack, MD.)

Technique

1. Identify the thyroid cartilage and the cricothyroid membrane directly caudally.
2. Cleanse skin surface with appropriate antibacterial medication.
3. Anesthetize skin surface with 1% lidocaine.
4. While stabilizing the trachea with the gloved nondominant hand, make a vertical incision through the skin overlying the cricothyroid membrane.
5. Dissect the tissues over the cricothyroid membrane in a horizontal direction, until the cricothyroid membrane is exposed.
6. Incise the cricothyroid membrane horizontally and insert a tracheal hook to stabilize and control the trachea.
7. If a tracheal dilator is available, insert it into the trachea to dilate and control the trachea to facilitate tube insertion.
8. Insert a cuffed tracheostomy tube, or a 6.0 ID ETT.
9. Inflate cuff and initiate ventilation.

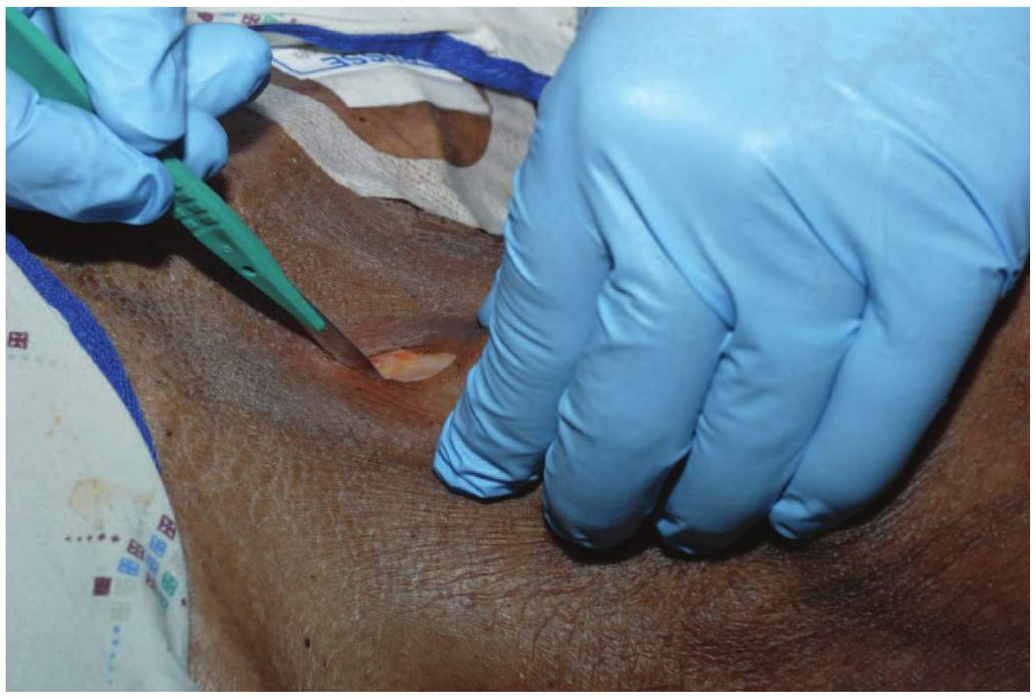


FIGURE 22.84 ■ **Incision over Cricothyroid Membrane.** Stabilize the proximal trachea, make a vertical incision over the cricothyroid membrane. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.85 ■ **Horizontal Incision through Cricothyroid Membrane.** Insert a tracheal hook through the incision to gain proximal control of the trachea. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 22.86 ■ **Insert Tracheal Dilator.** Rotate dilator cephalad. (Photo contributor: Lawrence B. Stack, MD.)

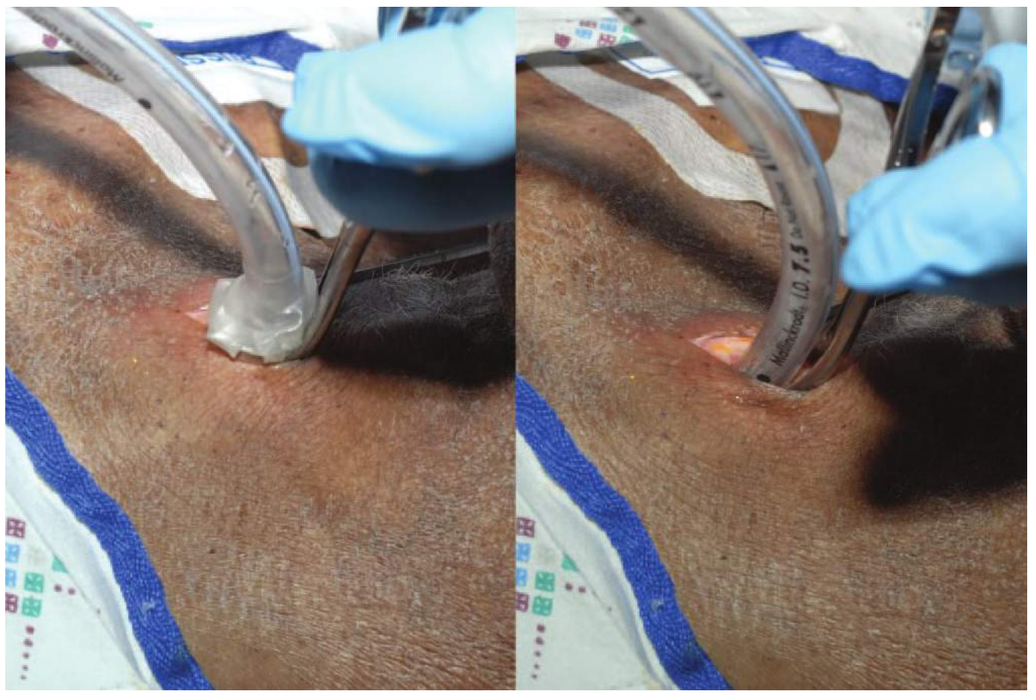
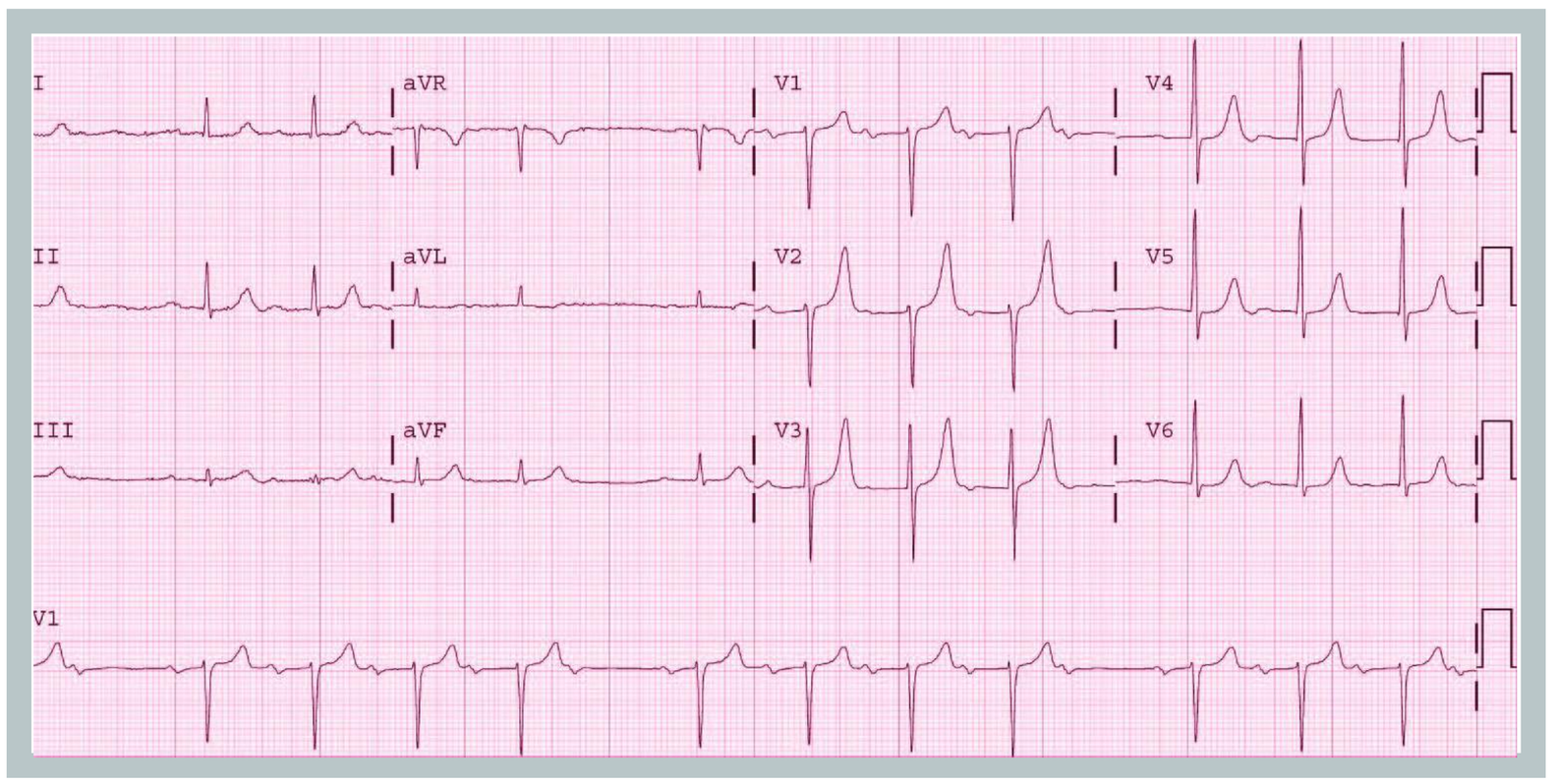


FIGURE 22.87 ■ **Insert ET or Tracheostomy Tube.** Insert a cuffed ETT or tracheostomy tube and remove dilator. (Photo contributor: Lawrence B. Stack, MD.)

Chapter 23

ECG ABNORMALITIES

James V. Ritchie
Michael L. Juliano
R. Jason Thurman



Second-Degree AV Block (Mobitz I, Wenckebach). The PR interval gradually increases until a P wave is not followed by a QRS and a beat is "dropped." The process then recurs. P waves occur at regular intervals, though they may be hidden by T waves. (ECG contributor: James Paul Brewer, MD.)

ACUTE ANTERIOR MYOCARDIAL INFARCTION

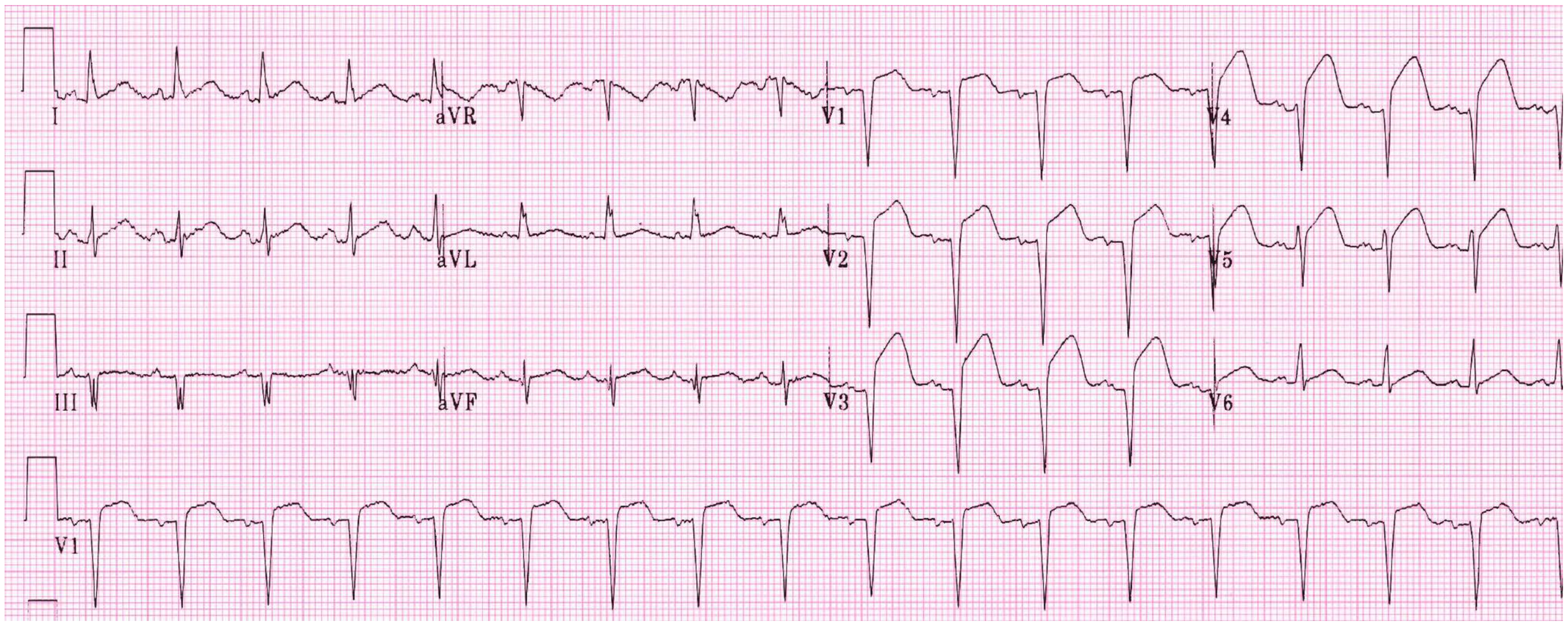


FIGURE 23.1A ■ **Acute Anteroseptal Myocardial Infarction.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment elevation in the anterior precordial leads.
- V_1-V_4: Anteroseptal injury.
- V_3-V_4: Anterior injury.
- V_3-V_6: Anterolateral injury. Leads I and aVL may also be involved, especially if the circumflex artery is affected (high lateral injury).
- Reciprocal ST segment depressions are often present in the inferior leads (II, III, aVF).

Pearls

1. The left anterior descending artery supplies blood to the anterior and lateral left ventricle and ventricular septum.
2. Normal R-wave progression (increasing upward amplitude with R wave > S wave at V_3 or V_4) may be interrupted.
3. The development of pathologic Q waves in any of the V leads other than V_1 strongly suggests that the injury has progressed to an infarction, as seen in this example.

FIGURE 23.1B ■ Pathologic ST-segment elevation beyond 1 mm (double arrow) with pathologic Q waves (arrow) in lead V_3. The ST segment demonstrates a convex upward, or "tombstone," morphology.

ACUTE INFERIOR MYOCARDIAL INFARCTION

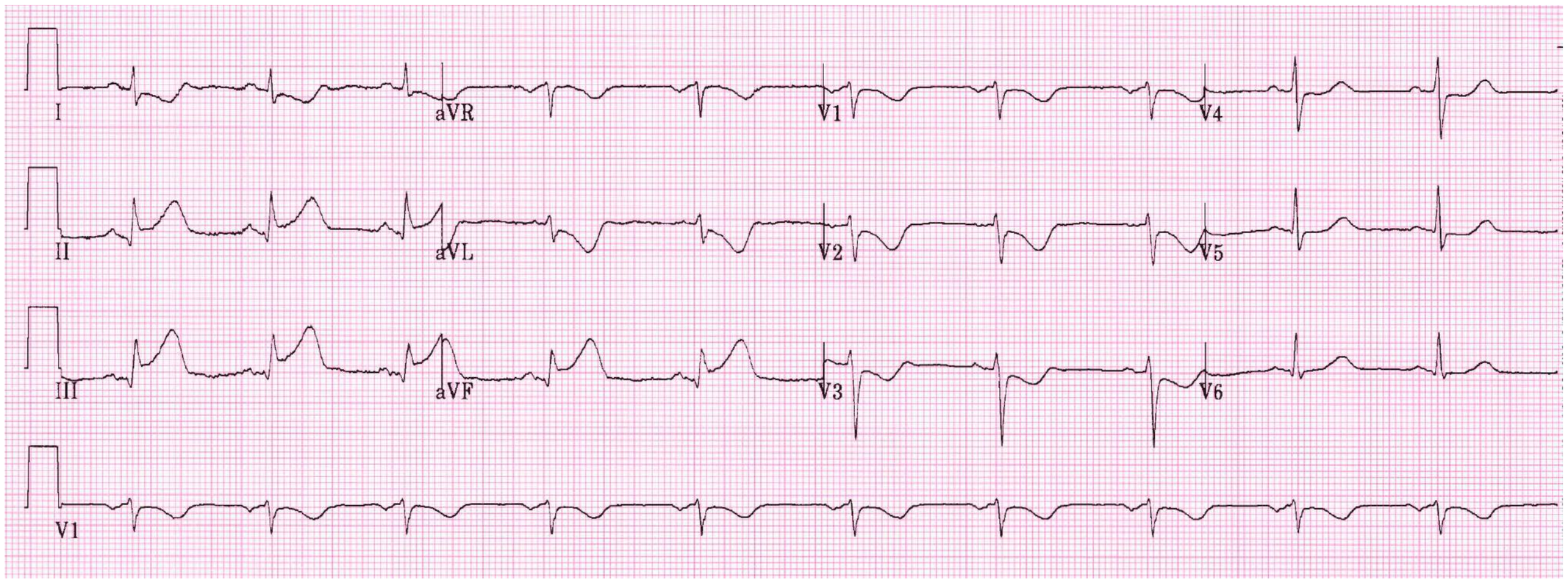


FIGURE 23.2A ■ **Acute Inferior-Posterior Myocardial Infarction.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment elevation in inferior leads (II, III, aVF)
- ST segment depressions in the anterior leads (V_1-V_3) and possibly high lateral leads (I, aVL)

Pearls

1. The right coronary artery supplies blood to the right ventricle, the sinoatrial (SA) node, the inferior portions of the left ventricle, and usually to the posterior portion of the left ventricle and the atrioventricular (AV) node.
2. Infarctions involving the SA node may produce sinus dysrhythmias including tachycardias, bradycardias, and sinus arrest.
3. Infarctions involving the AV node may produce AV blocks.
4. In the presence of acute inferior injury, a right-sided ECG should be obtained to look for right ventricular involvement. The administration of nitroglycerin in the presence of acute right ventricular infarction can precipitate profound hypotension, as these patients are preload-dependent.
5. Since the right coronary artery so often supplies the posterior left ventricle, look for evidence of a posterior infarction (as present in the example) and consider obtaining an ECG with posterior leads.

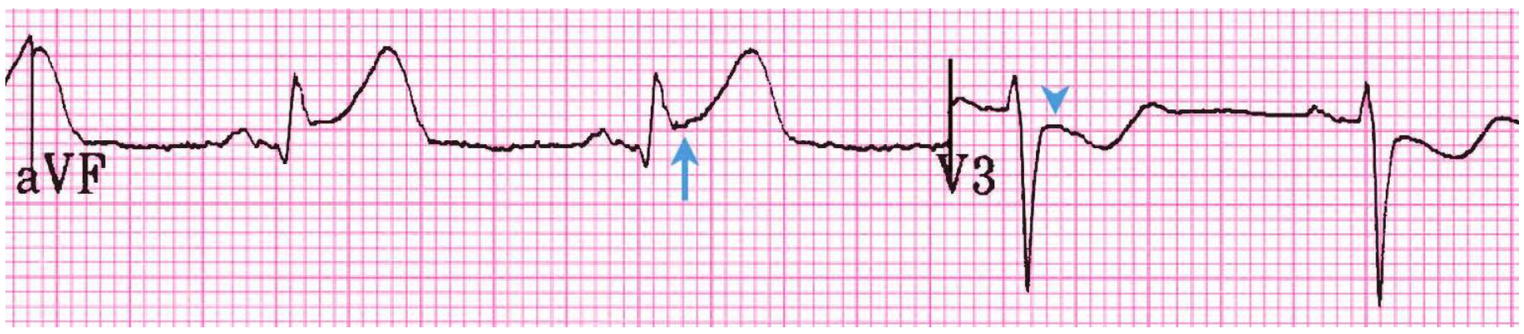


FIGURE 23.2B ■ ST-segment elevation is present in the inferior leads (II, III, aVF) (arrow), with reciprocal ST depression in the anterior leads (V_2-V_4) (arrowhead) and high lateral leads (I, aVL).

ACUTE RIGHT VENTRICULAR MYOCARDIAL INFARCTION

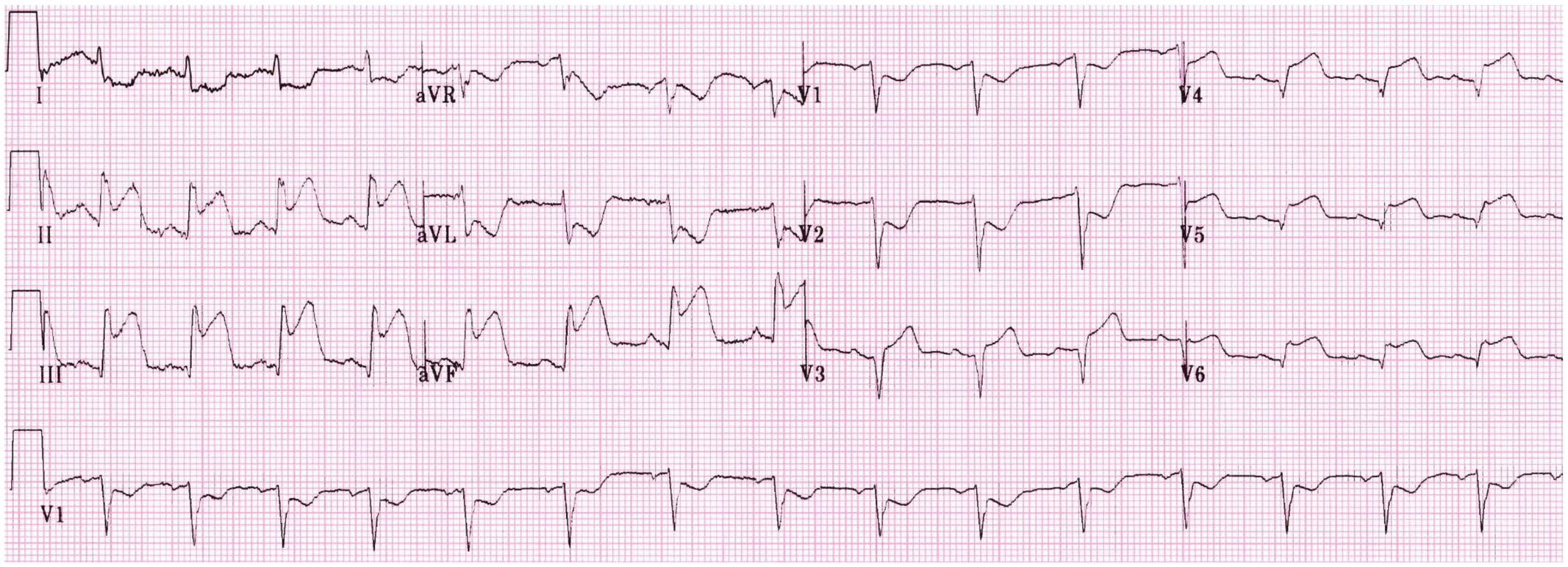


FIGURE 23.3A ■ **Right Ventricular Myocardial Infarction.** This ECG was obtained with right-sided lead placement. (ECG contributor: Thomas Bottoni, MD.)

ECG Findings

- ST elevation in right-sided V leads (V_4R, V_5R).
- ST elevation greater in lead III than lead II suggests RV MI.
- ST elevation in the normally obtained V_1 also strongly suggests RV MI.
- Often associated with inferior MI and/or posterior MI.

Pearls

1. The smaller muscle mass of the right ventricle produces a less intense injury pattern that is overwhelmed by the left ventricle in the normally obtained ECG. Placement of right-sided V leads, with V_1-V_6 in mirror-image locations on the right side of the chest, is important in detecting right ventricular injury.
2. The heart with an injured right ventricle is very preload-dependent. Beware of lowering preload with nitrates in any patient with suspected RV MI as severe hypotension may occur. Treat hypotension with volume.
3. Obtain a right-sided ECG in any patient with inferior or posterior MI, and in any patient with a significant hypotensive response to nitrates.

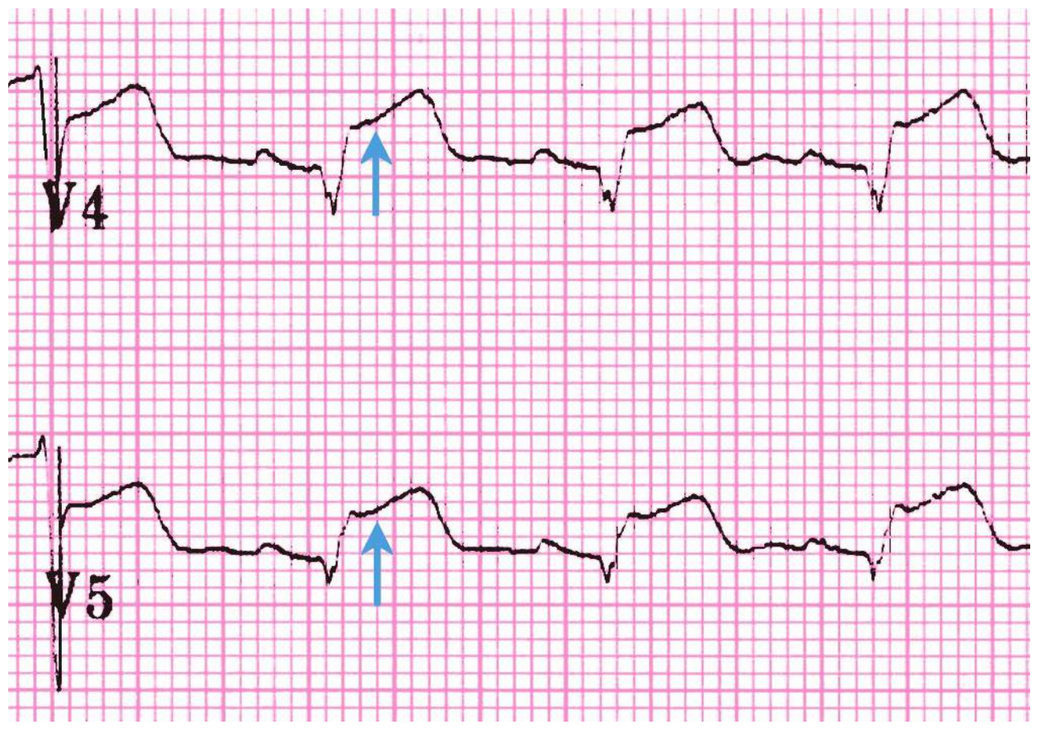


FIGURE 23.3B ■ ST elevation in V_4R and V_5R (arrows), with the V_4 and V_5 leads placed in their mirror-image locations on the right side of the chest. Any ST elevation seen in the right-sided precordial leads is significant.

ACUTE POSTERIOR MYOCARDIAL INFARCTION

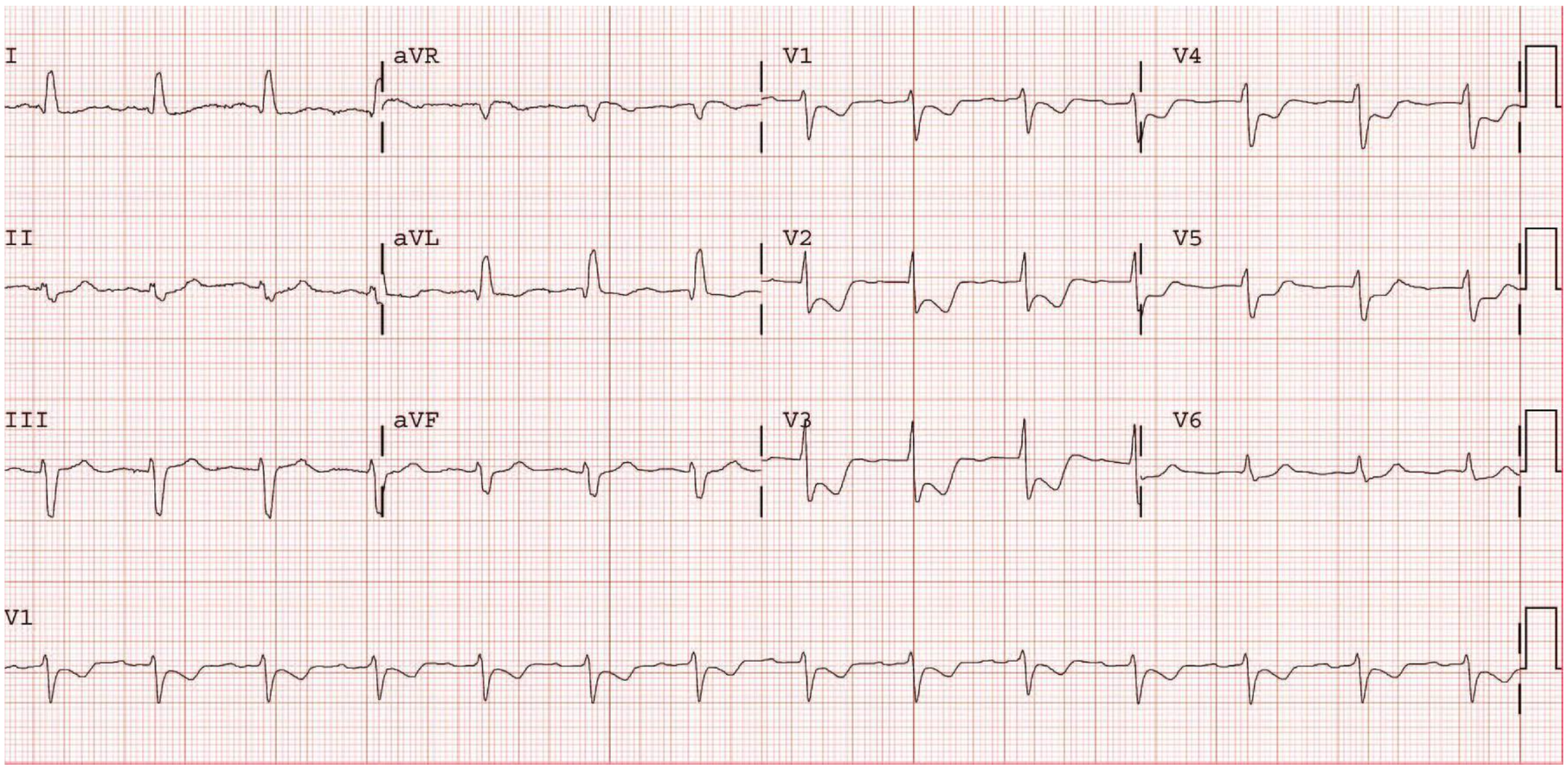


FIGURE 23.4A ■ **Acute Posterior Myocardial Infarction.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- With acute injury pattern—ST segment depression in lead V_1 and/or V_2 with acute injury pattern
- With infarction pattern—Small S wave and large R wave greater than 4 ms duration in lead V_1 or V_2 with infarction
- With infarction pattern—R-wave/S-wave ratio greater than 1 in lead V_1 or V_2 with infarction

Pearls

1. The posterior portion of the left ventricle has no EKG electrodes directly overlying it and is the last portion of ventricle to depolarize. It receives its blood supply from either the right coronary artery (in 85% of individuals) or the circumflex artery (in 15% of individuals).
2. V_1 and V_2 are primarily affected as the most anterior leads and indirectly assess the posterior left ventricle, though in an "inverted" orientation. Instead of observing downgoing Q waves and ST elevation, one expects to see large upgoing R waves and ST depression. By holding the EKG up to a backlight upside down and horizontally flipped, the more classic injury pattern can be observed by looking through the EKG paper (see Fig. 23.4C).
3. Posterior involvement may be confirmed with posterior leads. V_8 is located at inferior tip of left scapula; V_9 is positioned between V_8 and the spine at the same level.
4. Frequently, an inferior MI is also present with a posterior MI, since the right coronary artery serves both areas. In the above example, there is subtle ST elevation in the lateral leads, indicating posterior-lateral injury.

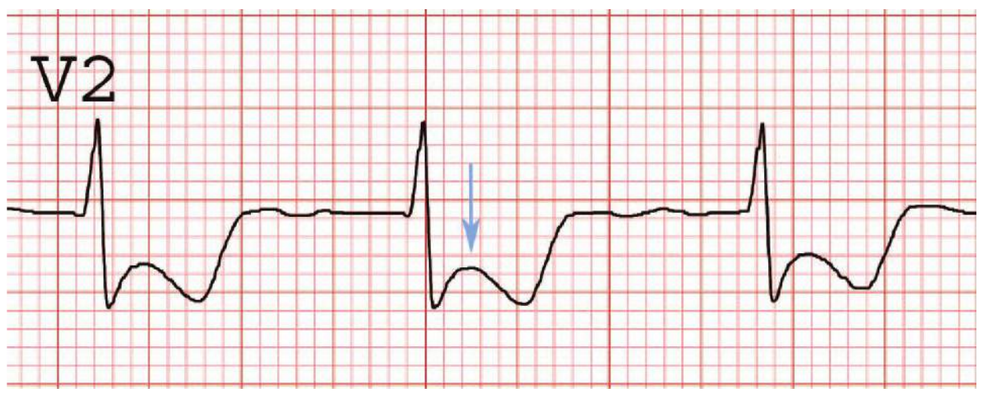


FIGURE 23.4B ■ This tracing demonstrates injury in the posterior LV, manifesting as acute ST depression in V_2 (arrow).

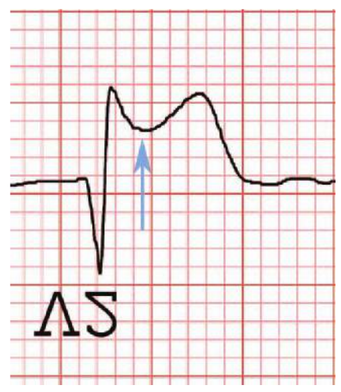


FIGURE 23.4C ■ By inverting and rotating the EKG, the characteristic ST-elevation injury pattern is easily seen (arrow). This can be done in practice by flipping the EKG upside down and looking through the printed EKG with a backlight.

FIGURE 23.4D ■ The ST depression is subtle and downsloping. However, the R-wave amplitude approximates that of the S wave, and the R-wave duration is significant (>4 ms). This is actually an "inverted Q wave" from a posterior infarction that has evolved since the initial tracing.

LEFT MAIN LESION

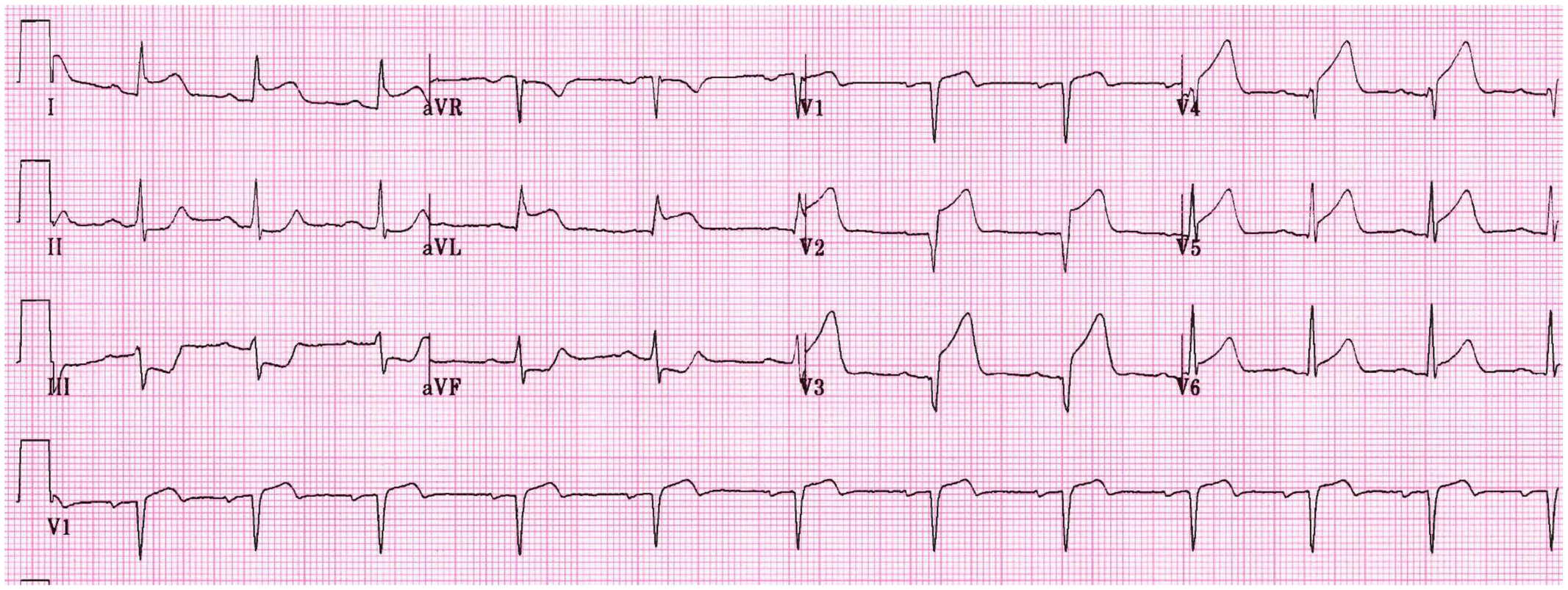


FIGURE 23.5A ■ **Left Main Coronary Artery Lesion (Widowmaker).** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment elevation in the precordial leads (V_2-V_6) and high lateral leads (I, aVL).
- Reciprocal ST segment depressions in the inferior leads (II, III, aVF).
- ST segment elevation in AVR coupled with reciprocal ST depressions may indicate AMI from left main disease.

Pearls

1. The left main coronary artery branches into the left anterior descending artery and the circumflex artery. It supplies blood to the ventricular septum and the anterior and lateral aspects of the left ventricle, usually sparing the posterior and inferior portion, which is most often served by the right coronary artery.
2. Normal R-wave progression (increasing R-wave amplitude across the precordial leads) may be interrupted.
3. Risk of cardiogenic shock is high since so much of the left ventricle is served by the left main coronary artery.
4. A left main coronary thrombosis is also known as the "widowmaker lesion."
5. Isolated ST segment elevation in lead aVR may also indicate acute myocardial ischemia from left main coronary artery disease.

FIGURE 23.5B ■ Significant ST elevation is present in the precordial leads (V_2-V_6) and high lateral leads (I and aVL) (arrows). In this example, significant Q waves have appeared, signaling infarction (arrowhead).

SGARBOSSA CRITERIA FOR AMI IN SETTING OF LBBB

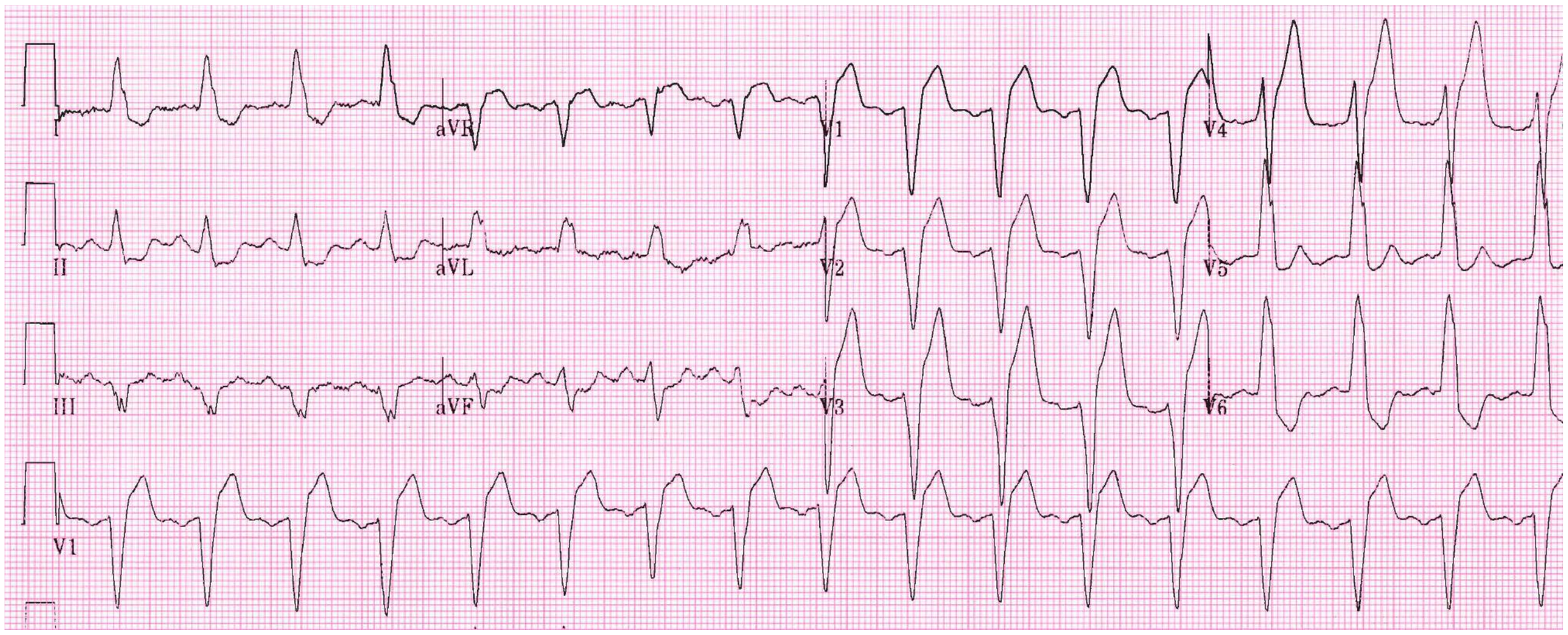


FIGURE 23.6A ■ **Acute Myocardial Infarction by Sgarbossa Criteria in the Setting of Underlying LBBB.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST elevation greater than or equal to 1 mm concordant with QRS deflection (score = 5)
- ST depression greater than or equal to 1 mm in leads V_1, V_2, V_3 concordant with QRS deflection (score = 3)
- ST elevation greater than or equal to 5 mm discordant with QRS deflection (score = 2)

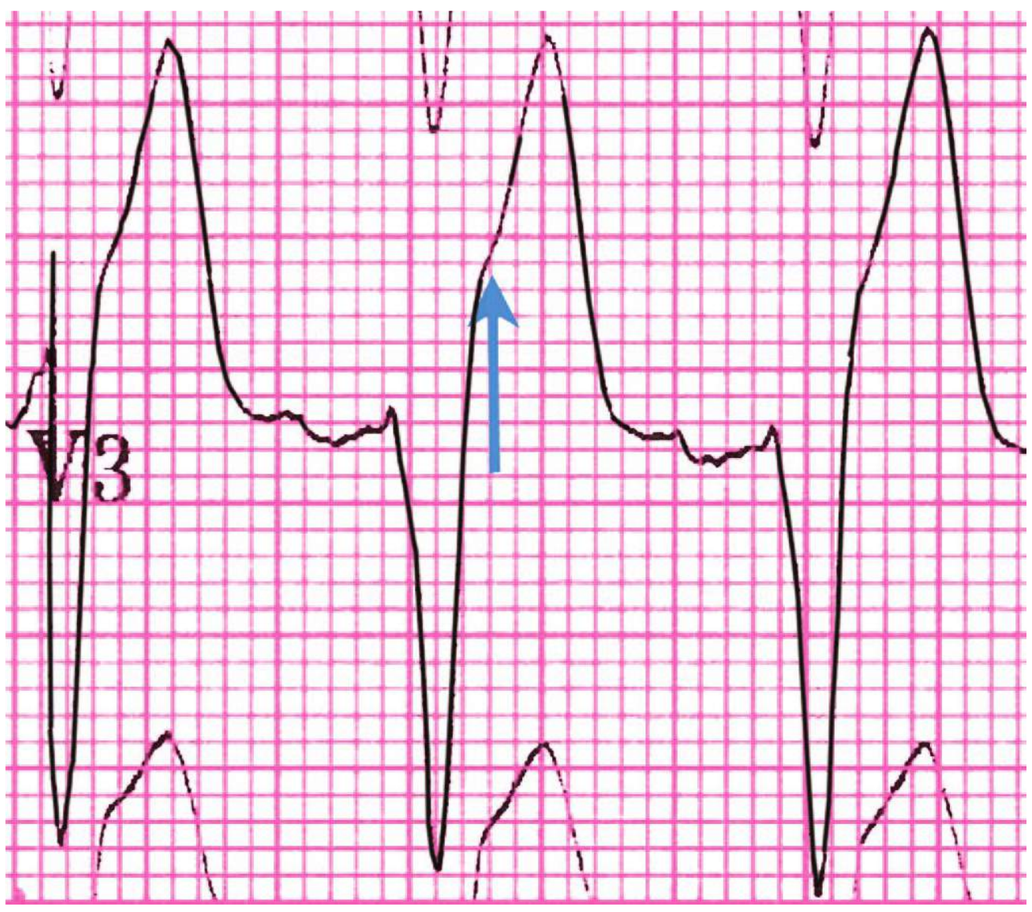


FIGURE 23.6B ■ The ST elevation is greater than 5 mm discordant from the primary QRS deflection (arrow).

Pearls

1. These scored criteria may be used to diagnose acute myocardial infarction in the setting of a left bundle branch block (LBBB). However, most myocardial ischemia in the setting of LBBB does not produce these changes. An absence of these findings should not be used as evidence against acute coronary syndrome.
2. Score of greater than or equal to 3 gives a specificity for myocardial infarction of 90%.
3. The first and third criteria listed above may also be used in ECGs with wide QRS complexes resulting from a pacemaker or idioventricular rhythm.

FIGURE 23.6C ■ The ST depression is greater than 1 mm concordant to the primary QRS deflection (arrow).

SUBENDOCARDIAL ISCHEMIA

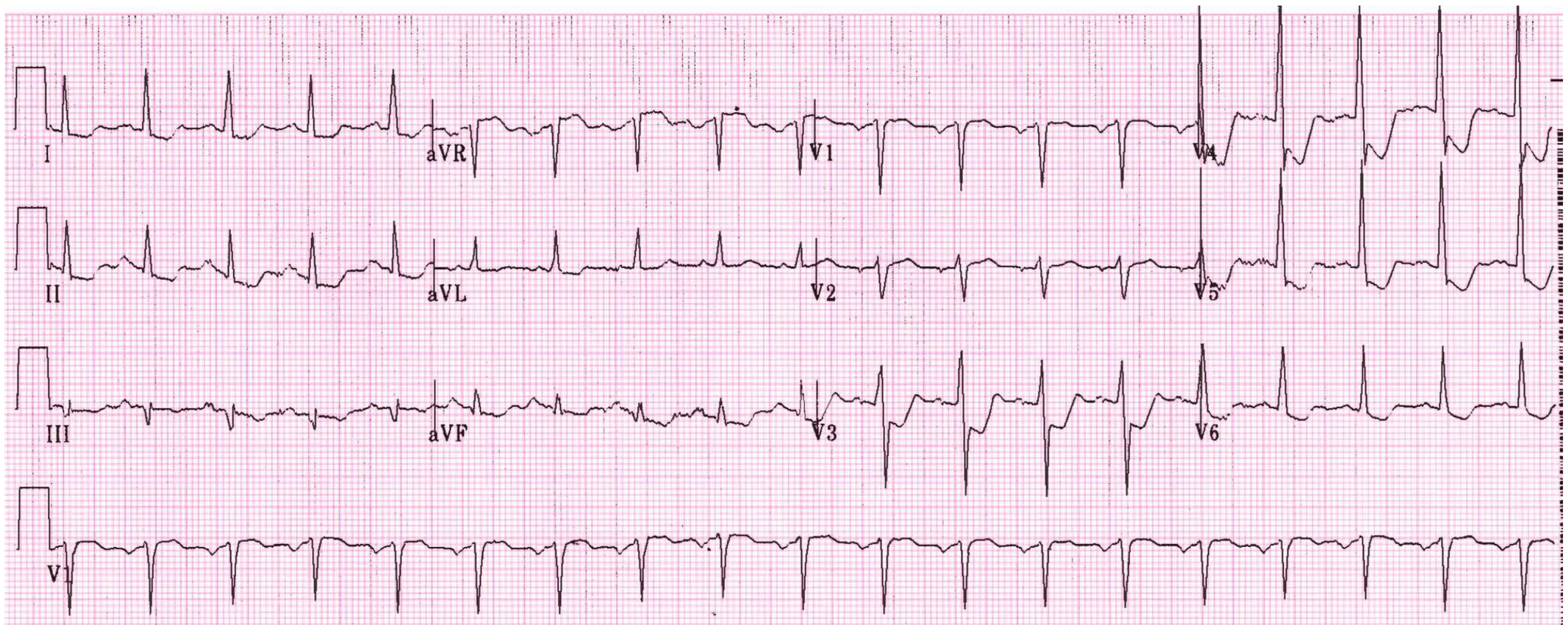


FIGURE 23.7A ■ **Subendocardial Ischemia.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment depression greater than or equal to 1 mm in anatomically adjoining leads.
- ST segments may be horizontal or downsloping with acute ischemia.

Pearls

1. Some ST depression in the lateral precordial leads (V_4-V_6) is common at higher heart rates, and is commonly seen during exercise treadmill tests, but such depression should not be downsloping unless ischemia is also present.
2. ST elevation in other leads suggests that the depression may represent reciprocal changes from acute injury rather than subendocardial ischemia.
3. Downsloping ST depression may also be seen in left ventricular hypertrophy (LVH), but this depression should not be dynamic, and should be stable with serial ECGs. ST depression from ischemia will be dynamic, changing with time on serial ECGs.
4. Isolated ST depression in leads V_1 and V_2 may represent posterior ischemia.

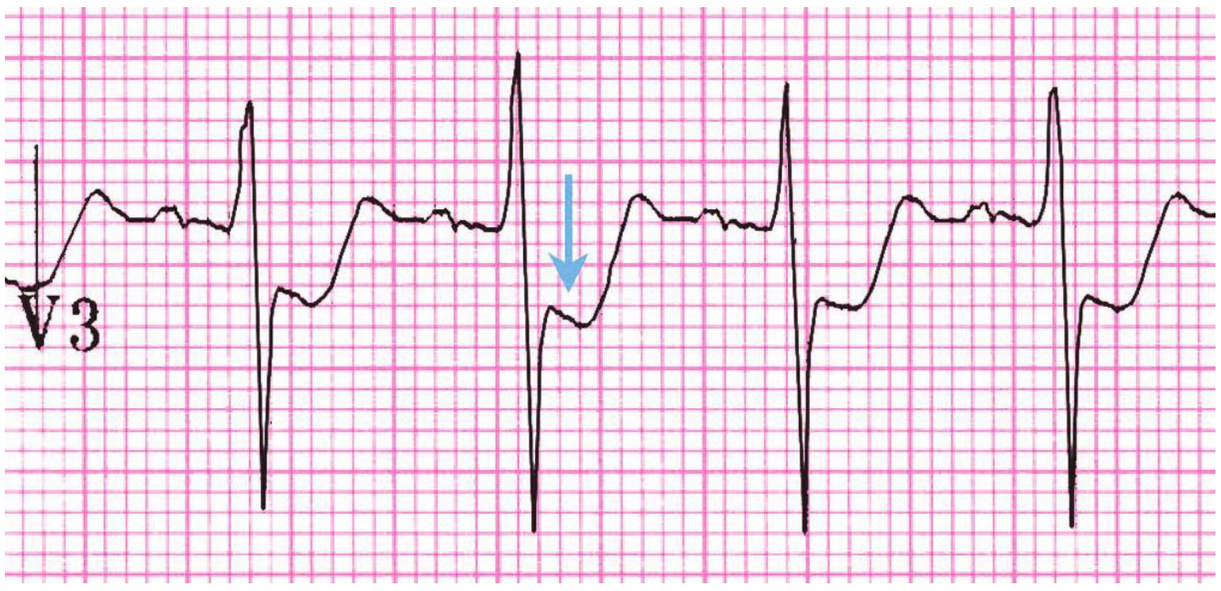


FIGURE 23.7B ■ Downsloping ST segments depressed greater than 1 mm (arrow). These changes were dynamic over time. The patient sustained a nontransmural myocardial infarction.

HYPERACUTE T WAVES

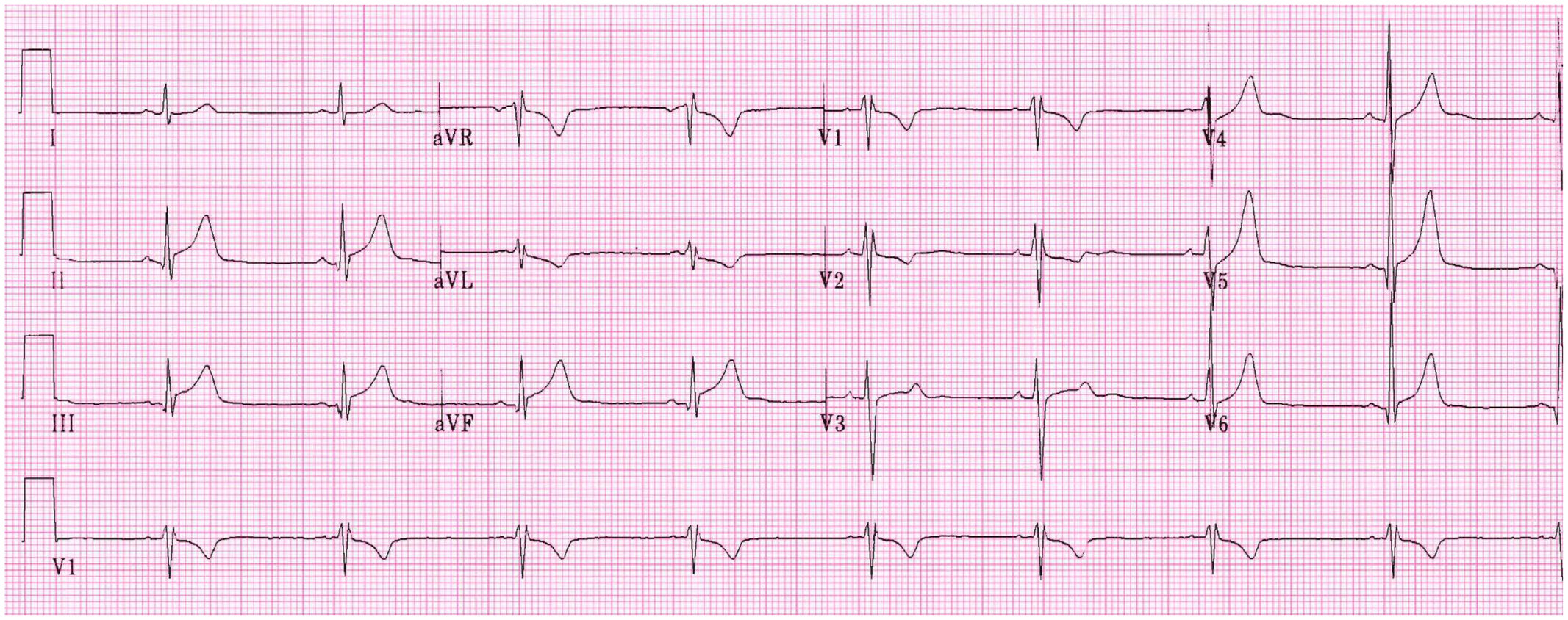


FIGURE 23.8A ■ **Hyperacute T Waves.** T waves in a patient with acute myocardial ischemia. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- T-wave amplitude/QRS amplitude greater than 75%.
- T waves greater than 5 mV in the limb leads.
- T waves greater than 10 mV in the precordial leads.
- T waves have asymmetric appearance.

FIGURE 23.8B ■ T-wave height is greater than 10 mm in V_5 (double arrow) and is asymmetric. This height was transient and was significantly diminished in a tracing obtained 15 minutes later. Note also in the 12-lead ECG example above the presence of inferior ST elevation.

Pearls

1. Hyperacute T waves occur very early (within minutes) during myocardial injury and are transient.
2. The term "hyperacute T waves" is reserved for the early stages of myocardial infarction. "Prominent T waves" can also be seen with LVH, early repolarization, or with hyperkalemia.
3. Serial ECGs are useful in distinguishing transient hyperacute T waves from other causes of tall, peaked T waves.

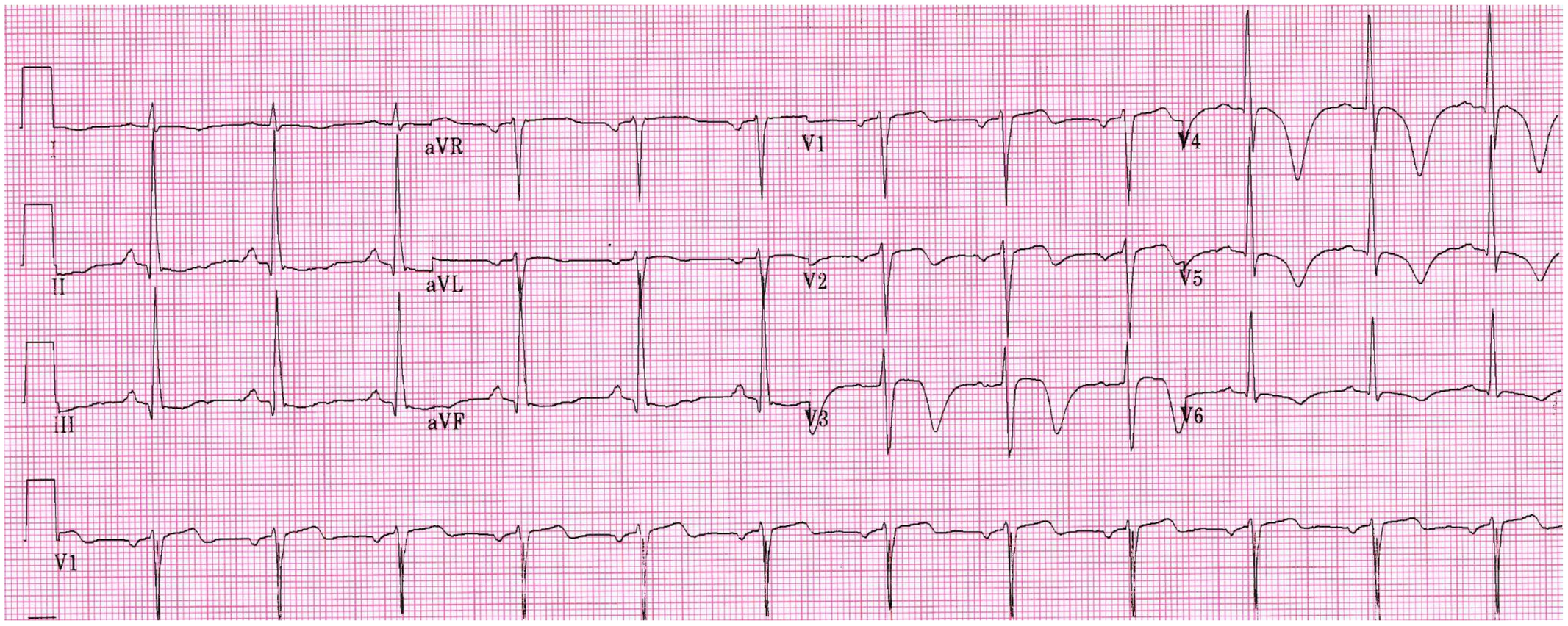


FIGURE 23.9A ■ **Wellens Waves.** Wellens waves are present and are indicative of a high-grade LAD lesion. (ECG contributor: James V. Ritchie, MD.)

ECG Findings (Two Classic Types)

- Biphasic T waves in anterior and/or lateral leads
- Deeply inverted, symmetrical T waves in the same leads

Pearls

1. These characteristic patterns of T-wave changes are closely associated with critical left anterior descending artery stenosis.
2. The changes are classically apparent on ECG after resolution of chest pain.
3. These changes are transient, and often are not associated with cardiac enzyme elevations.
4. Wellens waves are not associated with changes in R-wave progression.
5. Serial electrocardiograms may assist in differentiating Wellens waves from stable, nonspecific findings.

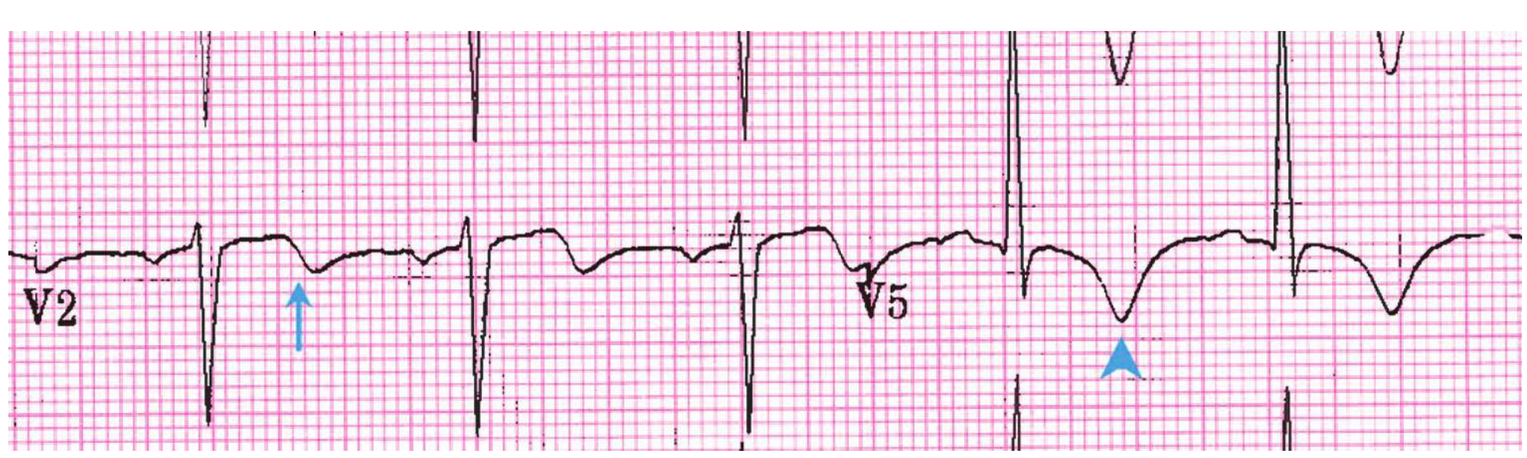


FIGURE 23.9B ■ Biphasic T waves with the later segment inverted, as in V_2 above (arrow), or deep symmetric inverted T waves, as in V_5 above (arrowhead). These findings in the precordial leads, in the setting of suspected ACS, strongly suggest an underlying high-grade LAD lesion.

"CEREBRAL" T WAVES

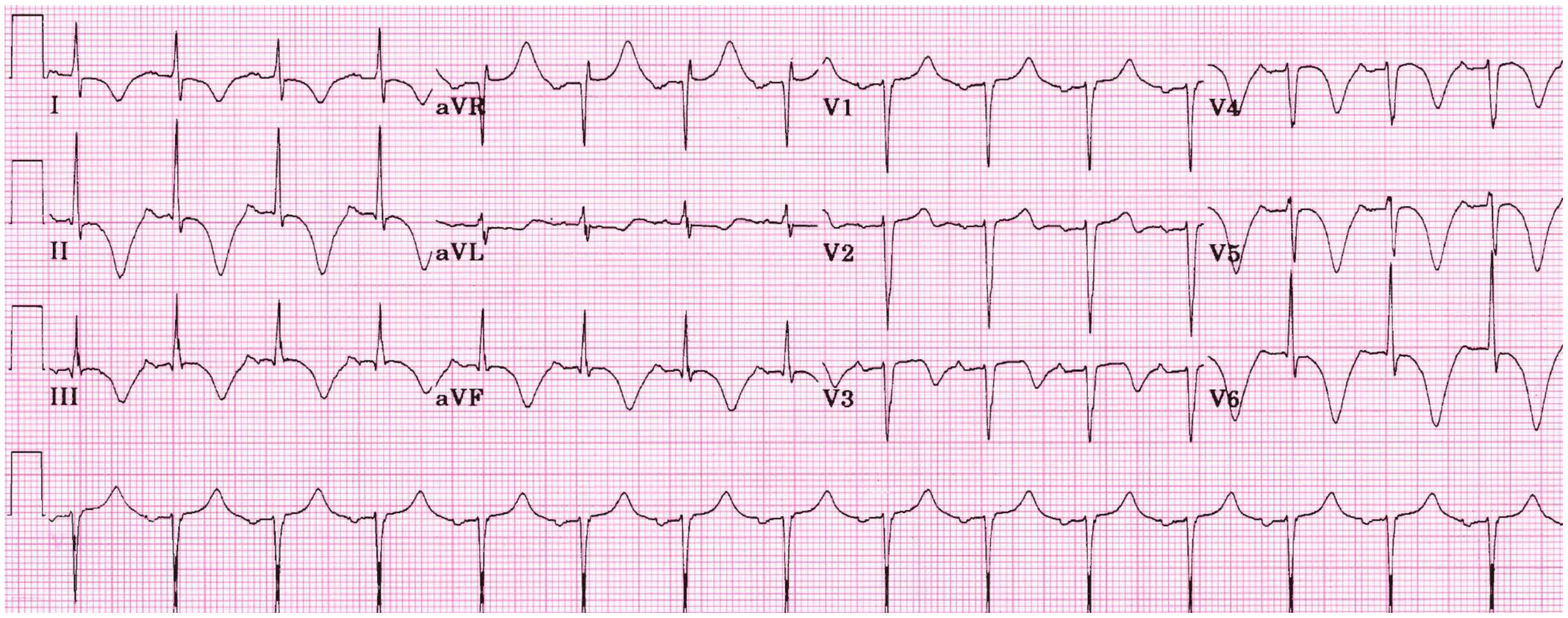


FIGURE 23.10A ■ **Cerebral T Waves.** This ECG was obtained on a patient with a severe acute hemorrhagic CVA. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Inverted, wide T waves are most notable in precordial leads (can be seen in any lead).
- QT interval prolongation.

Pearls

1. These are associated with acute cerebral disease, most notably an ischemic cerebrovascular event or subarachnoid hemorrhage.
2. They may be accompanied by ST segment changes, U waves, and/or any rhythm abnormality.
3. Differential diagnosis includes extensive myocardial ischemia.
4. Strongly suspect an intracranial etiology in a patient with altered mental status and these electrocardiographic findings.

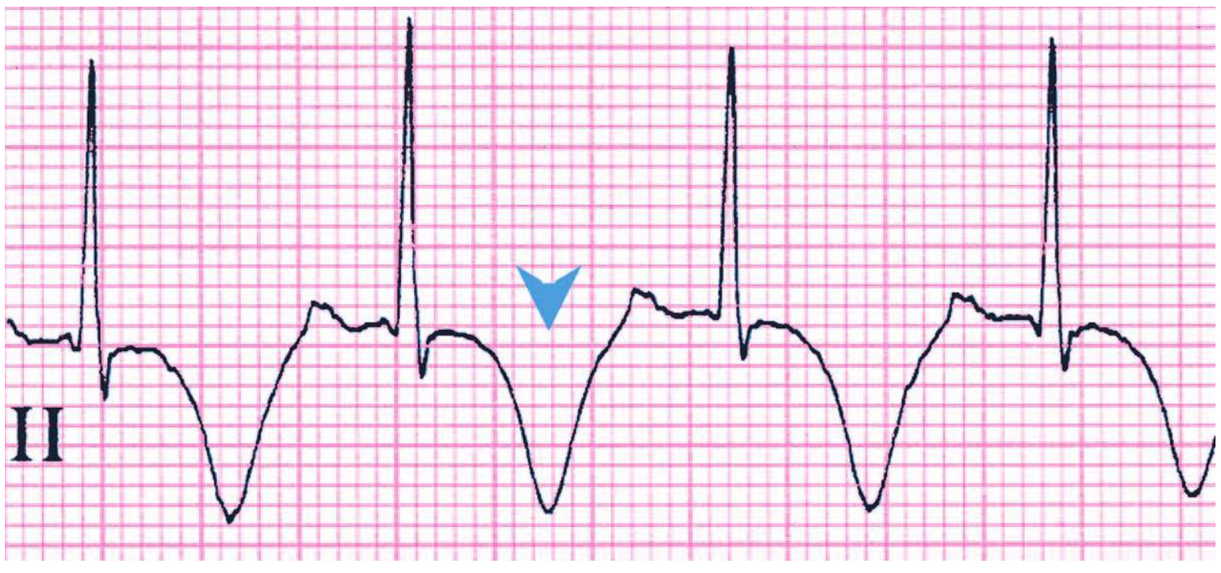


FIGURE 23.10B ■ Deep, symmetrical, inverted T waves (arrowhead) with a prolonged QT interval.

EARLY REPOLARIZATION

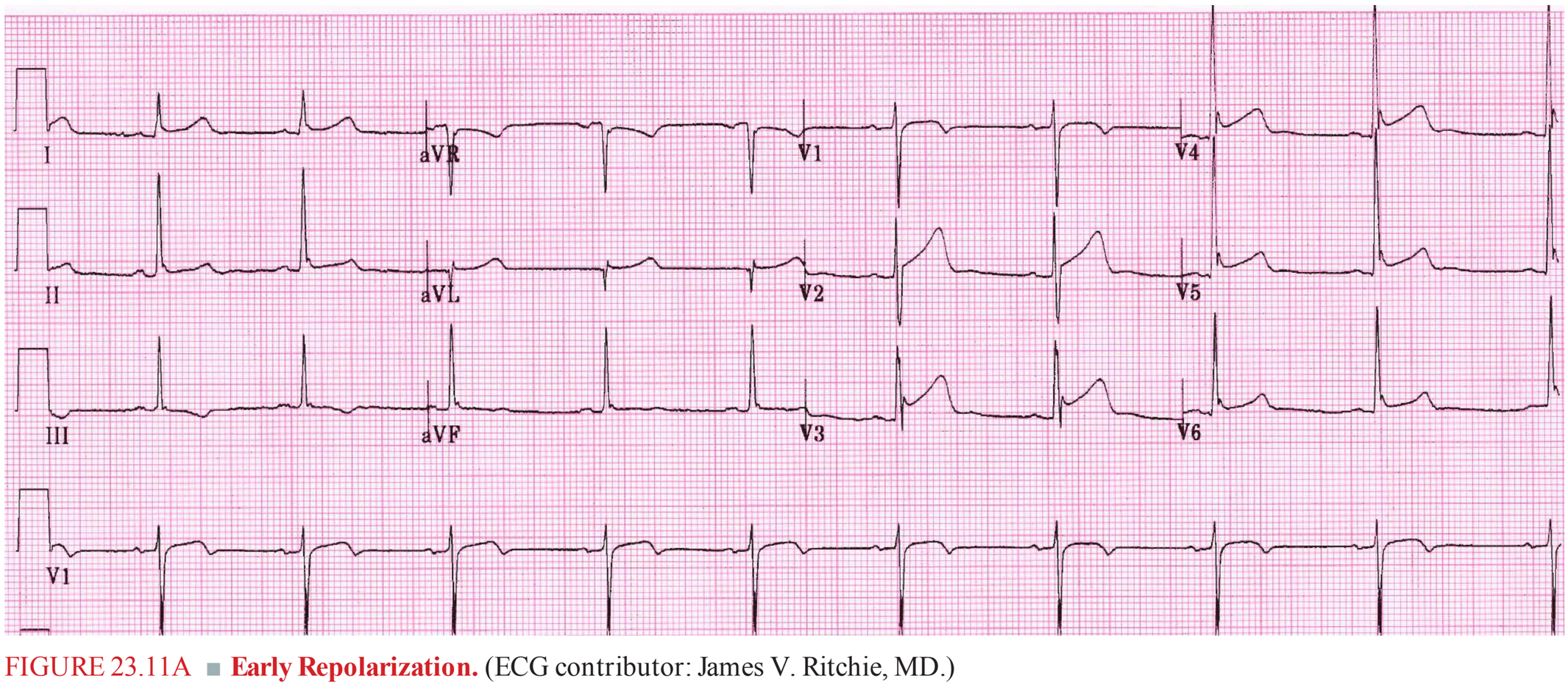


FIGURE 23.11A ■ **Early Repolarization.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST elevation, usually in the anterior leads.
- J-point elevation, but usually less than one-third the total height of the T wave.
- ST segment is "concave upward," or "holds water," or "is smiling at you."
- J-point notch strongly suggests early repolarization, but is not always present.

Pearls

1. This is a normal variant and is especially common in young healthy males, but also may be present in other groups. However, any clinical suspicion for ongoing myocardial ischemia should prompt further investigation
2. Q waves and reciprocal ST-segment depression in other leads should not accompany early repolarization. If present, they strongly suggest ischemia as the cause for the ST elevation.

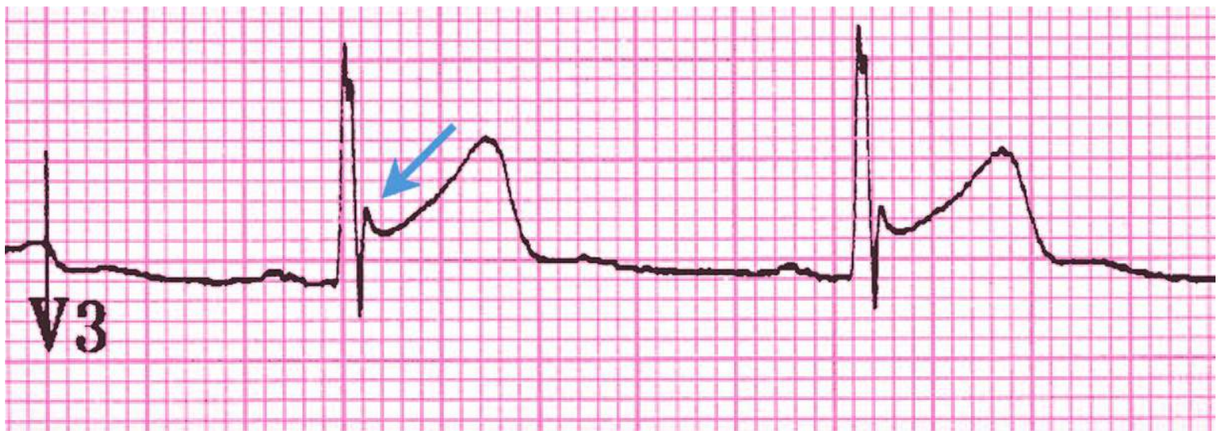


FIGURE 23.11B ■ ST elevation in precordial leads, with a concave-upward ST segment and a J-point notch (arrow).

LEFT VENTRICULAR ANEURYSM

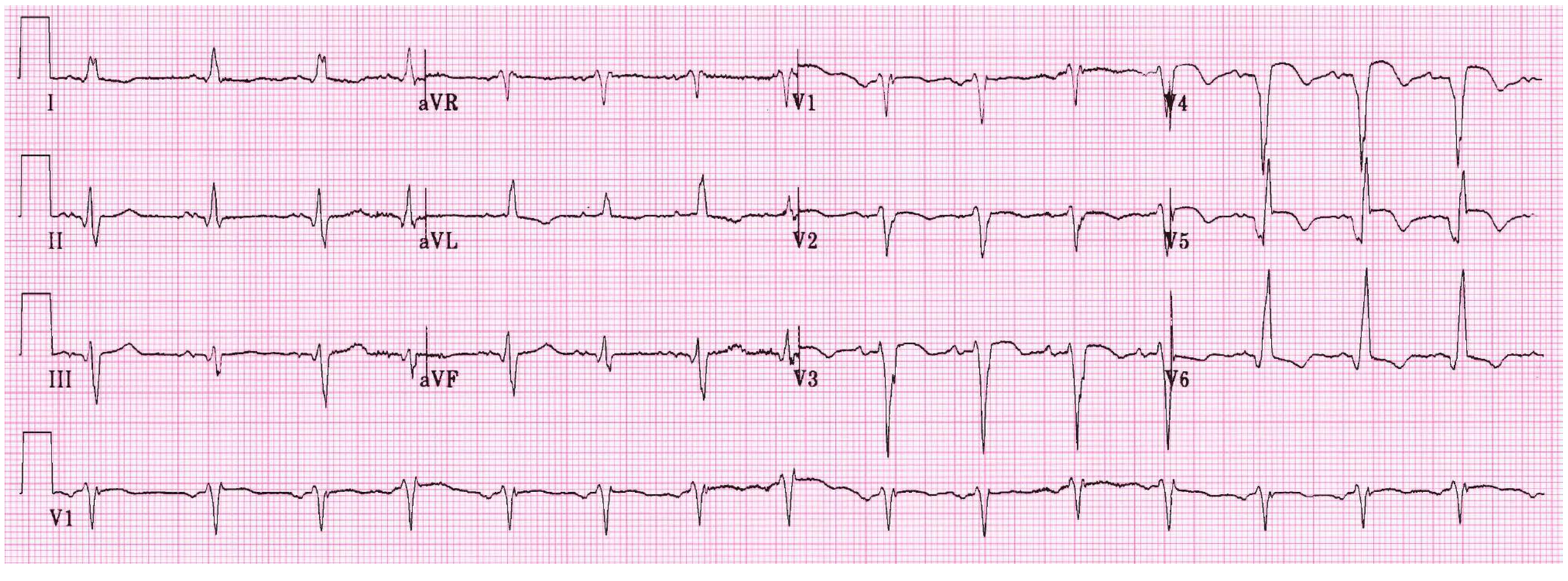


FIGURE 23.12A ■ **Left Ventricular Aneurysm.** This ECG was obtained on an asymptomatic patient with history of MI 2 years prior. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST elevation in anterior contiguous leads
- Deep pathologic Q waves in anterior leads

Pearls

1. ST segment elevation which occurs in the setting of a myocardial infarction should resolve within days under normal circumstances.
2. Persistent ST-segment elevation occurring for weeks or longer after a myocardial infarction is suspicious for ventricular aneurysm.
3. Ventricular aneurysms may follow a large myocardial infarction in the anterior portion of the heart.
4. The aneurysm consists of scarred myocardium, which does not contract but bulges outward during systole; complications include congestive heart failure, myocardial rupture, arrhythmias, and thrombus formation.
5. Suspect an LV aneurysm when these findings appear in the ECG of a patient who does not demonstrate symptoms suggesting ACS. However, one should also be vigilant for the presence of "silent" ACS.

FIGURE 23.12B ■ Persistent ST elevations (arrow) and deep, pathologic Q waves (arrowhead) in an asymptomatic patient with a history of anterior myocardial infarction 2 years earlier.

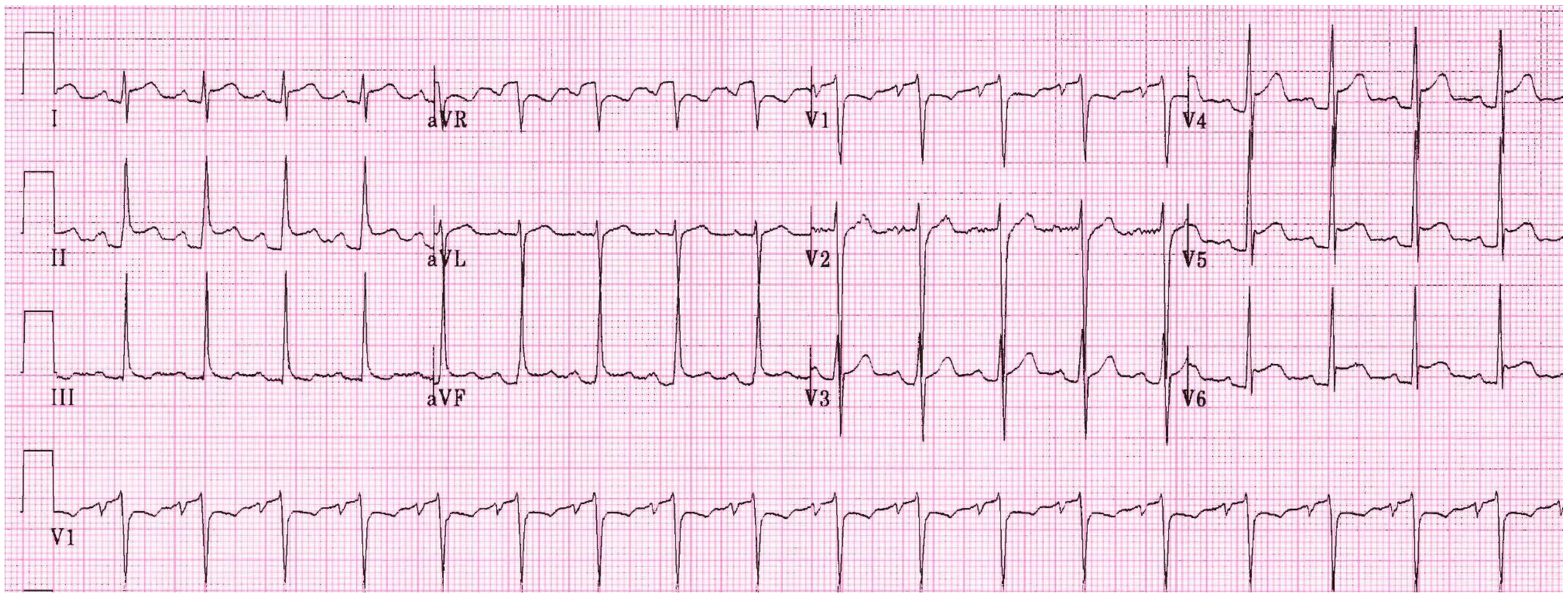


FIGURE 23.13A ■ **Acute Pericarditis.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Diffuse ST elevation in noncontiguous leads
- PR depression
- T-wave flattening or inversion
- PR elevation in lead aVR

Pearls

1. Pericarditis may produce inflammation of the epicardium. This is most often demonstrated on the ECG as a widespread injury pattern.
2. Pericarditis does not produce abnormal Q waves. The presence of abnormal Q waves must prompt consideration of acute or old coronary syndrome, including Dressler syndrome or postinfarct pericarditis.
3. Pericarditis may be focal, resulting in regional rather than diffuse EKG changes.
4. Benign early repolarization and myocarditis may also appear as ST elevation in many noncontiguous leads.

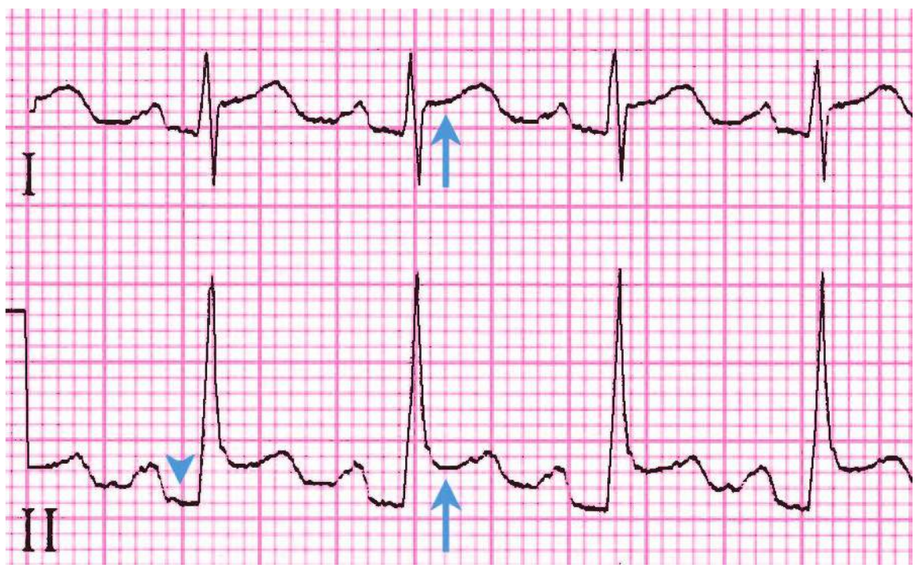


FIGURE 23.13B ■ ST elevation in noncontiguous leads I and II (arrows) with PR depression (arrowhead). No pathologic Q waves or reciprocal changes are present.

FIRST-DEGREE AV BLOCK

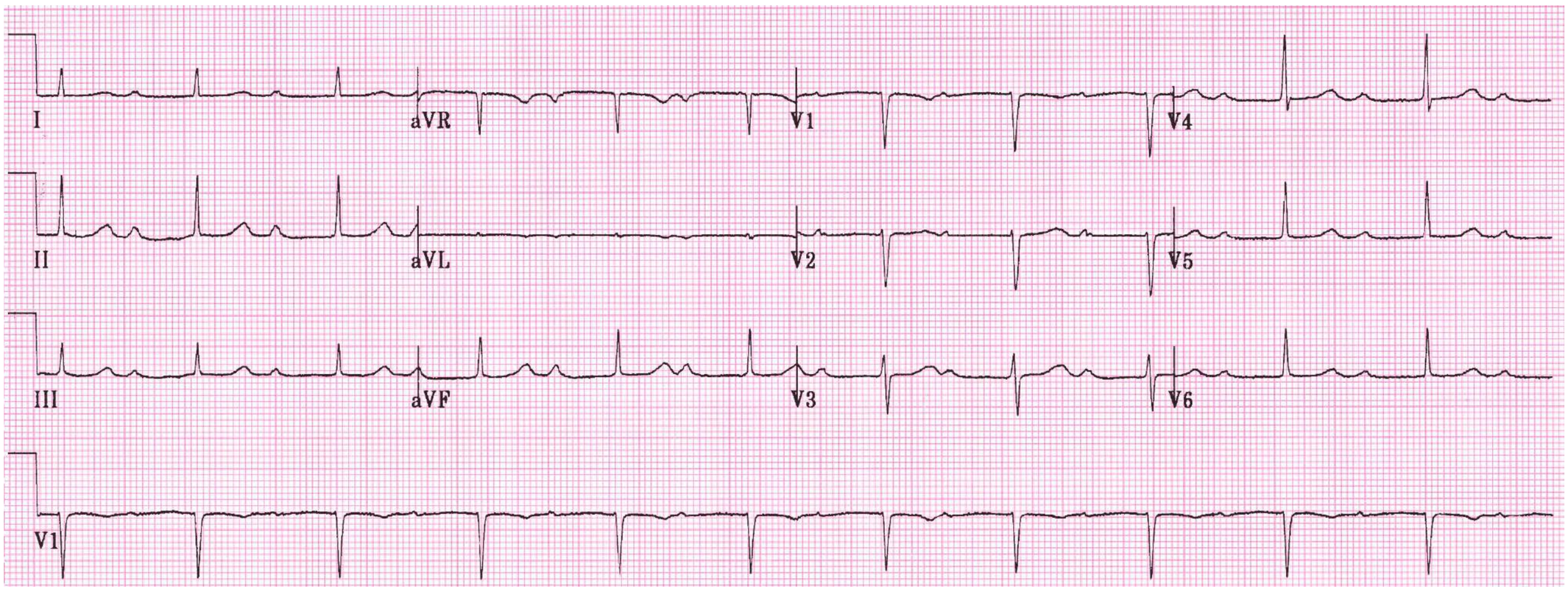


FIGURE 23.14A ■ **First-Degree AV Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- A PR interval greater than 200 ms (normal 120-200 ms) with no significant variation in PR intervals between beats.
- Each P wave is followed by a QRS complex.

Pearls

1. This type of heart block usually does not affect heart function and can be considered nonpathologic (especially in athletes or patients with higher vagal tone).
2. First-degree block may also be due to heart disease (myocarditis, rheumatic fever) or drugs (digoxin, amiodarone, β-blockers, calcium channel blockers).

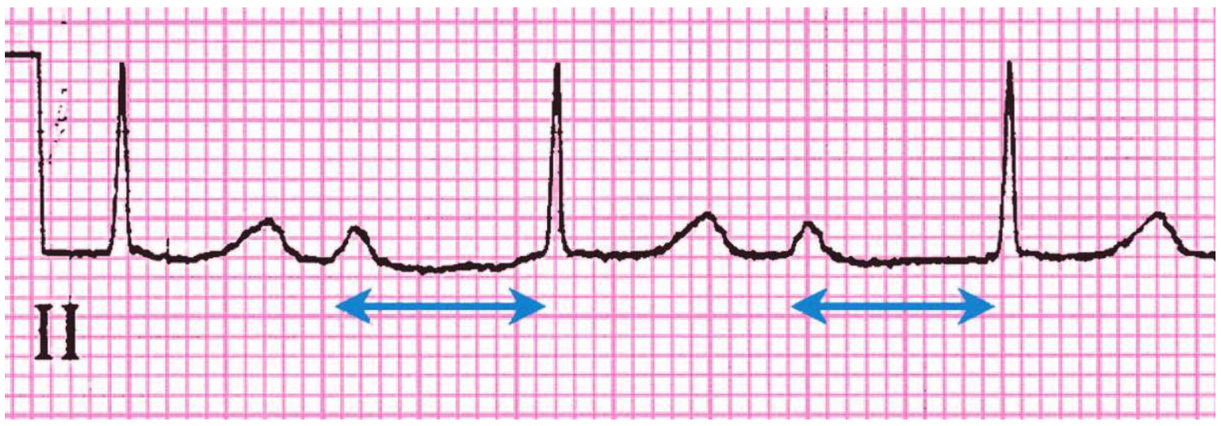


FIGURE 23.14B ■ The PR interval is fixed (double arrows) and is longer than 0.2 seconds, or five small blocks.

TYPE 1 SECOND-DEGREE AV BLOCK (MOBITZ I, WENCKEBACH)

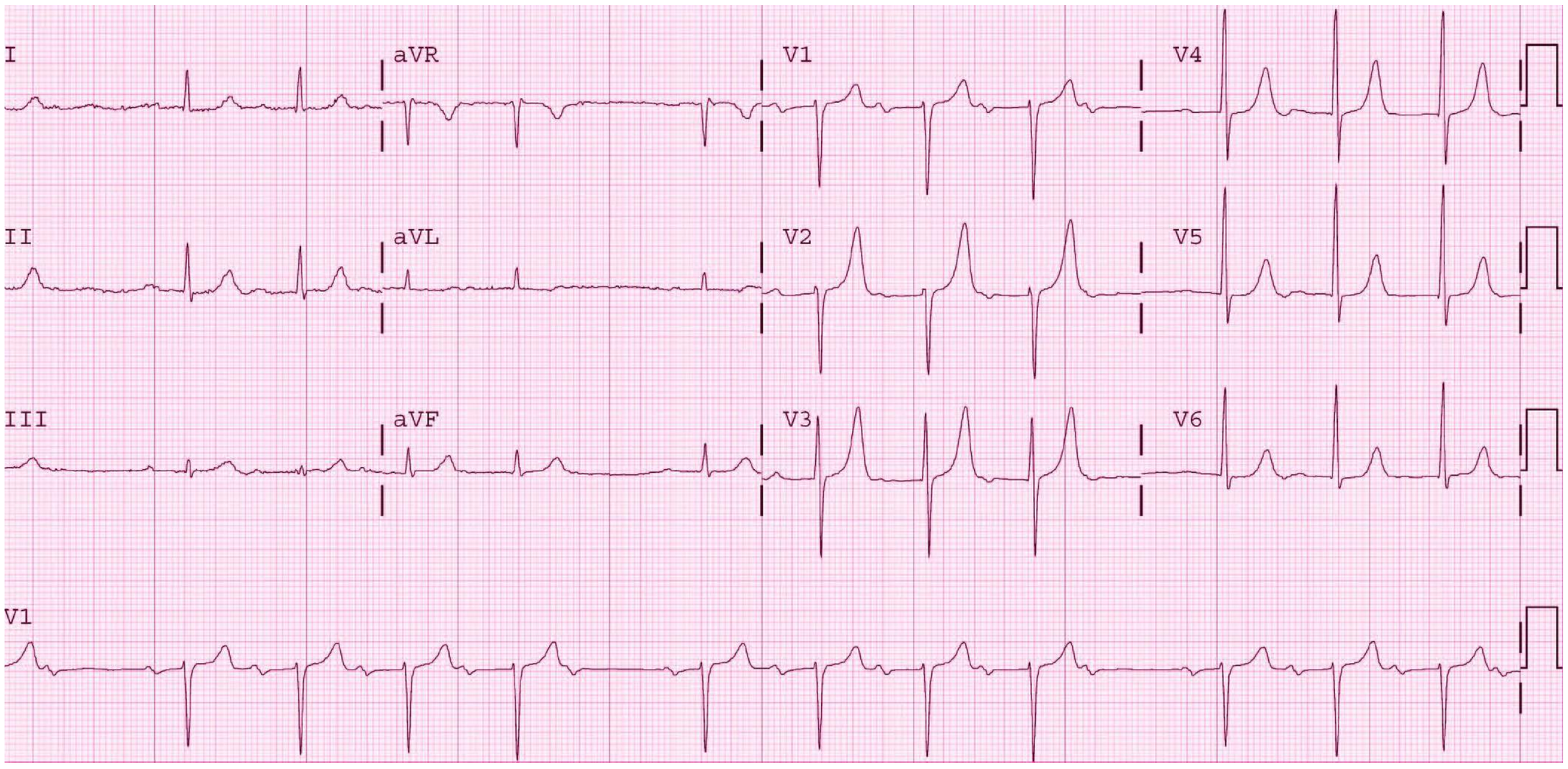


FIGURE 23.15A ■ **Second-Degree AV Block (Mobitz I, Wenckebach).** (ECG contributor: James Paul Brewer, MD.)

ECG Findings

- Progressive PR-interval prolongation throughout the cardiac cycle until a P wave occurs without a QRS complex ("dropped" beat).
- After the dropped QRS complex, the cycle continues again with the PR interval of the first beat in the cycle always shorter than the PR interval of the last beat in the previous cycle.
- P wave may be hidden by the preceding T wave.

Pearls

1. The number of P-QRS complexes prior to the "dropped" beat may vary.
2. A clue to the diagnosis of Mobitz type I heart block can be found in the appearance of grouped QRS complexes.
3. This type of block is normally asymptomatic, and may be seen in athletes.
4. These patients have low risk of progression to complete heart block, and usually do not require a pacemaker. However, Mobitz type I heart block may be caused by inferior myocardial infarction or drugs (digoxin, amiodarone, β-blockers, calcium channel blockers).

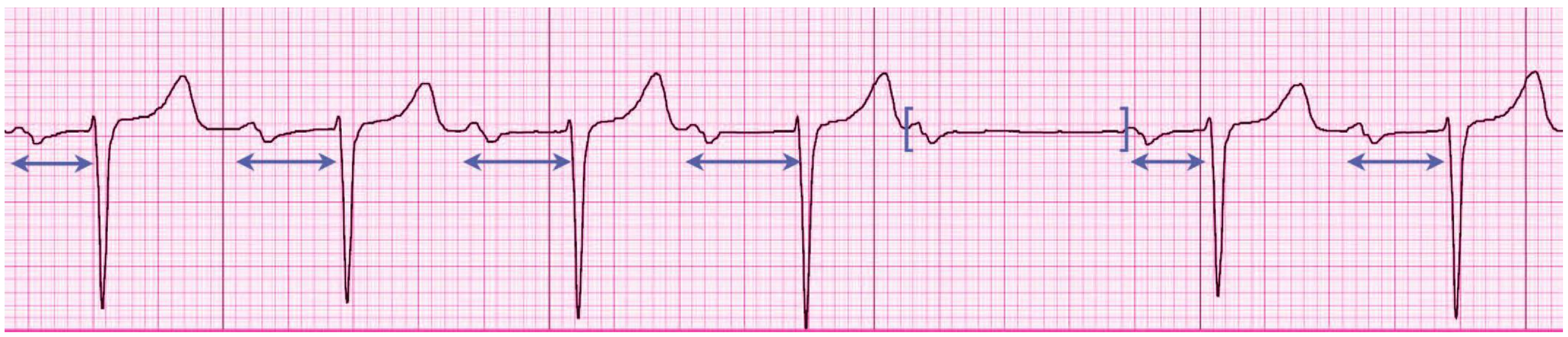


FIGURE 23.15B ■ The PR interval gradually increases (double arrows) until a P wave is not followed by a QRS and a beat is "dropped" (brackets). The process then recurs. P waves occur at regular intervals, though they may be hidden by T waves.

TYPE 2 SECOND-DEGREE AV BLOCK (MOBITZ II)

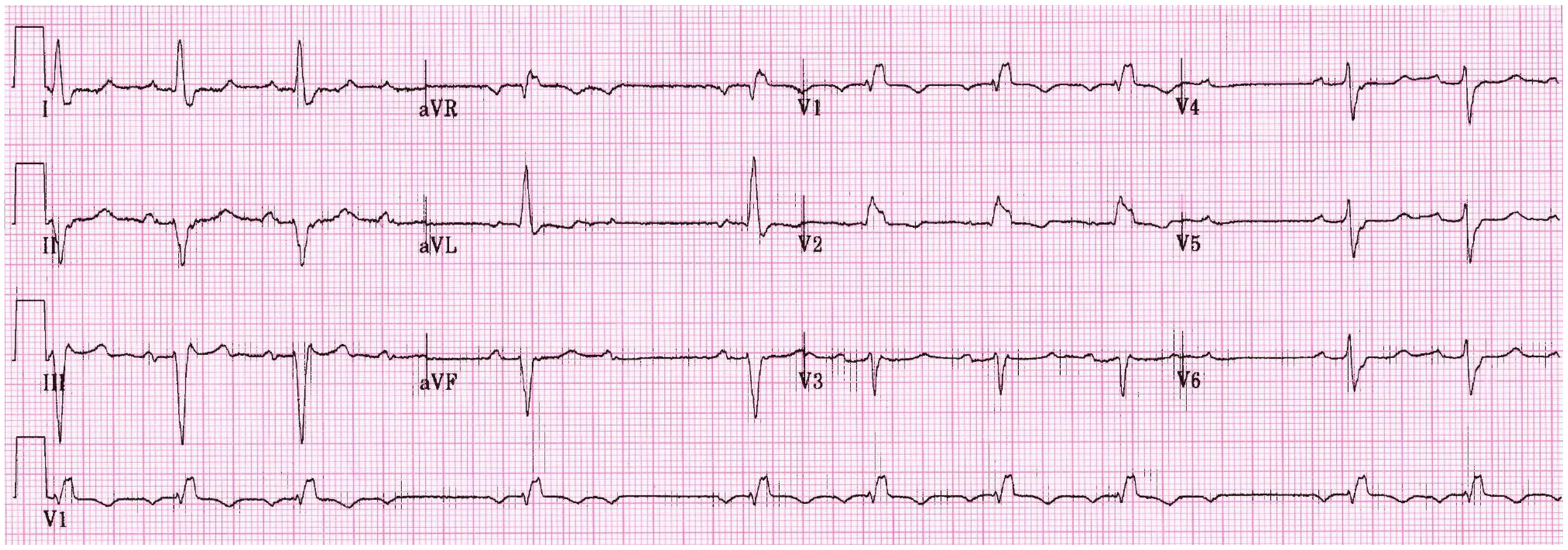


FIGURE 23.16A ■ **Type II Second-Degree AV Block (Mobitz II).** (ECG contributor: Michael L. Juliano, MD.)

ECG Findings

- The PR interval remains constant and does not increase (as seen with Mobitz type I) with each cardiac cycle prior to the "dropped" QRS complex.
- P-P interval is constant, and R-R interval is constant until the dropped beat.
- R-R interval encompassing the "dropped" QRS should be roughly equal to two P-P intervals.

Pearls

1. This type of heart block is associated with disease of the conduction system distal to the AV node. A pacemaker is usually indicated.
2. Mobitz type II block can accompany myocardial infarction and has a high chance of progression to a complete heart block.

FIGURE 23.16B ■ The PR interval is constant (double arrows) until the dropped beat (brackets).

THIRD-DEGREE (COMPLETE) AV BLOCK

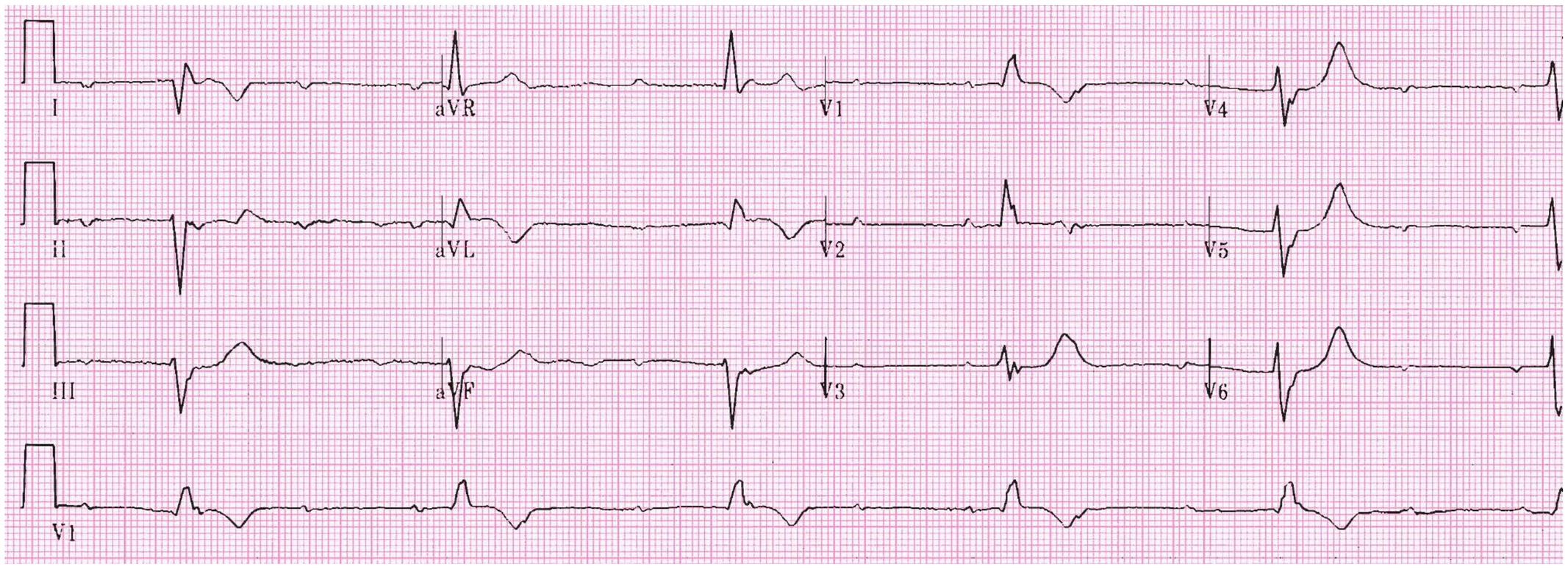


FIGURE 23.17A ■ **Third-Degree AV Block (Complete Heart Block).** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Atrial and ventricular electrical activities are entirely disassociated.
- The P-P and R-R intervals remain constant.
- P waves may be hidden in the QRS complex or may distort the shape of the T wave.
- The atrial rate is usually faster than the ventricular rate.

Pearls

1. Third-degree block is also called complete heart block because no impulses are conducted from the atria to the ventricles.
2. AV rate and QRS morphology depend upon the location of the escape pacemaker.
3. A node escape rate is typically 40 to 60 bpm, with a narrow QRS complex.
4. Ventricular escape rate is usually 20 to 40 bpm, with a widened QRS complex.
5. Complete heart block may be caused by myocardial infarction, conduction system disease, or drugs such as digoxin.
6. Complete heart block may dramatically decrease cardiac output, cardiac pacing is often required.

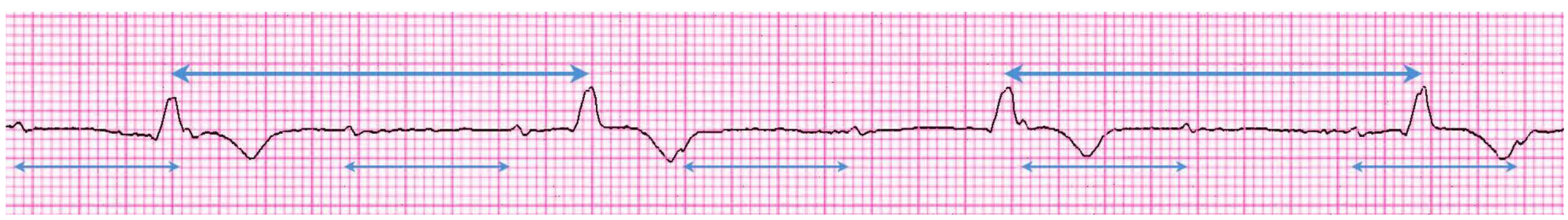


FIGURE 23.17B ■ The P-P interval is uniform (lower double arrows) and the R-R interval is uniform (upper double arrows), but the P waves and QRS complexes are disassociated.

QT INTERVAL PROLONGATION

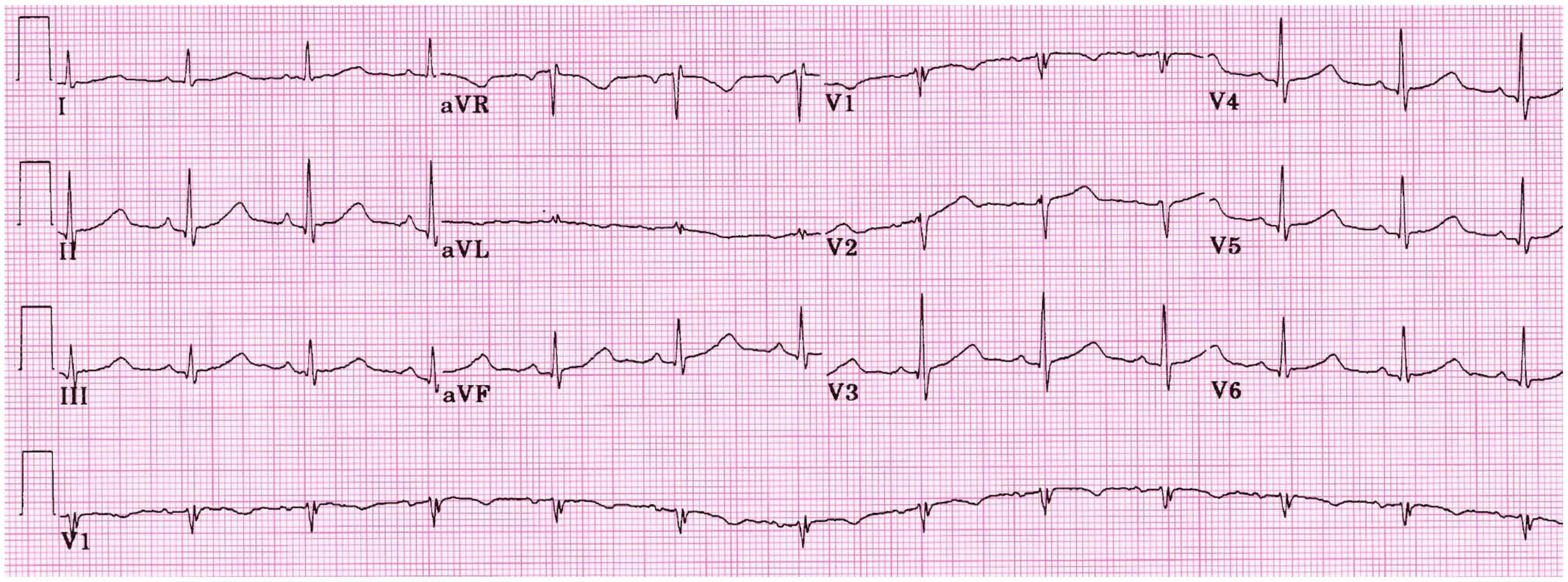


FIGURE 23.18A ■ **Prolonged QT Interval.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Normal QTc interval is less than 440 ms.
- QTc interval greater than 440 ms is considered prolonged.
- QTc interval greater than 550 ms is markedly prolonged.

Pearls

1. The QT interval is measured form the beginning of the QRS complex to the termination of the T wave (or U wave if present) and should be measured in the EKG lead with the longest-appearing QT interval that has a distinct T wave with a clear termination point.
2. The QT interval will increase with bradycardia and decrease with tachycardia, thus it is important to use the corrected QT interval (QTc = QT/μR-R interval) for heart rates other than 60 (in which the QTc = QT).
3. Prolonged QT intervals may be congenital. The vast majority are acquired, usually due to medications or electrolyte abnormalities (hypokalemia, hypomagnesemia, hypocalcemia, and hyperphosphatemia).
4. Numerous medications may prolong the QT interval in therapeutic or toxic doses.
5. Prolongation of the QT interval predisposes to torsades de pointes.
6. Look carefully for prolonged QT intervals in patients who present with syncope.

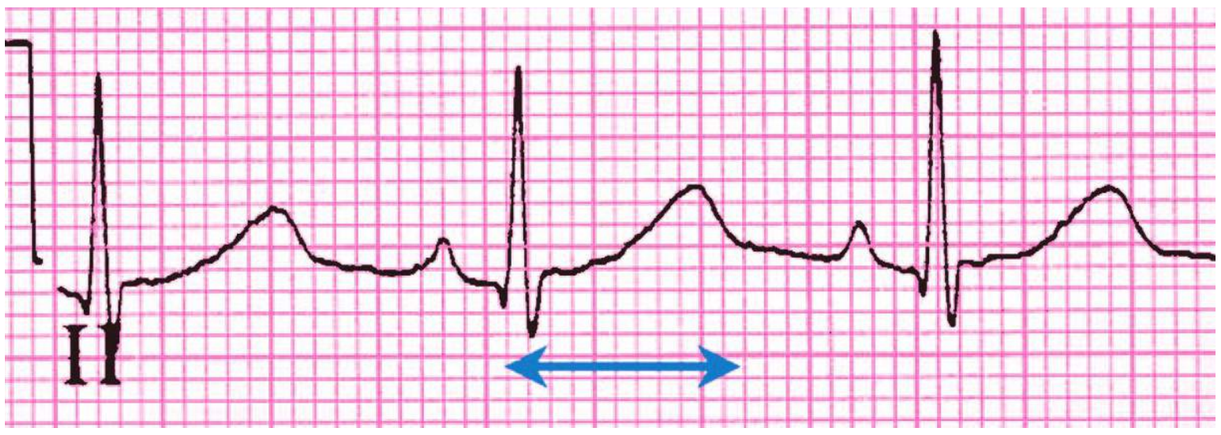


FIGURE 23.18B ■ **QT Interval Prolongation.** QT of 440 ms, QT_c of 498 (double arrow). Note the QT interval is measured from the beginning of the QRS complex to the termination point of the T wave.

RIGHT BUNDLE BRANCH BLOCK

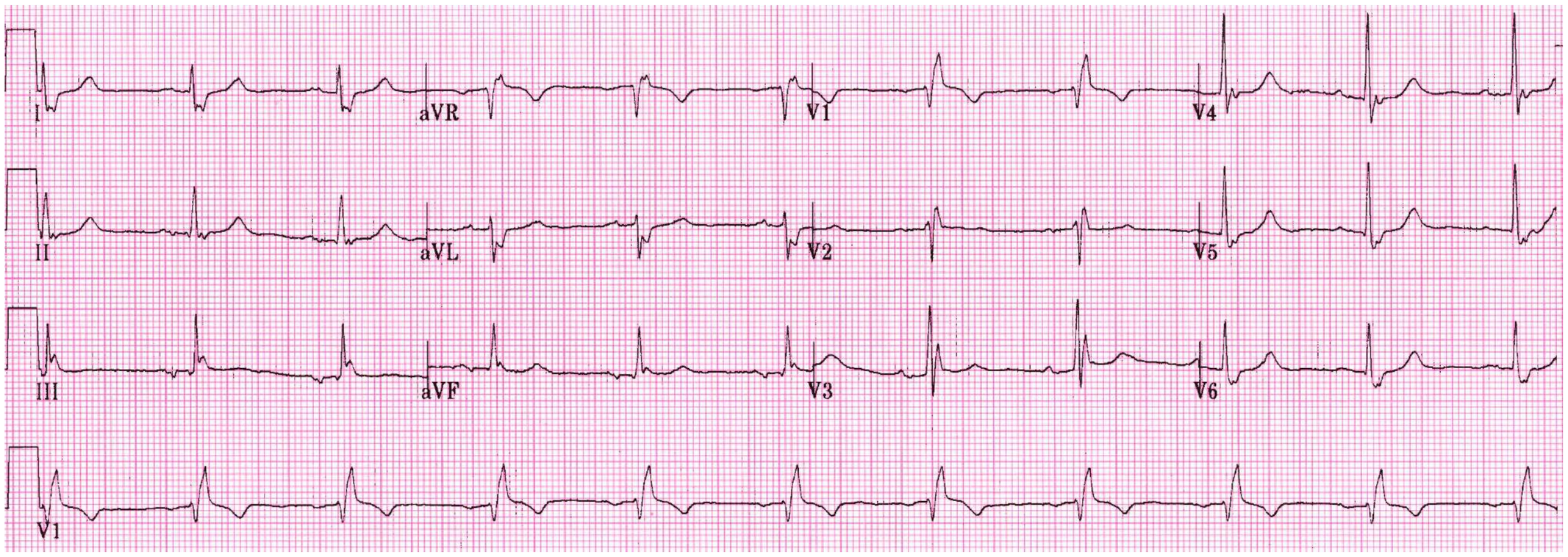


FIGURE 23.19A ■ **Right Bundle Branch Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Wide QRS complex, at least 120 ms (three small blocks).
- QRS complex has sR' or rsR' in leads V_1 and V_2.
- Slurred S wave V_6 and I.

Pearls

1. The signal exiting the AV node is carried rapidly to the LV through the intact left bundle, but is delayed into the right ventricle, where depolarization must propagate cell-to-cell. Since the RV myocardial mass is much smaller than that of the LV, this delay in depolarization is best seen in the leads overlying the right ventricle, leads V_1 and V_2.
2. Acute right heart strain, as may occur with pulmonary embolism, may result in new onset right bundle branch block (RBBB).

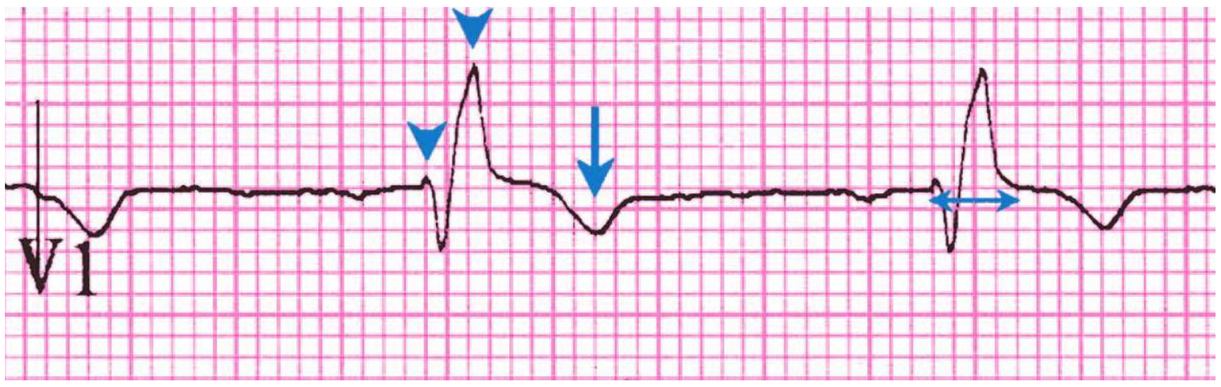


FIGURE 23.19B ■ rsR' pattern in V_1 (arrowheads), with T wave downgoing (arrow). QRS duration greater than 120 ms (double arrow).

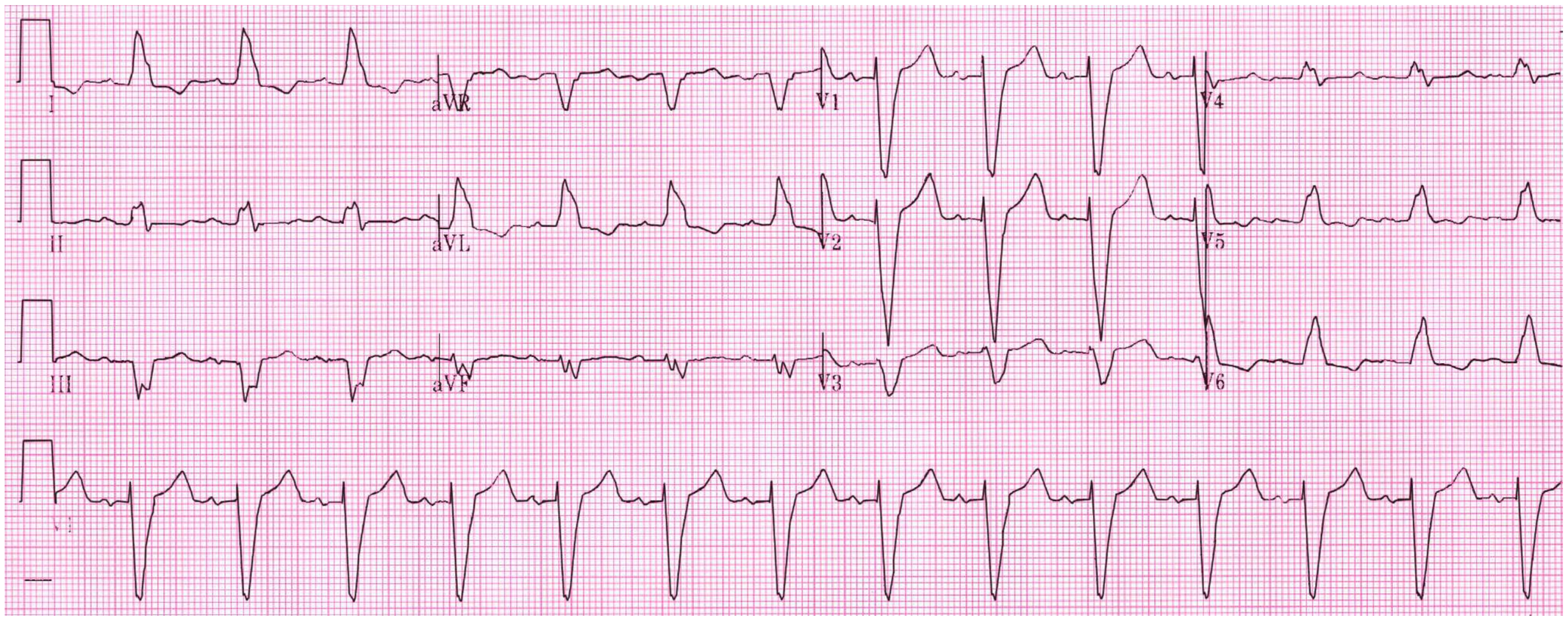


FIGURE 23.20A ■ **Left Bundle Branch Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Wide QRS complex, at least 120 ms (three small blocks).
- T wave appears on the opposite side of the baseline from the QRS complex.
- The QRS precordial axis is normal or deviated to the left.
- QRS complex deflection is predominately downward in lead V_1 and upward in lead V_6.

Pearls

1. The signal exiting the AV node does not proceed through the left ventricular conduction system. It must propagate more slowly cell-to-cell through the myocardium, starting in the septum. Therefore, the QRS is wider and the bulk of the depolarization signal is deflected toward the far lateral aspect of the heart.
2. Acute myocardial infarction may produce a new onset LBBB on ECG. Therefore, patients with new onset LBBB with a clinical presentation consistent with acute coronary syndrome should be treated as having an acute ST segment elevation myocardial infarction (STEMI).

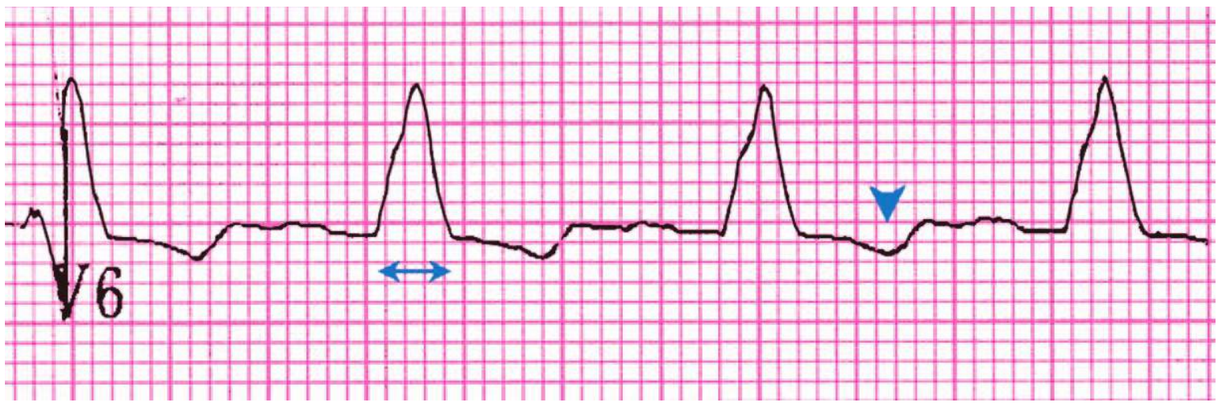


FIGURE 23.20B ■ The QRS is wider than 120 ms (double arrow). The T-wave deflection is in the opposite direction from the QRS deflection (arrowhead).

LEFT ANTERIOR FASCICULAR BLOCK

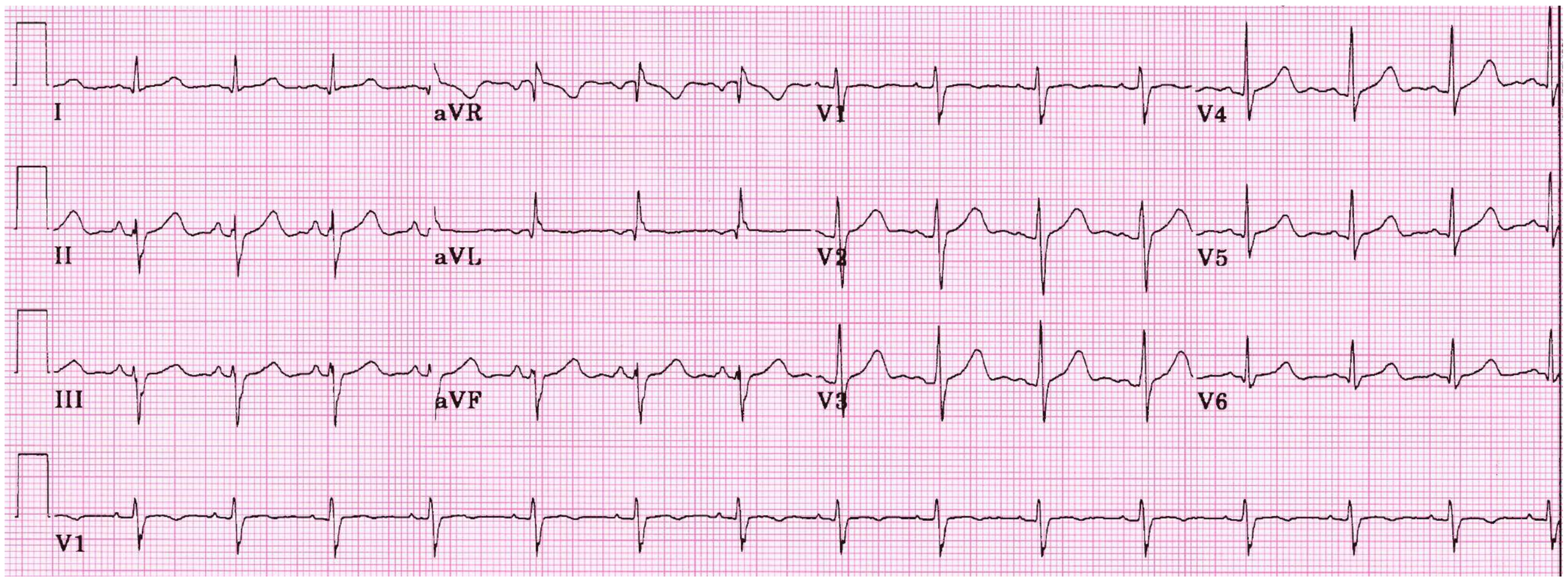


FIGURE 23.21A ■ **Left Anterior Fascicular Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- QRS complex widening, usually 90 to 120 ms
- Left axis deviation beyond minus 45 degrees with no other cause (such as inferior myocardial infarction)
- Small R wave and large S wave in the inferior leads
- Slurred S wave in V_5 and V_6

Pearls

1. The signal exiting the AV node is carried rapidly to the inferior aspect of the LV and all of the RV through the intact left posterior fascicle and right bundle, where quick depolarization occurs. However, conduction to the high lateral and upper portions of the left ventricle is slower and must proceed cell-to-cell due to the blocked left anterior fascicle. Therefore, the latter portion of the QRS depolarizes toward the upper lateral myocardium, manifested as strong left axis deviation.
2. Left anterior fascicular block is the most common intraventricular conduction disturbance associated with acute anterior myocardial infarction, with the left anterior descending artery usually involved.

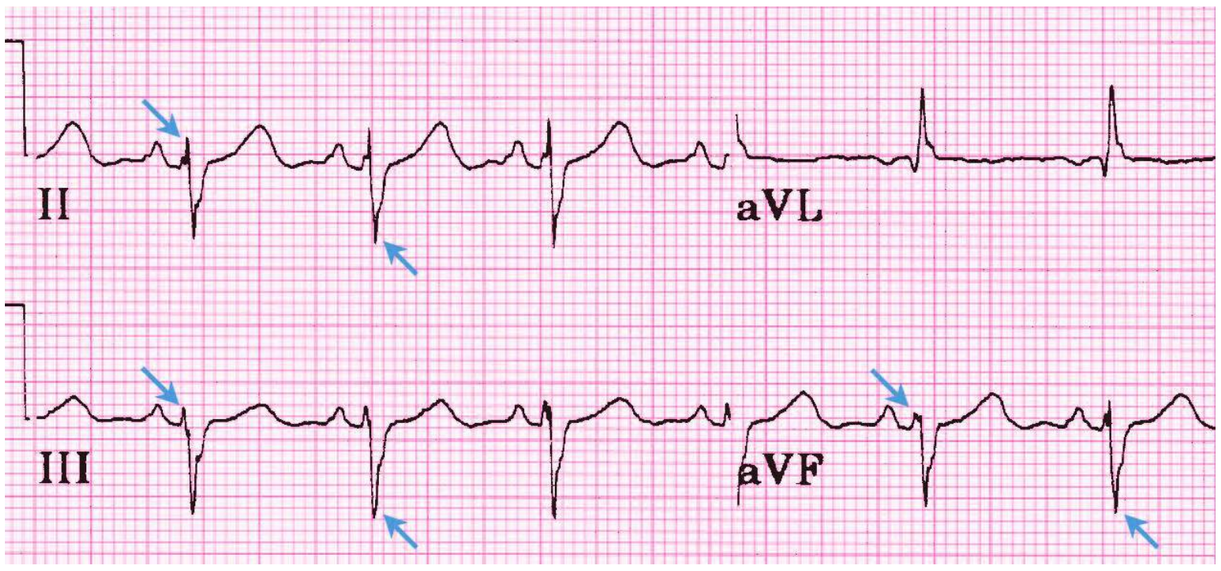


FIGURE 23.21B ■ Small R waves, large S waves in all inferior leads (arrows), with QRS axis deviated left beyond minus 45 degrees.

LEFT POSTERIOR FASCICULAR BLOCK

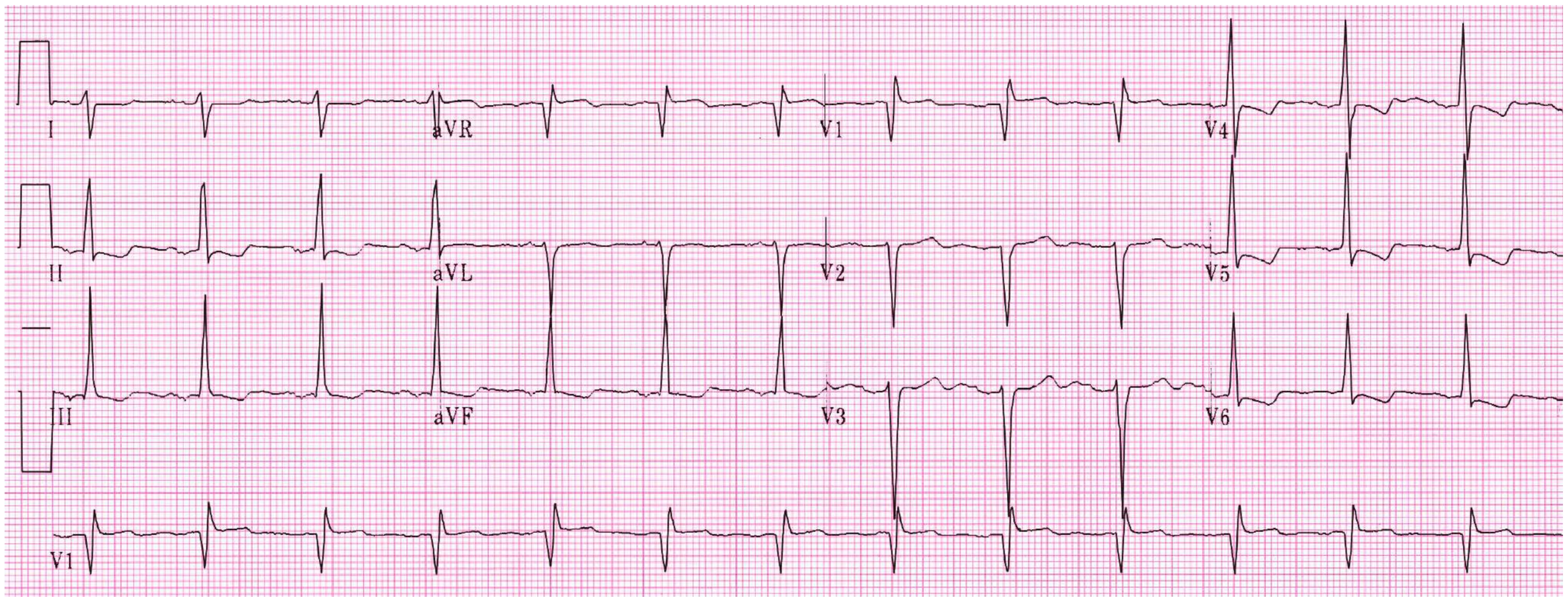


FIGURE 23.22A ■ **Left Posterior Fascicular Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- QRS complex widening to 90 to 120 ms.
- Right axis deviation must be beyond 100 degrees and must have no other cause (such as lateral myocardial infarction).
- Small R wave and large S wave in the high lateral leads, I and aVL.
- Slurred S wave in V_5 and V_6.
- This example also contains unrelated ST changes.

Pearls

1. The signal exiting the AV node is carried rapidly to the upper aspect of the LV and all of the RV through the intact left anterior fascicle and right bundle, where depolarization is rapid. However, conduction to the inferior portion of the left ventricle is slower and must proceed cell-to-cell due to the blocked left posterior fascicle. Therefore, the latter portion of the QRS depolarizes toward the inferior myocardium, manifesting as strong right axis deviation.
2. Left posterior fascicular block may be associated with acute inferior myocardial infarction as well as with multiple cardiomyopathic conditions.

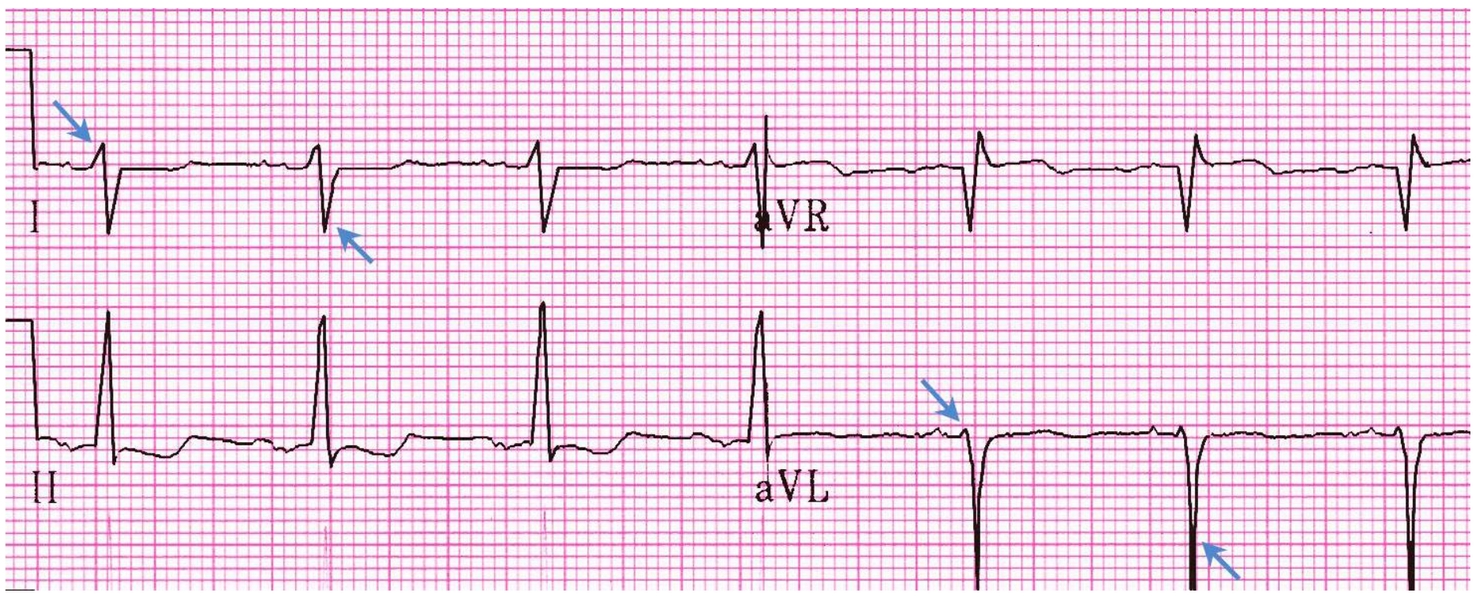


FIGURE 23.22B ■ Small R waves and large S waves in leads I and aVL (arrows).

TRIFASCICULAR BLOCK

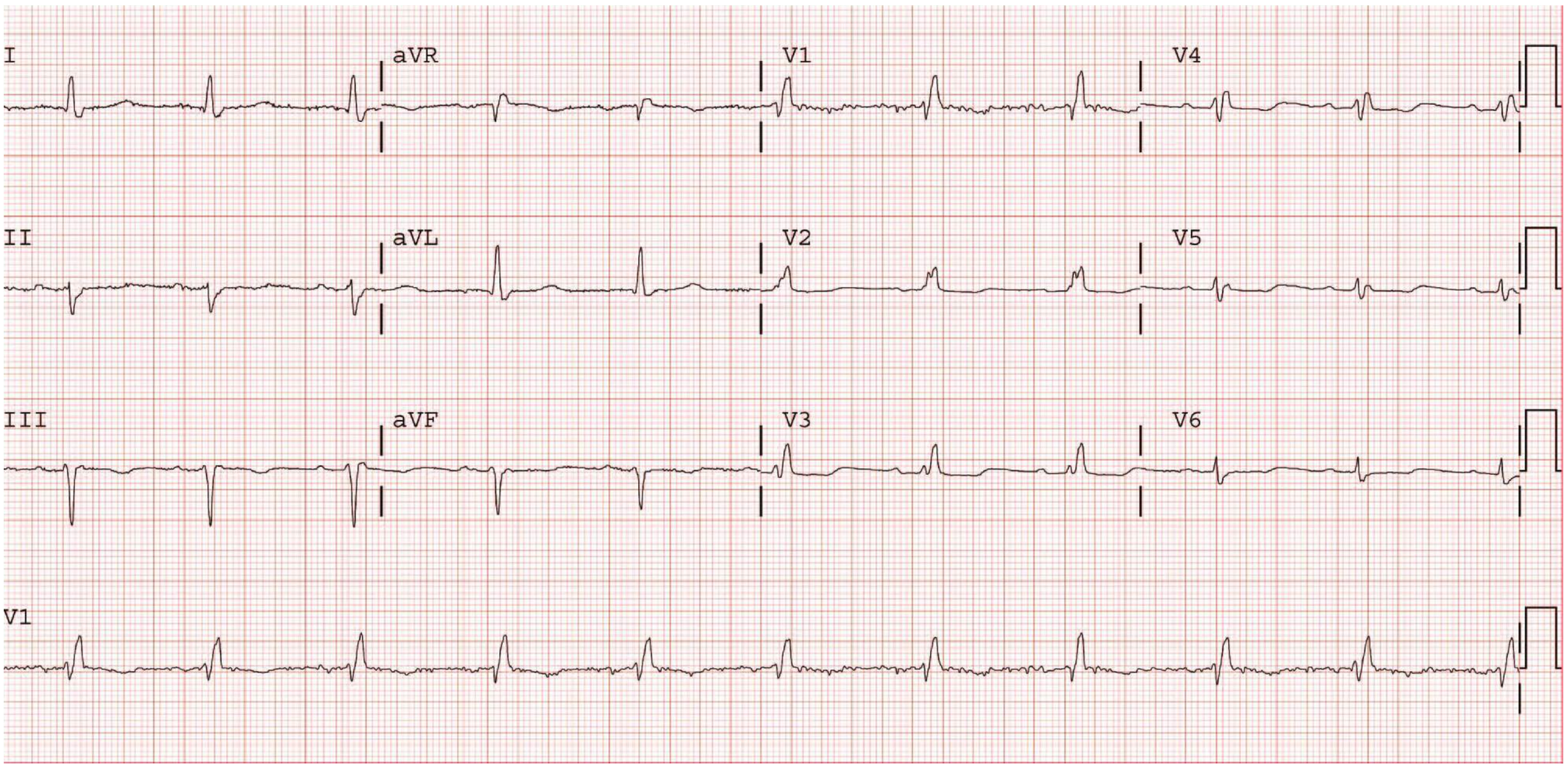


FIGURE 23.23A ■ **Trifascicular Block.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- RBBB with left anterior or posterior hemiblock and first-degree AV block
- Somewhat of a misnomer, not a true blocking of three fascicles: two fascicles blocked coupled with PR interval prolongation
- LBBB plus first-degree AV block sometimes referred to as trifascicular block as well, but again only two fascicles truly blocked in this scenario

Pearls

1. Trifascicular block may be a precursor to complete heart block: Pacemaker evaluation is warranted, especially if symptomatic, but incidence of progression to complete heart block is low.
2. AV blocking agents can potentiate degree of block.
3. Can be due to systemic diseases (Chagas disease) but usually indicative of primary advanced conduction system abnormalities secondary to coronary artery disease.

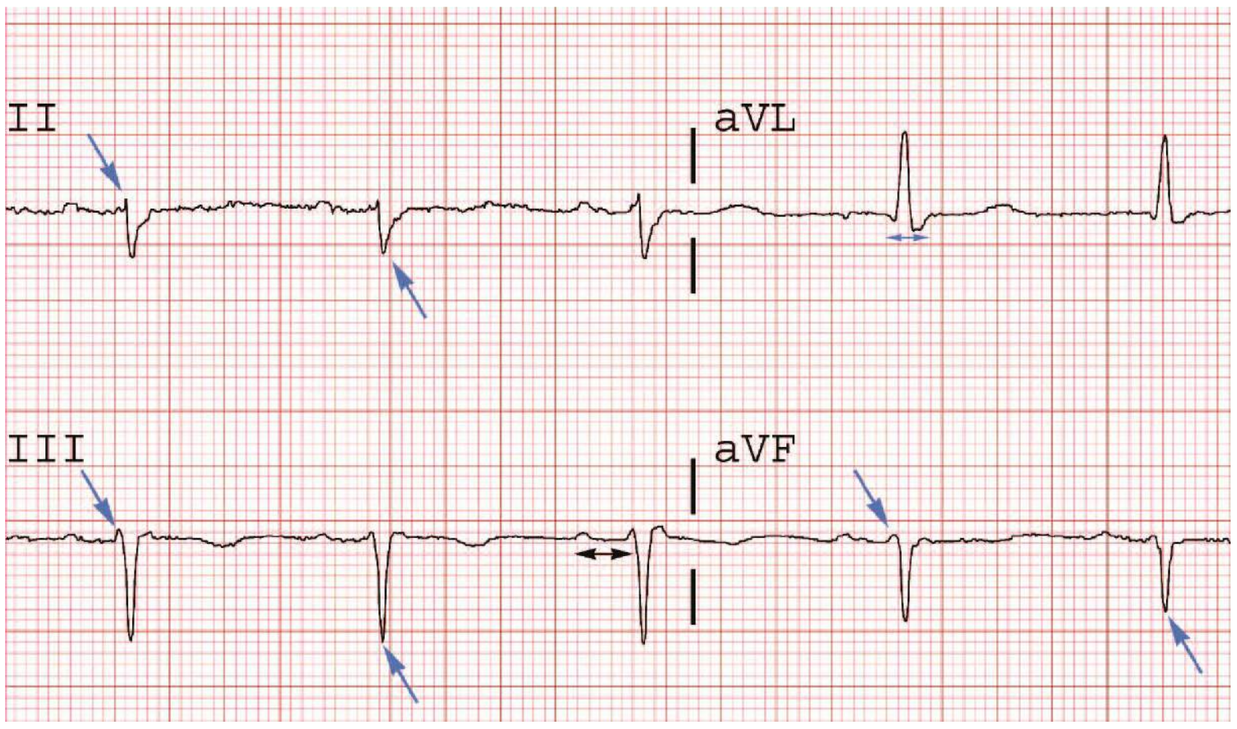


FIGURE 23.23B ■ The QRS is wide with RBBB pattern (blue double arrow). Left posterior fascicular block is present (diagonal arrows) along with a prolonged PR interval and first-degree AV block (black double arrow).

ASHMAN PHENOMENON

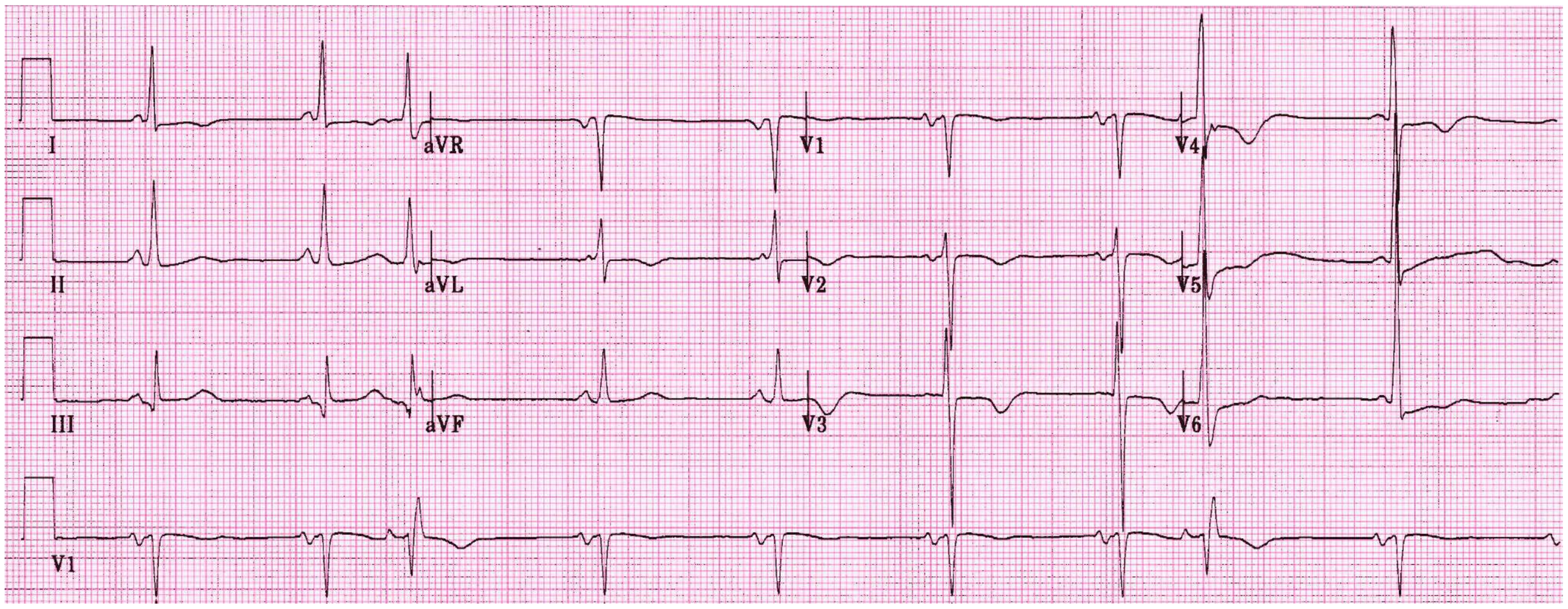


FIGURE 23.24A ■ **Ashman Phenomenon.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Aberrant ventricular conduction, usually with RBBB pattern.
- Altered durations of the refractory period of the bundle branch or ventricular tissue are present, commonly due to atrial fibrillation, atrial ectopy, and atrial tachycardia

Pearls

1. After depolarization, tissue repolarizes during its refractory period. Refractory period changes with the preceding cardiac cycle, with longer R-R intervals producing longer refractory periods and shorter R-R intervals producing shorter refractory periods.
2. A longer R-R interval lengthens the following refractory period. When an early or premature (ectopic) depolarization reaches the ventricular conduction system before it has completely repolarized, aberrant conduction may occur and be manifest on the ECG with a bundle branch block (BBB) pattern.
3. Ashman phenomenon most commonly appears with an RBBB pattern, since the right bundle has a longer refractory period than the left bundle.
4. Ashman phenomenon is often seen in atrial fibrillation, when a long R-R interval is followed by a much shorter R-R interval.
5. In the setting of a premature atrial beat (as seen in this example), the earlier in the cycle the PAC occurs and the longer the preceding R-R interval is, the more likely aberrant conduction of the beat will occur.

FIGURE 23.24B ■ After a relatively long R-R interval (double arrow), a PAC (diagonal arrow) is followed by an aberrantly conducted QRS with RBBB morphology (arrowhead). After a short pause (single arrow), the next beat is conducted normally as it has occurred outside of the refractory period set by the previous beat.

JUNCTIONAL RHYTHM

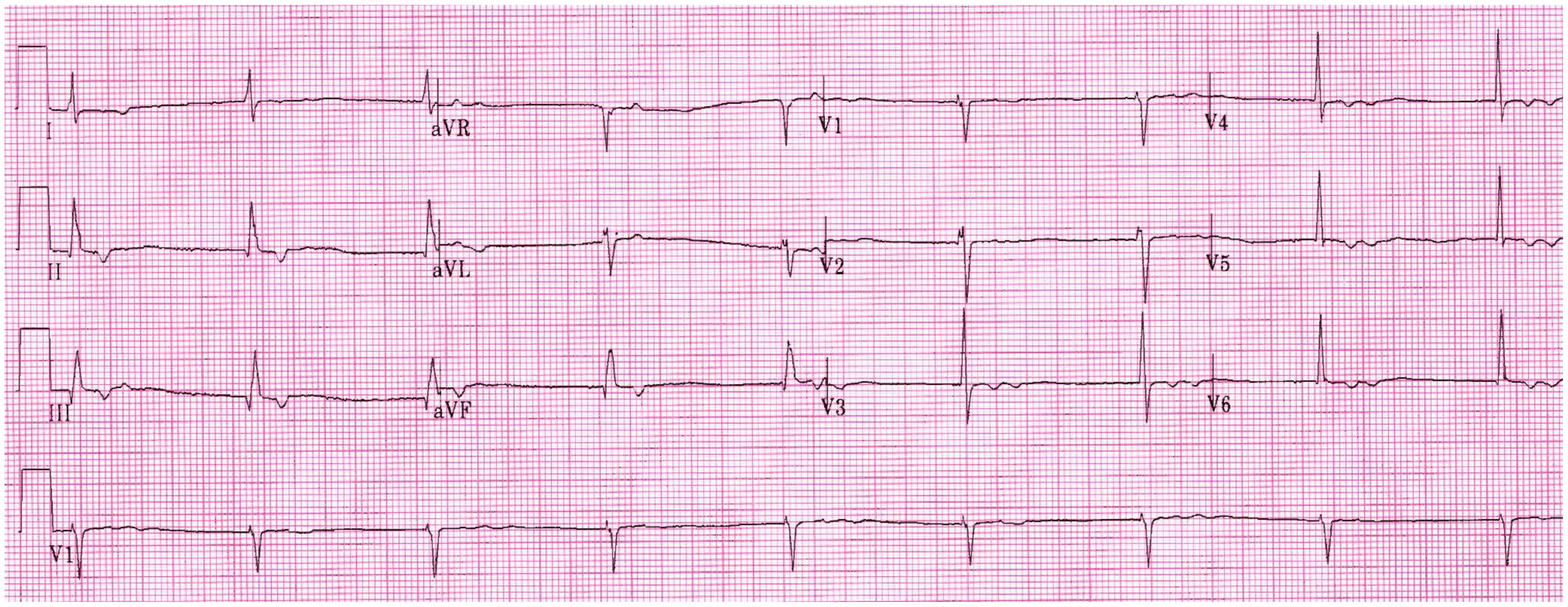


FIGURE 23.25A ■ **Junctional Rhythm with Retrograde P Waves.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- The QRS complex is narrow, with a rate typically between 40 and 60 bpm.
- P waves are absent, retrograde, very slow, or unrelated to the QRS complex.

Pearls

1. When the atria fail to initiate a cardiac rhythm, or when no pacing signal reaches the lower AV node, the AV node or His bundle usually picks up the pacemaking responsibility.
2. P waves may be conducted retrograde and buried in the T wave as seen in this example. In the case of a complete AV block, the P waves have no relation to the QRS complex.
3. If a BBB is also present, the QRS may be wide, and may be difficult to discern from a primary ventricular rhythm.

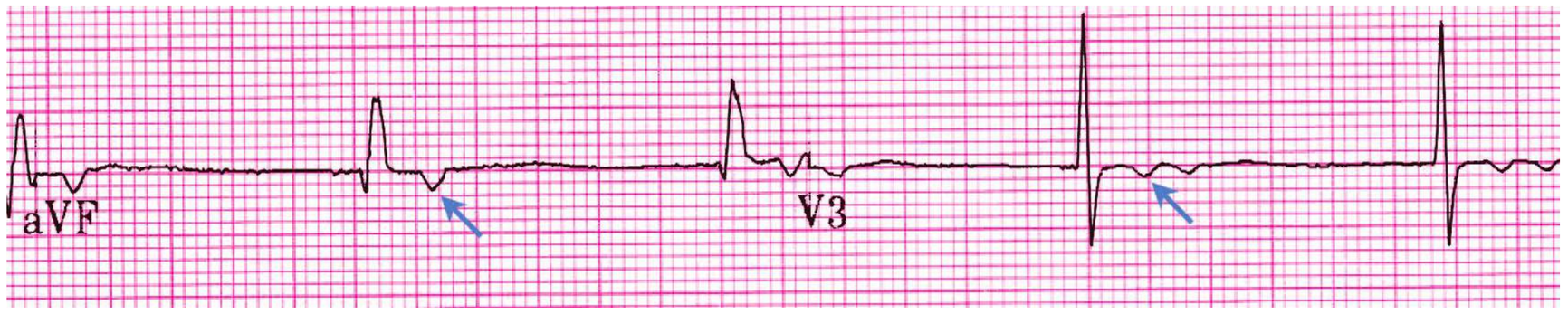


FIGURE 23.25B ■ The QRS is narrow. P waves are not present before the QRS. In this example, the signal which originated in the His bundle is conducted retrograde through the AV node into the atria, and retrograde P waves are apparent in the ST segment (arrows).

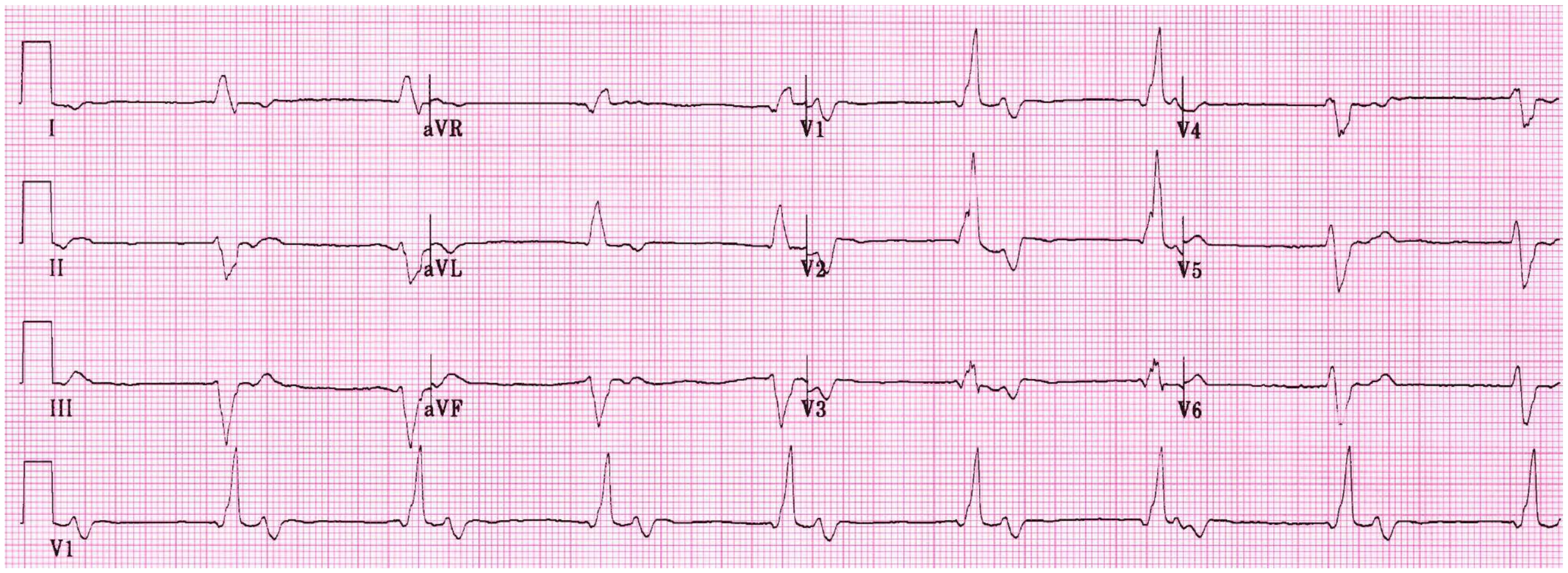


FIGURE 23.26A ■ **Ventricular Rhythm with Retrograde P Waves.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- The QRS complex is wider than 120 ms, with a rate typically between 20 and 40 bpm.
- The T wave is discordant relative to the QRS.

Pearls

1. When the atria and the AV node fail to initiate a cardiac rhythm, or when no pacing signal reaches the ventricle, the ventricular tissue usually picks up the pacemaking responsibility.
2. P waves may also be conducted retrograde and buried in the T wave as seen in this example. In the case of a complete AV block, the P waves have no relation to the QRS complex.

FIGURE 23.26B ■ Wide-complex (double arrow) regular QRS at a rate of approximately 50 bpm. Retrograde P waves are seen in this example (arrow).

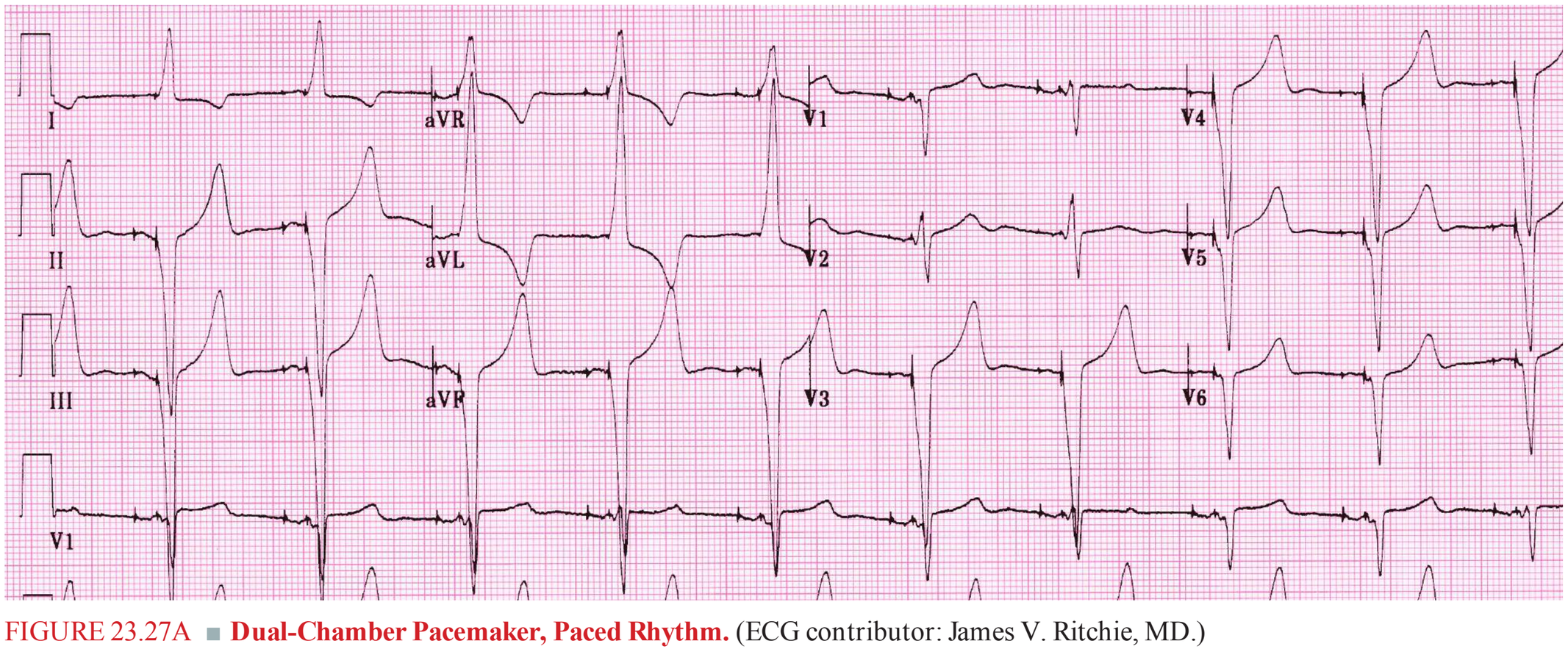


FIGURE 23.27A ■ **Dual-Chamber Pacemaker, Paced Rhythm.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Very narrow signal of no discernable width, immediately followed by a P wave (if an atrial lead) or a QRS complex (if a ventricular lead). The narrow pacer "spike" amplitude varies, and can be larger than the QRS or may be indiscernible.
- A QRS complex initiated by a pacer spike will be wide, with morphology similar to a PVC or idioventricular rhythm. The axis is unlike typical BBBs, because the signal usually originates low in the right ventricle.

Pearls

1. Pacemakers are designated by the "five letter" system. In this system, the letter "A" denotes atrium, "V" denotes ventricle, "D" denotes dual (both chambers), and "O" denotes neither. The first three letters are the most commonly used:
   A. First letter—designates chamber(s) paced
   B. Second letter—designates chamber(s) sensed
   C. Third letter—designates pacemaker response to sensed electrical activity: T: triggered—fires even when beat sensed, I: inhibitory—holds when beat sensed, D: dual—atrial triggered and ventricle inhibited
   D. Fourth letter—extra options: P: programmable, M: multiprogrammable, C: communicating, R: rate adaptation, O: none
   E. Fifth letter—cardioverting options: P: pacing, S: shocking, D: dual (P+S), O: none
2. The two most common pacemaker malfunctions are failure to pace and failure to sense.
3. Some ECG machines perceive small pacer spikes as artifact and do not reproduce them on the printed tracing.

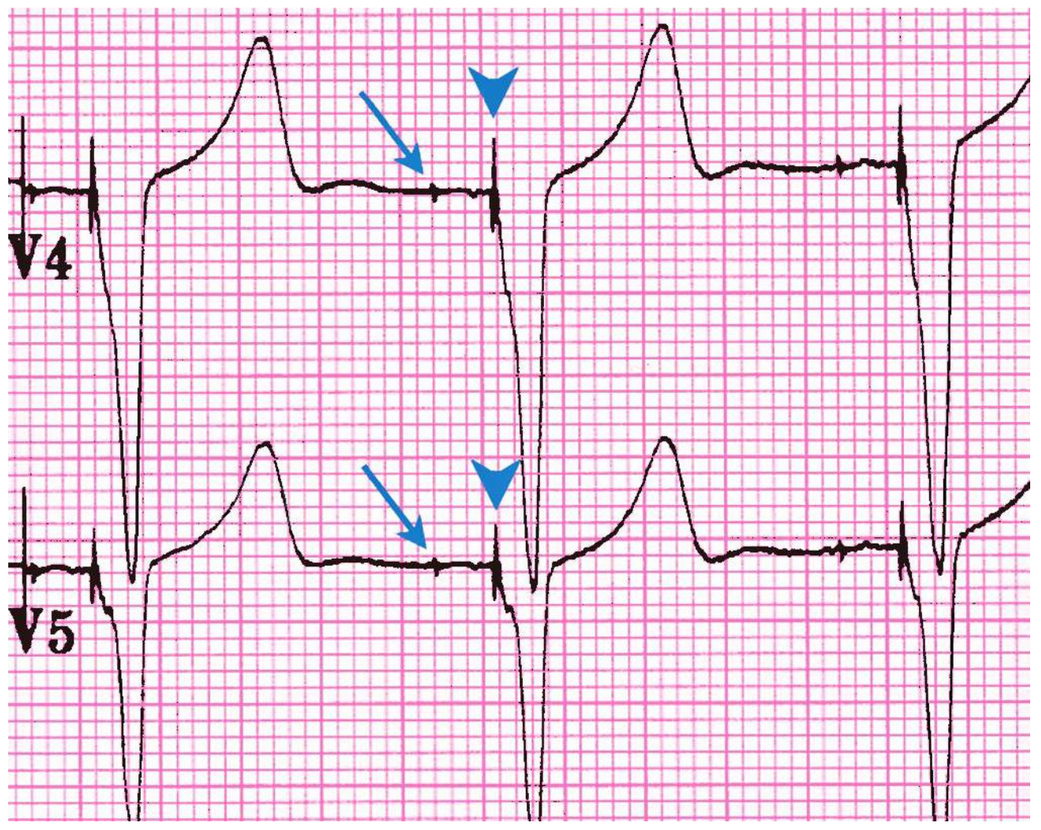


FIGURE 23.27B ■ Tiny pacer spikes (arrows) precede the P waves, and somewhat larger pacer spikes precede the QRS complexes (arrowheads). The QRS complexes are wide, with discordant T waves.

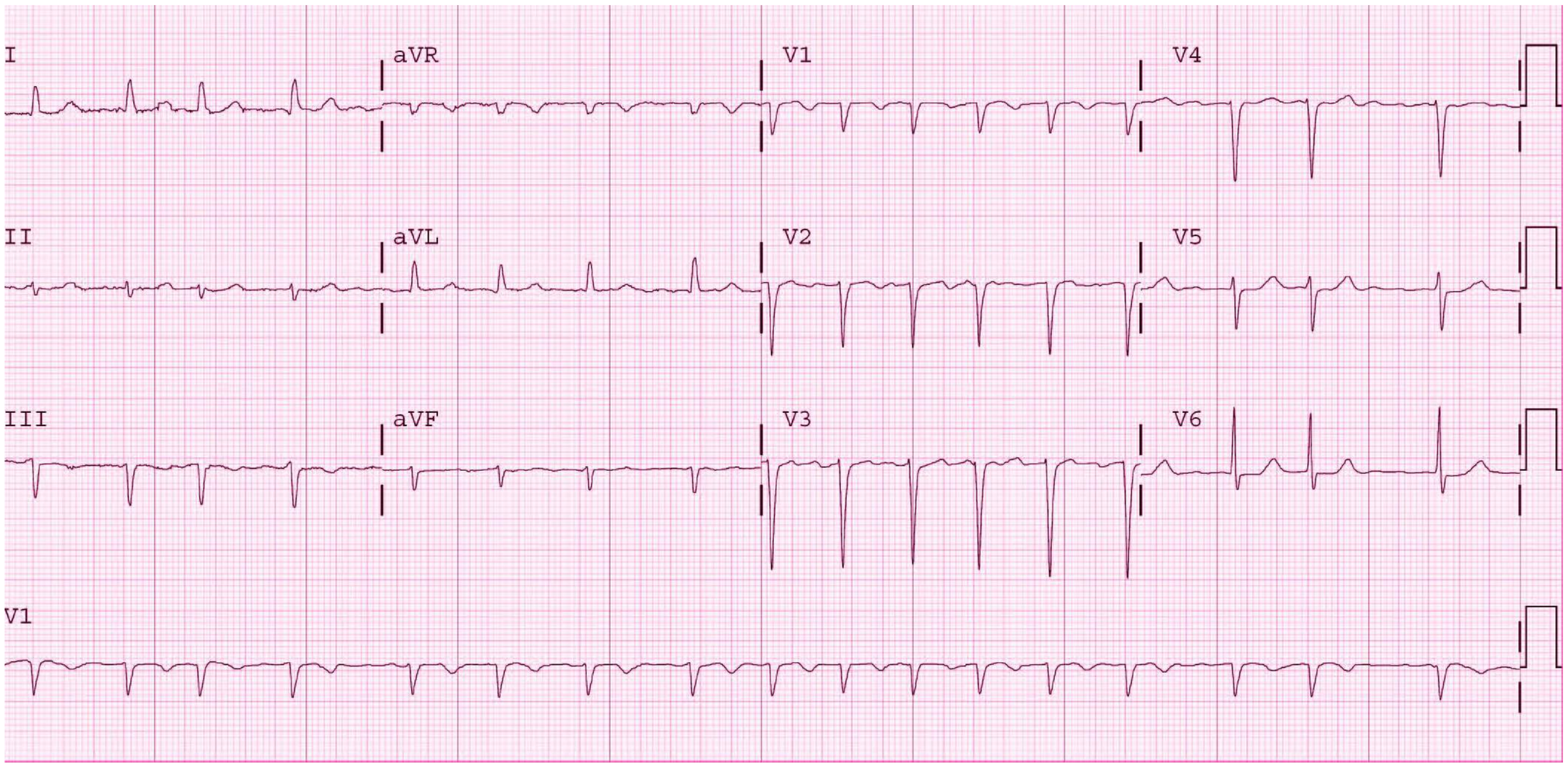


FIGURE 23.28A ■ **Atrial Fibrillation.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- Electrical activity in the atria is chaotic. The ECG baseline, representing the ongoing chaotic atrial activity, is unorganized. The resulting "rumbling" baseline may have large or indiscernibly small amplitude.
- The AV node has a refractory period, and therefore does not conduct every signal it receives from the atria. Thus, ventricles depolarize variably creating varying R-R intervals and an "irregularly irregular" pattern.

Pearls

1. Most "irregularly irregular" rhythms are due to atrial fibrillation, but other rhythms may produce similar findings. These include multifocal atrial tachycardia, atrial flutter with variable AV block, and frequent PJCs.
2. Therapy is geared toward either rate control of the ventricles or rhythm control and cardioversion (chemically or electrically).
3. Synchronized cardioversion may be indicated if a patient is unstable, but the risk of clot embolization must be carefully considered when planning nonemergent electrical cardioversion of atrial fibrillation.

FIGURE 23.28B ■ R-to-R interval varies in an "irregularly irregular" pattern (double arrows). The baseline "rumble," representing "F waves," may be very fine or even indiscernible.

FIGURE 23.28C ■ The baseline "rumble" may be very coarse resembling atrial flutter waves.

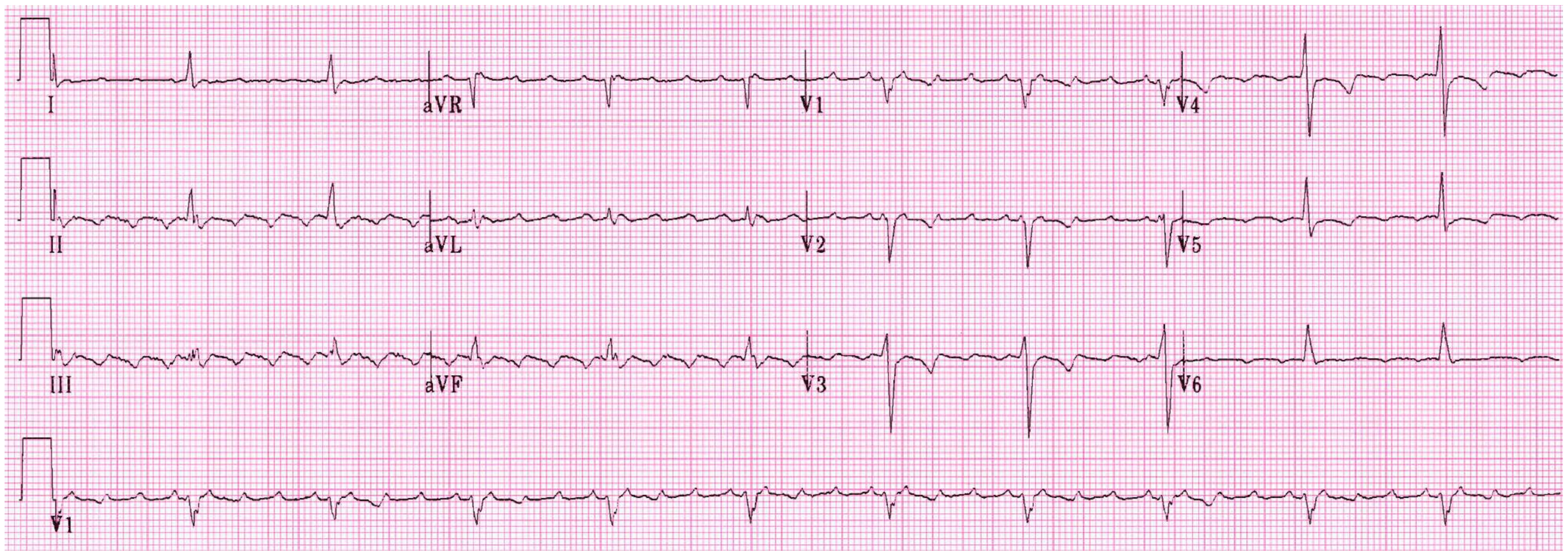


FIGURE 23.29A ■ **Atrial Flutter.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Electrical activity in the atria is ongoing and regular, self-propagating in a roughly circular movement.
- Flutter waves appear in a rapid sine wave or "sawtooth" pattern, usually in the inferior leads.
- Atrial activity in lead V_1 often appears as rapid P waves at a rate approximating 300 bpm.

Pearls

1. The AV node's refractory period prevents 1:1 conduction to the ventricles. Usually, conduction is blocked at a ratio of 1:2 to 1:4. The QRS complexes should appear with regular periodicity. However, AV conduction may be variable from beat-to-beat creating irregular R-to-R intervals.
2. A conduction ratio of 2:1 is usually difficult to discern, because the two flutter peaks between QRS complexes may look like normal P and T waves. A ventricular rate of 140 to 160 bpm should prompt consideration of the possibility of atrial flutter with 2:1 block.
3. Conditions that cause rapid repetitive tremors (such as Parkinson disease, rigors, shivering, or hepatic tremor) may mimic flutter waves on EKG (known as pseudoflutter).

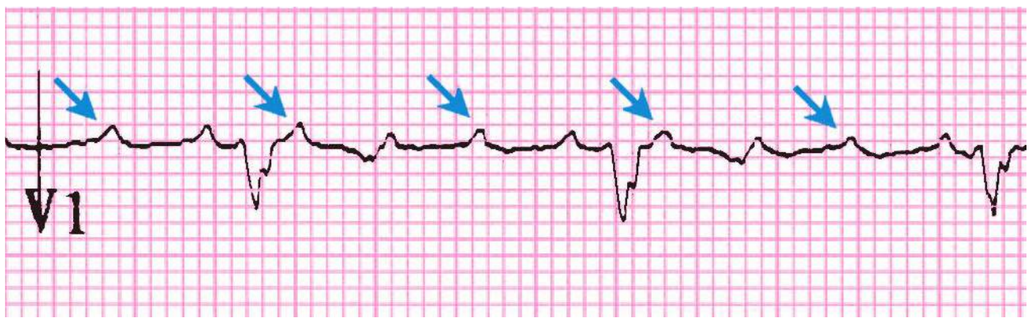


FIGURE 23.29B ■ Atrial flutter with 4:1 block. The flutter waves (arrows marking every other flutter wave) may be mistaken for P and T waves.

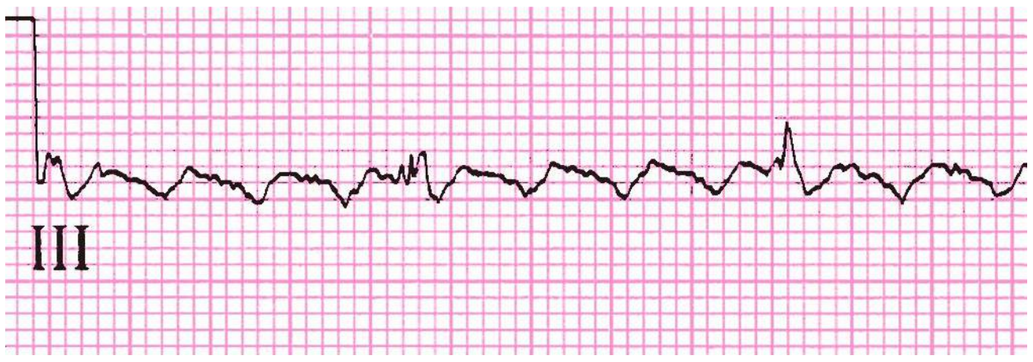


FIGURE 23.29C ■ The "sawtooth" pattern is most apparent in the inferior leads.

MULTIFOCAL ATRIAL TACHYCARDIA

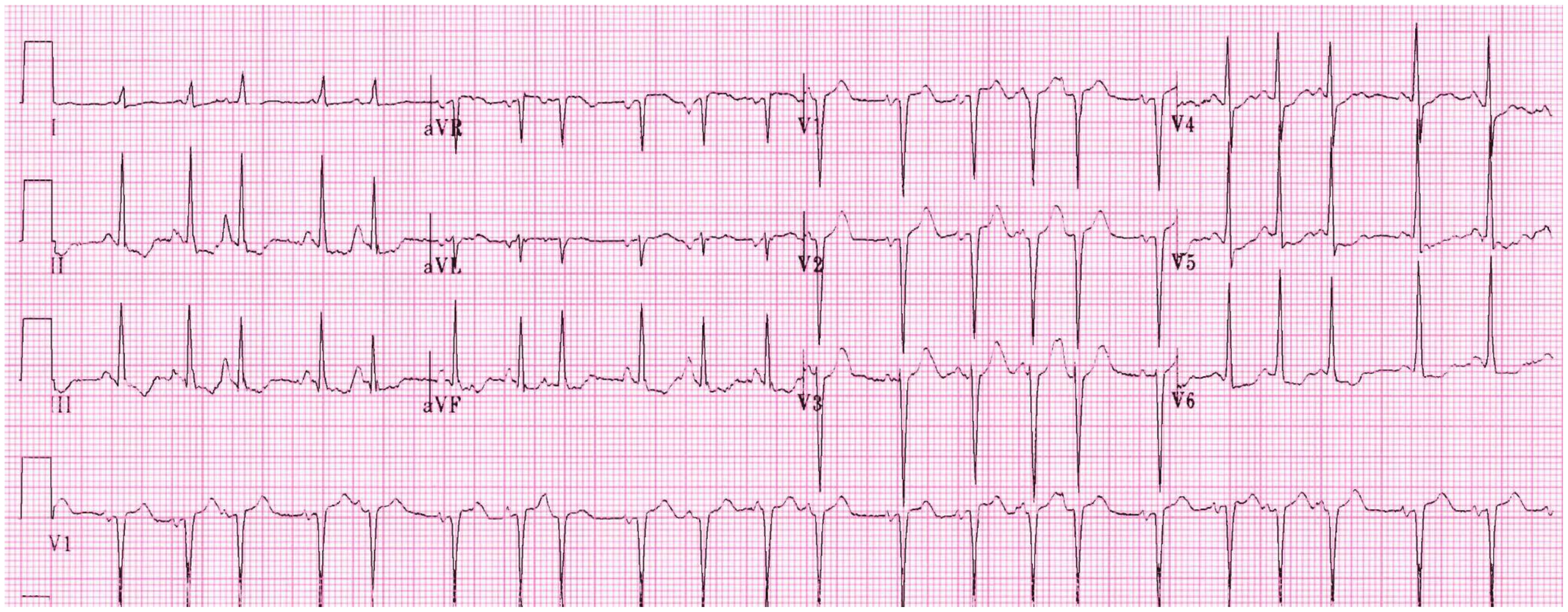


FIGURE 23.30A ■ **Multifocal Atrial Tachycardia.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Multiple P-wave morphologies with heart rate greater than 100 bpm
- A chaotic R-R interval
- Varying PR intervals

Pearls

1. Multiple atrial foci are capable of acting as pacemakers. When irritated by stretching, medications, or certain acute medical conditions, these foci compete in pacing the atria.
2. The different atrial foci produce P waves of different morphologies.
3. Since the atrial foci vary in distance to the AV node, PR intervals vary.
4. Multifocal atrial tachycardia (MAT) usually results from exacerbation of another condition which produces distention or irritation of the atria. The most common cause of MAT is COPD exacerbation. Treatment of the underlying condition should correct the arrhythmia.

FIGURE 23.30B ■ Multiple P morphologies (arrowheads), varying PR intervals (lower double arrows), and varying R-R intervals (upper double arrows) with heart rate greater than 100 bpm.

SUPRAVENTRICULAR TACHYCARDIA (SVT)

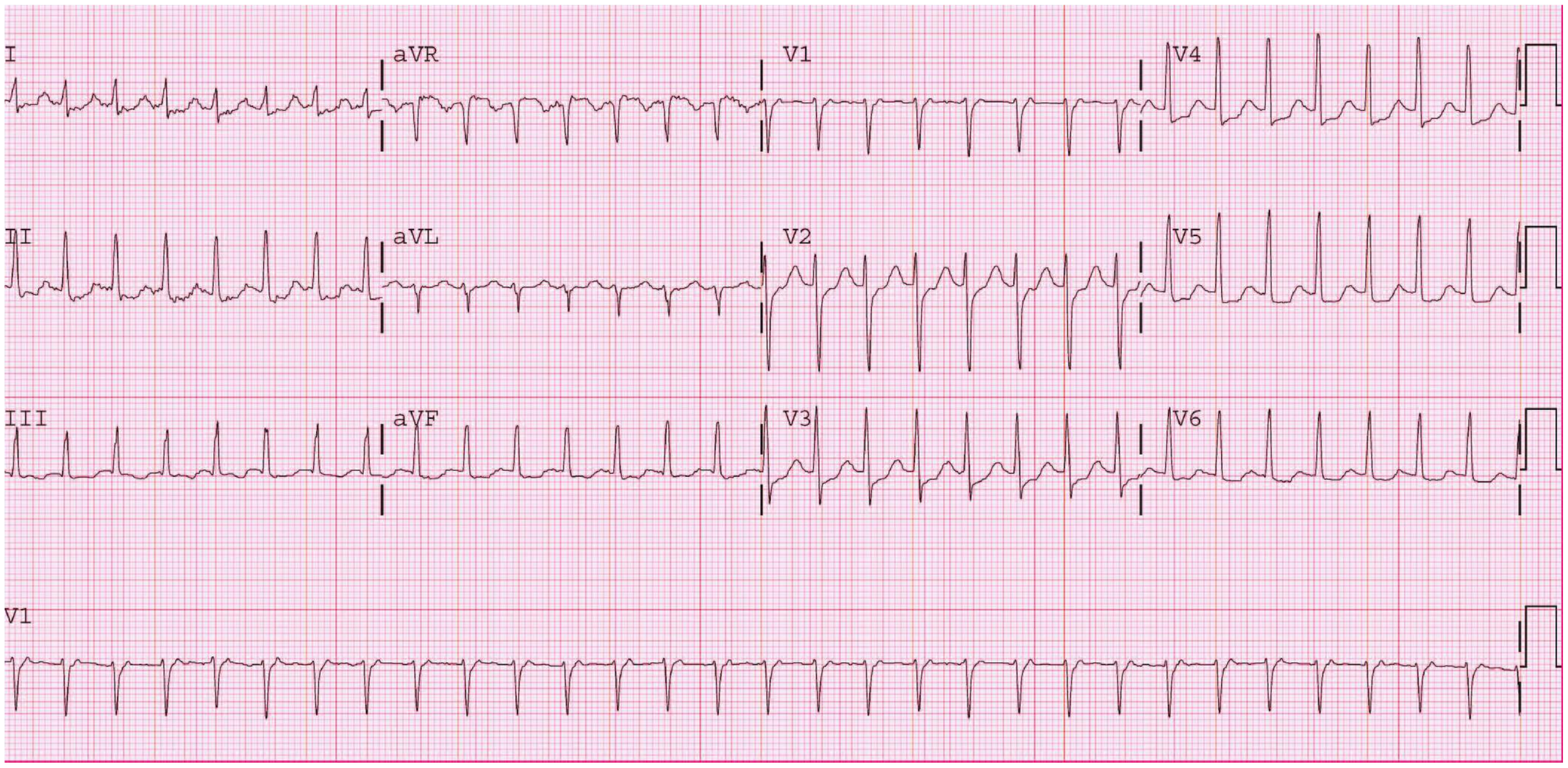


FIGURE 23.31A ■ **Supraventricular Tachycardia (AVNRT).** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- Tachycardia, rate usually greater than 140 bpm
- Narrow QRS complex
- Absent, retrograde, or unusual P waves

Pearls

1. SVT occurs when the SA node rhythm is superseded by a faster rhythm, usually originating in the AV node.
2. Three common types are:
   A. Atrial tachycardia—originates from an ectopic focus in the atrium. P waves may have an unusual morphology or may be hidden by the preceding T wave.
   B. AV nodal reentrant tachycardia (AVNRT) occurs when an electrical impulse continues around the AV node in a circular pattern causing rapid depolarizations of the ventricles. Since the AV node is the origin of the atrial depolarization, the P-wave deflection should be inverted if seen (eg, downgoing in II, III, aVF).
   C. AV reentrant tachycardia (AVRT) as seen with bypass tracts outside of the AV node (WPW). Instead of intranodal reentrant activity as seen with AVNRT, an accessory tract provides the reentrant pathway to propagate the tachycardia.
3. Normal deflections seen in the ST segments are relatively wide based. When you see "tight little turns" in the ST segment, you should strongly consider the presence of retrograde P waves.

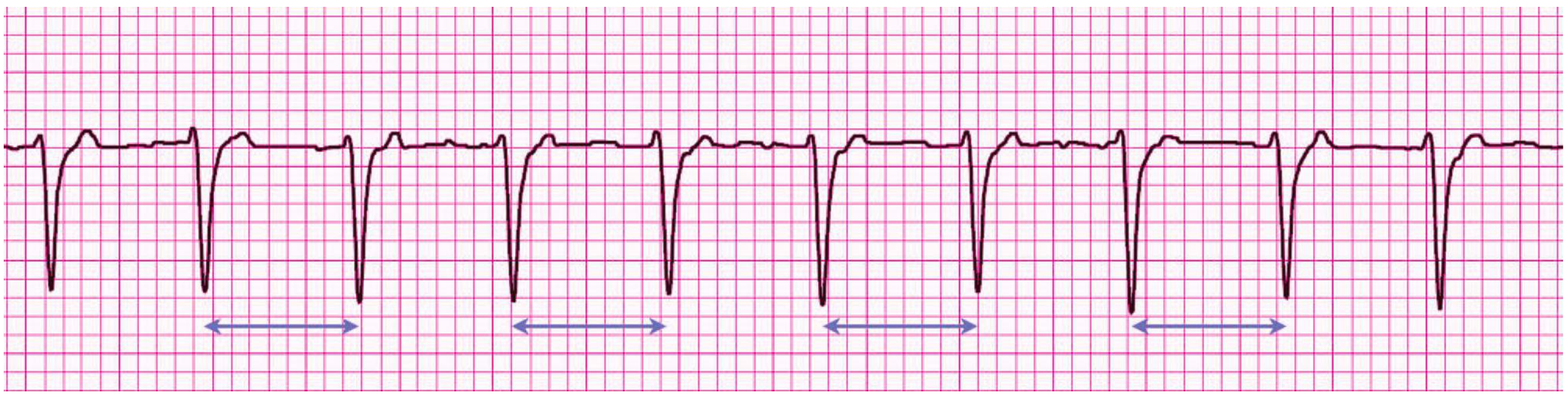


FIGURE 23.31B ■ A narrow-complex tachycardia, with no clear P waves preceding the QRS. R-R intervals are regular (double arrows), differentiating this from fine atrial fibrillation. This rhythm converted to a normal sinus rhythm after the administration of IV adenosine.

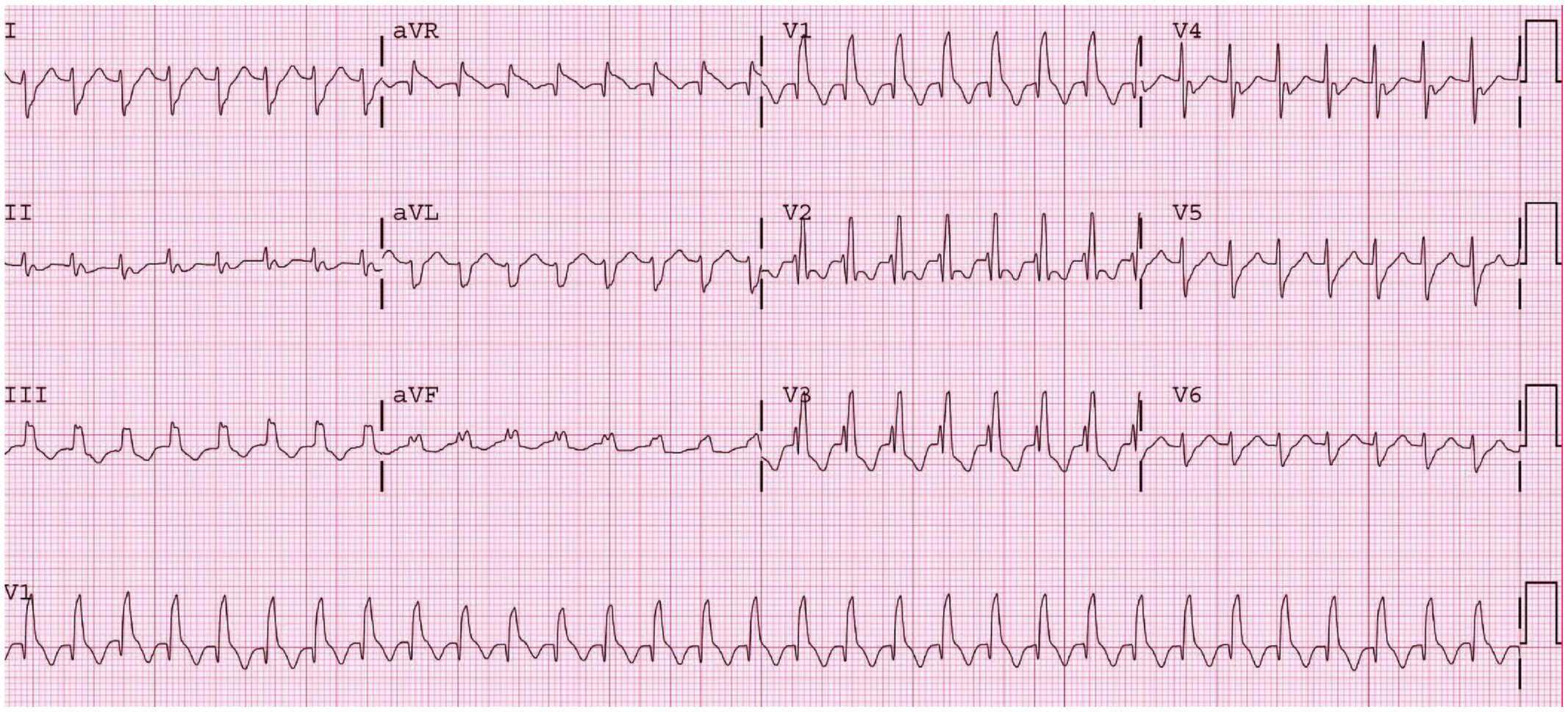


FIGURE 23.32A ■ **Supraventricular Tachycardia with Aberrant Conduction, Underlying RBBB.** (ECG contributor: Walter Clair, MD.)

ECG Findings

- Tachycardia (usually >140 bpm) with a wide QRS complex
- No "capture" or "fusion" beats or AV dissociation as seen with ventricular tachycardia
- QRS morphology consistent with one of the BBB patterns

Pearls

1. When a person with a chronic wide-complex (aberrant) BBB enters an SVT, the ECG will display a wide-complex tachycardia.
2. The rapid rate of an SVT may "outrun" the ventricular conducting system's ability to repolarize quickly, producing a rate-related BBB. The signal then must propagate cell-to-cell, producing a wide-complex tachycardia. A typical bundle branch pattern usually results.
3. An irregularly irregular or chaotic R-R interval, even if subtle, strongly suggests atrial fibrillation or flutter as the culprit SVT. In contrast, the R-R interval of ventricular tachycardia is almost never chaotic.
4. Ventricular rates of 140 to 160 bpm should prompt consideration of atrial flutter with a 2:1 block.

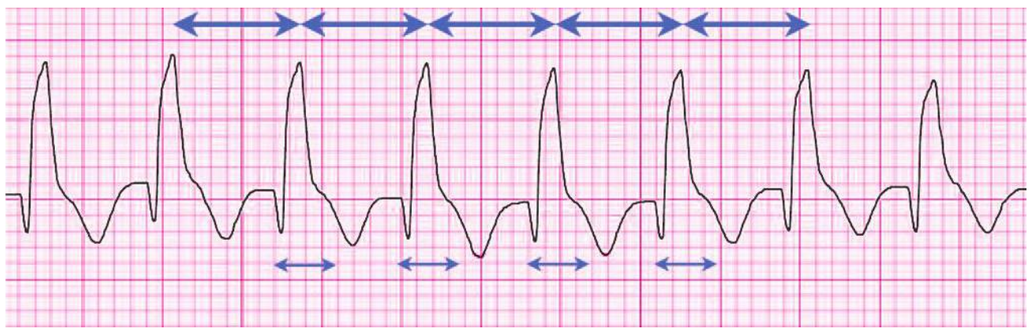


FIGURE 23.32B ■ Wide-complex tachycardia with a rate of 188 bpm. This patient has sudden onset of SVT with a known underlying RBBB. QRS complexes are wide (lower double arrows) and R-R intervals are regular (upper double arrows).

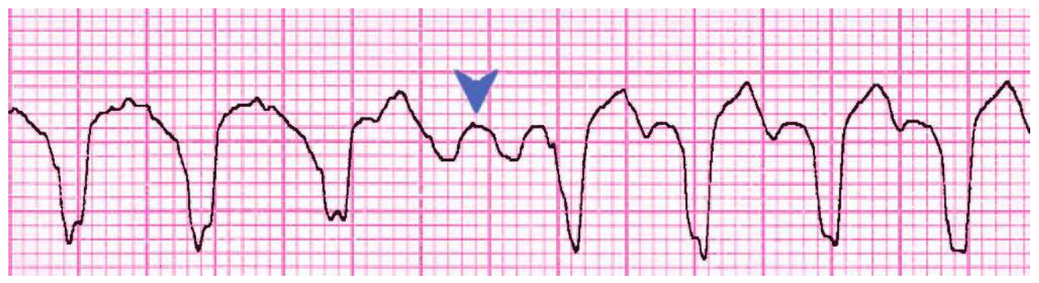


FIGURE 23.32C ■ Wide-complex tachycardia at approximately 150 bpm. The R-R interval is regular, except for one pause, when characteristic atrial flutter waves are apparent (arrowhead).

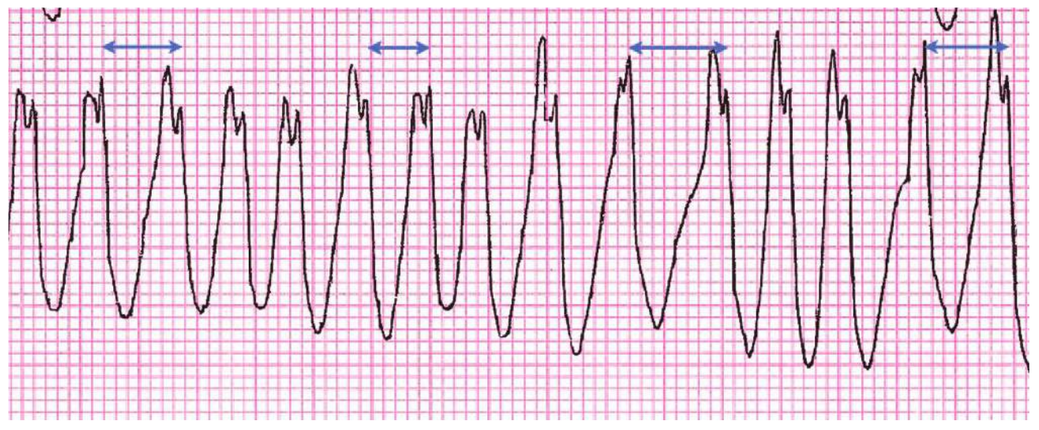


FIGURE 23.32D ■ Irregularity in the R-R interval, as seen most easily in the baseline (double arrows), strongly suggests the presence of rapidly conducted atrial fibrillation with aberrancy.

VENTRICULAR TACHYCARDIA

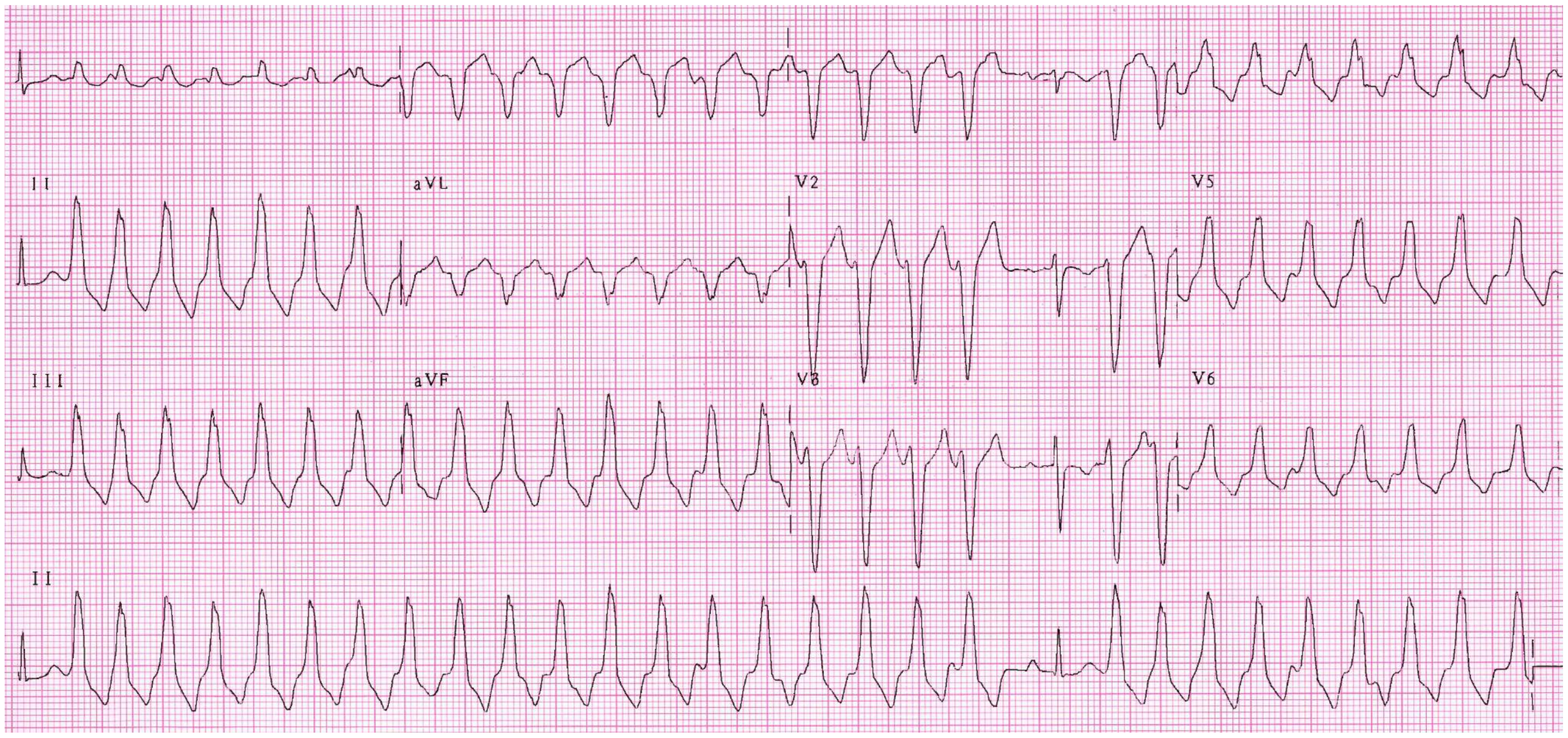


FIGURE 23.33A ■ **Ventricular Tachycardia with Capture Beat.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Tachycardia (usually >120 bpm) with a wide QRS complex.
- AV dissociation is present; P waves may appear periodically in the T wave or baseline.
- "Capture" beats may occur if atrial depolarization occurs prior to the intrinsic firing of the ventricle.
- "Fusion" beats may occur if atrial depolarization passes through the AV node at the same time as the intrinsic ventricle depolarization, producing a QRS that appears to be different or narrower than the other VT QRS complexes.

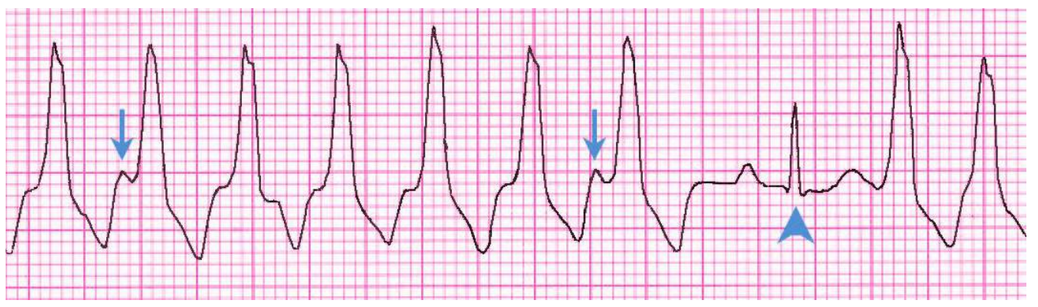


FIGURE 23.33B ■ A wide-complex tachycardia. AV dissociation is apparent, as P waves occasionally appear superimposed in the ST segment or just prior to the QRS (arrows). A capture beat occurs following a lapse in the VT (arrowhead).

Pearls

Findings more suggestive of VT versus aberrant SVT or antidromic WPW:

1. Apparent AV dissociation, capture, or fusion beats.
2. An unusual QRS axis, between 180 and 270 degrees.
3. Precordial concordance, in which QRS complexes in the precordium are all upgoing or all downgoing.
4. A completely upgoing QRS in V_1.
5. Predominately downgoing QRS in V_4, V_5, and V_6.

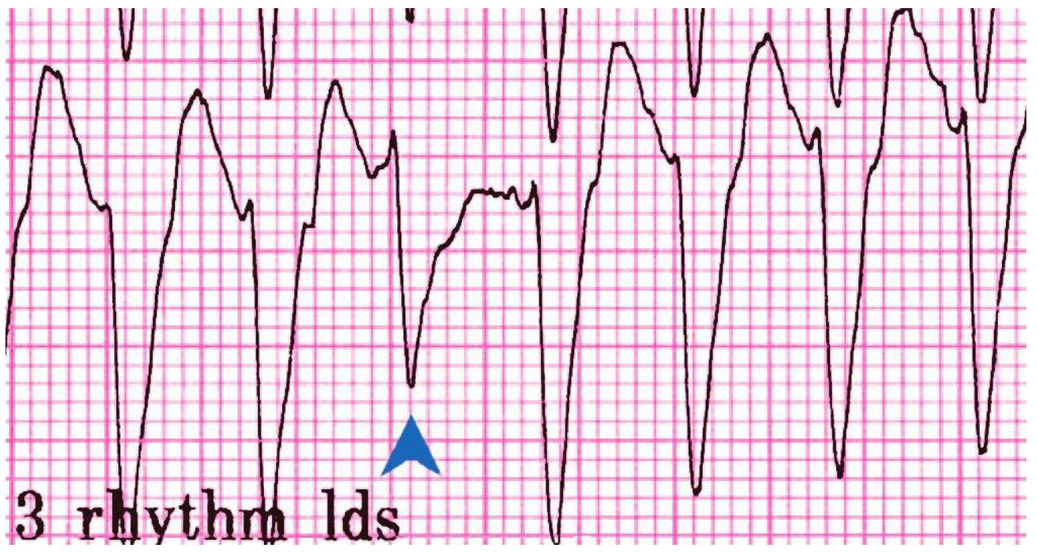


FIGURE 23.33C ■ Another example of ventricular tachycardia, featuring a fusion beat (arrowhead). (ECG contributor: Marc Mickiewicz, MD.)

VENTRICULAR FLUTTER

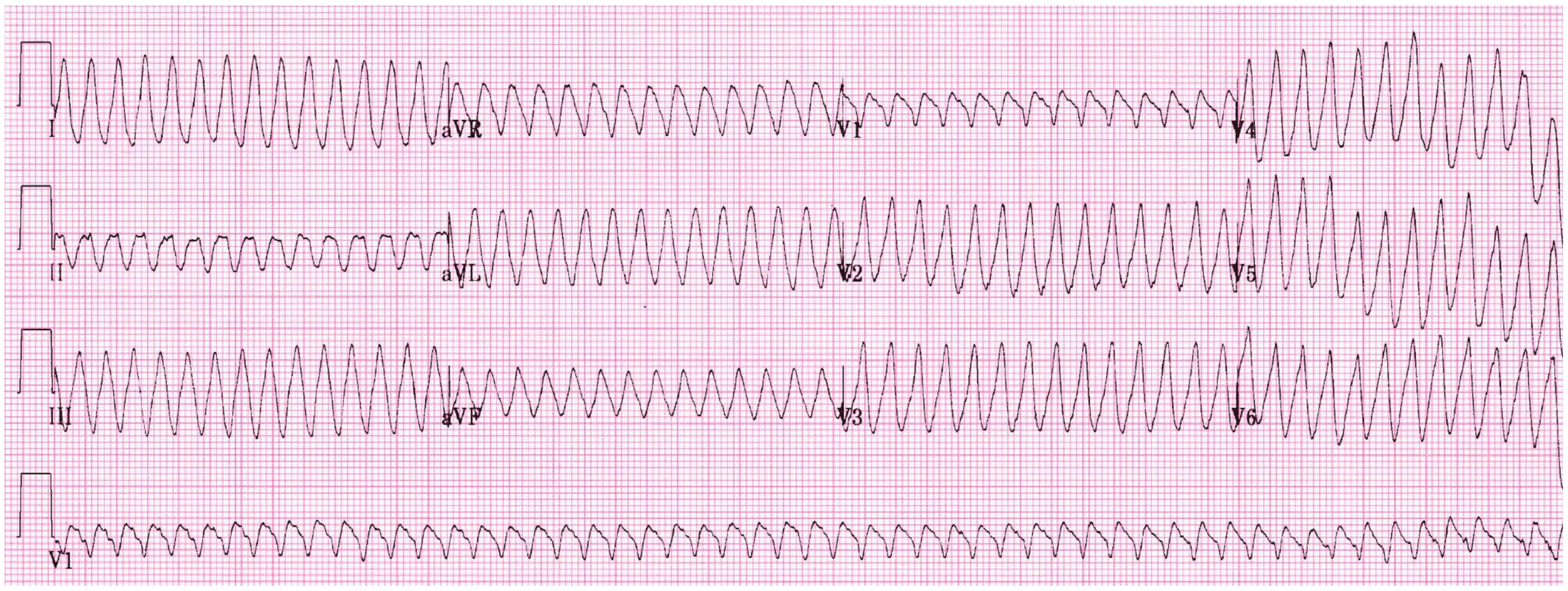


FIGURE 23.34A ■ **Ventricular Flutter.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Tachycardia with a wide monomorphic QRS complex.
- Ventricular rate may be very rapid (300 bpm).
- Sine wave appearance with regular large oscillations.

Pearls

1. Imagine an atrial flutter sawtooth with much larger amplitude.
2. When you see a very rapid wide-complex tachycardia (>240 bpm), consider ventricular flutter or WPW with atrial fibrillation or flutter.
3. WPW with atrial flutter may be indistinguishable from ventricular flutter.
4. Ventricular flutter is treated as ventricular tachycardia and usually leads to ventricular fibrillation if not promptly corrected with antiarrhythmic medications or electrical cardioversion.
5. Patients with such a rapid rate are almost always unstable. Emergent cardioversion is indicated. If the patient appears to be stable enough for chemical cardioversion, choose a medication which is safe to use with WPW, such as procainamide or amiodarone.

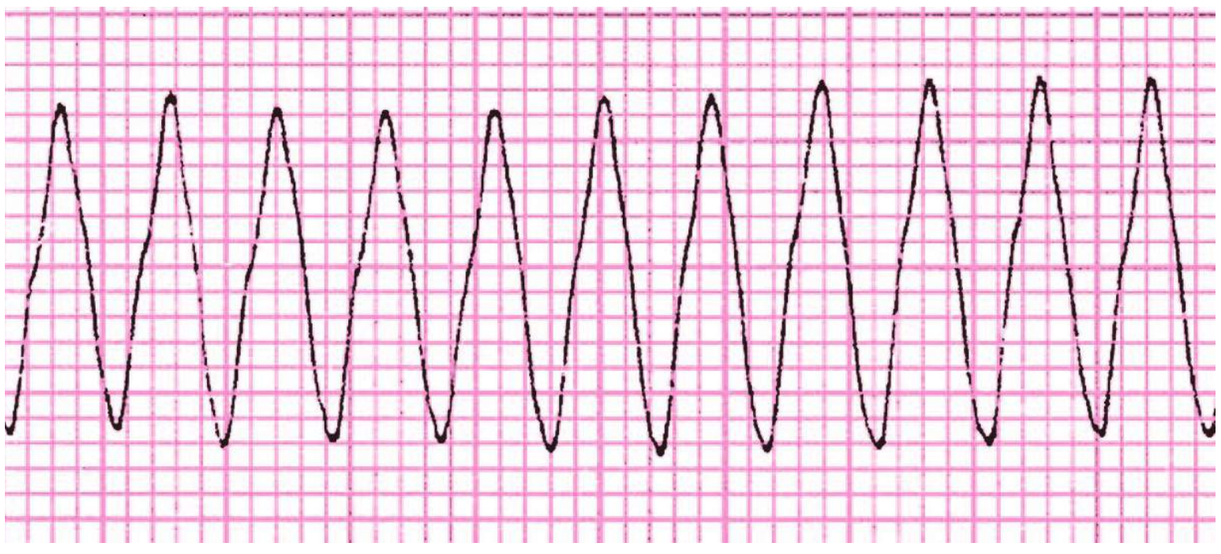


FIGURE 23.34B ■ Very rapid, regular, wide-complex tachycardia with sine-wave appearance. The rate in this example is 330 bpm. Differential diagnosis includes WPW with atrial flutter.

TORSADES DE POINTES

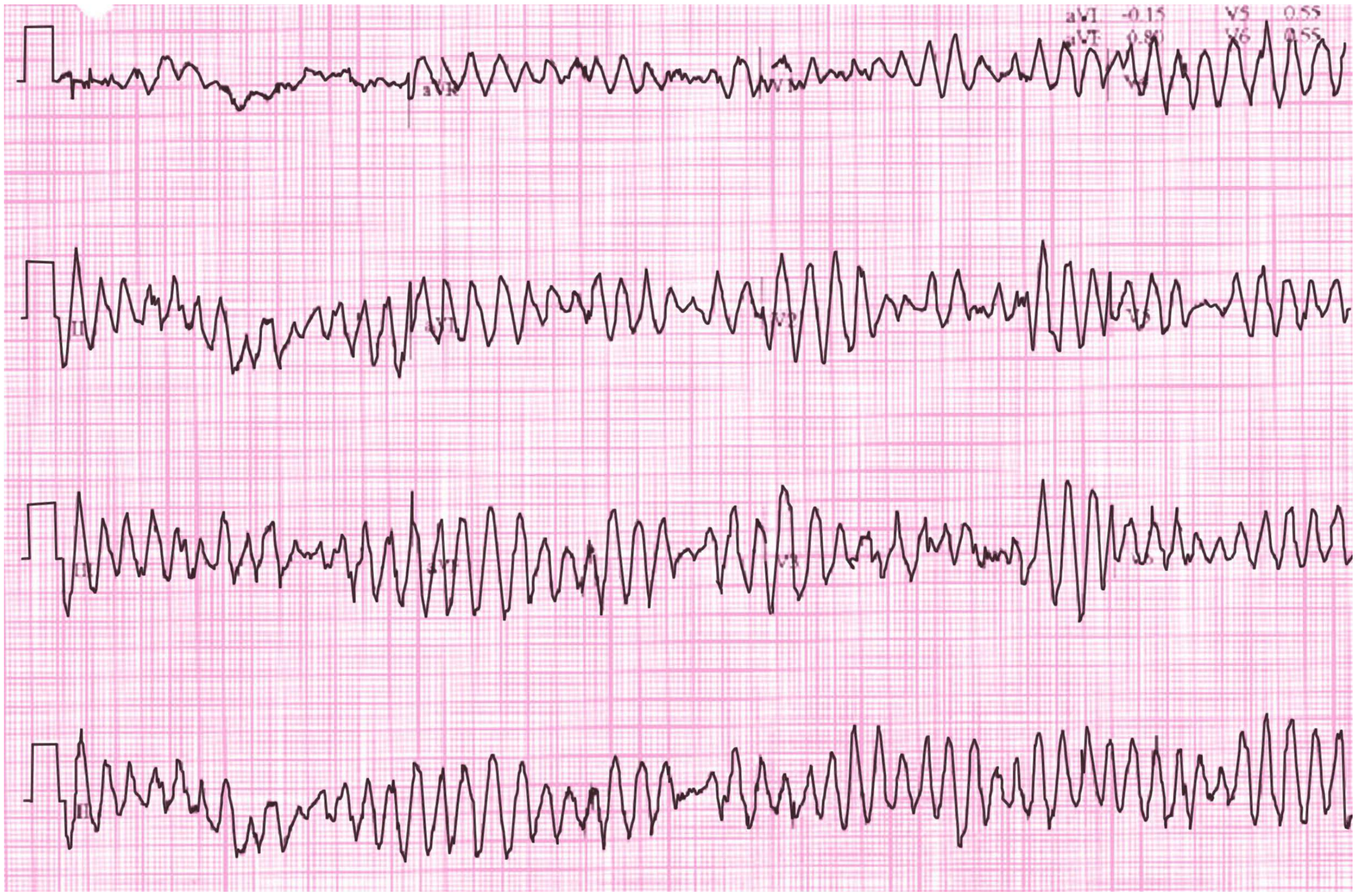


FIGURE 23.35A ■ **Torsades de Pointes.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Wide-complex tachycardia with QRS polymorphism.
- QRS morphology changes gradually throughout the tracing, appearing to rotate around the baseline to the opposite direction.

Pearls

1. The cyclic rotation of the QRS complex gives rise to the term torsades de pointes (French meaning "twisting of the points").
2. Common precipitating factors are etiologies which prolong the QT interval, such as medications, electrolyte abnormalities (hypocalcemia, hypomagnesemia, hypokalemia), and hereditary disorders.
3. Torsades can also occur in the setting of myocardial ischemia without prolongation of the QT interval.
4. Torsades is treated as a ventricular tachycardia, usually requiring defibrillation. Magnesium is used as an adjunct therapy to normal ACLS protocols.

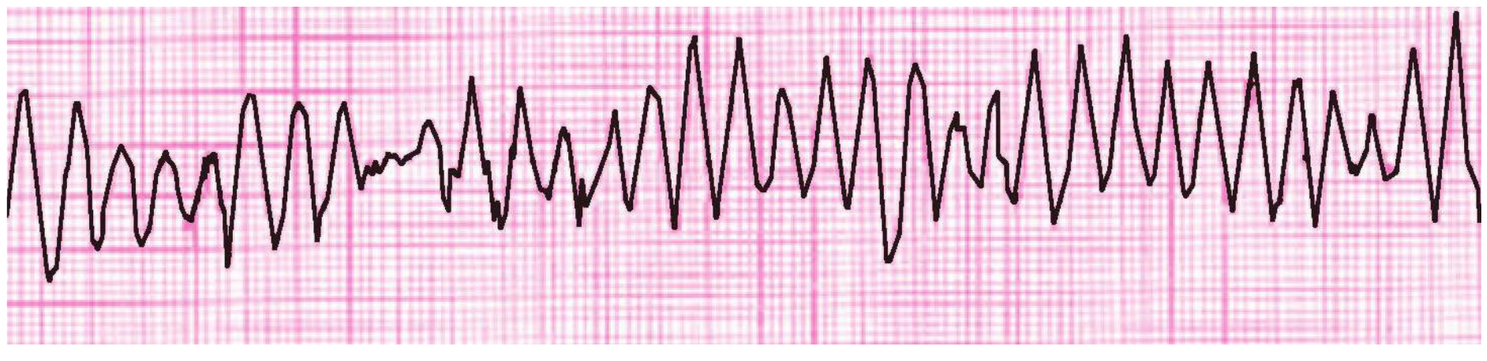


FIGURE 23.35B ■ Very rapid wide-complex tachycardia with sine-wave appearance and fluctuations in the amplitude of the QRS complexes consistent with torsades de pointes.

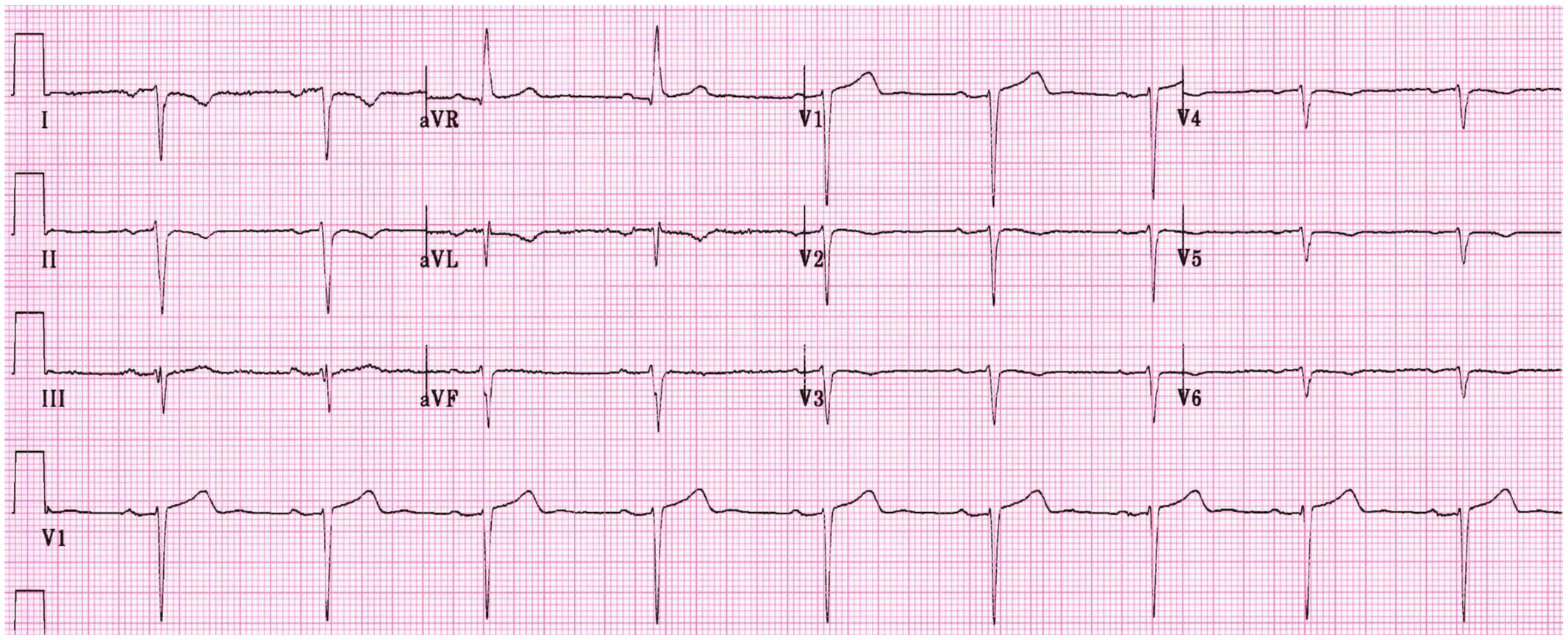


FIGURE 23.36A ■ **Dextrocardia.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- P, QRS, and T are downgoing in lead I, a mirror image of normal.
- QRS deflections in V_4 to V_6 are small and downgoing.

Pearls

1. The orientation of the heart in the chest cavity is reversed with the predominant electrical activity moving left to right (as opposed to right to left).
2. Normally placed precordial leads in a patient with dextrocardia are actually placed over the thinner right ventricle instead of the left ventricle.
3. Reversing all EKG leads should produce an essentially normal EKG.
4. A "reversed" lead I ('downward' QRS) and aVR ('upward' QRS) with normal-appearing V leads strongly suggests accidental limb lead reversal.

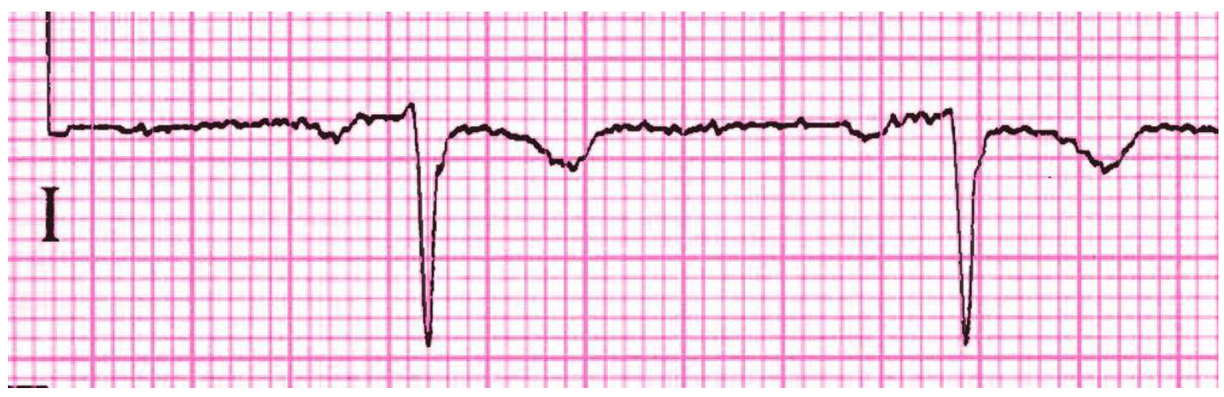


FIGURE 23.36B ■ The P wave, QRS, and T wave are downgoing in lead I. Differential diagnosis includes limb lead reversal and dextrocardia. The 12-lead ECG above represents dextrocardia as evidenced in the abnormal precordial leads.

LEFT VENTRICULAR HYPERTROPHY

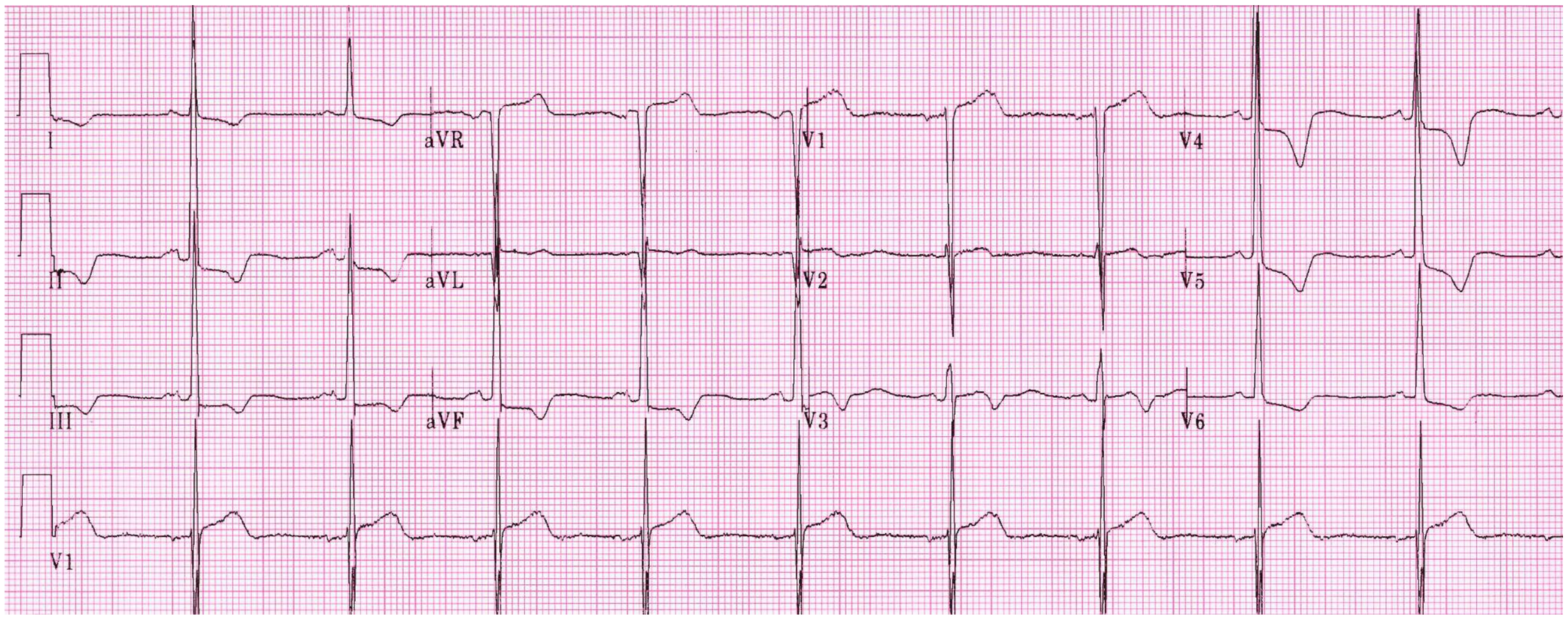


FIGURE 23.37A ■ **Left Ventricular Hypertrophy with Strain Pattern.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Large S waves in anterior precordial leads
- Large R waves in lateral precordial leads
- R wave in aVL + S wave in V_3 greater than 28 mm in males, greater than 20 mm in females
- S wave in V_1 + R wave in V_5 or V_6 greater than 35 mm if age over 40, greater than 40 mm if age 30 to 40, greater than 60 mm if age 16 to 30
- R wave in aVL greater than 11 mm
- T waves deflected opposite to QRS complex (strain pattern)

Pearls

1. The muscular left ventricle normally dominates the QRS morphology.
2. LVH can produce related changes such as left atrial abnormality. It may also produce ST-T wave changes, particularly in opposite deflections of the T wave with respect to the main deflection of the QRS complex. These ST deflections, often referred to as "LVH with strain," may be confused with ischemia.
3. LVH is often a sign of disease states such as systemic hypertension or aortic stenosis.
4. LVH may manifest on the ECG in many different ways. Several different systems for diagnosing LVH by ECG have been promoted. No one system is adequately sensitive and specific enough to warrant exclusion of all others.

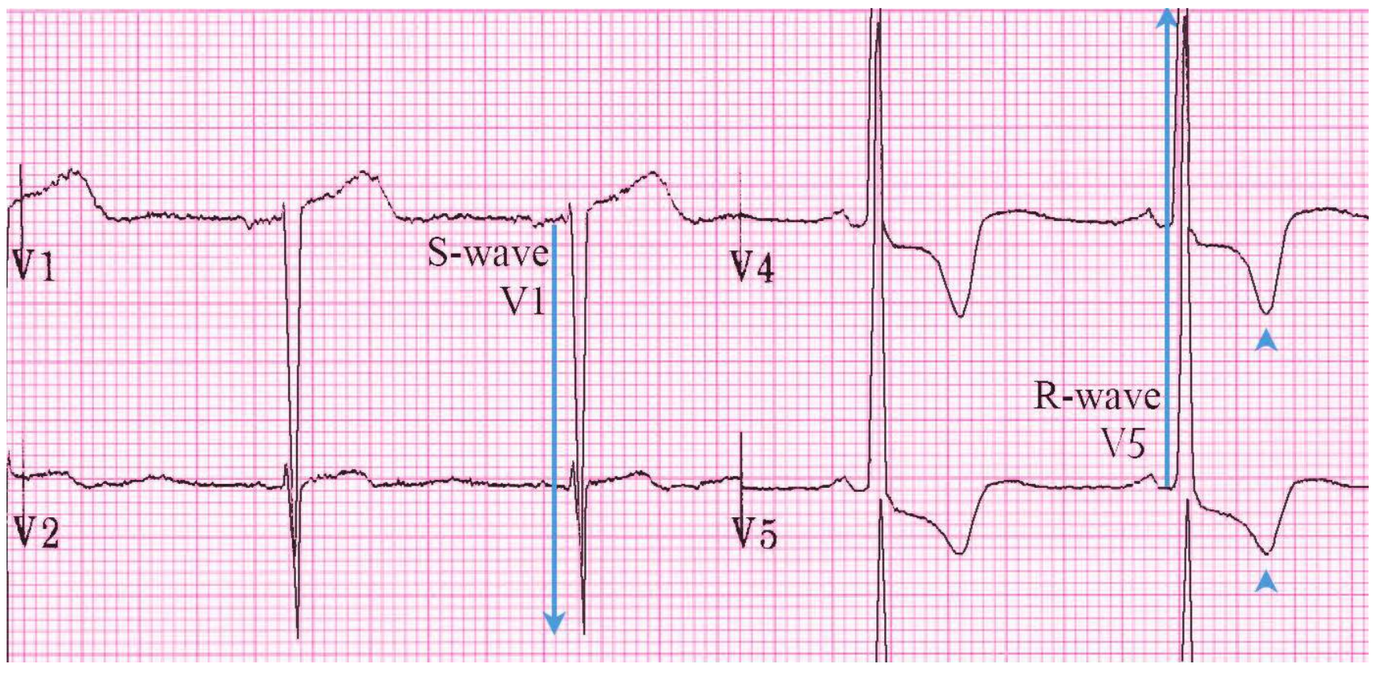


FIGURE 23.37B ■ The QRS deflections are very large. The R wave in V_5 plus the S wave in V_1 total approximately 75 mm (arrows). ST downsloping to inverted T waves in V_4 and V_5 (arrowheads) may also be seen, a finding often referred to as "LVH with strain."

RIGHT VENTRICULAR HYPERTROPHY

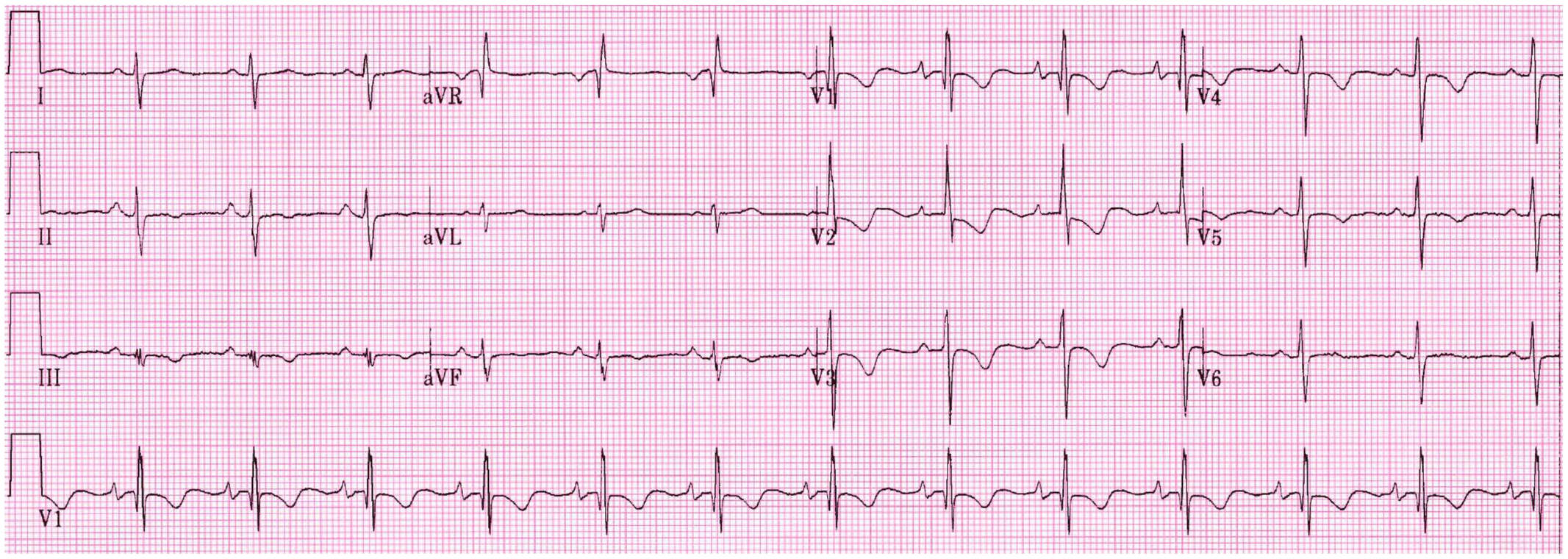


FIGURE 23.38A ■ **Right Ventricular Hypertrophy.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- S-wave amplitude increases in lateral leads (V_5, V_6, I, aVL).
- R-wave amplitude increases in aVR, V_1, V_2, and may exceed the S-wave amplitude (especially in lead V_1).
- Right axis deviation (> +90 degrees).
- T-wave inversions in relation to QRS complex.

Pearls

1. Hypertrophy of the right ventricle causes characteristic EKG changes as the predominant electrical signal of the left ventricle is overcome.
2. As right ventricular hypertrophy (RVH) persists, right atrial enlargement (RAE) may occur as seen in the example (P-wave amplitude in V_1 >1.5 mm).
3. Congenital heart disease, pulmonic or mitral stenosis, and pulmonary hypertension are common causes of RVH.

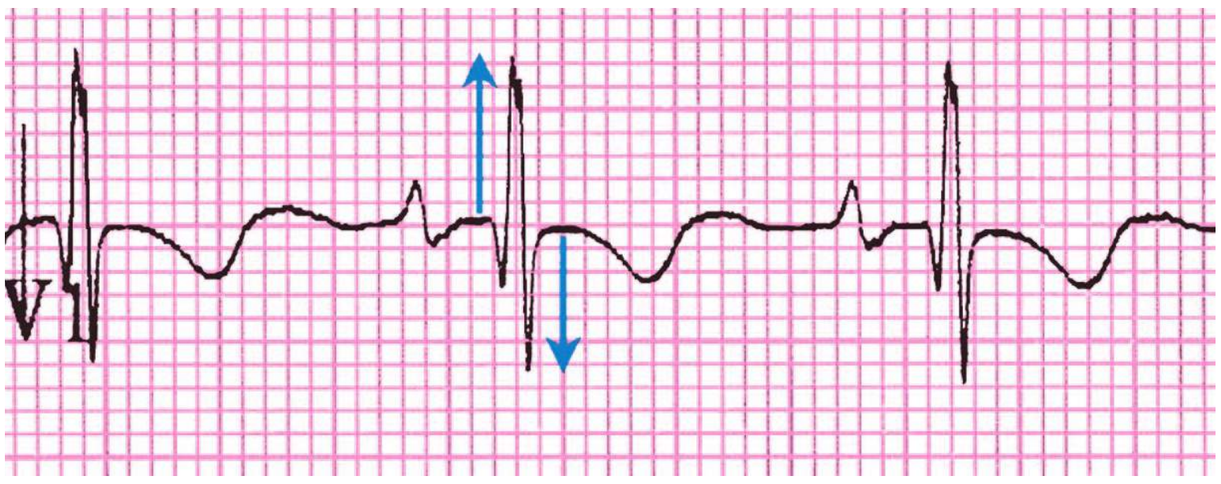


FIGURE 23.38B ■ The R-wave amplitude exceeds the S-wave amplitude (arrows) in lead V_1. In addition, the P-wave upward deflection exceeds 1.5 mm, indicating concomitant right atrial enlargement.

LEFT ATRIAL HYPERTROPHY

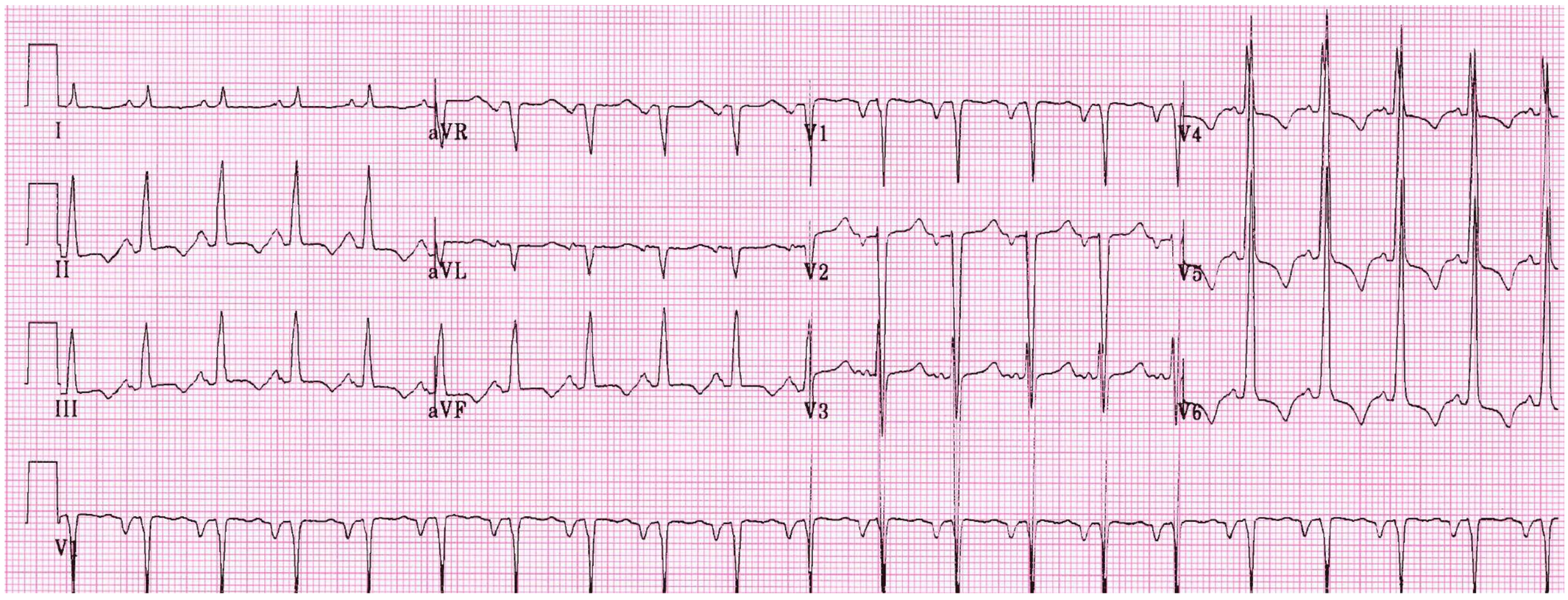


FIGURE 23.39A ■ **Left Atrial Hypertrophy with LVH Present.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Increased duration (width) of the P wave without affecting its upward amplitude (as commonly seen with right atrial abnormalities)
- Negative P-wave deflection in lead V_1, with width and depth greater than 0.04 seconds (one small box)
- Wide P wave in lead II
- Notched P wave in II, III, or aVF with duration greater than or equal to 0.12 seconds (also known as P-mitrale)

Pearls

1. Normal P-wave morphology has an amplitude of less than 2.5 mV (2.5 vertical boxes) and a duration (width) of less than 120 ms (three small boxes).
2. The left atrium depolarizes after the right atrium and therefore has the most effect on the second portion of the P wave.
3. Causes of left atrial abnormality or P-mitrale include valvular heart disease (mitral and aortic), CAD, cardiomyopathy, hypertension, and LVH.

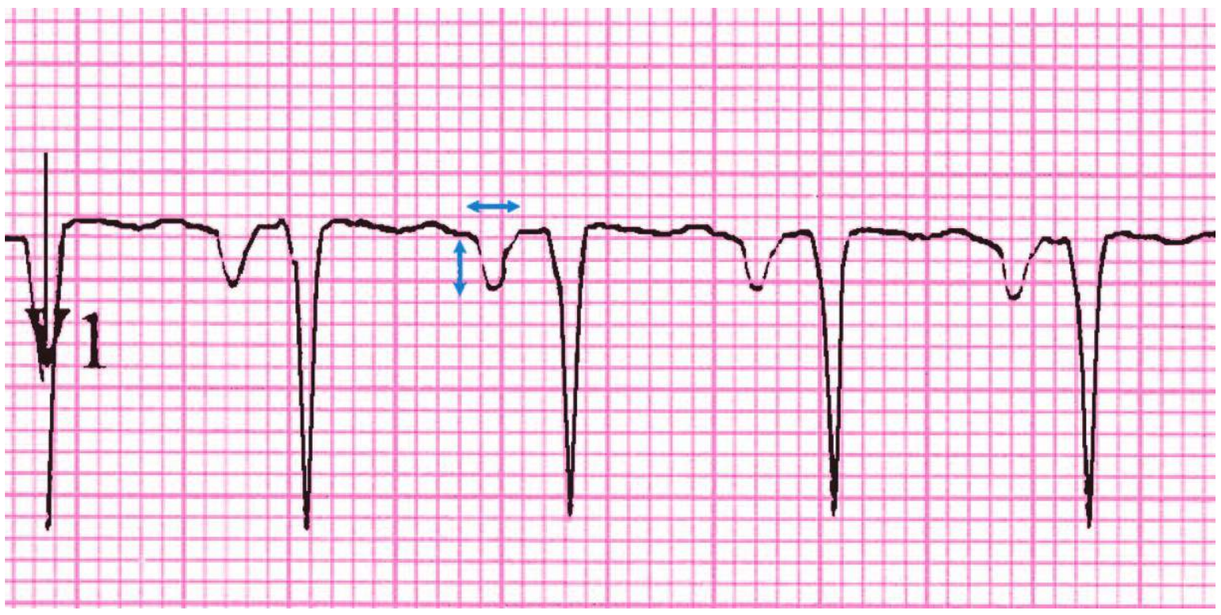


FIGURE 23.39B ■ The P wave in V_1 is downgoing. The downgoing segment is wider and deeper than one small block (double arrows).

RIGHT ATRIAL HYPERTROPHY

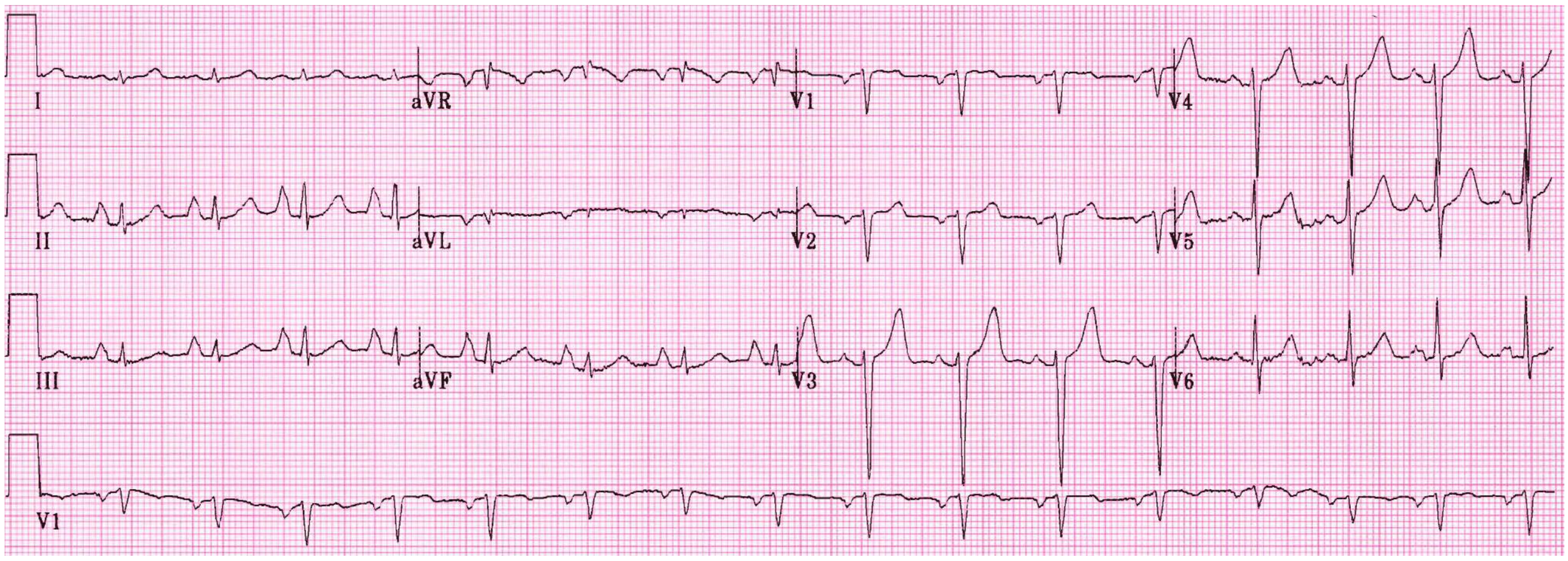


FIGURE 23.40A ■ **Right Atrial Hypertrophy.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Increased amplitude of P wave without affecting duration (as commonly seen with left atrial abnormalities)
- Peaked P waves (>2.5 mm) in leads II, III, aVF (also known as P pulmonale)
- P-waves upward deflection greater than 1.5 mm in lead V_1 or V_2

Pearls

1. Normal P-wave morphology has amplitude of less than 2.5 mV (2.5 small vertical boxes) and duration (width) of less than 120 ms (three small boxes).
2. The right atrium depolarizes before the left atrium and therefore has the most effect on the first portion of the P wave.
3. RAE is often associated with RVH, COPD, some congenital heart diseases, and pulmonary hypertension, and may be seen transiently in pulmonary embolus.

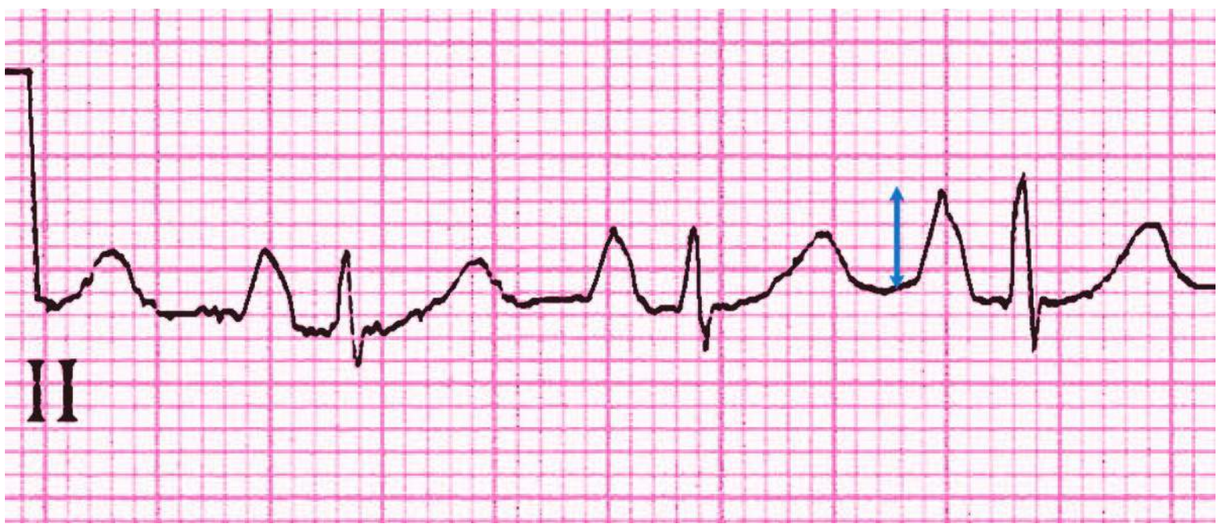


FIGURE 23.40B ■ The P wave in lead II (an inferior lead) is greater than 2.5 mm in amplitude (double arrow).

HYPERTROPHIC CARDIOMYOPATHY

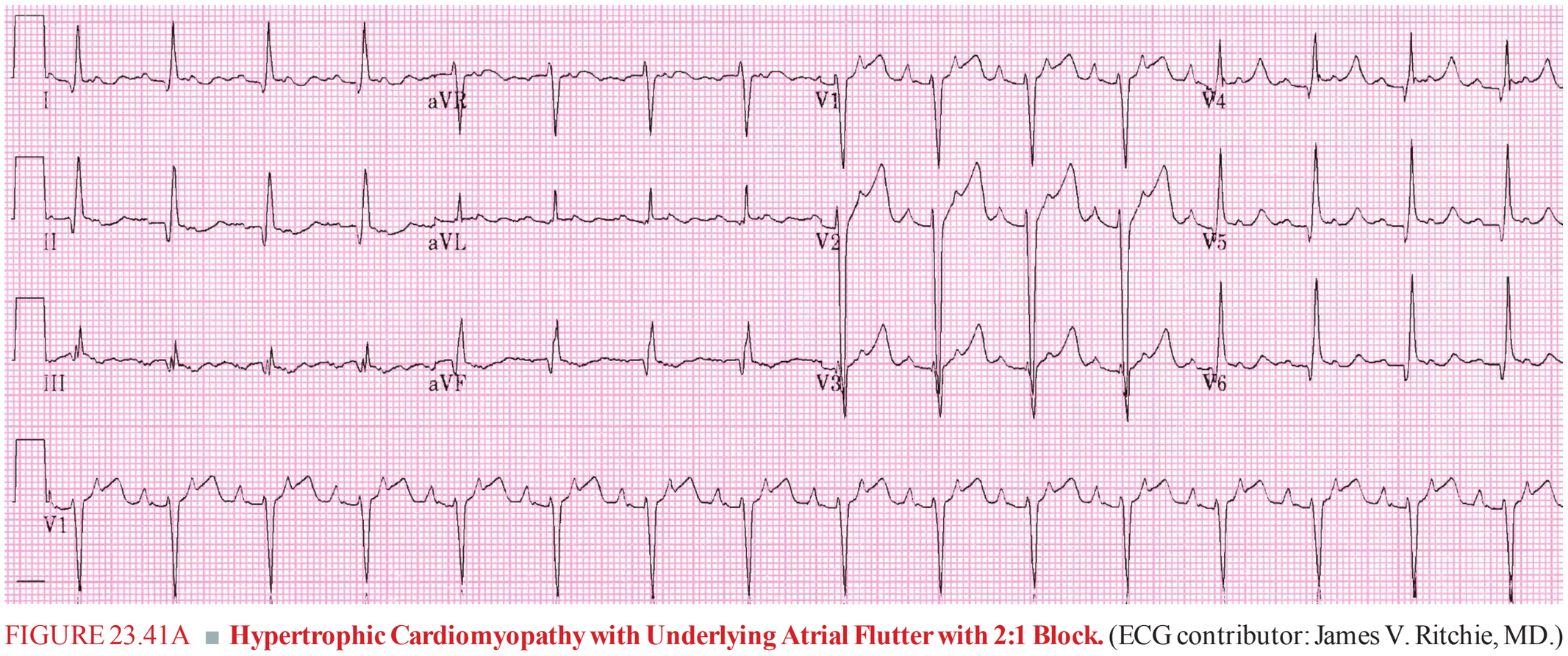


FIGURE 23.41A ■ **Hypertrophic Cardiomyopathy with Underlying Atrial Flutter with 2:1 Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- High-voltage QRS suggesting LVH
- Left atrial abnormality
- Prominent Q waves, especially in the lateral precordial leads
- Deep S waves in anterior precordial leads
- Poor R-wave progression across precordium
- Lateral T-wave inversions

Pearls

1. The ventricular myocardium hypertrophies abnormally, either concentrically or focally. Left ventricle outflow obstruction from the hypertrophy may lead to LVH without dilation.
2. Hypertrophic cardiomyopathy (HCM) is also known as idiopathic hypertrophic subaortic stenosis (IHSS), hypertrophic obstructive cardiomyopathy (HOCM), and muscular subaortic stenosis (MSS).
3. ECG changes are variable and usually do not include all listed above.
4. Always consider this condition in young athletes with syncope or unusual dyspnea.
5. HCM is associated with a systolic ejection murmur that diminishes with increases in preload (having the patient squat) and augments with decreases in preload (Valsalva maneuver).

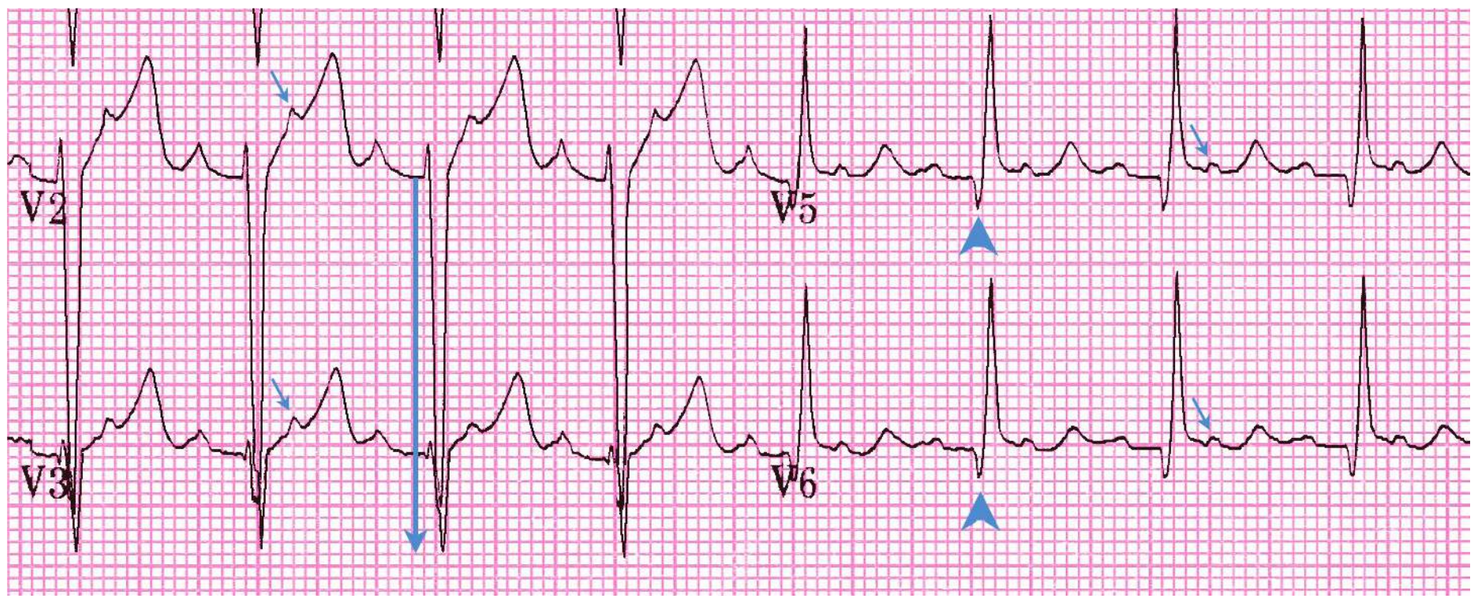


FIGURE 23.41B ■ Deep S-wave voltage (28 mm S in V_2, large arrow), and narrow Q waves in V_5 and V_6 (arrowheads). This patient also has atrial flutter with 2:1 block. The additional P waves appear in the ST segments (small arrows).

BRUGADA SYNDROME

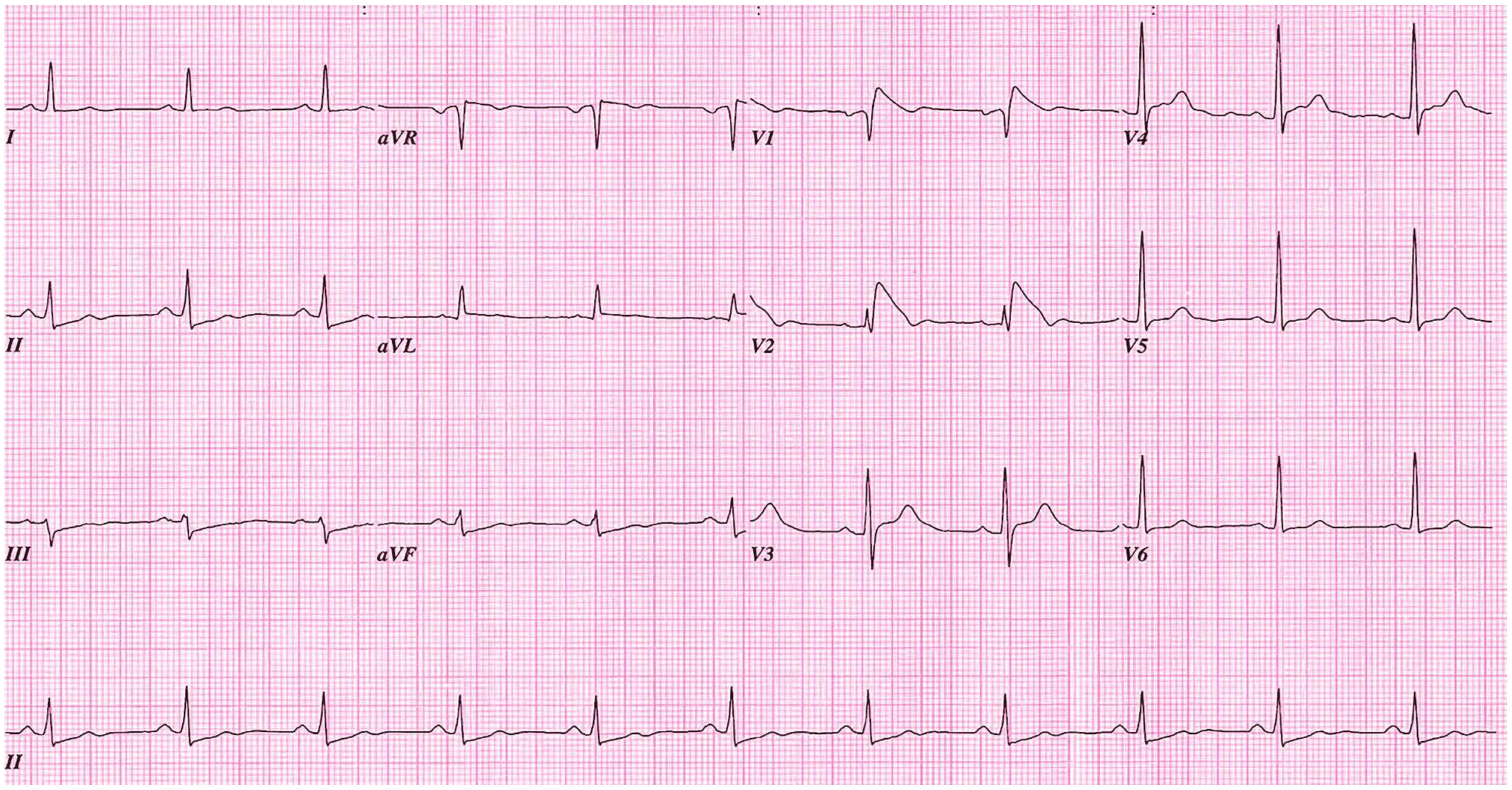


FIGURE 23.42A ■ **Brugada Syndrome.** (ECG contributor: Michael L. Juliano, MD.)

ECG Findings

- Incomplete or complete RBBB pattern in leads V_1, V_2, and sometimes V_3
- ST elevation of at least 1 mV in leads V_1, V_2, and sometimes V_3 followed by T inversion (convex pattern) or upright T (concave, or "saddle back" pattern)

Pearls

1. This syndrome was first described in individuals who experienced sudden cardiac death with structurally normal hearts, but congenitally abnormal ion channels in myocyte cell membranes have been associated with the disease.
2. Concern for spontaneous ventricular dysrhythmia is high.
3. Consultation with a cardiologist is recommended for electrophysiological testing and intracardiac defibrillator placement.

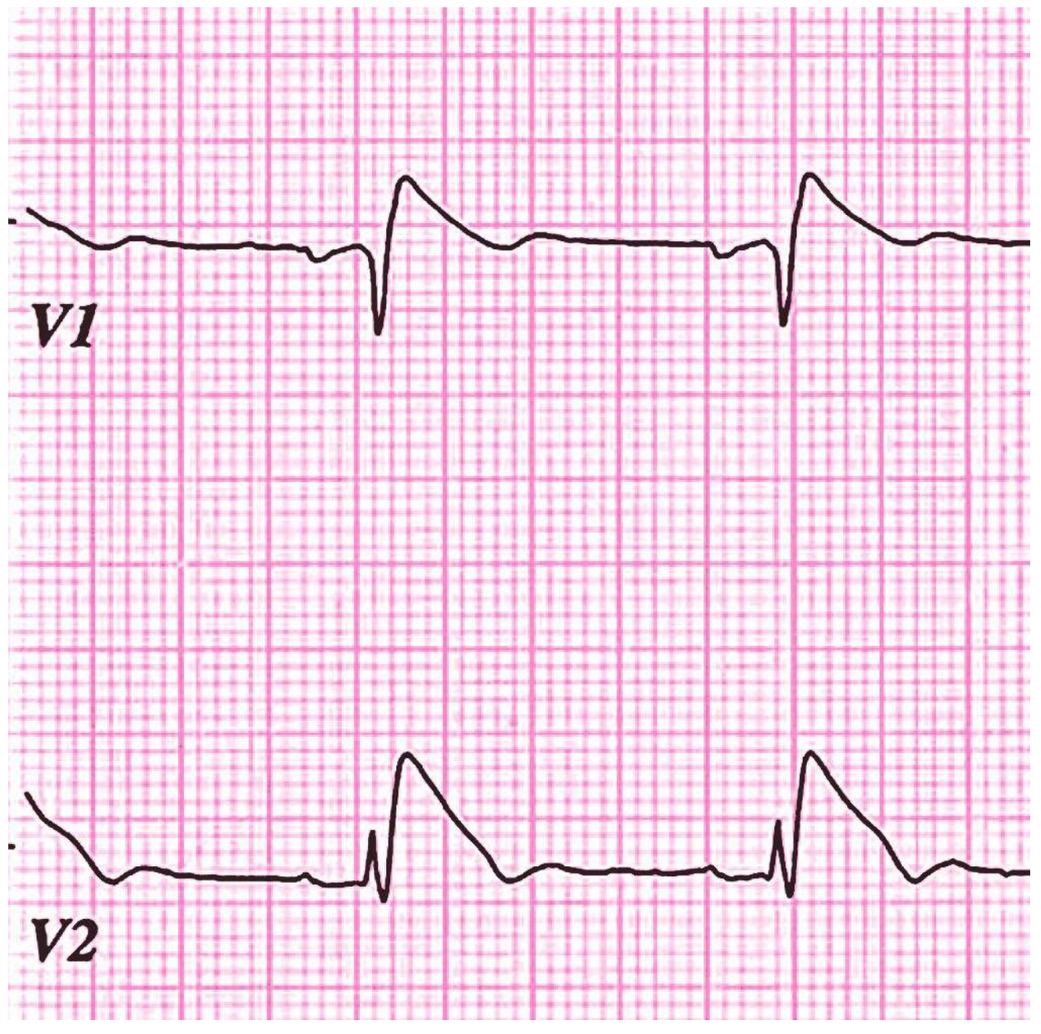


FIGURE 23.42B ■ RBBB pattern with ST elevation (type 1 Brugada syndrome).

WOLFF-PARKINSON-WHITE SYNDROME

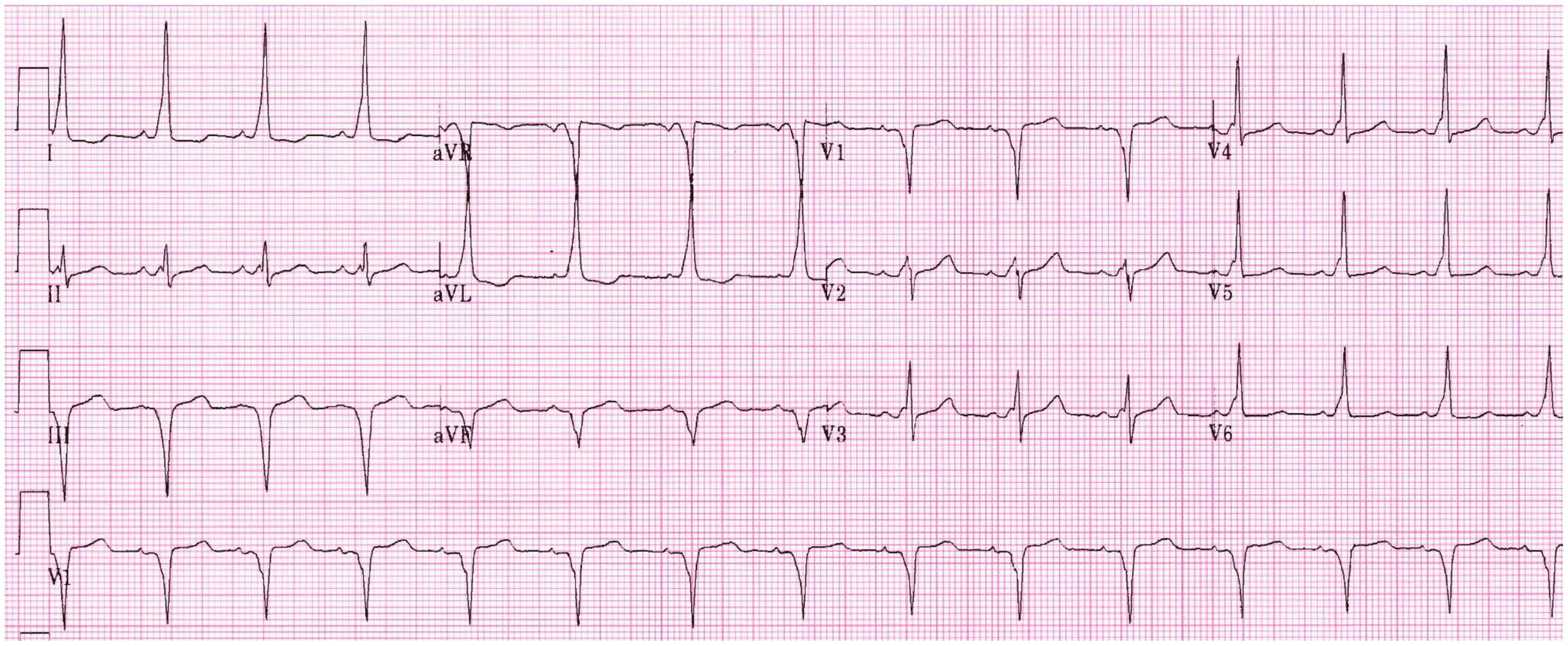


FIGURE 23.43A ■ **Wolff-Parkinson-White Syndrome.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Normal P waves
- Shortened PR interval
- Prolonged QRS interval
- Delta waves (slurring of the initial upstroke of R wave)

Pearls

1. Accessory tracts from the atria to the ventricles lead to depolarization of ventricles without using the AV node as the primary connecting route.
2. Tachycardia associated with WPW may be mistaken for ventricular tachycardia. Suspect WPW if the QRS complex is wide and tachycardia is extreme (ventricular rate > 240).
3. Do not treat this tachycardia with AV nodal blocking agents (calcium channel blockers, β-blockers, digoxin). This may lead to unopposed ventricular stimulation through the accessory tract and may worsen the tachycardia.
4. Procainamide, amiodarone, and cardioversion are accepted methods for conversion of a tachycardia associated with WPW.
5. Depolarization via the accessory pathway may produce "pseudo-Q waves" as shown in leads III and aVF in this example.

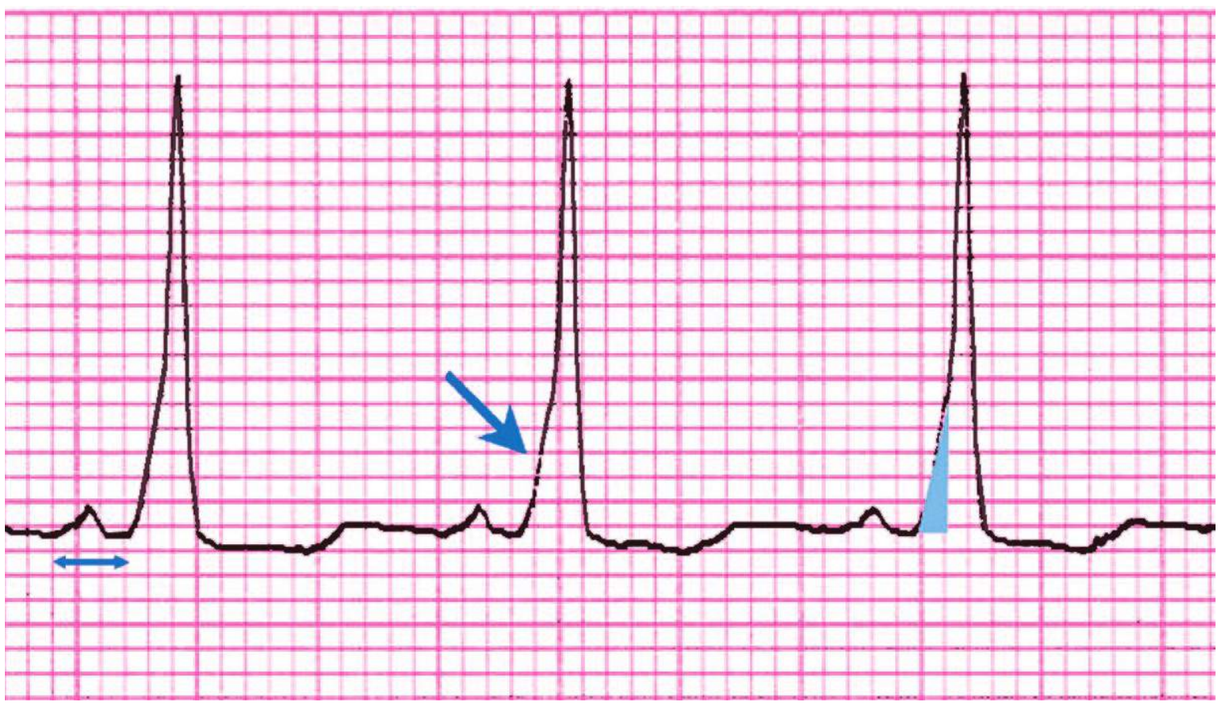


FIGURE 23.43B ■ The PR interval is shortened (double arrow) and a delta wave (upsloping initial QRS segment) is seen (arrow, shaded area).

HYPOTHERMIA

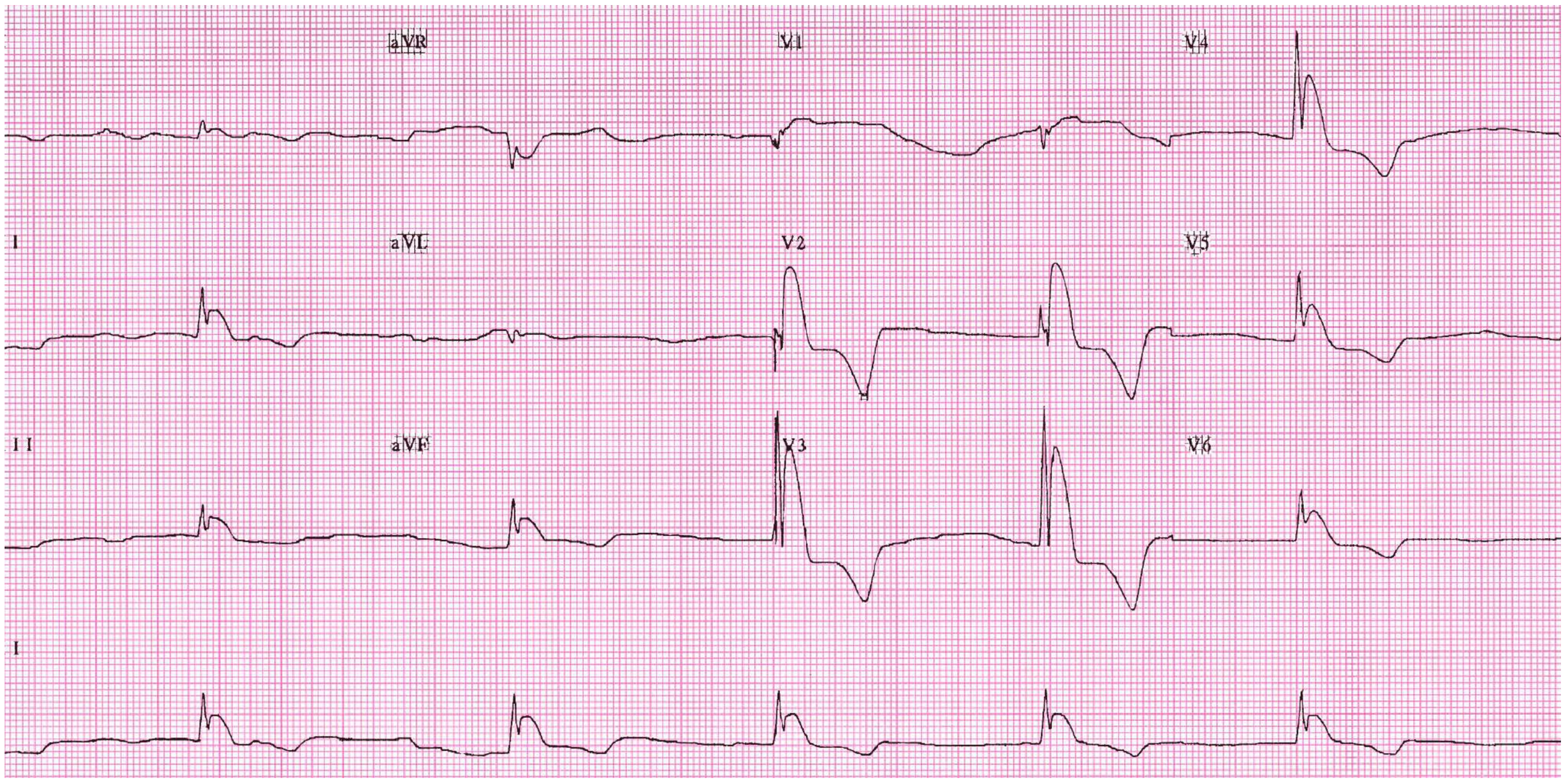


FIGURE 23.44A ■ **Hypothermia with Osborne Waves ("J" Waves) Present.** (ECG contributor: Michael L. Juliano, MD.)

ECG Findings

- Sinus bradycardia or atrial fibrillation with slow ventricular response.
- PR, QRS, and QT intervals are typically prolonged.
- Osborne or "J" wave (a positive deflection of the terminal portion of the QRS complex). The J wave may be subtle or large and "humped."

Pearls

1. The hypothermic patient's rhythm slows, proceeding from sinus bradycardia to atrial fibrillation with slow response and may proceed to other arrhythmias including ventricular fibrillation and asystole.
2. The amplitude of the "J" wave corresponds to the degree of hypothermia.
3. Myocardial damage and EKG changes associated with hypothermia are not necessarily due to low temperature. They may be indirectly caused by systemic circulatory issues such as hypoperfusion.
4. Defibrillation and many medications may be ineffective in the hypothermic patient. Rapid rewarming is indicated as an initial and critical resuscitative measure.

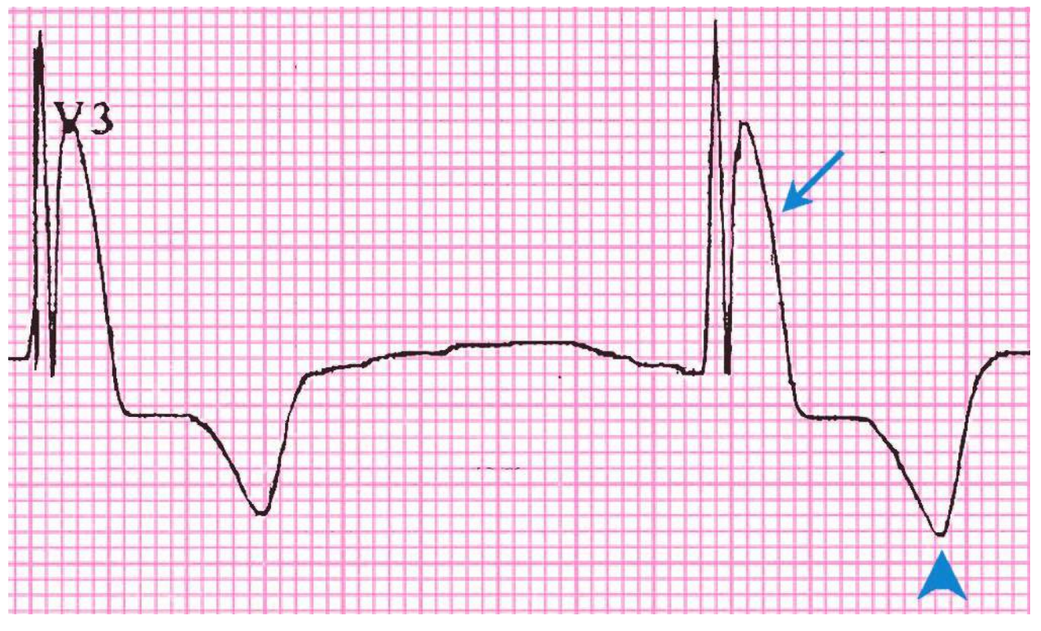


FIGURE 23.44B ■ A large Osborn wave (J wave) (arrow) follows the QRS, and is distinct from the T wave (arrowhead).

FIGURE 23.44C ■ This is a more typical appearance of a J wave (arrow).

HYPERKALEMIA

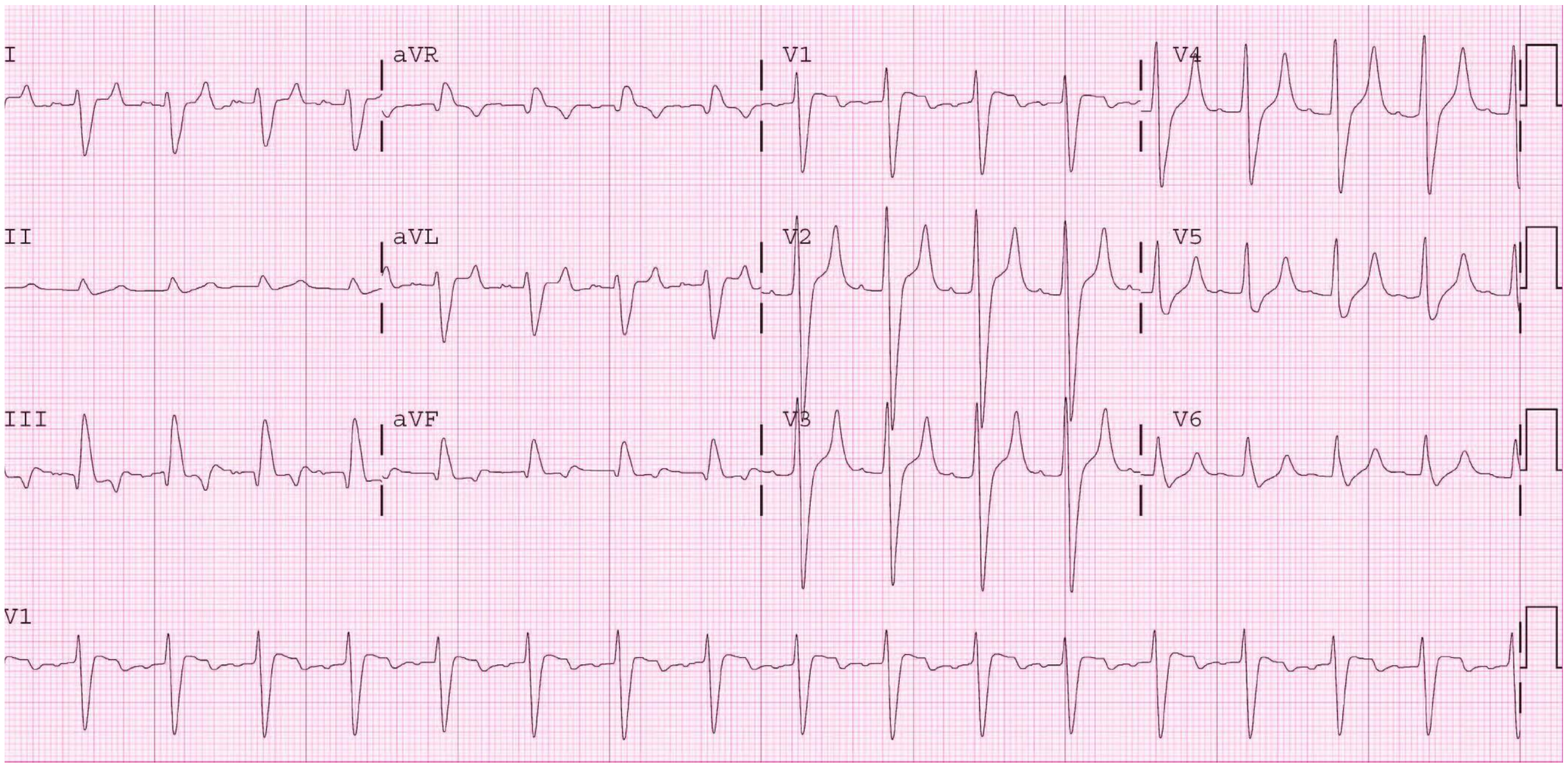


FIGURE 23.45A ■ **Hyperkalemia (K 7.1).** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

Findings are variable but tend to correlate with increasing serum potassium levels following the order below:

- Peaked T waves, tented with a narrow base (may be >10 mm high in precordial leads and/or >6 mm in limb leads)
- QT interval shortening
- QRS complex widening
- PR interval prolongation
- Decreased P-wave amplitude
- As potassium levels approach and exceed 8.0 mEQ/L:
  - Indiscernible P waves
  - Sine wave appearance of QRS-T complex
  - Left or right bundle branch pattern
  - Ventricular tachycardia, fibrillation, or asystole

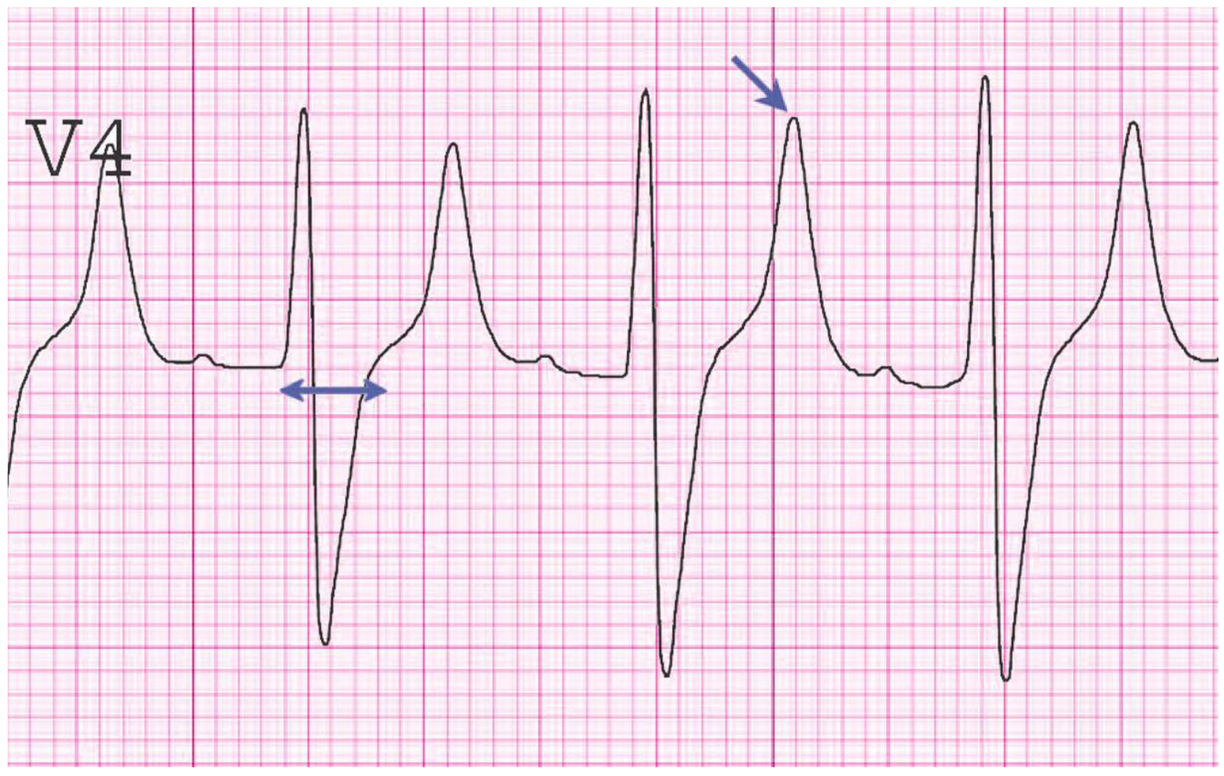


FIGURE 23.45B ■ Peaked T waves (arrow), widened QRS (double arrow), and subtle flattening of the P waves are seen in this patient with a serum K of 7.1.

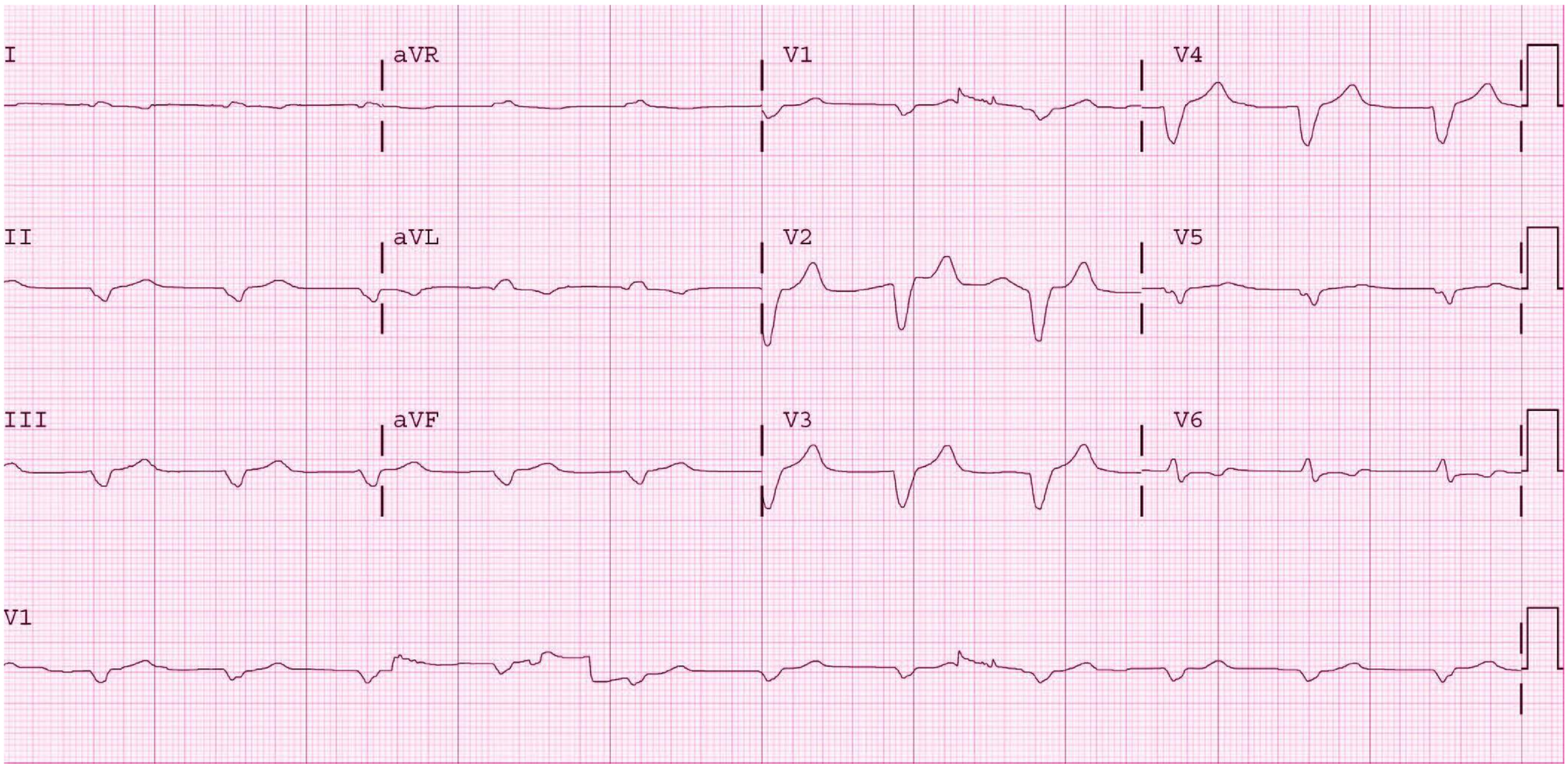


FIGURE 23.46A ■ **Severe Hyperkalemia (K 8.5).** (ECG contributor: Ben Heavrin, MD.)

Pearls

1. As the QRS complex widens, it appears more "blunted" than expected when compared with an LBBB.
2. Acute treatment for hyperkalemia includes insulin and glucose, sodium bicarbonate, and β-agonists in an attempt to drive potassium into the cell. Intravenous calcium may be used to stabilize the myocardium but has no effect on serum potassium levels. These are temporizing measures which must be followed by definitive treatment of the underlying problem, which may include the need for dialysis.

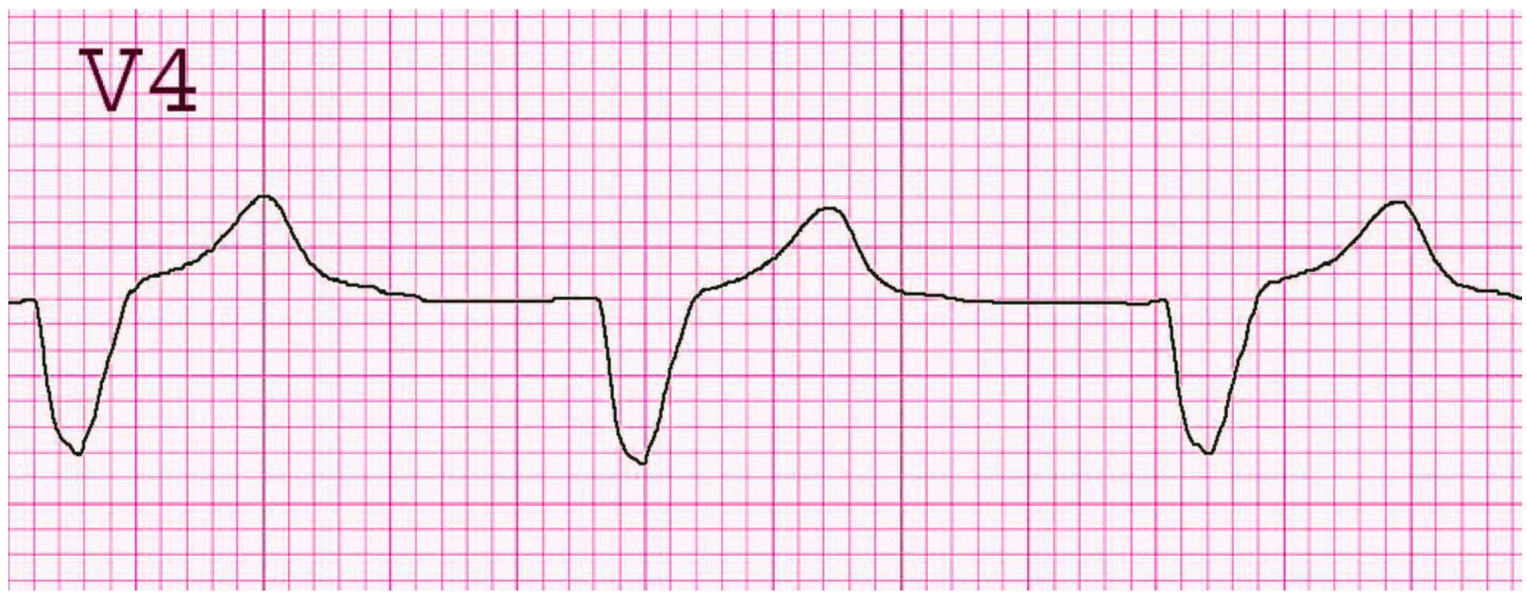


FIGURE 23.46B ■ Wide, blunted QRS with near sine-wave appearance. No P waves visible. Serum K was 8.5 in this patient. These abnormalities resolved with rapid treatment.

HYPOKALEMIA

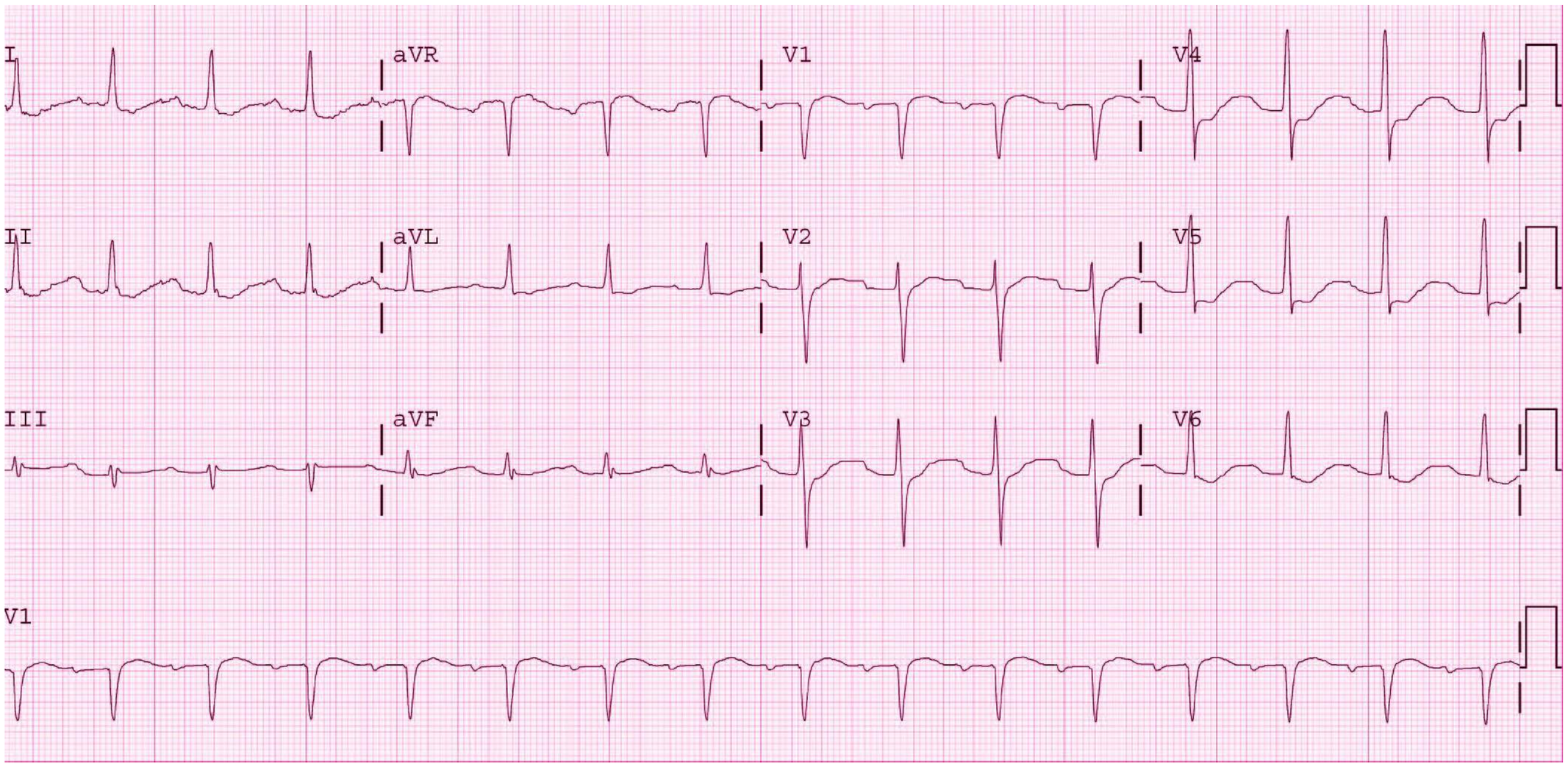


FIGURE 23.47A ■ **Hypokalemia.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- Flattened or inverted T waves
- Prominent U waves
- ST segment depression
- Conduction disturbances

Pearls

1. Hypokalemia can produce varied ECG changes associated with the repolarization phase of the cardiac cycle.
2. Unlike hyperkalemia, in hypokalemia there is no direct correlation with the potassium level and the severity of ECG changes. However, more ECG changes may become apparent as the potassium level falls.
3. Suspect hypomagnesemia if the ECG does not normalize after potassium replacement.

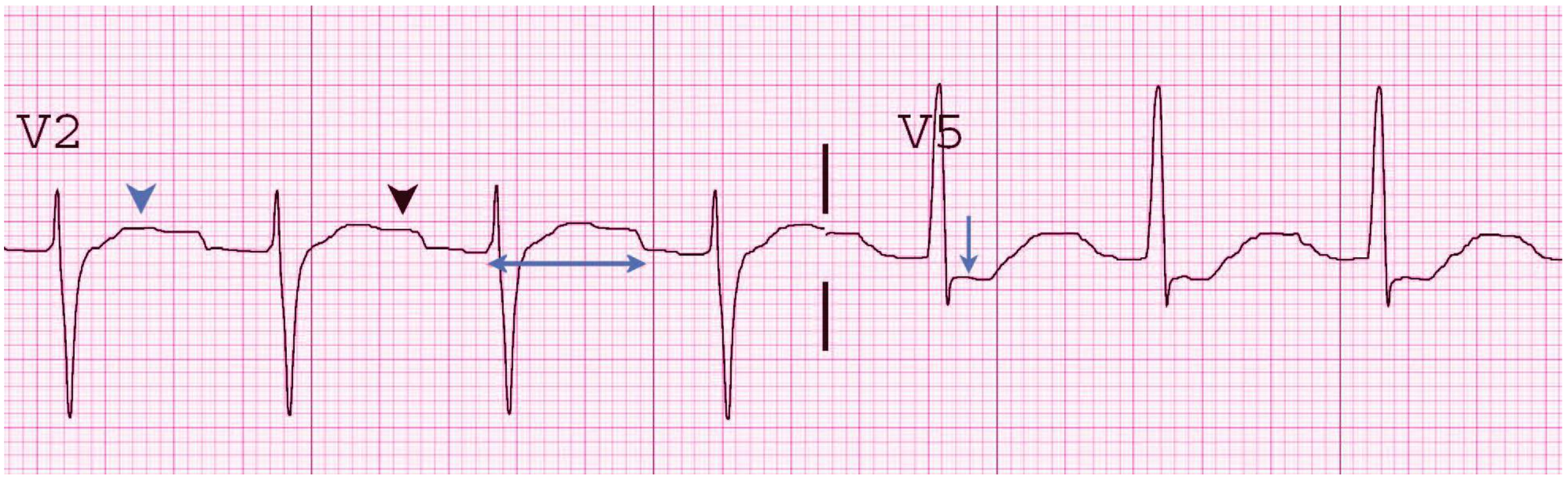


FIGURE 23.47B ■ This EKG demonstrates multiple findings consistent with hypokalemia: flattened T waves (blue arrowhead), U waves (black arrowhead), prolonged QT (QU) intervals (double arrow), and ST-segment depression (arrow). This patient's potassium level was 1.9.

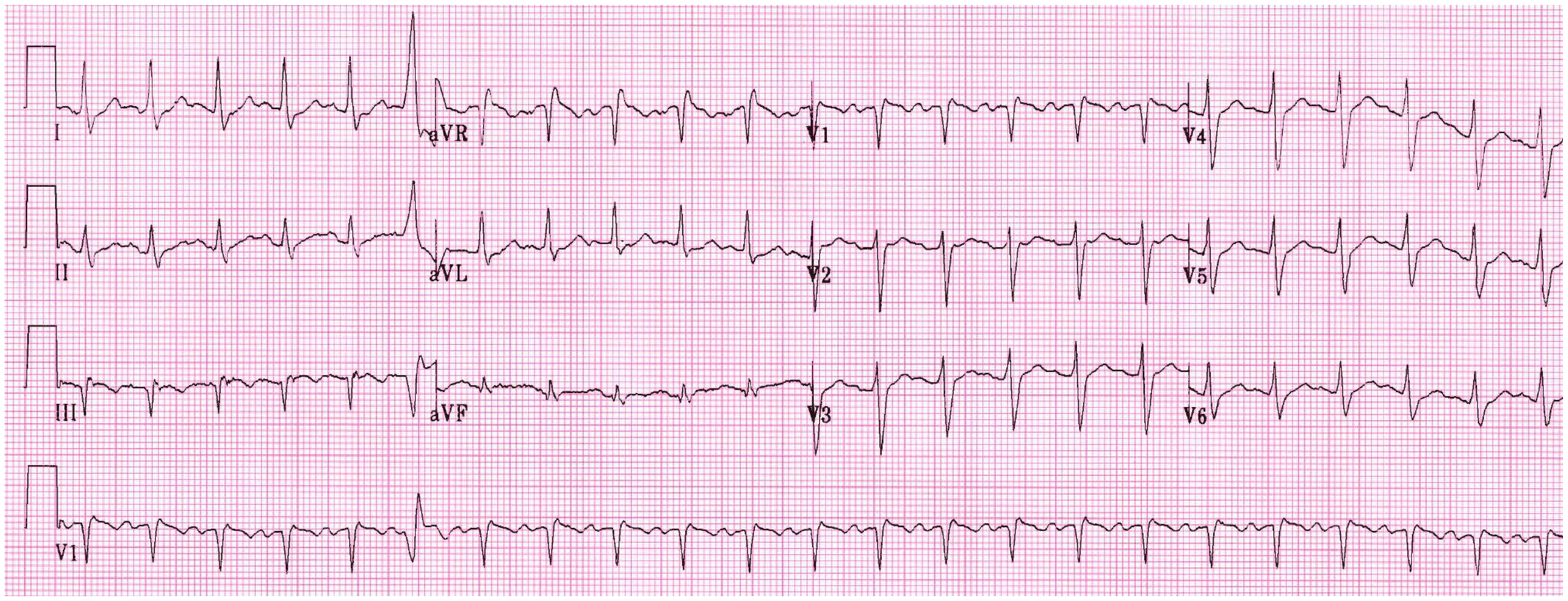


FIGURE 23.48A ■ **Sinus Tachycardia and S1Q3T3 Pattern in a Patient with Acute Pulmonary Embolism.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Sinus tachycardia, nonspecific ST-T changes
- Precordial T-wave inversions
- Prominent S wave in lead I, Q wave in lead III, and inverted T wave in III (S1/Q3/T3)
- Incomplete or complete RBBB, P pulmonale (lead II)
- May see right axis deviation

Pearls

1. No EKG pattern is diagnostic for pulmonary embolism. Small-to-moderate emboli may not affect the EKG.
2. With large emboli, increased resistance to pulmonary arterial flow produces right ventricle overload and dilation.
3. Increased right atrial pressures may produce "P pulmonale" (tall P waves >2.5 mm in lead II) or atrial dysrhythmias.

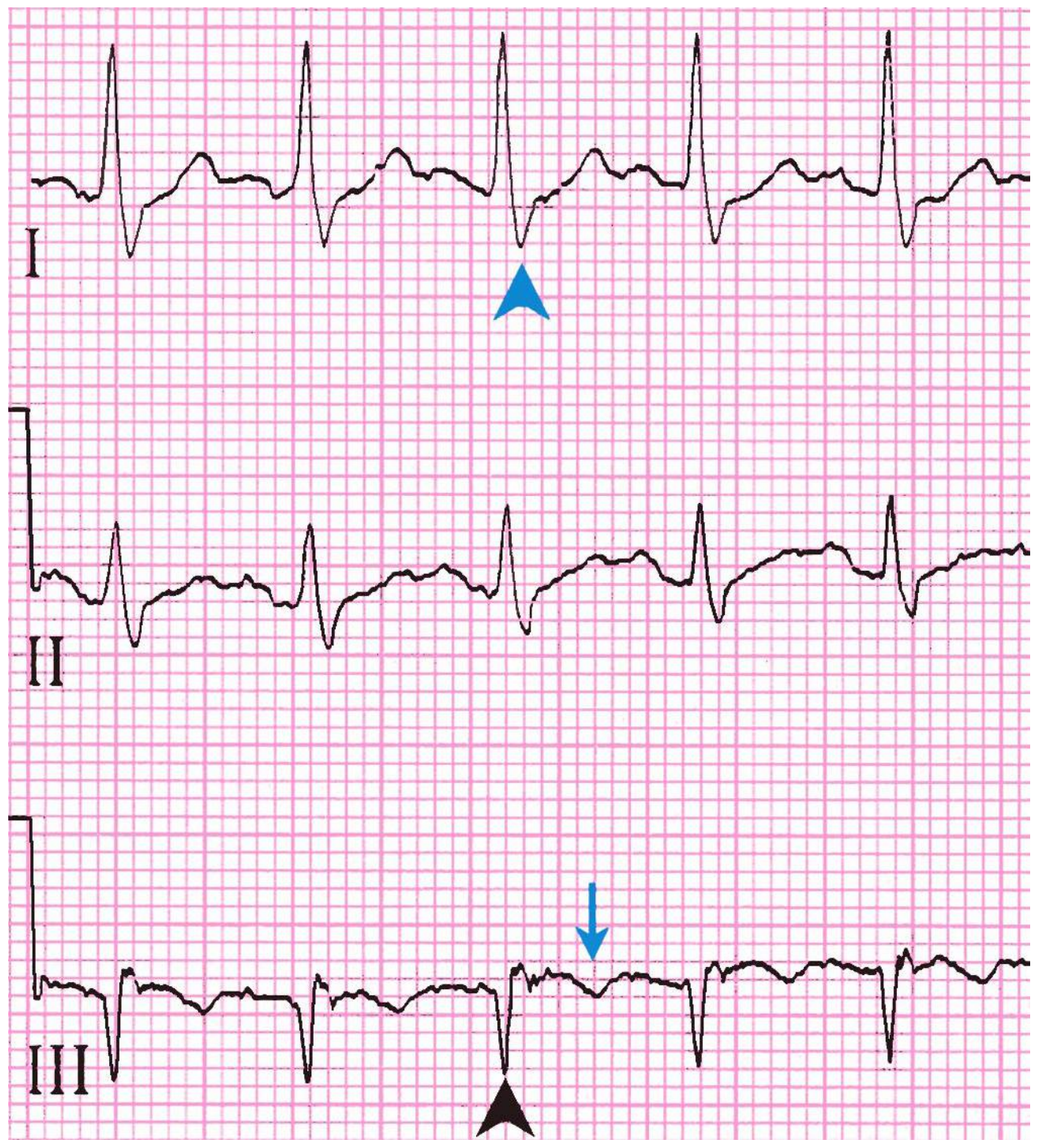


FIGURE 23.48B ■ S wave is apparent in lead I (blue arrowhead), Q wave in lead III (black arrowhead), and inverted T wave in lead III (blue arrow).

PERICARDIAL EFFUSION

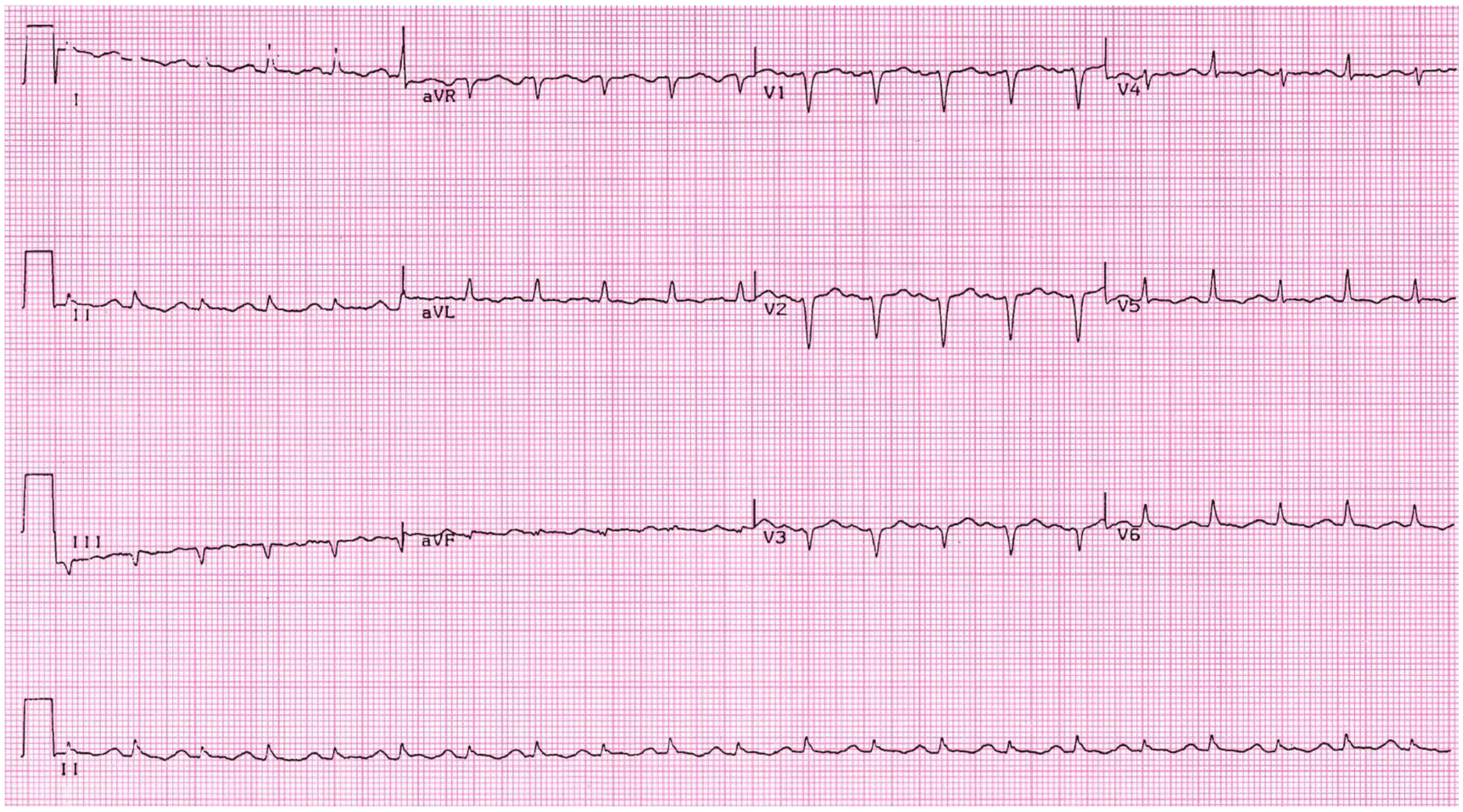


FIGURE 23.49A ■ **Pericardial Effusion with Electrical Alternans.** (ECG contributor: Kevin E. Zawacki, MD.)

ECG Findings

- Sinus tachycardia
- Low voltage of QRS complex (QRS averaging <5 mm height in limb leads, or <10 mm height in precordial leads)
- Electrical alternans (beat-to-beat change in electrical axis and/or amplitude of the QRS complex)

Pearls

1. A physiologically significant pericardial effusion compresses the heart, and affects the ability of the heart to fill properly. This typically results in a reflex sinus tachycardia to maintain cardiac output.
2. Pericardial effusion may be caused by pericarditis, malignancy, uremia, trauma, iatrogenic injury (CVL placement), aortic dissection with retrograde involvement of the pericardium, and free wall rupture after a myocardial infarction.
3. Initial treatment of physiologically significant pericardial effusion is with intravenous fluid bolus to increase preload. Pericardiocentesis should be reserved for hemodynamically threatening effusions due to a high associated morbidity. Surgical pericardial window may be necessary, especially in malignant effusions.
4. Electrical alternans is an uncommon finding. Pericardial effusion should be suspected in the setting of a sinus tachycardia and low voltage.

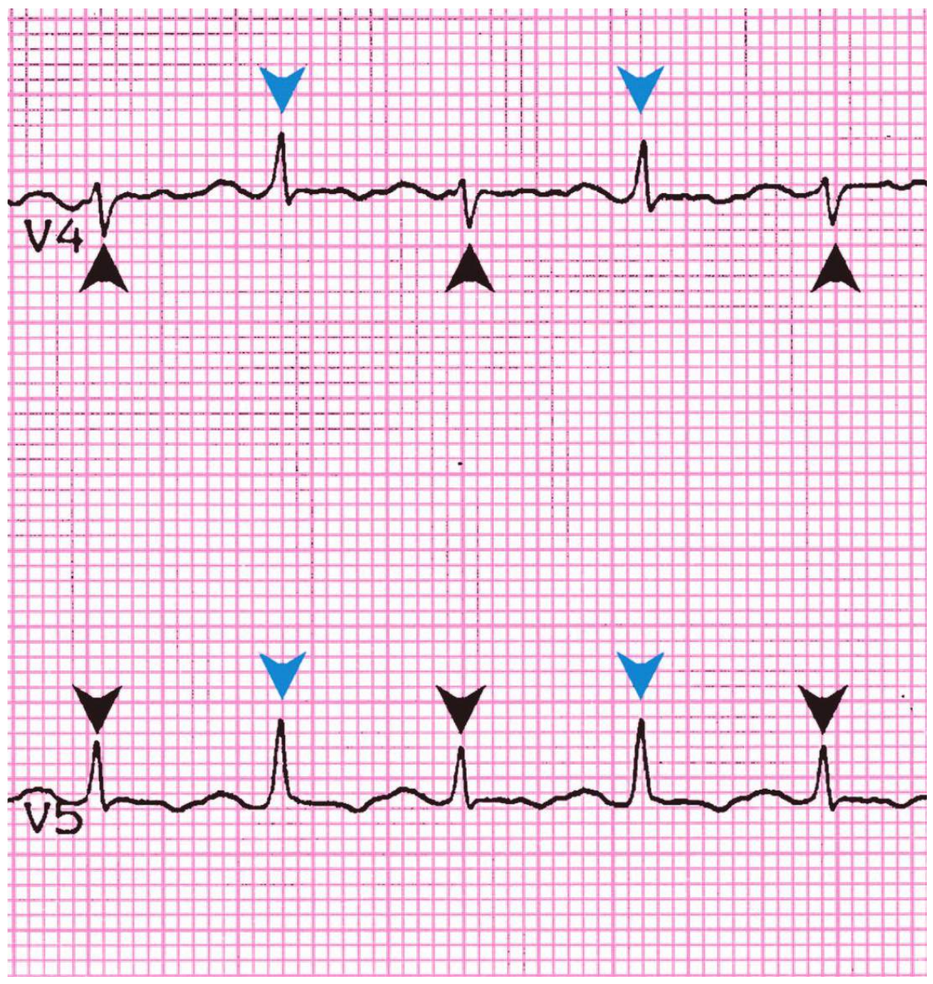


FIGURE 23.49B ■ Low voltage, sinus tachycardia, electrical alternans (arrowheads) demonstrate beat-to-beat alternating QRS electrical axis and/or amplitude. Electrical alternans is often best seen in the anterior precordial leads V_3 and V_4.

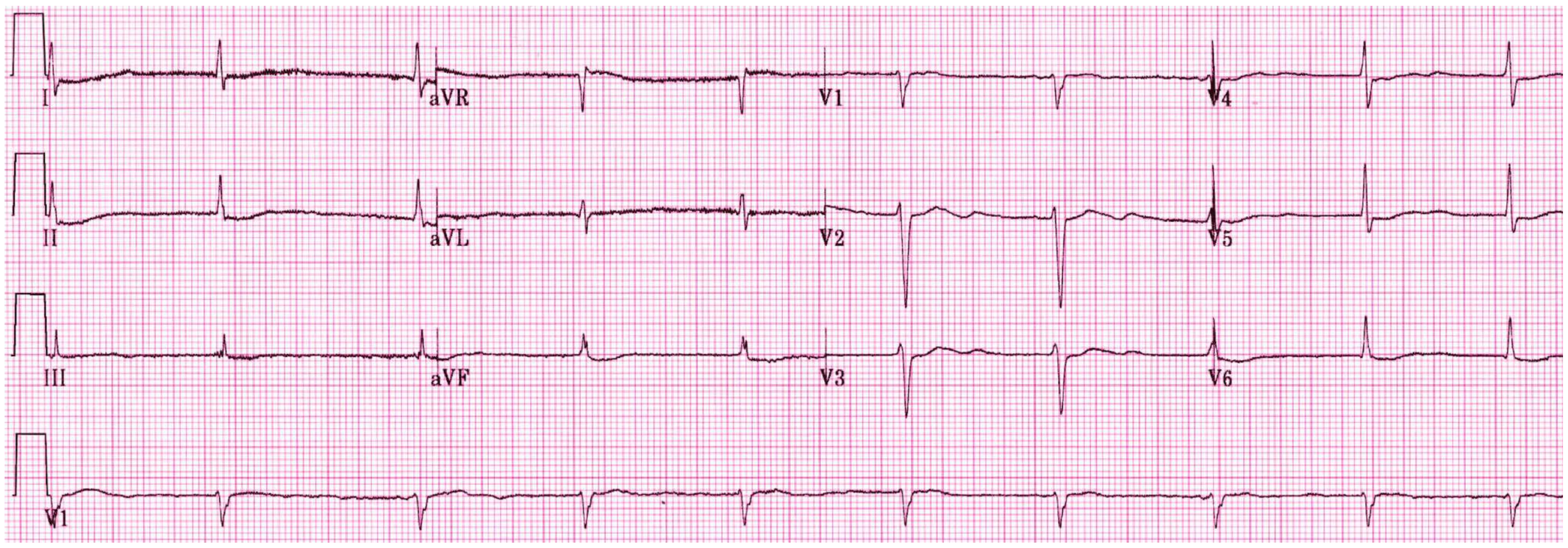


FIGURE 23.50A ■ **Digoxin Effect with Evidence of Toxicity.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment shortening and depression leading to a "scooped" appearance.
- QT interval shortening.
- PR interval prolongation.
- Decreased T-wave amplitude.
- Premature ventricular complexes are the most common dysrhythmia.
- Bradydysrhythmias, various heart blocks, especially with findings consistent with increased automaticity (atrial tachycardia with block, atrial fibrillation with slow ventricular response, accelerated junctional rhythms).
- Bidirectional ventricular tachycardia may rarely be seen (see Fig. 17.72).

Pearls

1. ECG changes associated with digoxin can be seen from therapeutic or toxic levels.
2. ST-segment changes may be exaggerated by myocardial disease or tachycardia.
3. An acute overdose of a digoxin is usually associated with hyperkalemia which may increase the height of the T wave.
4. Avoid calcium for treatment of hyperkalemia in the setting of digoxin toxicity as this may potentiate some adverse effects of digoxin.
5. A digoxin overdose can lead to almost any dysrhythmia, but it commonly blocks the transmission of impulses through the AV node leading to bradycardic rhythms and accelerated escape rhythms.

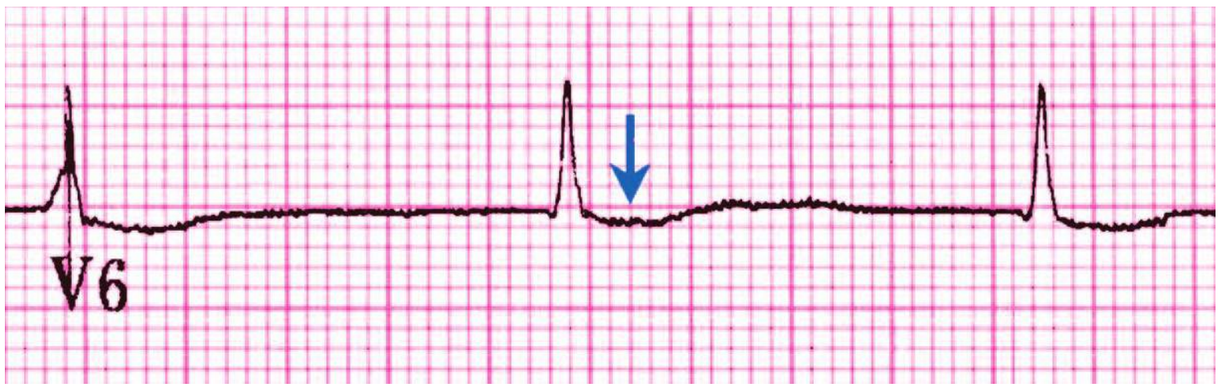


FIGURE 23.50B ■ The "sagging" appearance of the ST segment (arrow) is characteristic of digoxin therapy, and is not a sign of toxicity. However, this patient also has a sign of chronic digoxin toxicity. Atrial fibrillation is present, but the R-to-R interval has become regular. Digoxin toxicity has produced a total AV block but has also excited the AV node, producing a relatively accelerated junctional escape rate.

TRICYCLIC ANTIDEPRESSANT EFFECT

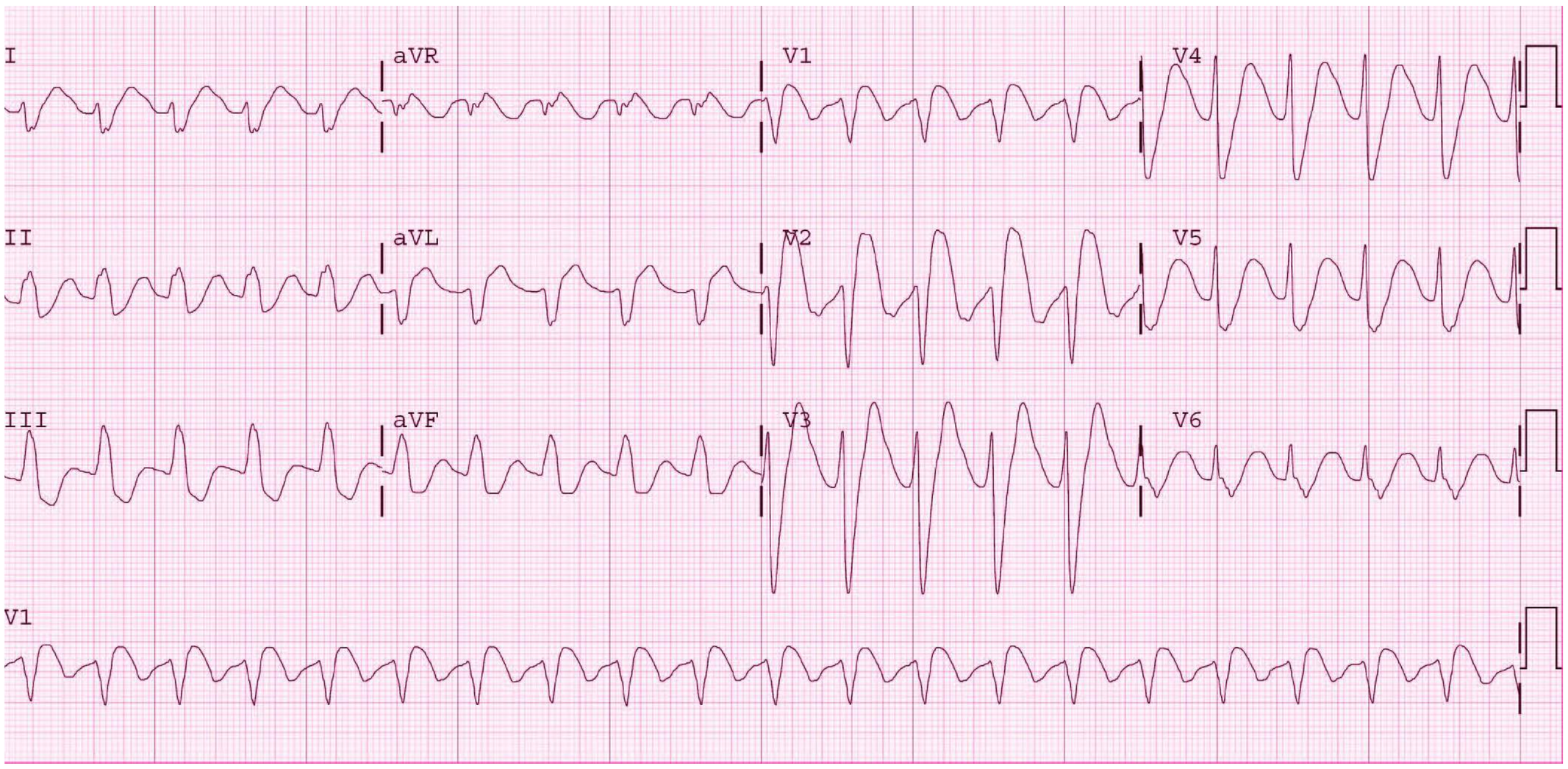


FIGURE 23.51A ■ **Tricyclic Antidepressant Toxicity.** (ECG contributor: Saralyn R. Williams, MD.)

ECG Findings

- Tachycardia
- QRS complex widening
- QT prolongation
- Prominent terminal R wave in aVR or V_1
- Prominent S in Lead I

Pearls

1. Tricyclic antidepressants (TCAs) produce their effects by several mechanisms. Anticholinergic effects may induce tachycardia and sodium channel blockage may lead to QRS widening.
2. The QRS widening seen in a TCA overdose has a nonspecific pattern and is typically unlike any BBB morphology.
3. ECG effects are rate-dependent and become more pronounced with tachycardia and acidosis.

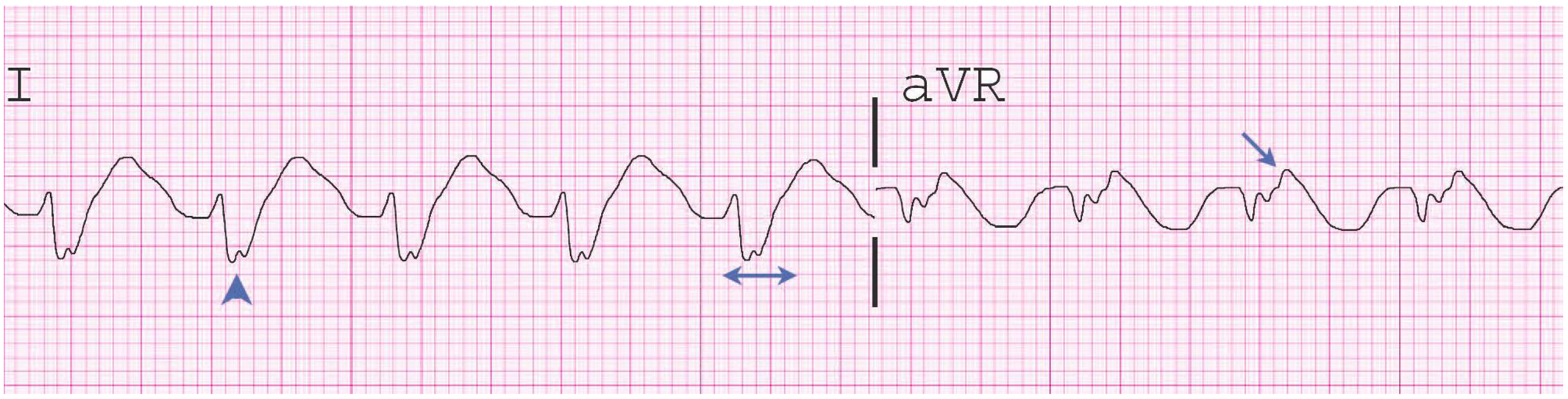


FIGURE 23.51B ■ Prominent S wave in lead I (arrowhead) with prominent terminal R wave in aVR (arrow). The QRS complex is wide (double arrow), the QT interval is prolonged, and the patient is tachycardic.

LIMB LEAD REVERSAL

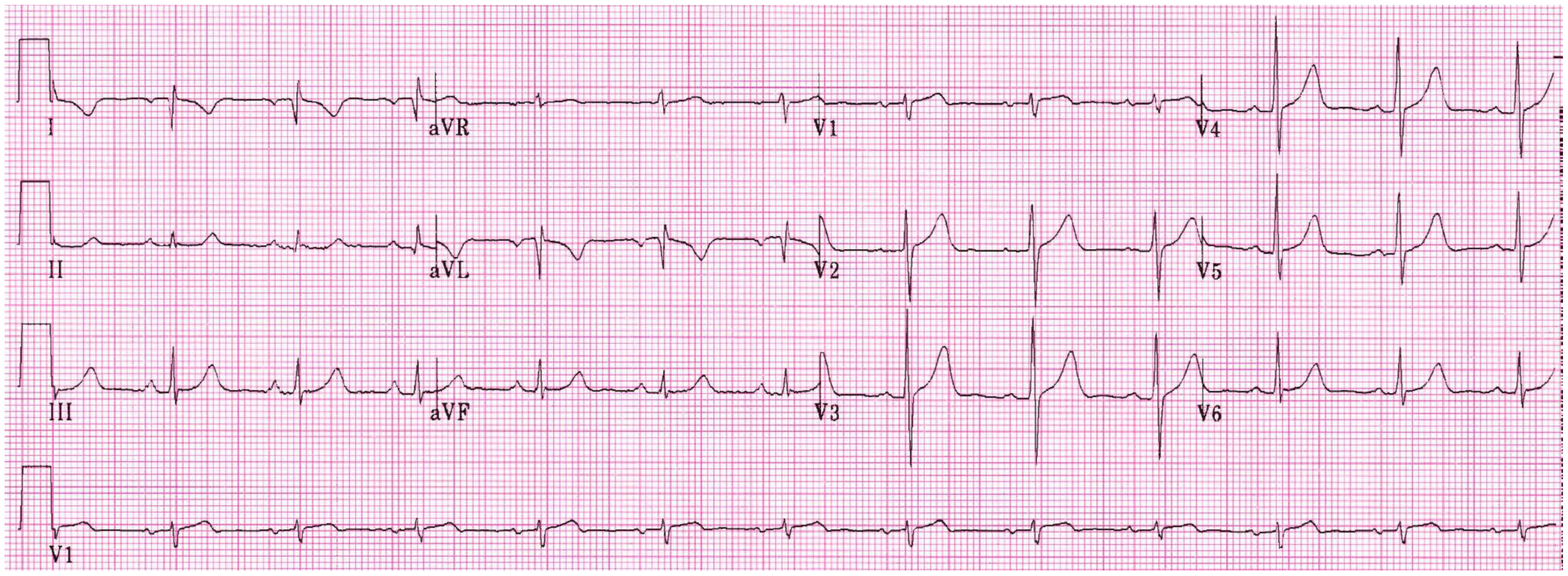


FIGURE 23.52A ■ **Limb Lead Reversal.** (ECG contributor: Michael L. Juliano, MD.)

ECG Findings (dependent on which leads are reversed)

- Reversal of the left arm (LA) and right arm (RA) most common
- P, QRS, and T predominantly downgoing in lead I
- P, QRS, T upgoing in lead aVR
- Precordial leads unaffected
- Reversal of the leg leads (left leg [LL] and right leg [RL])
- Does not commonly produce EKG changes because RL is used as a grounding electrode
- Reversal of LA-LL
- Transposition of leads I and II and leads aVF and aVL with reversal of lead III
- Reversal of RA-RL
- Transposition of aVR and aVL and inversion of lead II
- Incorrect precordial lead placement
- Isolated reversal of the usual R-wave progression from V_1 to V_6

Pearls

1. If the ECG seems to have an unusual axis or appearance, especially when compared with a prior ECG on the same patient, consider a lead misplacement and repeat the tracing, confirming correct lead positions.
2. A "reversed" lead I with normal-appearing V leads strongly suggests accidental limb lead reversal as opposed to dextrocardia. Dextrocardia features a "reversed" lead I, while QRS deflections in V_4 to V_6 appear small and downgoing.

FIGURE 23.52B ■ The P wave, QRS, and T wave are inverted in lead I in this EKG. Normal-appearing V leads in the 12-lead ECG above suggest limb lead reversal rather than dextrocardia. The arm leads were indeed reversed, and correction produced a normal-appearing tracing.

LOW VOLTAGE

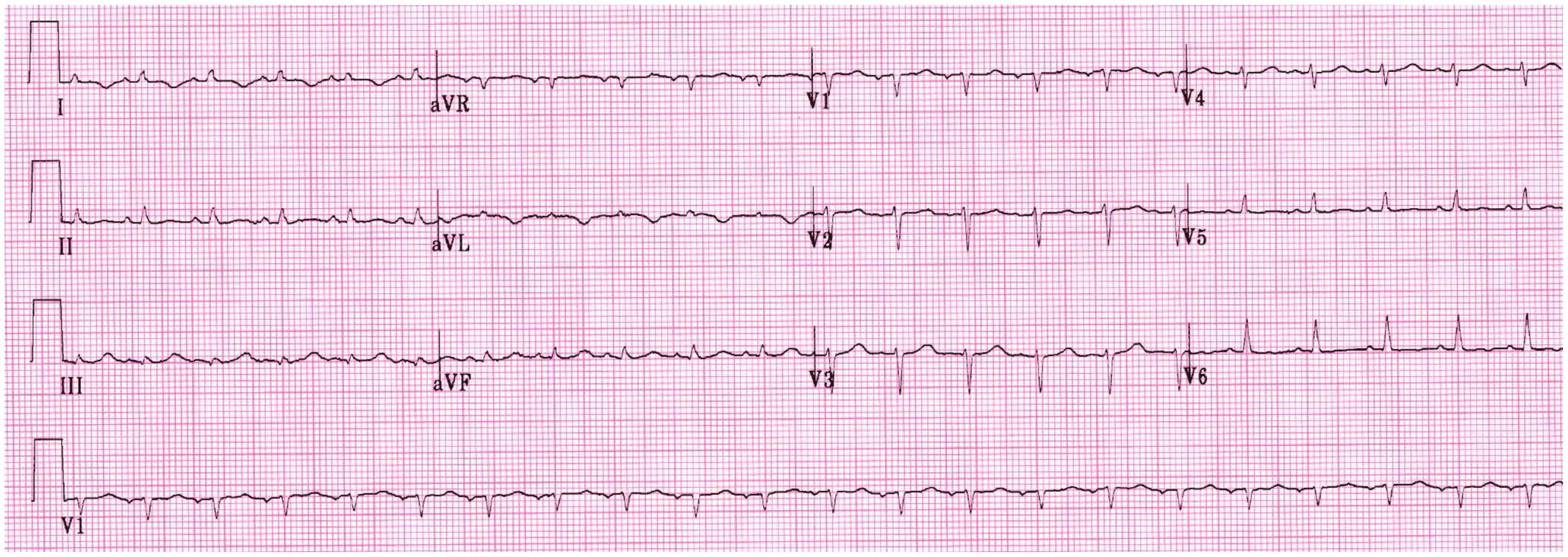


FIGURE 23.53A ■ **Low-Voltage EKG.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- QRS amplitude of less than or equal to 5 mV in all limb leads or a sum of all limb lead QRS amplitude less than or equal to 30 mV and/or QRS amplitude less than or equal to 10 mV in all precordial leads

Pearls

1. Differential diagnosis includes normal variant, low standardization of the ECG machine, pericardial or pleural effusion, obesity/anasarca, COPD/emphysema, cardiac infiltrate (tumor, amyloid), myocardial infarction, myocarditis, cardiomyopathy, adrenal insufficiency, or hypothyroidism.
2. Always check the calibration markings at the left of the ECG to check for low standardization of the ECG machine as an etiology for the observed tracing.

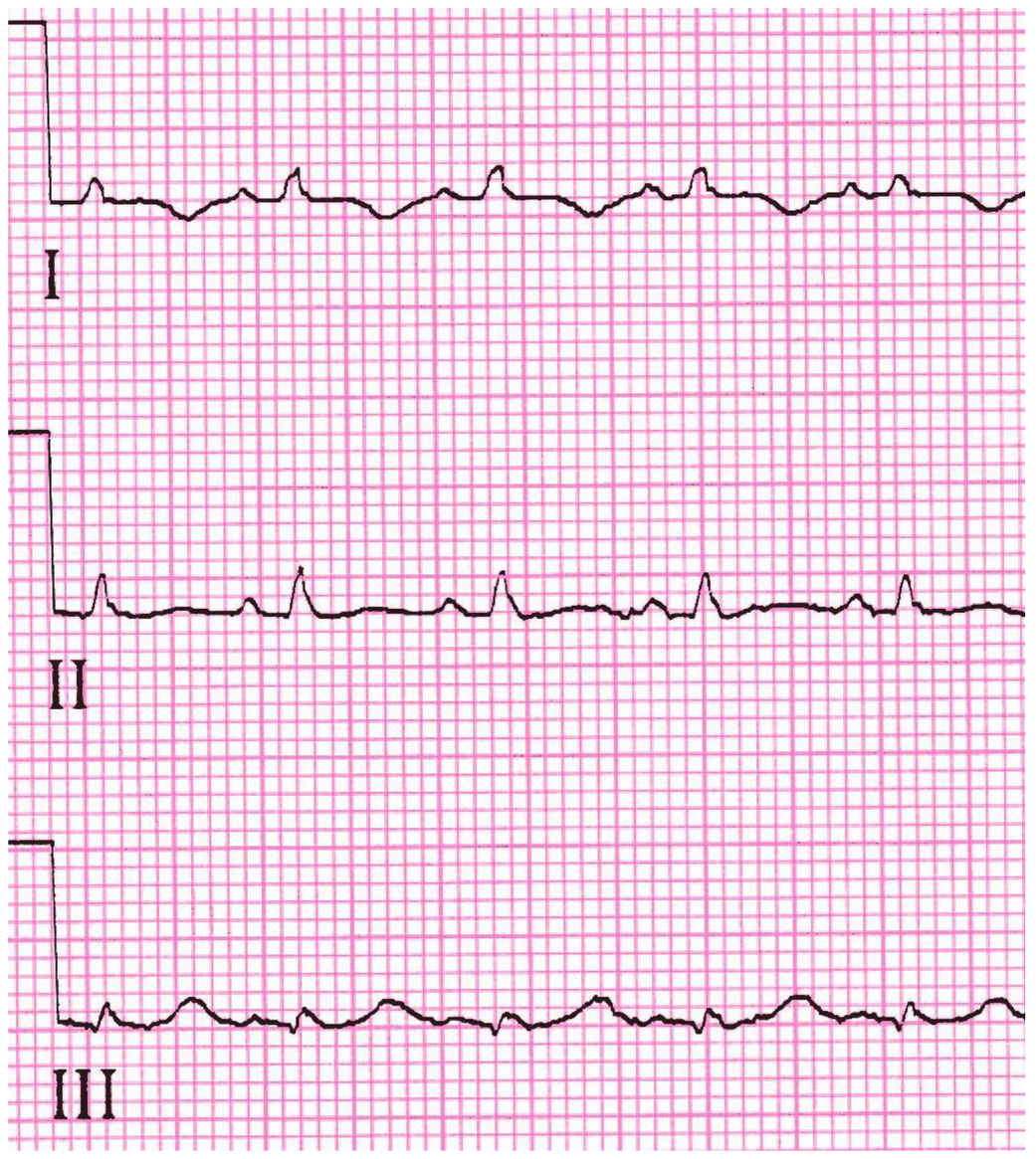


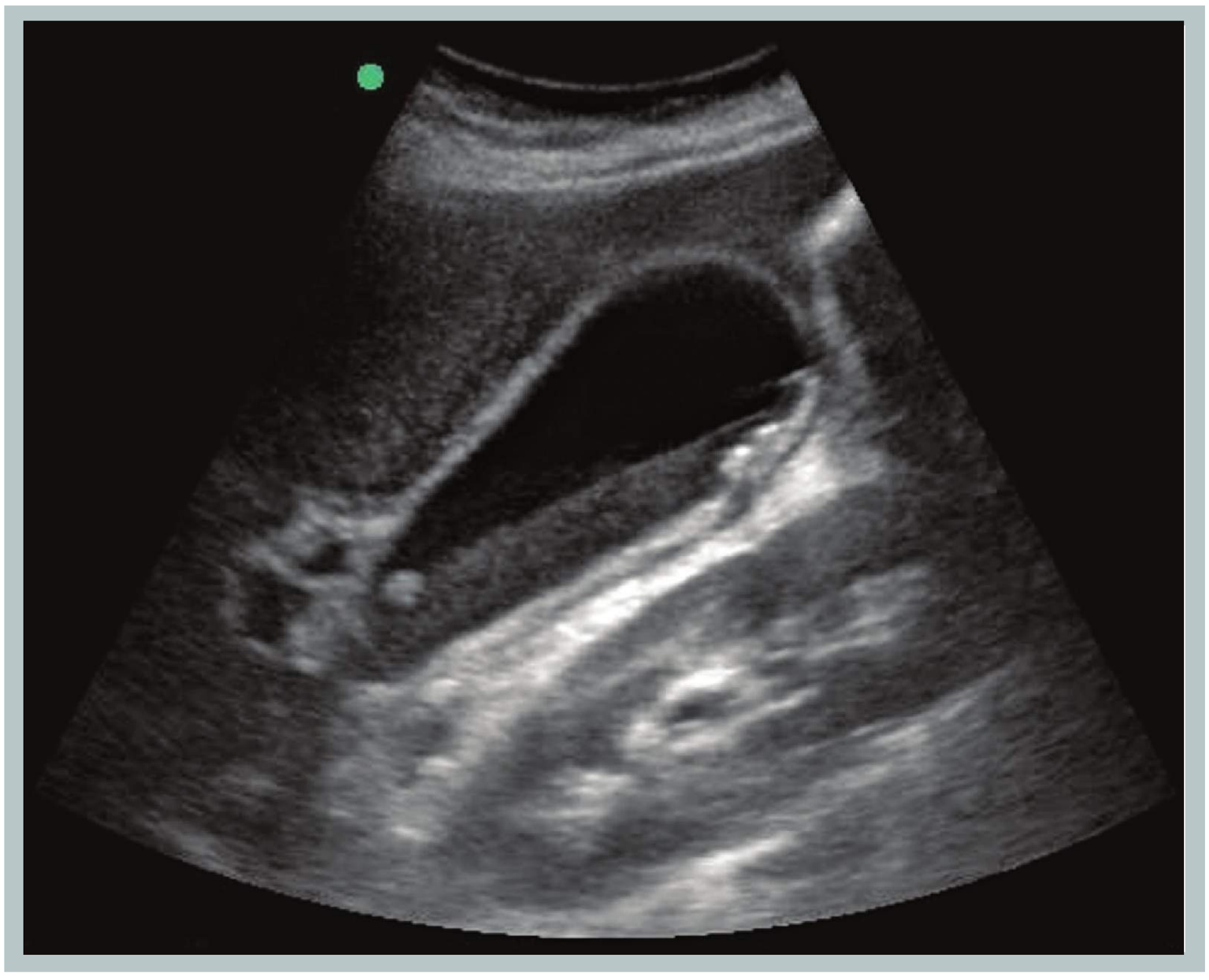
FIGURE 23.53B ■ QRS height is less than 5 mm in limb leads in this normally calibrated tracing.

Chapter 24

EMERGENCY ULTRASOUND

Jeremy S. Boyd
Jordan D. Rupp
Robinson M. Ferre

Scan QR code or go to www.ematlas.com for video related to chapter



Acute Cholecystitis. Longitudinal image of a gallbladder demonstrating gallstone, biliary sludging, and pericholecystic fluid. (Ultrasound contributor: Jeremy S. Boyd, MD.)

EMERGENCY ULTRASOUND: INTRODUCTION

Emergency ultrasound (EUS) is the use of point-of-care ultrasound by clinicians in the emergency department (ED) to answer focused clinical questions and/or assist in performing invasive procedures. It is not an extension of the physical exam by means of advanced technology, but rather uses ultrasound technology to sonographically assess patients for the presence or absence of pathologic conditions that commonly present to the ED. The use and scope of EUS has grown and evolved since the FAST exam was first introduced as part of the evaluation of the trauma patient. For more than a decade, EUS has been a required component of Emergency Medicine Residency training with additional training offered as part of a dedicated EUS fellowship programs. These dedicated EUS fellowship training programs have surged in recent years to meet the demand, with over 90 such programs currently operating.

EUS can be used by clinicians across the spectrum of diseases that present to the ED. Properly trained emergency clinicians use ultrasound to answer focused questions regarding the patient's presenting complaint or condition. Rather than a comprehensive approach, the EUS examination is goal-directed and focused, often seeking to answer dichotomous, "yes/no," questions such as "Is there fluid within the peritoneum?"

This text is not intended to provide an in-depth description of the limitations, efficacy, sensitivity, or appropriateness of EUS. Every application described herein is intended as a rapid visual review for those trained in basic EUS applications. Applicable protocols are based on the imaging guidelines of the American College of Emergency Physicians and American Institute of Ultrasound in Medicine (AIUM), as well as the authors' collective experience. Basic information—transducer recommendations, scanning protocols, anatomic schematics—is presented within each application to represent both image acquisition and normal and pathological findings.

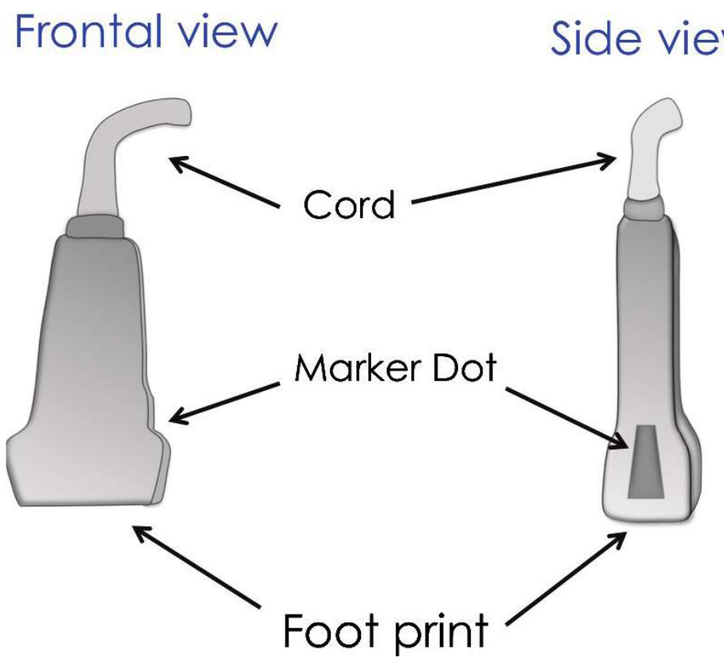


FIGURE 24.1 ■ Probe anatomy demonstrating location of transducer indicator/probe marker/marker dot and foot print. (Illustration courtesy of Robinson M. Ferre, MD.)

Key Terms

Sonographic windows—Anatomical locations on the body where an ultrasound probe is placed in order to view internal organs.

Transducer indicator/probe marker/marker dot—Usually a bump or ridge on the ultrasound transducer that corresponds to a symbol on the ultrasound screen. Used to indicate which side of the image corresponds with the transducer's edge (Fig. 24.1).

Foot print—The part of the ultrasound probe that contacts the patient's skin (Fig. 24.1).

Hyperechoic/echogenic—Used to describe objects on the ultrasound screen that are bright and therefore reflect sound waves.

Hypoechoic/anechoic—Used to describe objects on the ultrasound screen that are dark and therefore transmit sound waves.

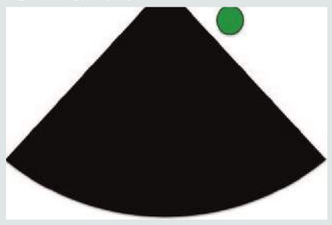
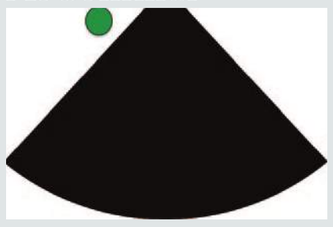
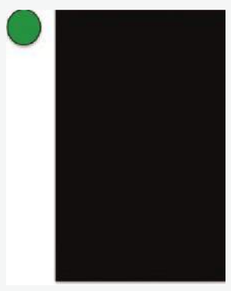
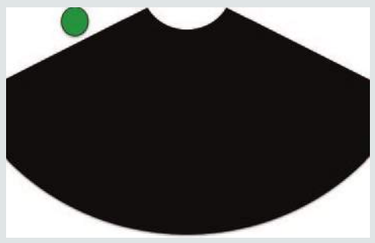
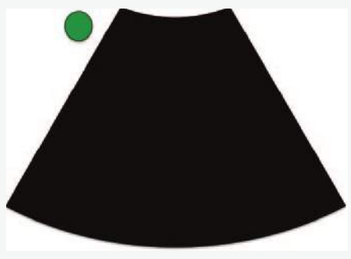
Echotexture—The characteristic appearance of specific organs when viewed using ultrasound. The liver has a characteristic echotexture.

Transducer movements—When performing sonography, specific terms are used to describe characteristic movements of the transducer used to obtain the image desired. Some examples of this include dragging, rocking, fanning, and rotating (Fig. 24.2).

Transducers

Emergency sonography is performed using a wide variety of transducers. Lower frequency transducers are used to visualize structures deep within the body, while higher frequency transducers allow better image resolution of superficial anatomic structures. The size and shape of transducers are also configured for imaging specific anatomic locations. Table 24.1 lists the basic transducer types frequently used in EUS.

TABLE 24.1 ■ TRANSDUCER, APPEARANCE, AND APPLICATIONS

Transducer	Image Appearance	Applications*
Phased array FIGURE 24.3A • Small footprint for imaging between ribs • Lower frequency (1-5 MHz) = better penetration, less resolution • Wide far-field view (wedge-shaped window)	Cardiac  FIGURE 24.3B Abdominal  FIGURE 24.3C	(+) Cardiac, abdominal, thoracic (−) Soft tissue, pregnancy, vascular
Linear array FIGURE 24.4A • Flat footprints in various sizes • High frequency (5-13 MHz) = better resolution of superficial structures • Rectangular window (at right)	 FIGURE 24.4B	(+) Procedural, vascular, soft tissue/MSK, superficial, thoracic (pleural), ocular (−) Abdominal, cardiac, pregnancy
Endocavitary probe FIGURE 24.5A • Curved footprint probe for examining internal structures • High frequency (5-8 MHz) = better resolution of nearby structures • Designed for intracavitary use, primarily transvaginal ultrasound • Wide wedge-shaped window (at right)	 FIGURE 24.5B	(+) Transvaginal pregnancy, intraoral (−) Any external exam
Curvilinear/convex array transducer FIGURE 24.6A • Wide, curved footprint with large surface area decreases discomfort with applied pressure to the abdomen • Lower frequency 2-5 MHz (but with better detail than phased array probe) • Wide wedge-shaped window (at right)	 FIGURE 24.6B	(+) Aorta, abdominal, transabdominal pregnancy (−) Cardiac

*(+) = best use; (-) = not ideal

(Photo contributor: Lawrence B. Stack MD, Illustrations courtesy of Robinson M. Ferre, MD.)

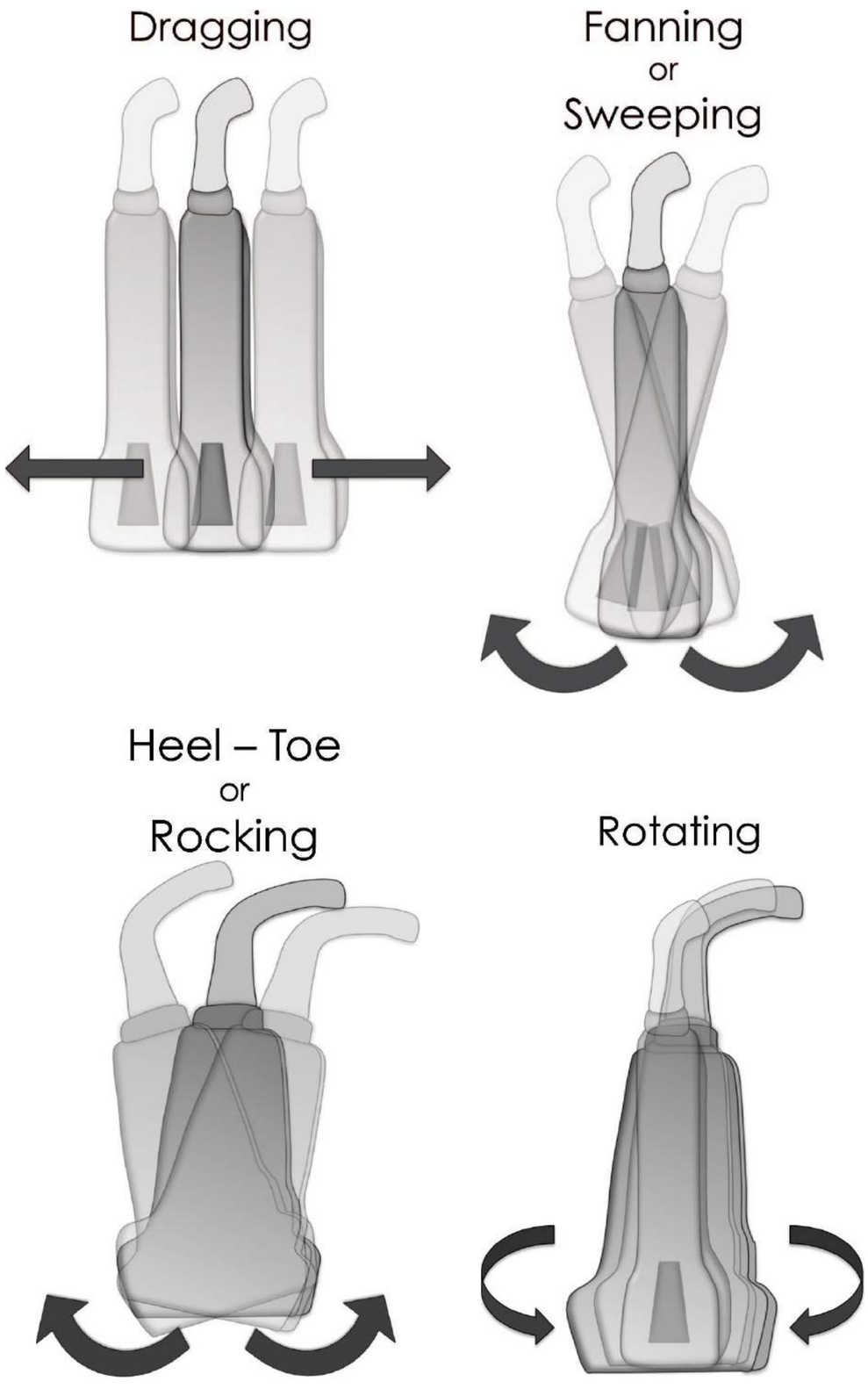


FIGURE 24.2 ■ **Transducer Movements**. (Illustration courtesy of Robinson M. Ferre, MD.)

Clinical Summary

The Extended Focused Assessment with Sonography in Trauma (E-FAST) is a protocolized series of sonographic views that attempt to identify the presence or absence of fluid or air in anatomic potential spaces such as the pericardium, the peritoneum, and the thorax. The goal of this thoracoabdominal survey is to identify or exclude immediate life threats in the trauma or critically ill patient. Though initially intended for the evaluation of the trauma patient, the E-FAST examination and its component views are also extremely valuable in the evaluation of several emergent complaints and clinical conditions including the patient with undifferentiated hypotension.

Indications

- Trauma (blunt and penetrating)
- Unexplained hypotension (traumatic and nontraumatic)
- Ectopic pregnancy (to evaluate for rupture)

The FAST examination uses four primary sonographic views to evaluate the patient. It is recommended that all four views are evaluated for a complete exam, but isolated views may be obtained when indicated. The extended component of the FAST exam (termed E-FAST) incorporates imaging of both the anterior and lateral hemithoraces to identify the presence or absence of a pneumothorax or hemothorax (Fig. 24.7). It is important to note that these are not static "single" views, but a series of images obtained in each plane as the transducer is moved or "fanned" through the area of interest.

Views for the E-FAST Examination

1. Pericardial (usually a subcostal/subxiphoid view—alternatively, one may obtain a parasternal long view of the heart if a subxiphoid view is unobtainable)
2. Right upper quadrant (RUQ; pouch of Morison)
3. Left upper quadrant (LUQ) (perisplenic view)
4. Suprapubic (pelvic view)
5. Right thorax
6. Left thorax

Patient Positioning

The E-FAST exam should be done with the patient in the supine position.

Recommended Transducers for the E-FAST Examination

- Convex array (abdominal, cardiac, and lung imaging)
- Phased array (abdominal, cardiac, and lung imaging)
- Linear array (lung only)

The E-FAST examination is a thoracoabdominal examination. Ideally, this is done using a single transducer that can image all three of these areas, but may result in some compromise of image quality and require the use of different probes for different components of the examination.

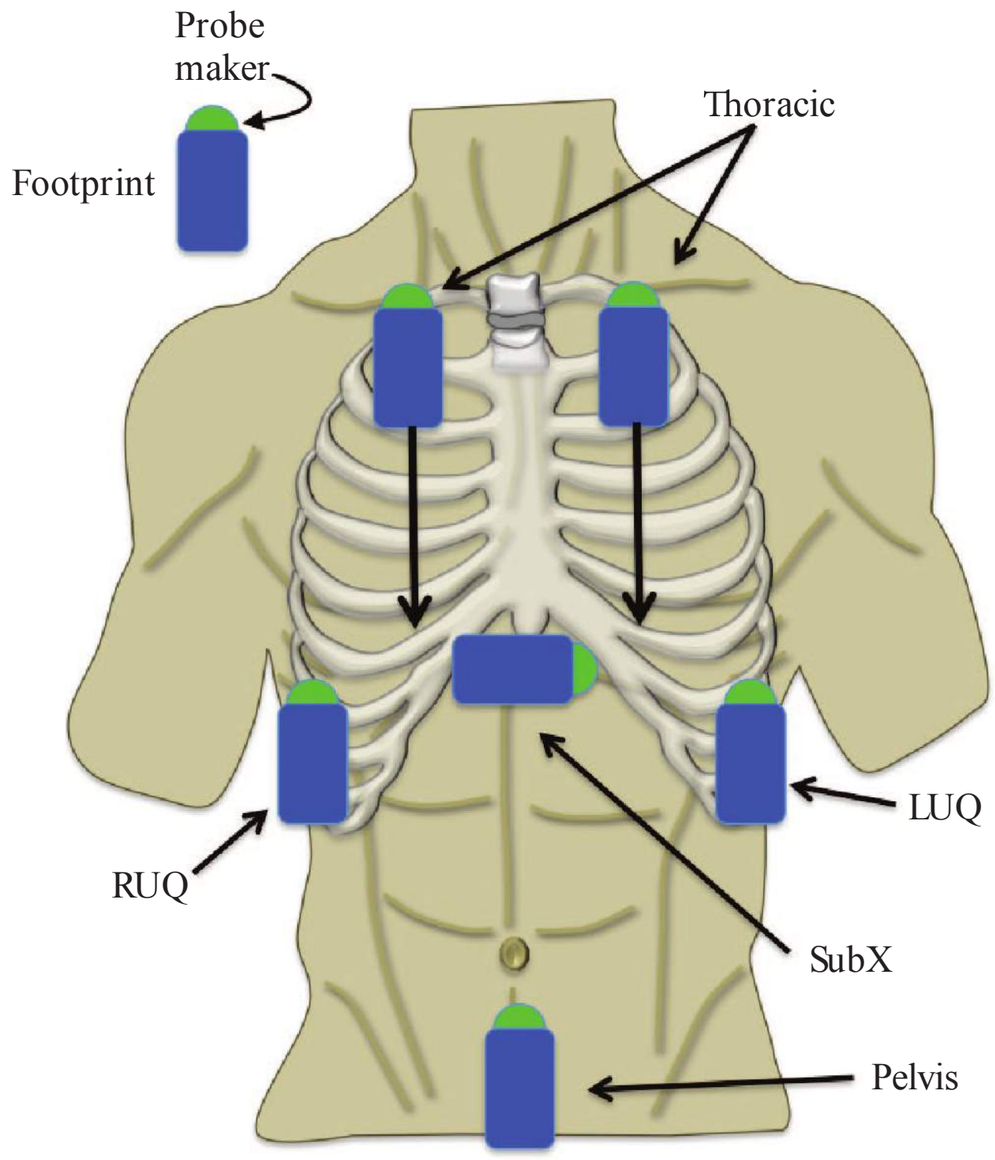


FIGURE 24.7 ■ **E-FAST Trauma Series.** Ultrasound transducer and probe marker positions for evaluation of pericardial fluid, hemoperitoneum, or pneumothorax. (Illustration courtesy of Robinson M. Ferre, MD.)

PERICARDIAL (SUBXIPHOID VIEW)

Technique

- Direct the transducer indicator to the patient's right (in abdominal or general preset). Note that the E-FAST exam is traditionally performed with abdominal preset settings, but the subxiphoid view is often performed with cardiac preset settings as part of a full focused cardiac ultrasound exam. When this is the case, the marker dot is typically on the opposite side of the screen and the indicator is oriented to the patient's left.
- With the transducer indicator pointing to the patient's right, the transducer is directed under the xiphoid process toward the left shoulder in a horizontal plane (Fig. 24.8).
- Pivot, sweep, and tilt the transducer as necessary to view all four cardiac chambers and pericardium.
- Identify the liver (if in view), heart, four cardiac chambers, and surrounding pericardium (Fig. 24.9).

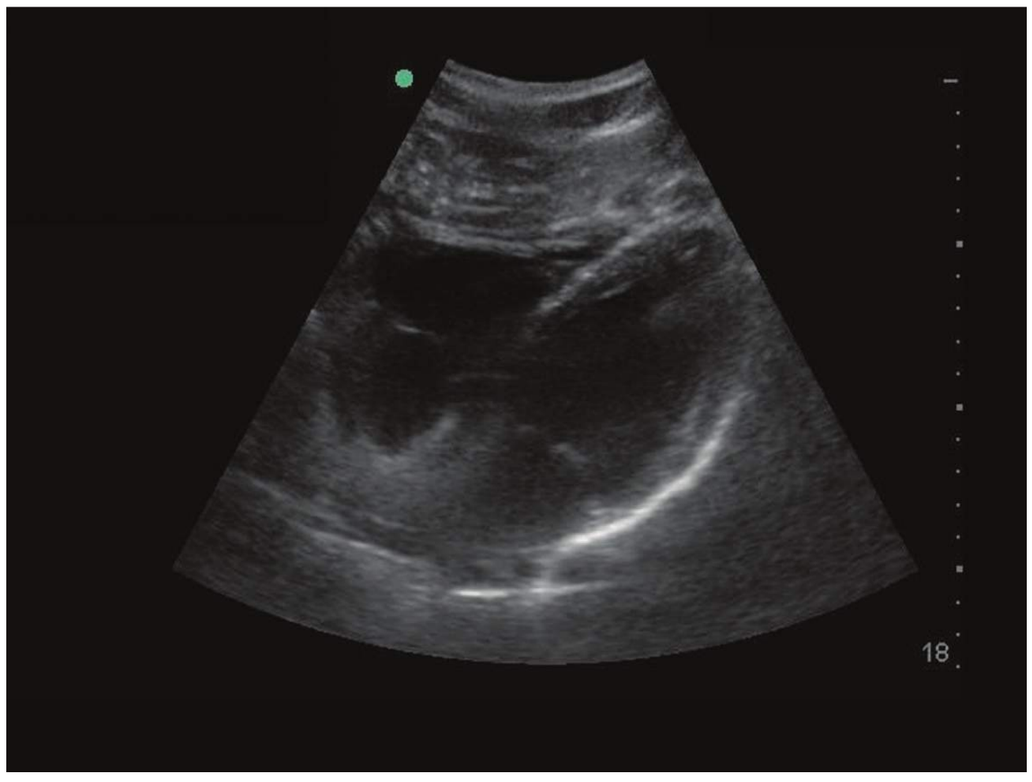
FIGURE 24.8 ■ **Subxiphoid View.** The transducer is directed under the xiphoid process toward the left shoulder in a horizontal plane. (Photo contributor: Lawrence B. Stack, MD.)

Abnormal Findings

- Hemopericardium (pericardial effusion): Pericardial fluid will appear as an anechoic (black) region noted between the pericardium and the right ventricle. As more fluid collects, the fluid will be seen completely surrounding all four chambers of the heart. Occasionally, internal echoes representing fibrin, clot, or cardiac tissue may be present within the pericardial space (Fig. 24.10).
- Asystole: No cardiac activity present.
- Hyperdynamic cardiac activity: Extensive cardiac contraction with near-total or complete collapse of the cardiac chambers, often associated with tachycardia and hypovolemia.

Pearls

1. When the view is obscured by gas, slide the transducer slightly to the patient's right subcostal region, and use the right lobe of the liver as the sonographic window.



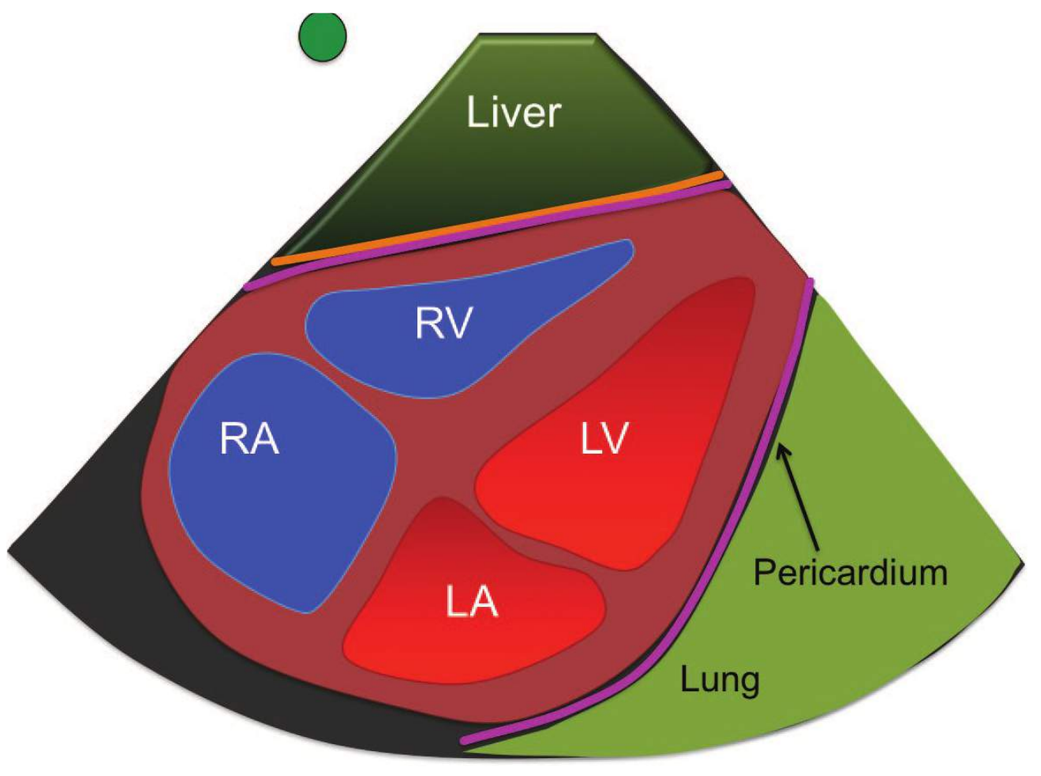


FIGURE 24.9 ■ **Subxiphoid View.** The heart, four cardiac chambers, and surrounding pericardium are seen in this view. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

2. If unable to view the heart in the true subxiphoid or subcostal window, move to a parasternal long-axis view (see "Focused Cardiac Ultrasound" below).
3. A frequent mistake in imaging is to direct the transducer posterior toward the spine rather than cephalad toward the shoulder. You will often require less than a 30-degree angle between the transducer and the skin to view the heart.
4. Start imaging with the depth/scale setting near its maximum (eg, 24-35 cm). This should allow you to image the anterior and posterior pericardium in your initial view. Gradually decrease the depth/scale (eg, 14-18 cm) to fill the entire sector image with the heart as you continue to optimize your image.
5. Hypotensive trauma patients with a pericardial effusion and findings such as right ventricular (RV) collapse and/or a distended inferior vena cava (IVC) warrant emergent pericardiocentesis (see Fig. 24.34).

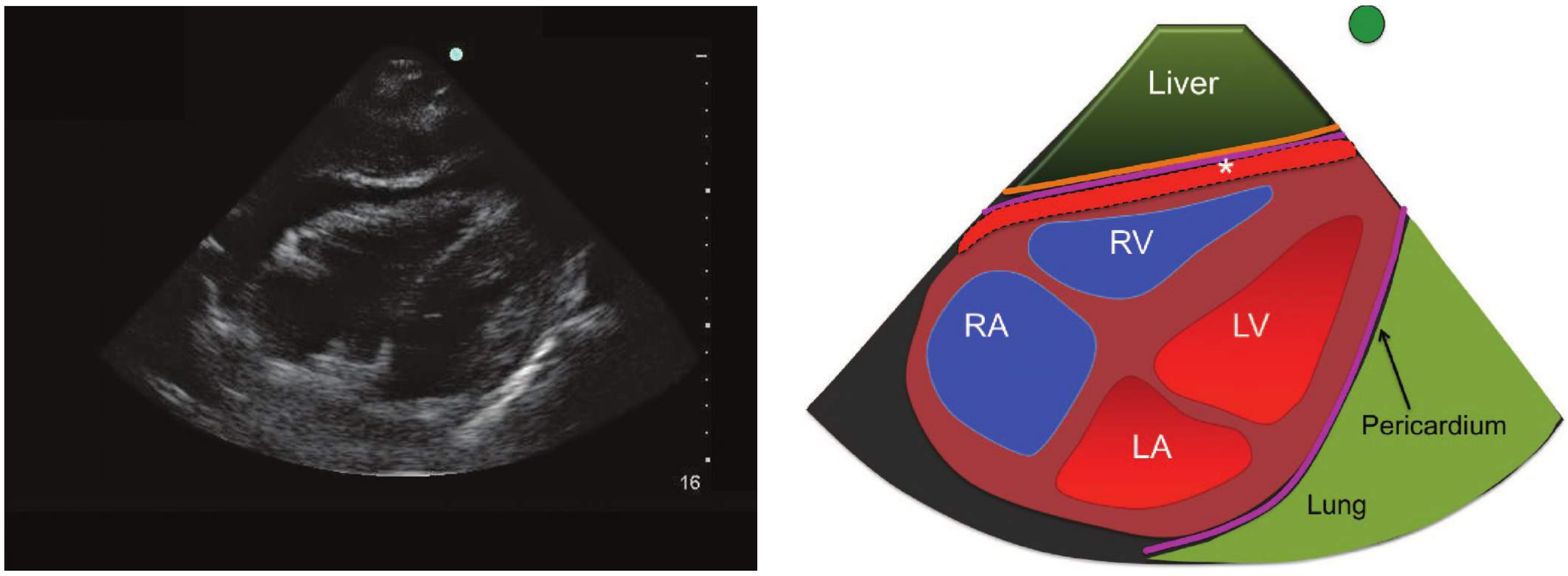


FIGURE 24.10 ■ **Hemopericardium**. Pericardial fluid appears as an anechoice (black) region noted between the pericardium and the right ventricle. Hemopericardium is denoted by the asterisks in the diagram. (Ultrasound contributor: Jim Fiechtl, MD; illustration courtesy of Robinson M. Ferre, MD.)

RIGHT UPPER QUADRANT (POUCH OF MORISON)

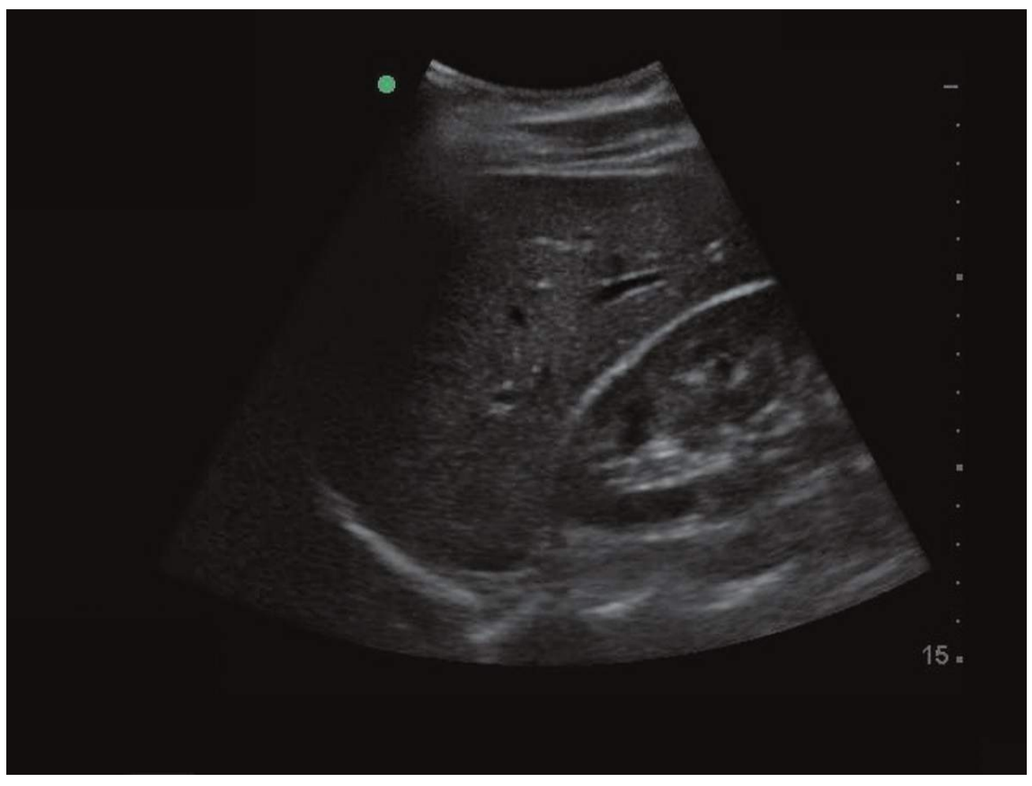
Technique

- With the indicator directed toward the patient's head, the transducer is oriented in a coronal section through the body in the midaxillary line, extending from the 9th through 12th ribs. Start between the 11th and 12th ribs initially, then move cephalad or caudal, anterior or posterior, to complete the evaluation (Fig. 24.11).
- Identify and evaluate the interface of the liver and right kidney. This region is the potential space known as the pouch of Morison. Normally, the liver and kidney are in direct contact with one another or separated by adipose tissue of heterogeneous echoes (Fig. 24.12).
- Evaluate the right paracolic gutter by identifying and evaluating the caudalmost portion of the hepatic parenchyma (inferior tip of the liver).

Abnormal Findings

- Hemoperitoneum: Anechoic (black) region between the liver and right kidney or in the paracolic gutter. Rarely is blood only seen collecting between the diaphragm and the liver in the subdiaphragmatic space (Fig. 24.13).
- Solid organ injury: Solid organ injury such as hepatic and renal lacerations as well as organ rupture have been described but are not the goal of this examination as ultrasound is an insensitive exam for solid organ injury.

FIGURE 24.11 ■ **Right Upper Quadrant View.** The transducer is oriented in a coronal section through the body in the midaxillary line extending from the 9th through 12th ribs. (Photo contributor: Lawrence B. Stack, MD.)



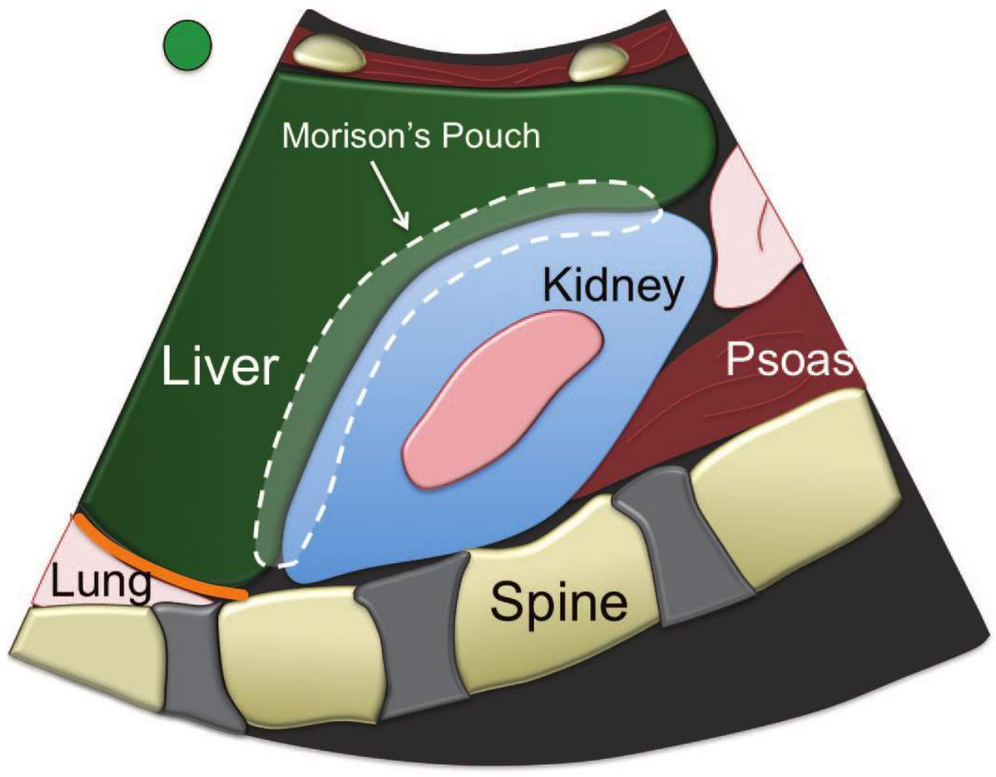
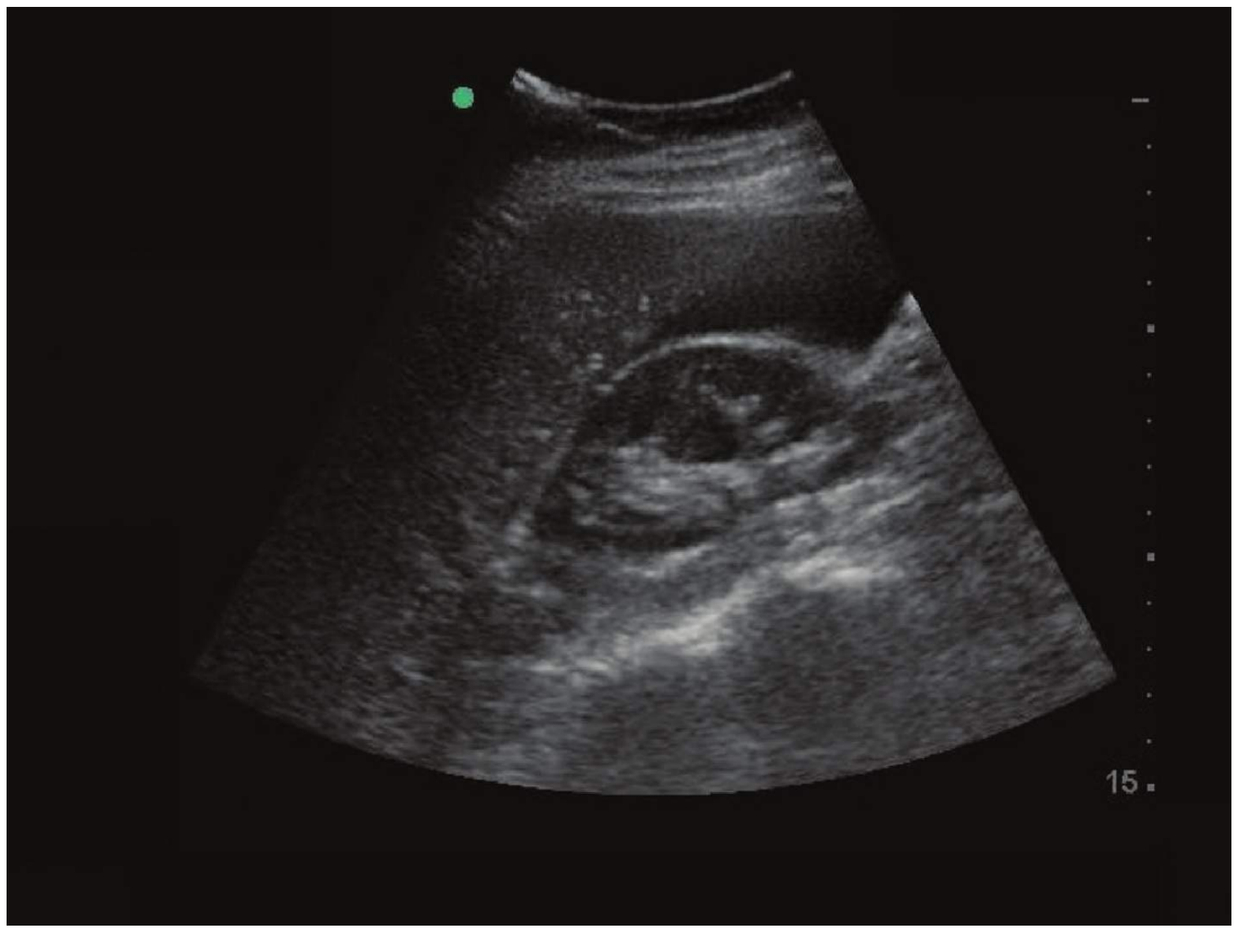


FIGURE 24.12 ■ **Normal Right Upper Quadrant View (Morison Pouch).** Identify and evaluate the interface of the liver and right kidney. This region is the potential space known as the pouch of Morison. Normally, the liver and kidney are in direct contact with one another or separated by adipose tissue of heterogeneous echoes. The reflection of the anterior border of the lumbar spine is visualized deep to the kidney and terminates at the costophrenic angle in the absence of fluid within the thoracic cavity. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)



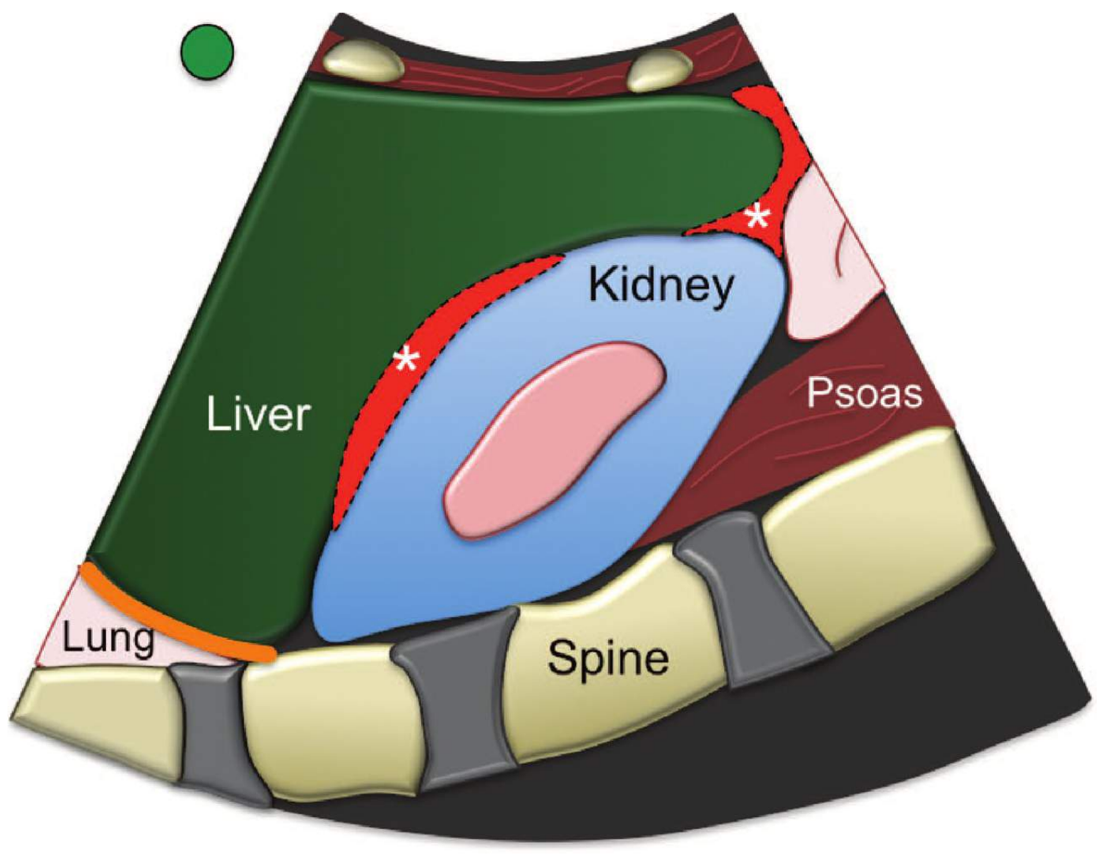


FIGURE 24.13 ■ **Hemoperitoneum (Right Upper Quadrant view).** An anechoic (black) region between the liver and right kidney is fluid (blood in this case) accumulating within the potential space. The diagram illustrates the typical locations of these peritoneal potential spaces, marked by asterisks. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

LEFT UPPER QUADRANT (PERISPLENIC VIEW)

Technique

- The transducer indicator is directed toward the axilla.
- With the indicator pointed toward the patients head, the transducer is oriented in a coronal section through the body in the mid to posterior axillary line extending from the 9th through 12th ribs. Start between the 11th and 12th ribs initially, then move cephalad or caudal, anterior or posterior, to complete the evaluation (Fig. 24.14). As a general rule of thumb, the perisplenic view is more posterior and cephalad than that of the RUQ view.
- Identify and evaluate the area surrounding the spleen, including its upper and lower poles, the interface with the diaphragm, and the interface with the left kidney. Normally, the surrounding tissues of the spleen and kidney are in direct contact with one another (Fig. 24.15).

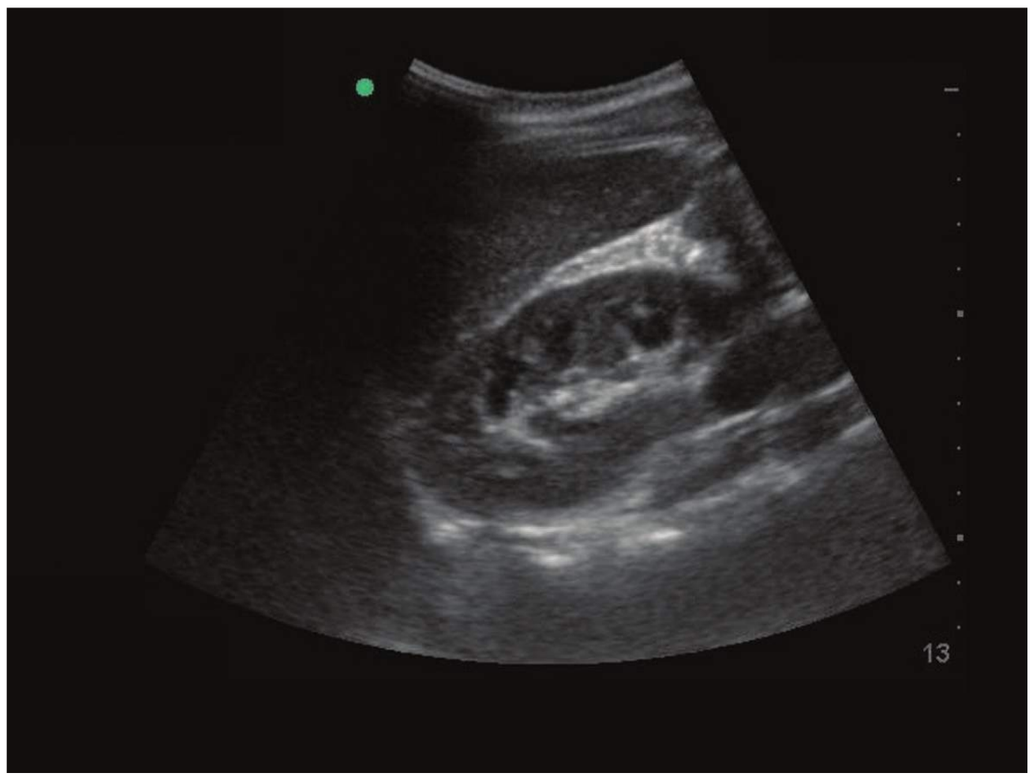
Abnormal Findings

- Hemoperitoneum: Anechoic (black) region around the spleen. This may be visible at the superior or inferior poles of the spleen, between the spleen and the diaphragm or between the spleen and left kidney. Unlike the pouch of Morison, blood cannot flow beyond the inferior pole of the spleen down the paracolic gutter due to the phrenicocolic ligament. Blood that collects in the perisplenic space must first pass out of the lesser peritoneal sac and into the greater peritoneal sac before it will be seen collecting along the left paracolic gutter (Fig. 24.16).
- Solid organ injury: Solid organ injury such as splenic and renal lacerations as well as organ rupture have been described but are not the goal of this examination as ultrasound is an insensitive exam for solid organ injury.

Pearls for RUQ and LUQ Views

1. The diaphragmatic recess includes a superior region, which is the inferior border of the right thorax (often referred to as the costophrenic angle), and an inferior region (subdiaphragmatic recess), which is the superior border of the abdomen. Fluid in the diaphragmatic recess

FIGURE 24.14 ■ **Left Upper Quadrant View.** The transducer is oriented in a coronal section through the body in the mid to posterior axillary line extending from the 9th through 12th ribs. (Photo contributor: Lawrence B. Stack, MD.)



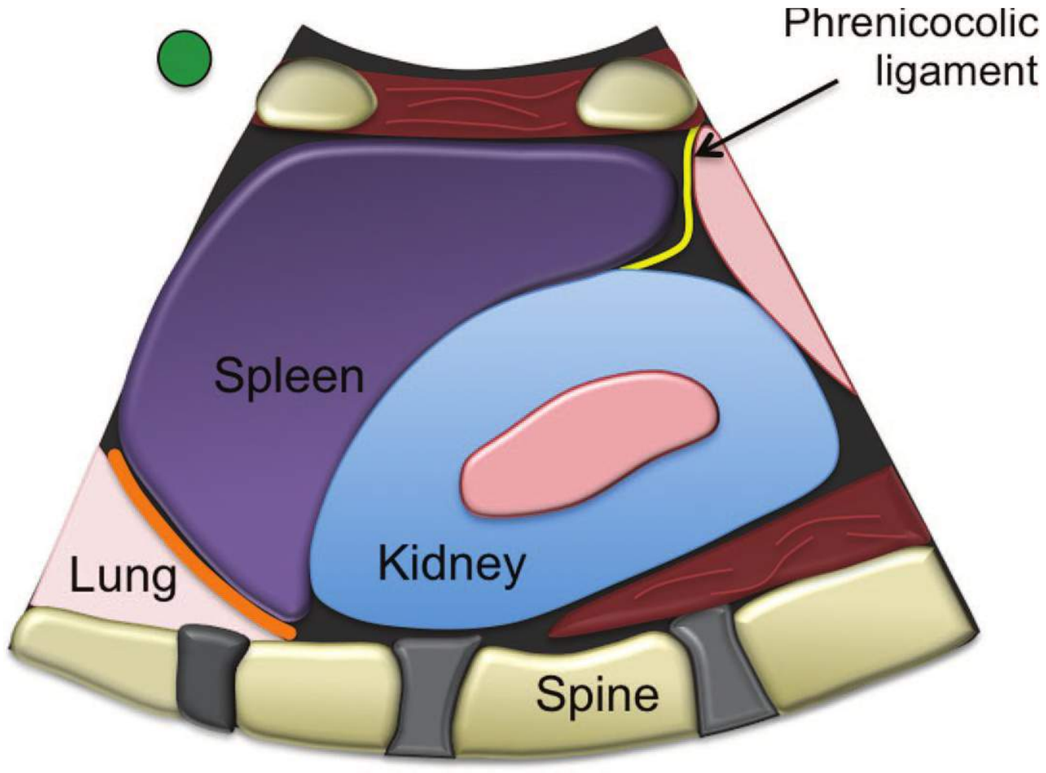
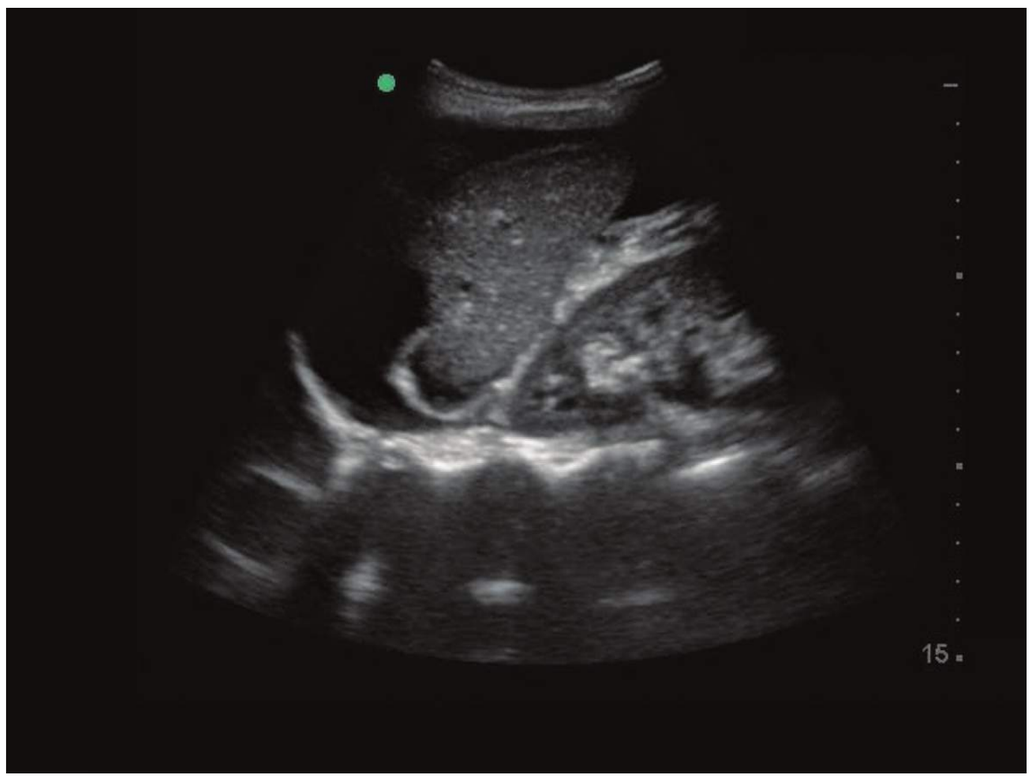


FIGURE 24.15 ■ **Normal Left Upper Quadrant View.** The region between the spleen and left kidney is a physiologic potential space (splenorenal recess); however, due to the phrenicocolic ligament (rarely seen on ultrasound but shown on illustration), free fluid is often shunted toward the subdiaphragmatic space between the diaphragm (orange line) and spleen. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

can represent a hemothorax when located superior/cephalad to the diaphragm or a hemoperitoneum (inferior to the diaphragm) in the setting of trauma.

2. If you are uncertain whether a finding is real or artifact, evaluate it in a second plane. Turn the transducer 90 degrees from your initial transducer position to see if the finding is still noted on the image. If the entire image is unchanged, it is less likely to be an artifact.
3. The liver is affixed to the diaphragm via the coronary ligament but the spleen lacks a similar attachment. Dependent fluid collects in the left subdiaphragmatic area more often than the right and this space should be evaluated sonographically during the examination.



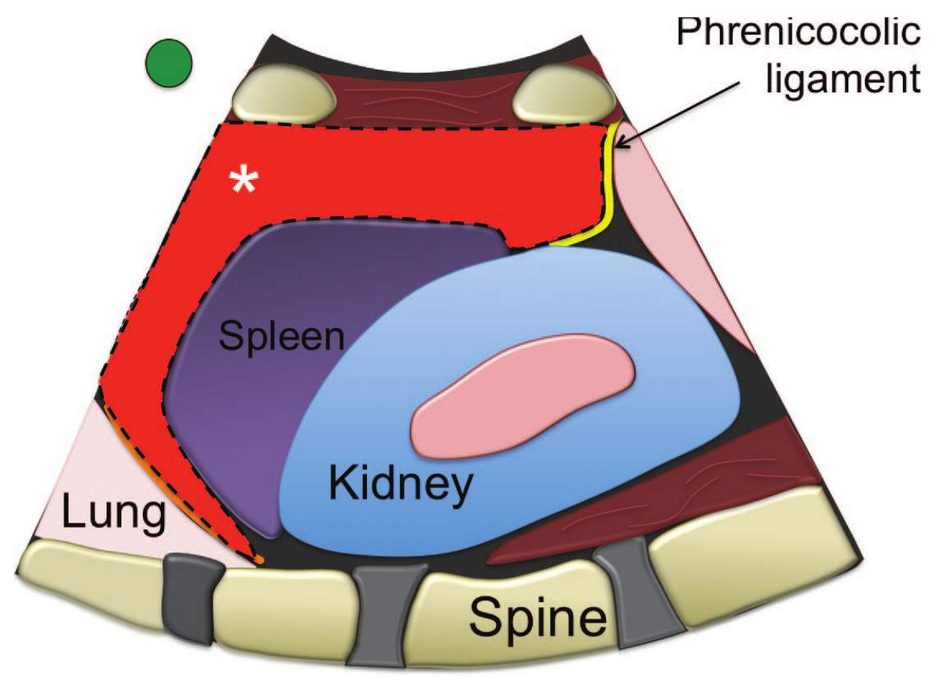


FIGURE 24.16 ■ **Hemoperitoneum, Left Upper Quadrant View.** In this view, fluid is seen in the subdiaphragmatic space between the diaphragm and the spleen. The diagram illustrates other common potential sites for free fluid, marked by asterisks. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

Technique

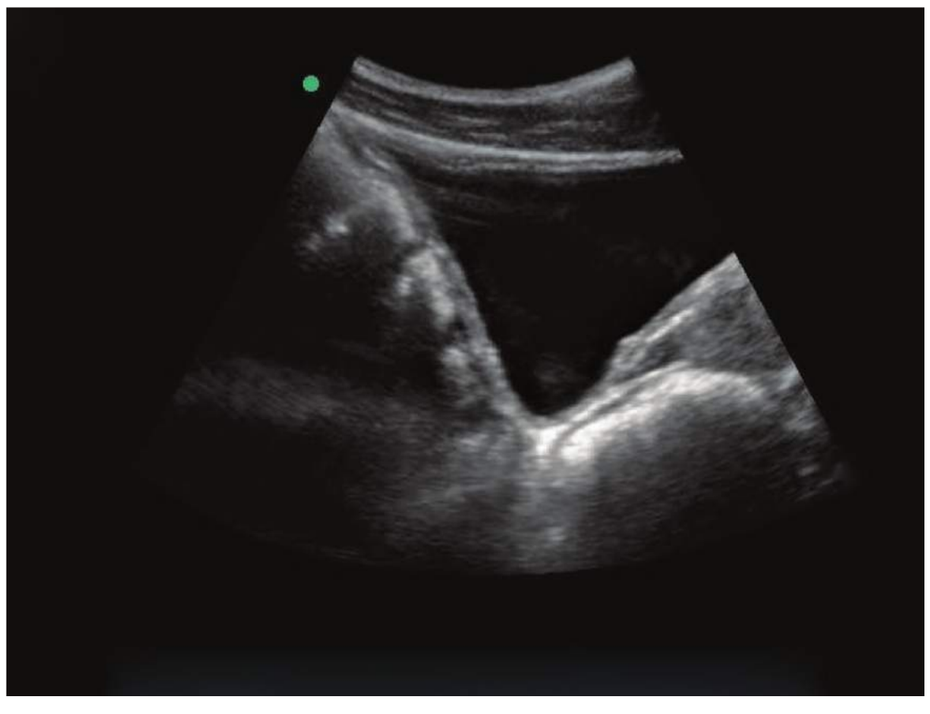
Sagittal View (Longitudinal)

- With the indicator oriented toward the patient's head, the transducer is placed just above the symphysis pubis and is directed into the pelvis (Fig. 24.17).
- Identify the bladder (triangular in this view when fully distended), uterus (pear-shaped if present), prostate, seminal vesicles, and rectum (Figs. 24.18 and 24.19).

Technique: Transverse View

- With the indicator oriented toward the patient's right, the transducer is placed about 1 to 2 cm above the symphysis pubis and the beam is angled through the bladder into the peritoneum (Fig. 24.20).

FIGURE 24.17 ■ **Suprapubic Sagittal View.** The transducer indicator is oriented toward the patient's head and the transducer is placed just above to the symphysis pubis and is directed into the pelvis. (Photo contributor: Lawrence B. Stack, MD.)



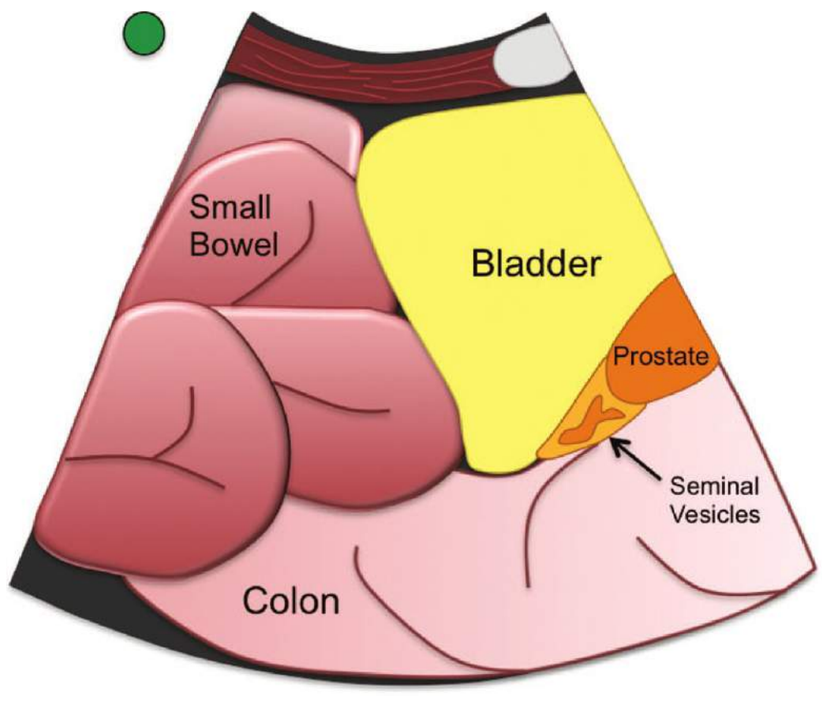


FIGURE 24.18 ■ **Normal Male Pelvis, Sagittal View.** The bladder appears triangular in this view. The prostate and seminal vesicles can be seen to the right of the screen, just deep to the bladder. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

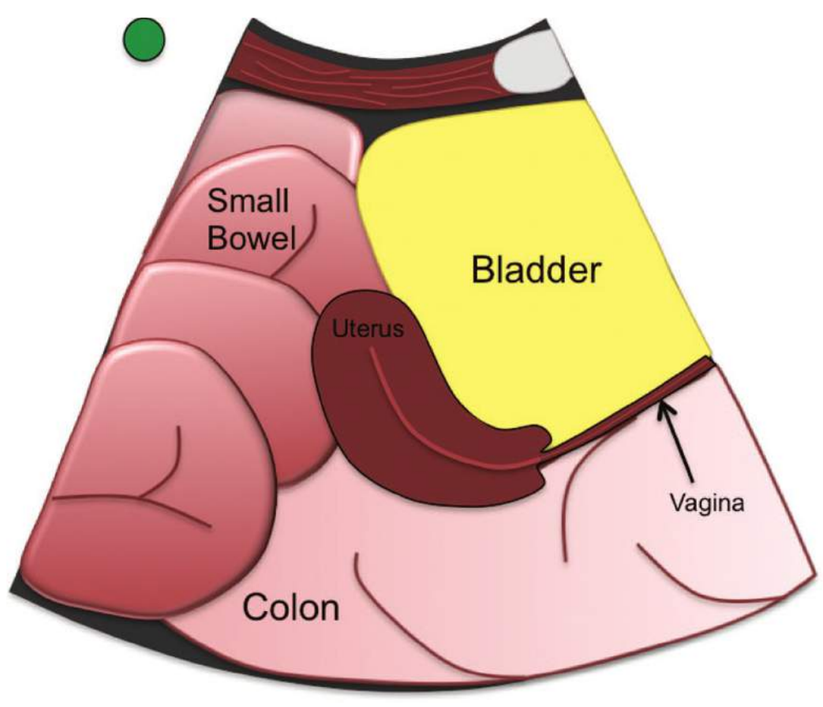


FIGURE 24.19 ■ **Normal Female Pelvis, Sagittal View.** The uterus appears pear-shaped in this view, and may be anteroflexed or retroflexed. The "pouch of Douglas" is a potential space between the uterus and the rectum, and is deep to the uterus. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

FIGURE 24.20 ■ **Suprapubic View, Transverse.** The indicator is oriented to the patient's right and the beam angled through the bladder into the peritoneum. (Photo contributor: Lawrence B. Stack, MD.)

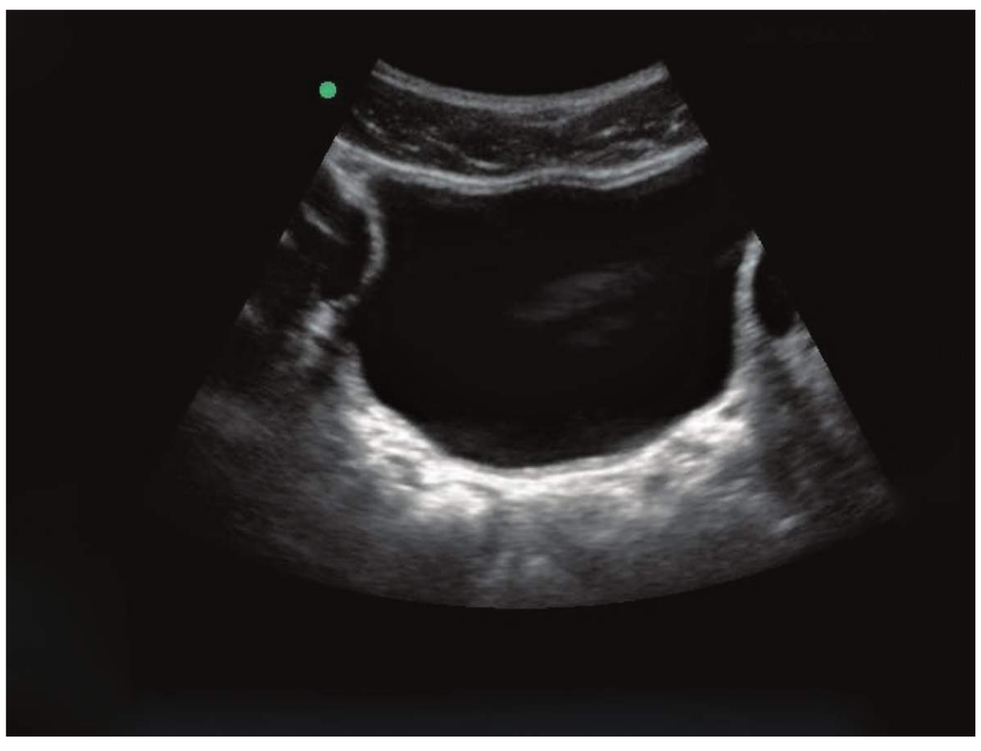
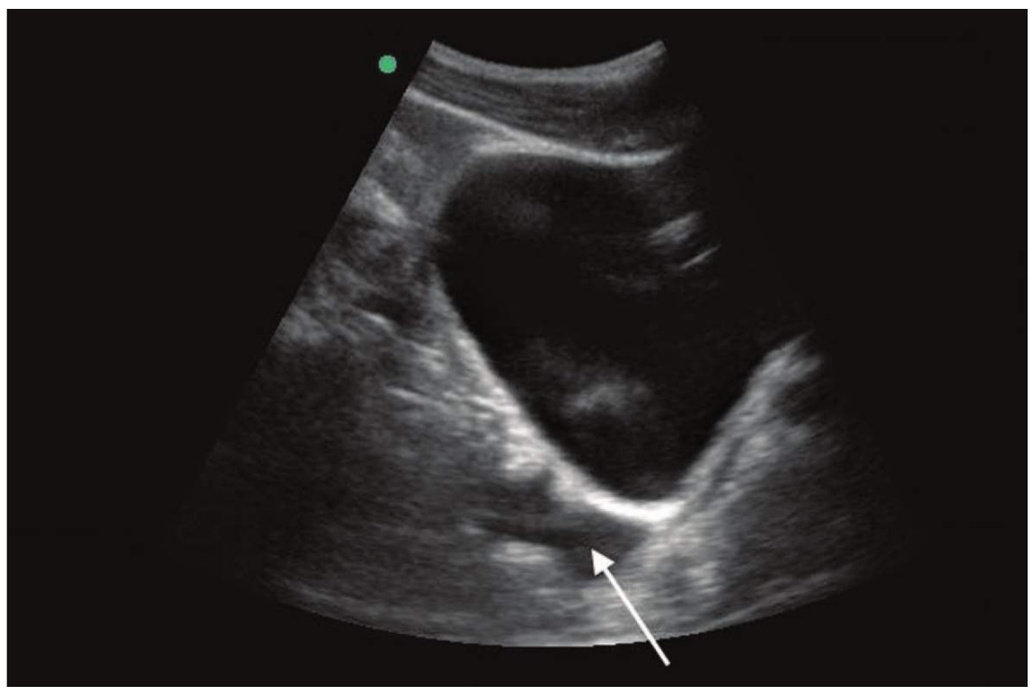


FIGURE 24.21 ■ **Suprapubic View, Transverse.** The bladder appears rectangular in this view. The uterus/cervix or prostate/seminal vesicles can be seen deep to the bladder. (Ultrasound contributor: Jeremy S. Boyd, MD.)

- Identify the bladder (rectangular in this view when fully distended), uterus (oval hyperechoic structure if present), prostate, seminal vesicles, and rectum (Fig. 24.21).

Abnormal Findings

- Hemoperitoneum (male pelvis): Free fluid in the male pelvis is seen as anechoic (black) areas filling the rectovesicular space, the potential space between the bladder and the rectum. The rectovesicular space is immediately cephalad to the extraperitoneal prostate and seminal vesicles. In the sagittal view, it is easier to see this junction that distinguishes the peritoneal cavity from the extraperitoneal pelvis. A large amount of free fluid may appear as loops of bowel floating lateral to the bladder in the transverse view and superior to the bladder in the sagittal view (Figs. 24.22 and 24.24).
- Hemoperitoneum (female pelvis): Free fluid in the female pelvis is seen as an anechoic (black) area in the rectouterine space, the potential space between the uterus and rectum. The vaginal stripe is an extraperitoneal structure and intraperitoneal fluid will not be seen collecting beneath it. A large amount of free fluid may appear as loops of bowel floating lateral to the bladder in the transverse view and superior to the bladder in the sagittal view (Figs. 24.23 and 24.24).



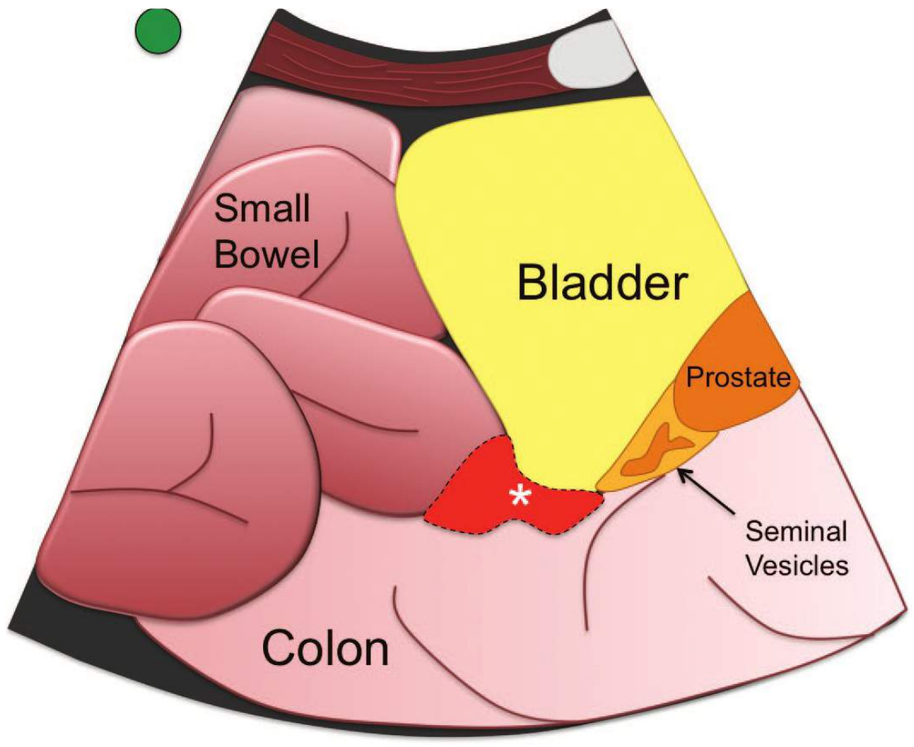


FIGURE 24.22 ■ **Hemoperitoneum, Sagittal Male.** Hemoperitoneum is seen deep to the bladder in the rectovesicular area on this sagittal view (arrow). Free fluid is seen as an anechoic (black) area in the rectovesicular space as demarcated by the asterisk. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Pearls

1. It is important to remember that the bladder is within the pelvis; therefore, the transducer must be directed cephalad through the bladder into the peritoneum to evaluate for intraperitoneal fluid or hemorrhage in the rectovesicular or rectouterine space. If the prostate or vaginal stripe can be seen on a transverse suprapubic view, then the beam is directed caudal to the peritoneum and is missing from the peritoneal cavity entirely.
2. When in the sagittal plane, rotate the transducer 90 degrees counterclockwise directing the transducer indicator toward the patient's right to obtain a transverse view.
3. An empty bladder will not be able to sufficiently displace the small bowel from the pelvis. This often prevents an adequate view of the dependent portions of the pelvis to assess for free peritoneal fluid. Filling the bladder through a Foley catheter or waiting for the bladder to distend will allow a better view for free intraperitoneal fluid that has collected in the pelvis.

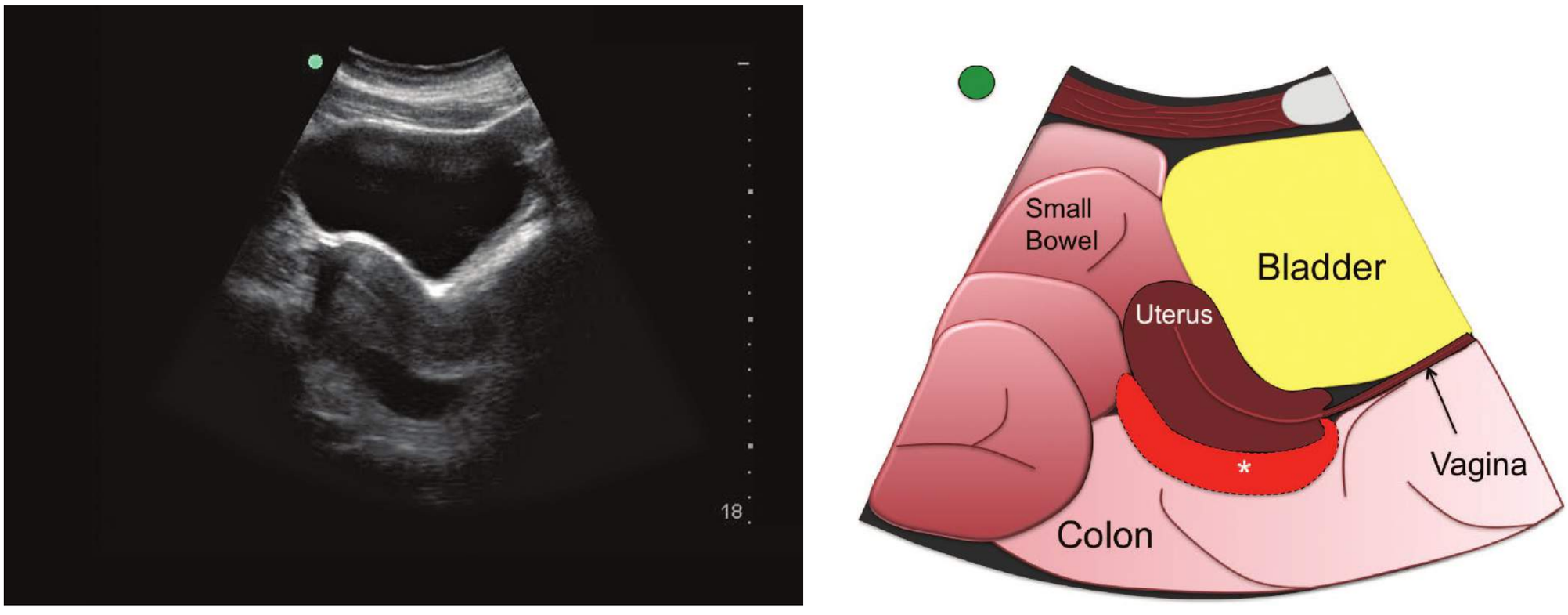


FIGURE 24.23 ■ **Hemoperitoneum, Sagittal Female.** Hemoperitoneum is seen deep to the uterus, in the pouch of Douglas. Free fluid is seen as an anechoic (black) area in the rectouterine space as demarcated by the asterisk. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

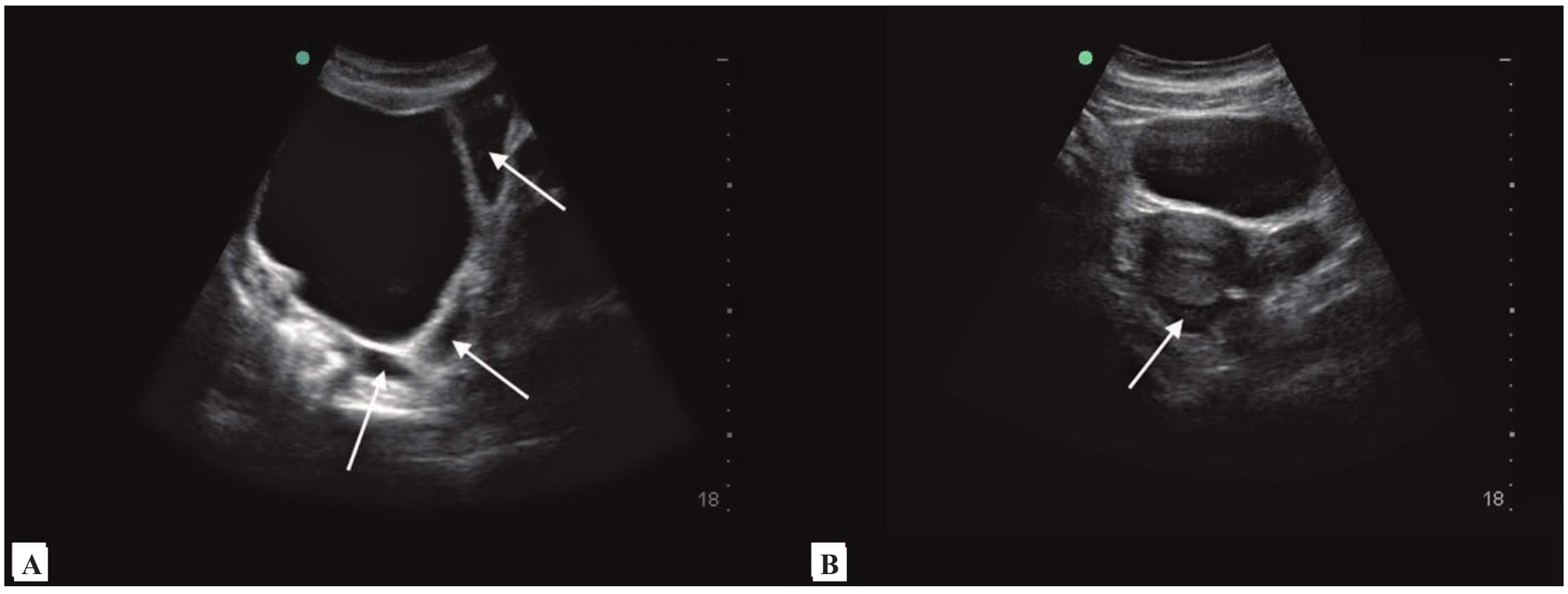


FIGURE 24.24 ■ **Hemoperitoneum, Transverse Male (A) and Female (B).** Hemoperitoneum (arrows) is seen deep and lateral to the bladder in the male pelvis (**A**), and deep to the uterus in the female pelvis (**B**). (Ultrasound contributor: Jeremy S. Boyd, MD; Robinson M. Ferre, MD.)

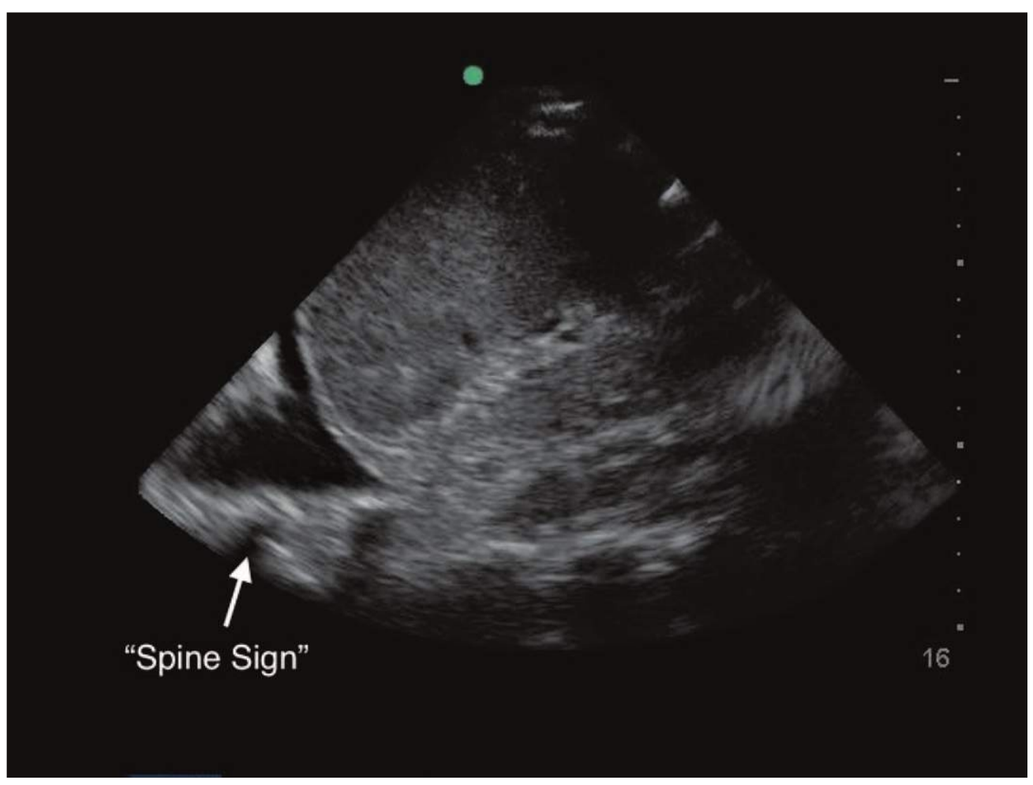
- Note that evaluation for thoracic fluid is best performed as an extension of the RUQ and LUQ views by sliding the transducer up into the chest in a coronal plane.

Technique

- A linear or convex transducer is preferred for evaluation of pneumothorax.
- A convex or phased array transducer is preferred for evaluation of hemothorax.
- The transducer indicator is oriented toward the patient's head.

Suspected Pleural Fluid (Hemothorax)

- With the indicator directed toward the patient's head, the transducer is placed in the fifth or sixth intercostal space in the midaxillary line.



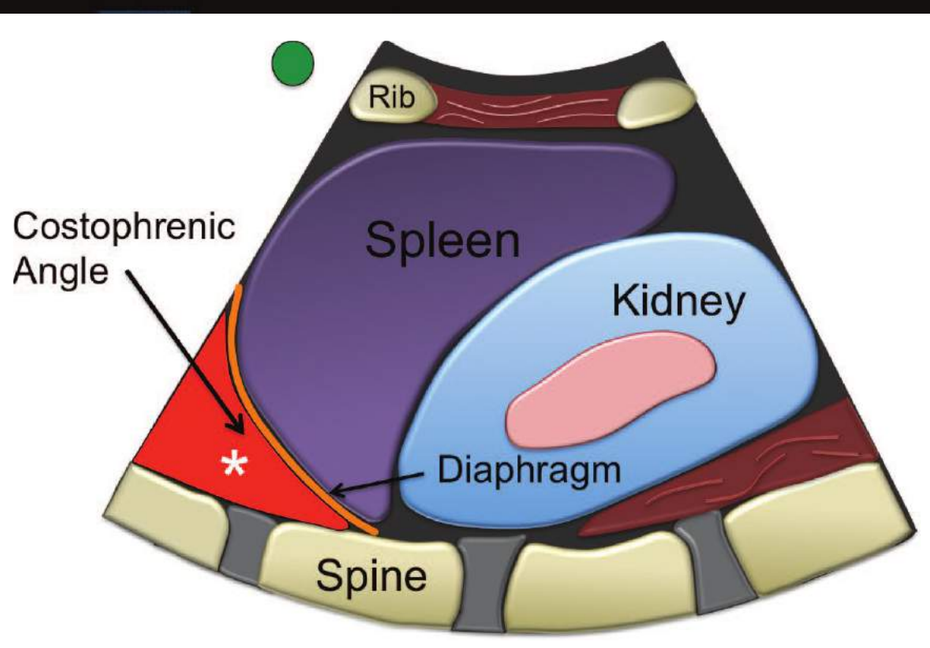


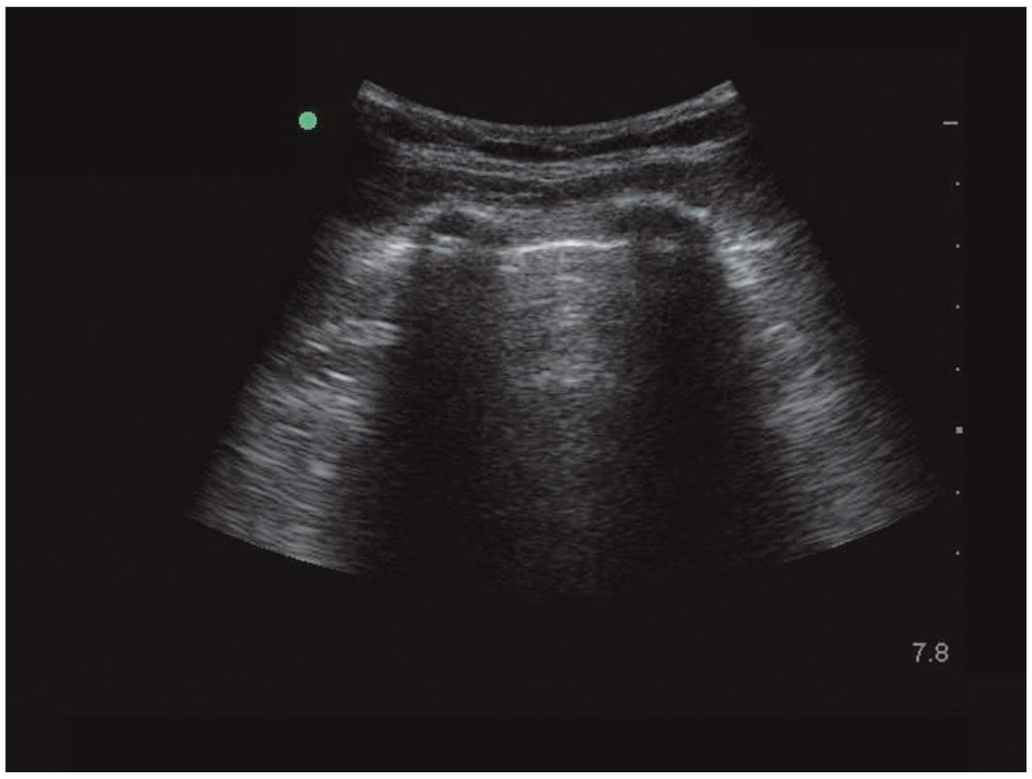
FIGURE 24.25 ■ **Hemothorax.** Fluid (in this case blood) is identified as an anechoic "wedge" above the diaphragm with loss of the normal mirroring artifact of the diaphragm allowing visualization of the spine extending beyond the costophrenic angle, also known as the "spine sign." (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

- Identify the liver (on the right) or the spleen (on the left), the diaphragm, and the vertebral bodies that lie at the bottom of the screen.
- In the fully inflated healthy lung, air prevents direct visualization of structures deep to the interface of the diaphragm and the visceral pleura of the lung. If fluid is present, it is identified as an anechoic (black) area that is caudal to the lung in the costophrenic recess. A hemothorax may have a simple anechoic appearance, but may also contain heterogeneous echoes from clotted blood or portion of lacerated lung tissue.
- The presence of pleural fluid will allow direct visualization of the lung and posterior structures, including the vertebral bodies of the thoracic spine often referred to as the "spine sign" (Fig. 24.25).

Suspected Pneumothorax

- When using ultrasound to evaluate for a pneumothorax, the transducer is placed in the superior most portion of the chest wall in a supine patient to assess for normal apposition of the lung. Unlike chest radiography, ultrasound must look for normal apposition of the visceral and parietal portions of the lung in a stepwise fashion. Indeed, it is important to note that ultrasound can only assess for the presence or absence of a pneumothorax on the portion of the lung, where the transducer is currently imaging. Thus, when using ultrasound, we assume that there is no prior history of pleural scarring or pleural adhesions.

FIGURE 24.26 ■ **Anterior Thorax View.** A linear transducer is placed below the clavicle in the midclavicular line dragged inferiorly to assess multiple rib spaces for pneumothorax. Other probe types may be used as well. (Photo contributor: Lawrence B. Stack, MD.)



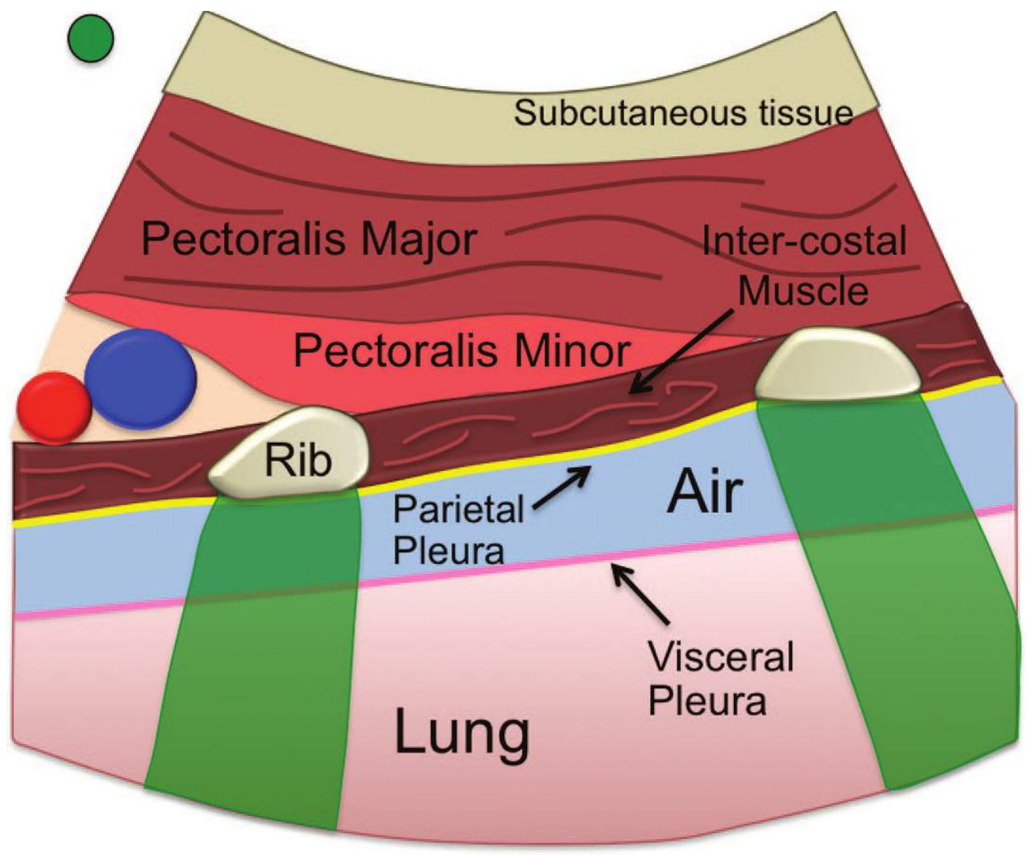


FIGURE 24.27 ■ **Pneumothorax**. A still image of a pneumothorax as seen on ultrasound using a curvilinear or convex array transducer. The diagram illustrates the layer of air between the pleura causing the absence of "lung sliding" seen in real-time imaging. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

- With the indicator directed toward the patient's head, the transducer is placed just below the clavicle in the midclavicular line. To provide maximum sensitivity for the presence of a pneumothorax, the probe should be dragged caudal in the midclavicular line until the diaphragm is seen just below the costal margin (Fig. 24.26).
- Once the probe is placed on the chest wall, identify the pectoralis and intercostal muscles and ribs with associated acoustic shadows that project to the bottom of the screen. The interface of the visceral and parietal pleura, known as the pleural line, will be seen immediately deep to the rib and intercostal muscle.
- The depth should be adjusted so that the pleural line is positioned in the middle or upper half of the screen.
- If there is normal apposition of the visceral and parietal pleura (no pneumothorax), sliding will be seen along the

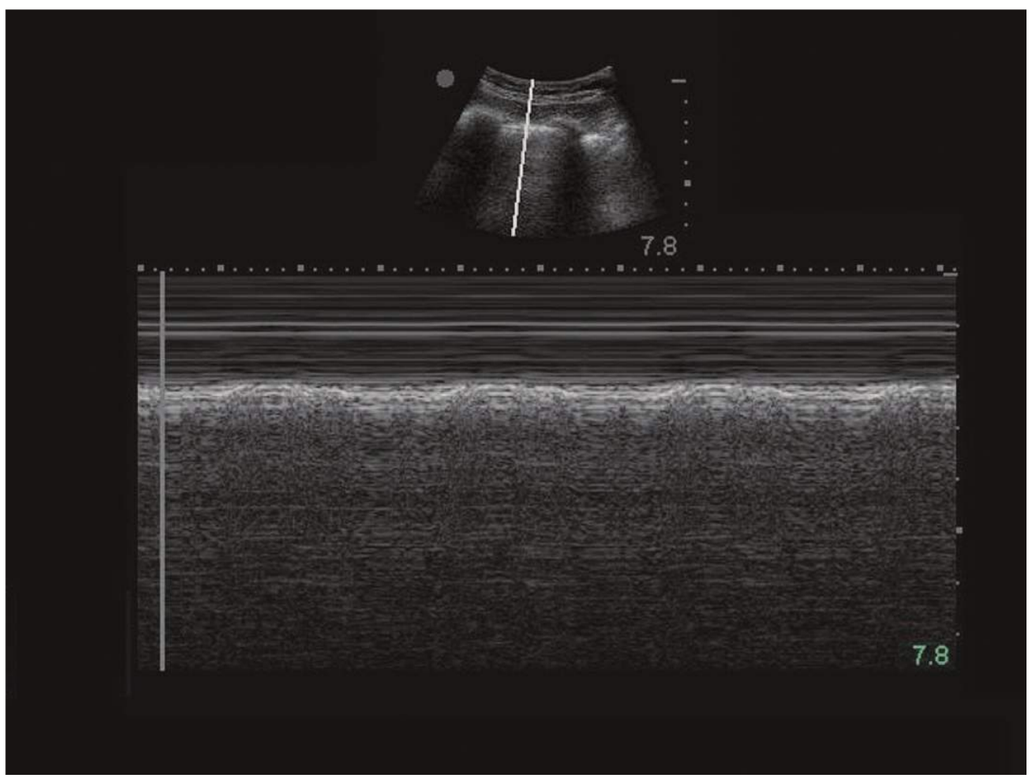


FIGURE 24.28 ■ **Seashore Sign.** The "seashore sign" describes the M-mode appearance on thoracic ultrasound when there is no pneumothorax. The near-field image resembles waves on a beach, and the far-field image resembles a sandy beach, an artifact caused by lung sliding. (Ultrasound contributor: Jeremy S. Boyd, MD.)

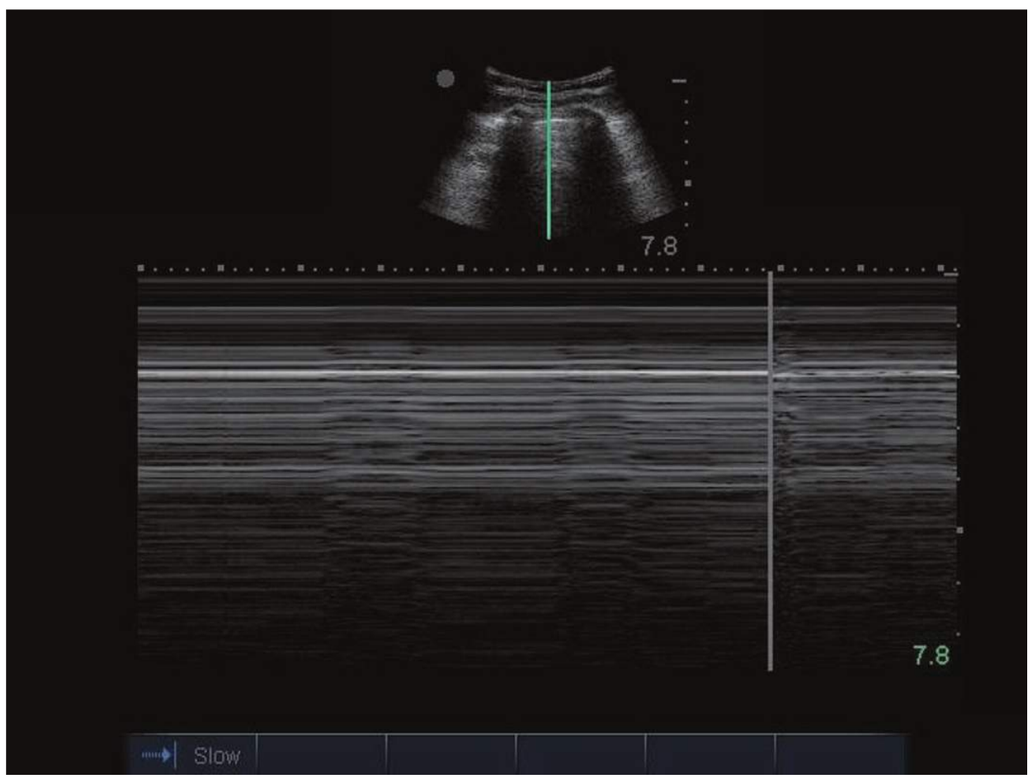


FIGURE 24.29 ■ **Barcode or Stratosphere Sign (Pneumothorax).** This describes the M-mode appearance on thoracic ultrasound when pneumothorax is present. The "sandy beach" is now gone due to the absence of lung sliding. (Ultrasound contributor: Jeremy S. Boyd, MD.)

pleural line during normal respiration. If a pneumothorax is present, no sliding will be seen (Fig. 24.27).

- B-lines, a type of comet tail artifact, are occasionally seen arising from the pleural line and will project to the bottom of the screen (see Lung Ultrasound). B-lines are only present if there is normal apposition of the visceral and parietal pleura. In essence, they "rule out" a pneumothorax at the portion of lung that is being imaged. If more than two B-lines are present in any one view, it indicates the presence of fluid within the extravascular lung tissue, which may indicate pulmonary contusion in the setting of trauma.
- M-mode can assist with the detection and documentation of normal lung sliding. The "seashore sign," described as "waves on the beach," is noted with M-mode when no pneumothorax is present (Fig. 24.28). Conversely, a "stratosphere sign" is seen when a pneumothorax is present, the result of the M-mode tracing only a reverberation artifact (Fig. 24.29).

Abnormal Findings

- Hemothorax: As visualized at the upper quadrants of the lateral chest, a hemothorax will appears as an anechoic (black) area between the diagphram and the lung within the costophrenic recess. Heterogeneous echoes may be present due to clotted blood, lacerated lung, or other material. The presence of pleural fluid will allow direct visualization of the lung and posterior structures, including the vertebral bodies of the thoracic spine often referred to as the "spine sign"(Fig. 24.25).
- Pneumothorax: The absence of sliding along the pleural line is indicative of a pneumothorax. If lung sliding is absent, one should search for the leading edge of the pneumothorax, also known as lung point. Estimation of the pneumothorax size is possible by evaluating progressively more lateral views of the thorax in the supine patient.

Pearl

1. Lung point is the point of transition from normal lung sliding to the absence of lung sliding that occurs at the leading edge of the pneumothorax. The finding of a "lung point" is 100% diagnostic of a pneumothorax (Fig. 24.30).

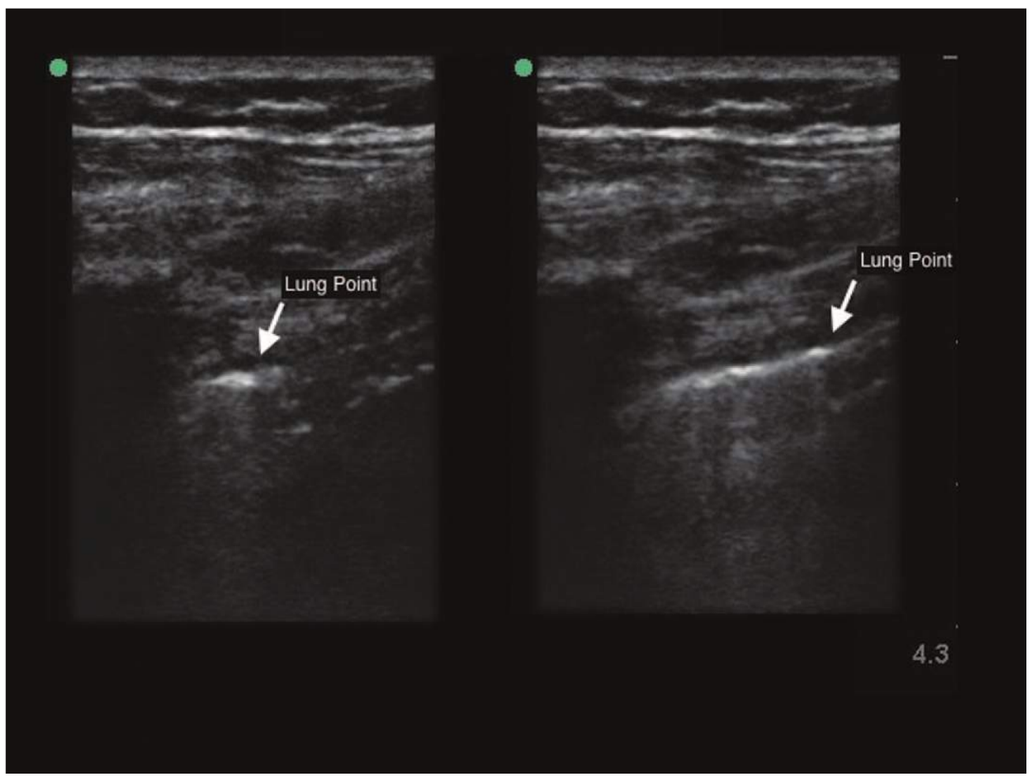


FIGURE 24.30 ■ **Lung Point.** The point of transition from normal lung sliding to the absence of lung sliding occurs at the leading edge of a pneumothorax. It is seen in this pair of images to move from left to right as the patient inhales. (Ultrasound contributor: Jeremy S. Boyd, MD.)

Clinical Summary

Focused cardiac ultrasound can yield significant diagnostic information for the patient presenting with cardiac arrest, shock, shortness of breath, and a host of other complaints or physical findings. Although the intricacies of comprehensive echocardiography are beyond the scope of practice of most emergency medicine providers, with experience one can incorporate focused bedside cardiac ultrasound into the diagnostic armamentarium safely to answer specific diagnostic questions such as cardiac activity, volume status, gross cardiac function, right heart strain, and the presence of pericardial effusion.

It is important to note that by convention, unlike abdominal sonography, cardiac ultrasound is viewed with the transducer indicator displayed on the right of the screen. This will require the indicator on the transducer to be directed toward the patient's left in an anatomically transverse view. This may be disorienting for many who have not performed cardiac ultrasound before. Most ultrasound systems include cardiac presets that automatically reverse the indicator orientation to the right of the display screen. The following section describes a sonographic approach for a conventionally oriented image using standard cardiac windows.

Indications

- Cardiac arrest, pulseless electrical activity (PEA)
- Penetrating thoracic/abdominal trauma
- Unexplained hypotension or shock
- Dyspnea
- Chest pain
- Acute myocardial infarction
- Suspected aortic dissection

Specific pathologic states investigated with bedside cardiac ultrasound include asystole, cardiac activity, pericardial effusion, acute heart failure, aortic root dilatation/dissection, and right heart enlargement.

The sonographic windows for focused cardiac ultrasound include the subxiphoid view presented within the trauma/FAST examination as well as parasternal and apical views. Focused cardiac ultrasound utilizes standard views that are familiar to cardiologists and sonographers alike. These five windows allow the emergency physician to effectively and efficiently evaluate emergent cardiopulmonary pathology.

Views for Focused Cardiac Ultrasound (Fig. 24.31)

1. Subxiphoid/subcostal (SUBX; see E-FAST Examination, Subxiphoid View)
2. Parasternal long-axis view (PSLA)
3. Parasternal short-axis view (PSSA)
4. Apical four-chamber view (AP4)
5. Subxiphoid long-axis view (IVC)

The technique and common findings for each of these views are presented in the next five topics.

Patient Positioning

- The patient should be optimally positioned in the left lateral decubitus position with the left arm above the head (allowing better access to the intercostal spaces) for all but the SUBX and IVC views, which should be performed with the patient supine. If this is not feasible, the patient may be supine for the entire study.

Recommended Transducer

- Phased array

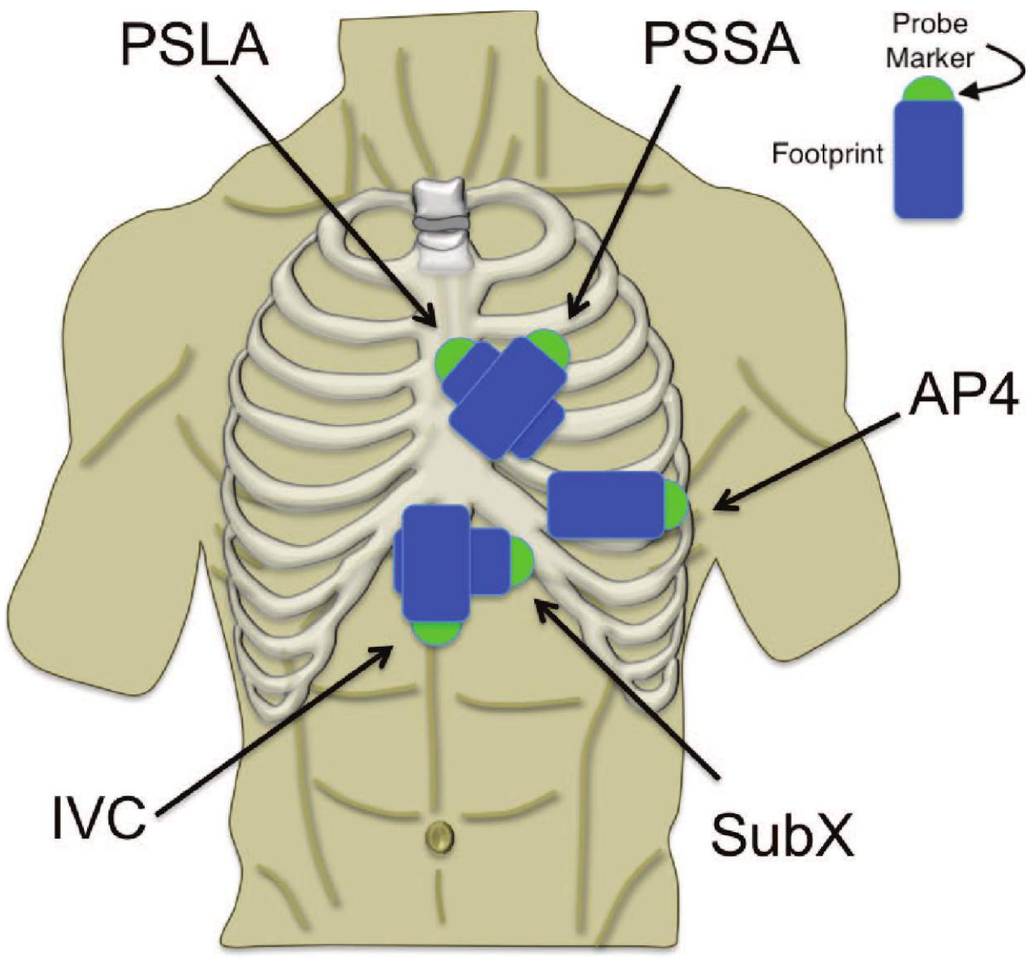


FIGURE 24.31 ■ **Focused Cardiac Ultrasound**. Transducer and probe marker positions for evaluation of the heart and IVC. (Illustration courtesy of Robinson M. Ferre, MD.)

Technique

- The transducer indicator should be directed toward the left in a cardiac preset.
- With the transducer indicator pointing to the patient's left, the transducer is placed inferior to the xiphoid process and directed cephalad toward the left shoulder in a horizontal plane (see Fig. 24.32).

FIGURE 24.32 ■ **Subxiphoid View.** With the transducer indicator pointing to the patient's left, the transducer is placed inferior to the xiphoid process and directed cephalad toward the left shoulder in a horizontal plane. (Photo contributor: Lawrence B. Stack, MD.)

- Pivot, sweep, and tilt the transducer as needed to view all four cardiac chambers.
- Identify the heart, four cardiac chambers, and surrounding pericardium (see Fig. 24.33).

Abnormal Findings

- Pericardial effusion: Pericardial fluid will appear as an anechoic (black) region noted between the pericardium and the right ventricle. As more fluid collects, the fluid will be seen completely surrounding all four chambers of the heart. Occasionally, internal echoes representing fibrin, clot, or cardiac tissue may be present within the pericardial space (Fig. 24.34).
- Asystole: No cardiac activity present.
- Hyperdynamic cardiac activity: Extensive cardiac contraction with near-total or complete collapse of the cardiac chambers, often associated with tachycardia and hypovolemia.

Pearl

See "Pearls" for the "E-FAST Examination, Subxiphoid View."

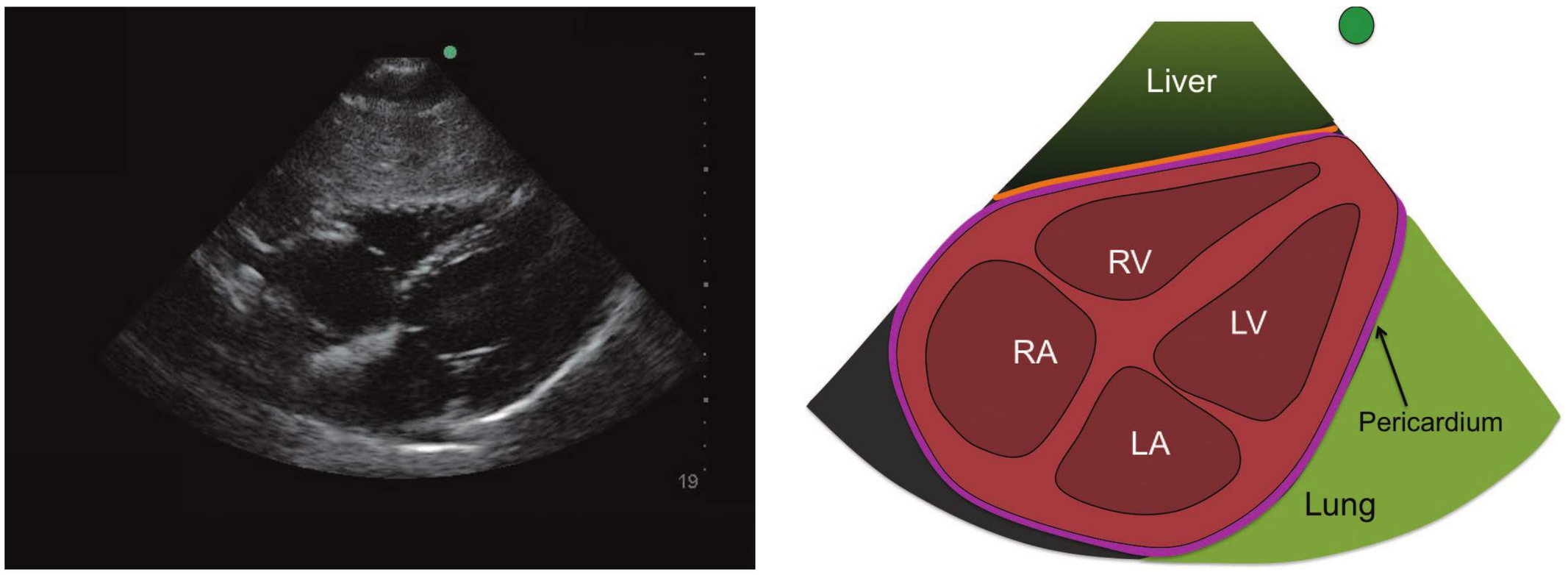
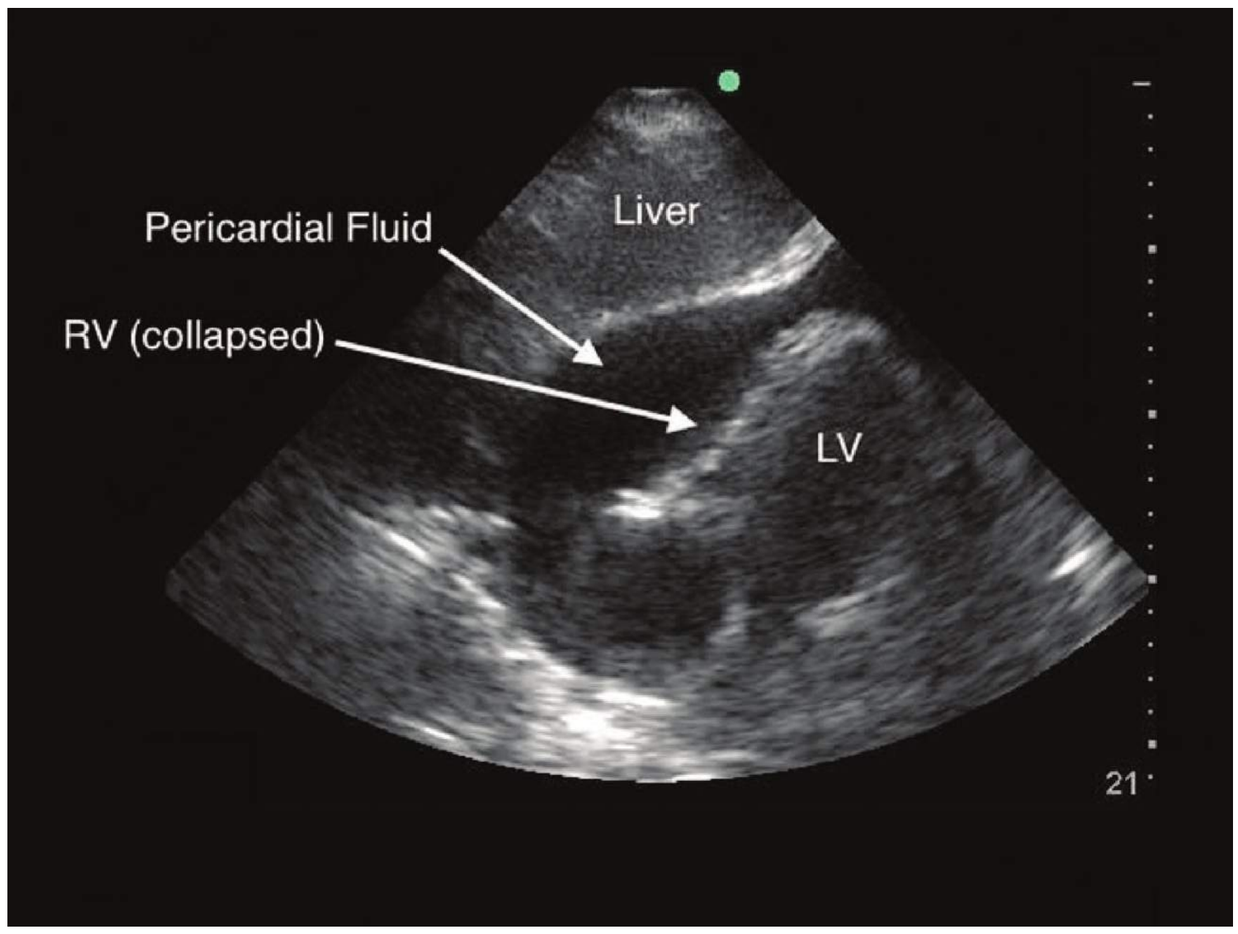


FIGURE 24.33 ■ **Subxiphoid View.** A normal view demonstrating the heart, four cardiac chambers, and surrounding pericardium. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)



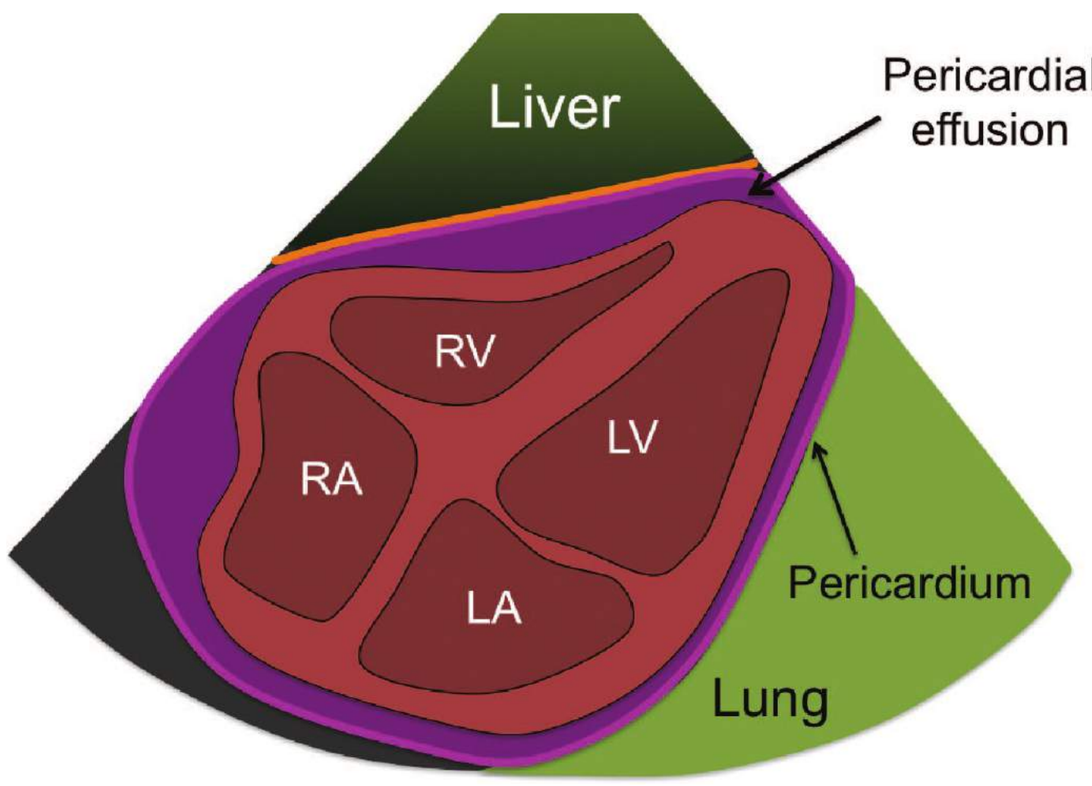


FIGURE 24.34 ■ **Pericardial Effusion**. Pericardial fluid will appear as an anechoic (black) region noted between the pericardium and the right ventricle. Hypotensive patients with a pericardial effusion and findings such as right ventricular collapse (illustration) and/or a distended IVC warrant consideration of emergent pericardiocentesis. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Technique

- The transducer indicator directed at the right clavicle or shoulder.
- The transducer is placed in the fourth or fifth left parasternal intercostal space and the beam is directed posteriorly (Fig. 24.35).
- Identify the right ventricle, left atrium, left ventricle, mitral valve, aortic valve, aortic root, aortic outflow tract, and surrounding pericardium (Fig. 24.36).

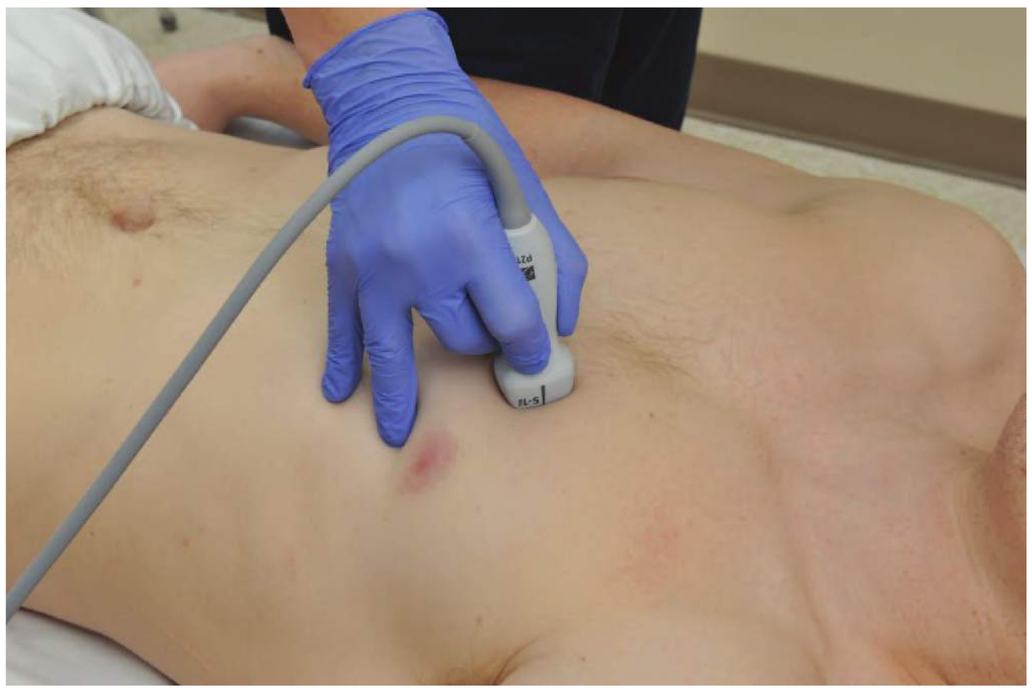


FIGURE 24.35 ■ **Parasternal Long-Axis View.** The transducer is placed in the fourth or fifth left parasternal intercostal space and the beam is directed posteriorly. (Photo contributor: Lawrence B. Stack, MD.)

Abnormal Findings

- Pericardial effusion: Anechoic (black) region noted between the hyperechoic (bright) pericardium and the walls of the heart (see Fig. 24.37). In this view, pericardial effusion may be distinguished from a left-sided pleural effusion by its relationship to the descending thoracic aorta.
- Aortic root dilatation: An aortic root measurement greater than 3.8 to 4.0 cm is abnormal and indicates aneurismal dilatation that may suggest aortic dissection in the appropriate clinical setting. Further evaluation is recommended.
- Dilated descending aorta: The transverse descending thoracic aorta can be seen in the far field in this view posterior to the left atrium. A descending thoracic aorta greater than 4.0 cm is abnormal and indicates aneurismal dilatation that may suggest aortic dissection in the appropriate clinical setting. Further evaluation is recommended.
- Poor left ventricular function: Gross estimates of left ventricular function may be made by observing the anteroposterior motion of the mitral valve, the increase in myocardial thickness with contraction, and the extent to which the anterior and posterior walls of the left ventricle approach each other in systole.

Pearls

1. A true parasternal long-axis view (a sagittal image through the heart) will visualize the aortic root within the image. If the aortic root is not present, you are likely in

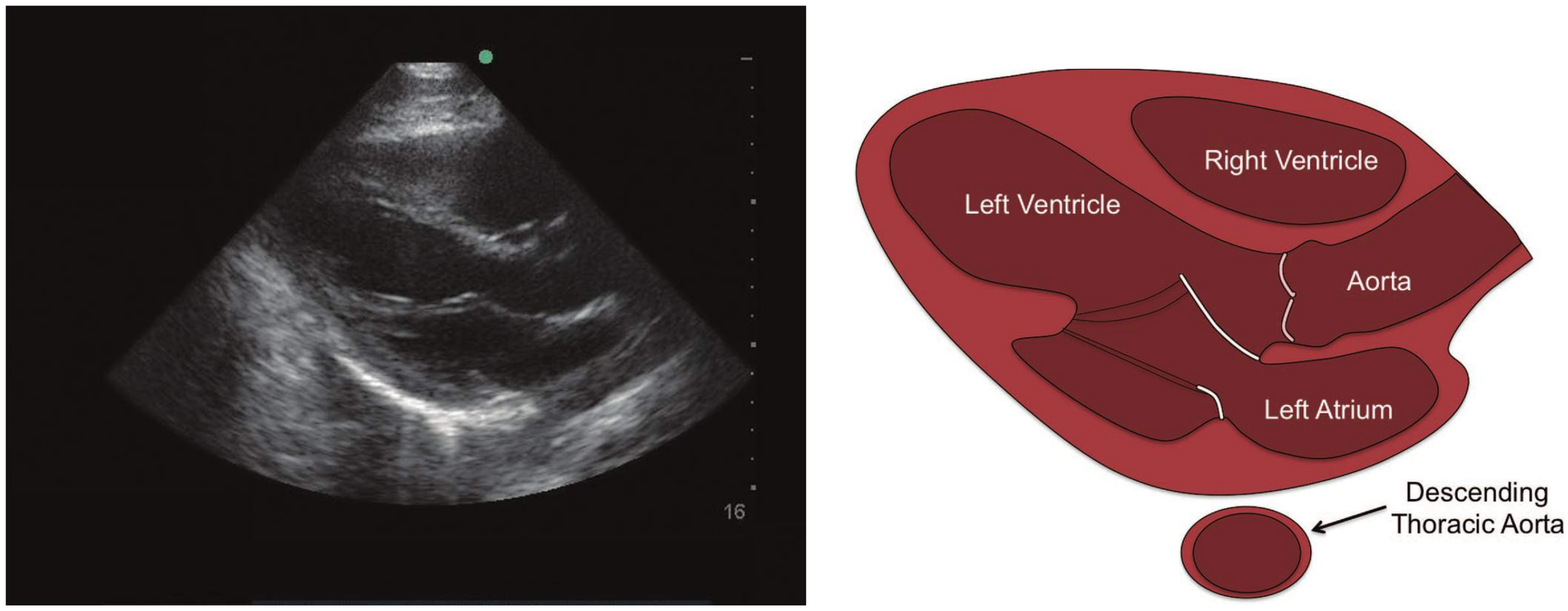


FIGURE 24.36 ■ **Parasternal Long-Axis View.** The right ventricle, left atrium, left ventricle, mitral valve, aortic valve, aortic root, aortic outflow tract, and surrounding pericardium are seen. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)

an oblique plane and will need to angle the transducer to optimize the image.

2. It is critical to make deliberate, slow, small adjustments of the transducer in imaging the heart, since even small movements at the skin surface can translate into large changes in beam angle at just 5 to 10 cm deep from the surface.
3. Normal spontaneous respiration is usually fine for cardiac imaging. Patients who are tachypneic can be very challenging, and verbally coaching the patient's breathing patterns is best. If you note a great deal of artifact due to lung interposition, place the patient in the left lateral decubitus position; have him or her inhale and slowly exhale while you scan. When you have an acceptable window, ask the patient to stop exhaling and hold his or her breath while you capture your images.
4. Remember that the parasternal long axis is approximated by a line running from the right acromioclavicular joint and the left antecubital fossa (when the arm is lying by the patient's side).

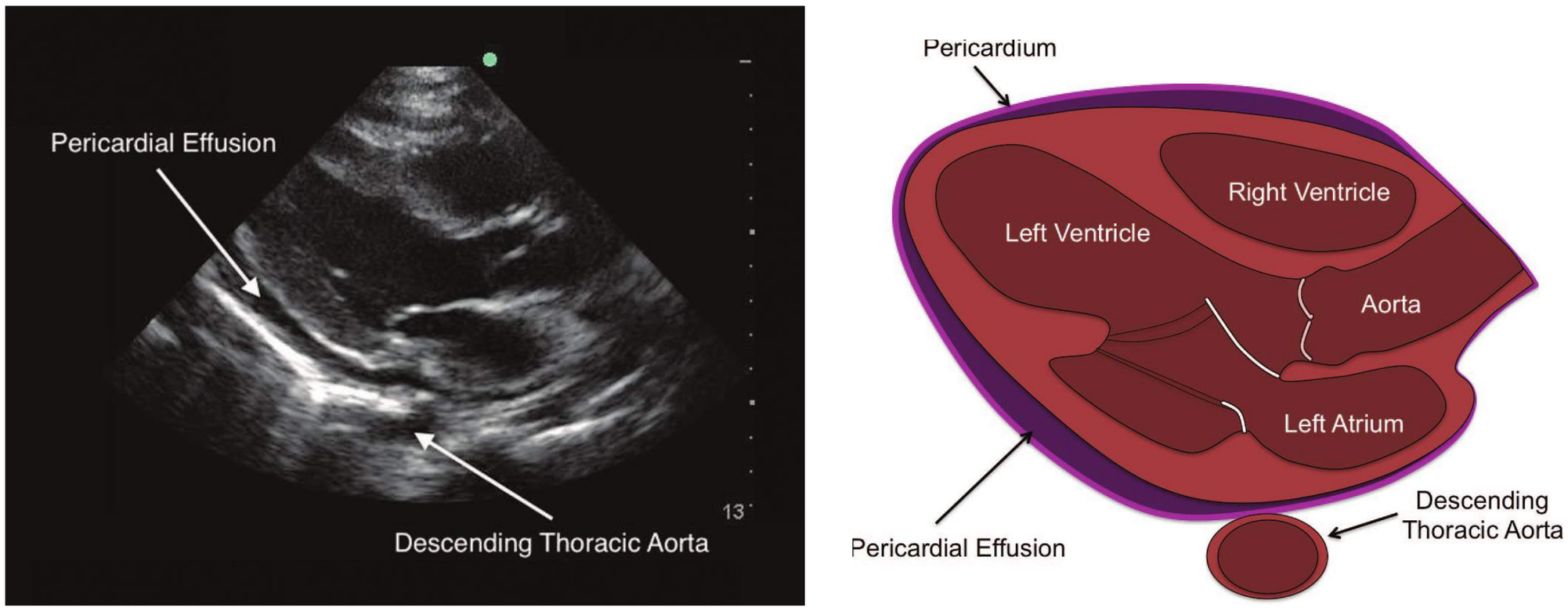


FIGURE 24.37 ■ **Parasternal Long-Axis View.** A pericardial effusion is seen as an anechoic (black) region between the hyperechoic (bright) pericardium and the walls of the heart. The image demonstrates a small pericardial effusion, while the illustration demonstrates the location of a larger (circumferential) effusion. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

PARASTERNAL SHORT-AXIS VIEW (PSSA)

Technique

- From the parasternal long-axis position, rotate the transducer 90 degrees clockwise (to the patient's left) or place the transducer in the fourth or fifth left parasternal intercostal space in a line connecting the left clavicle/shoulder and the right hip (Fig. 24.38).

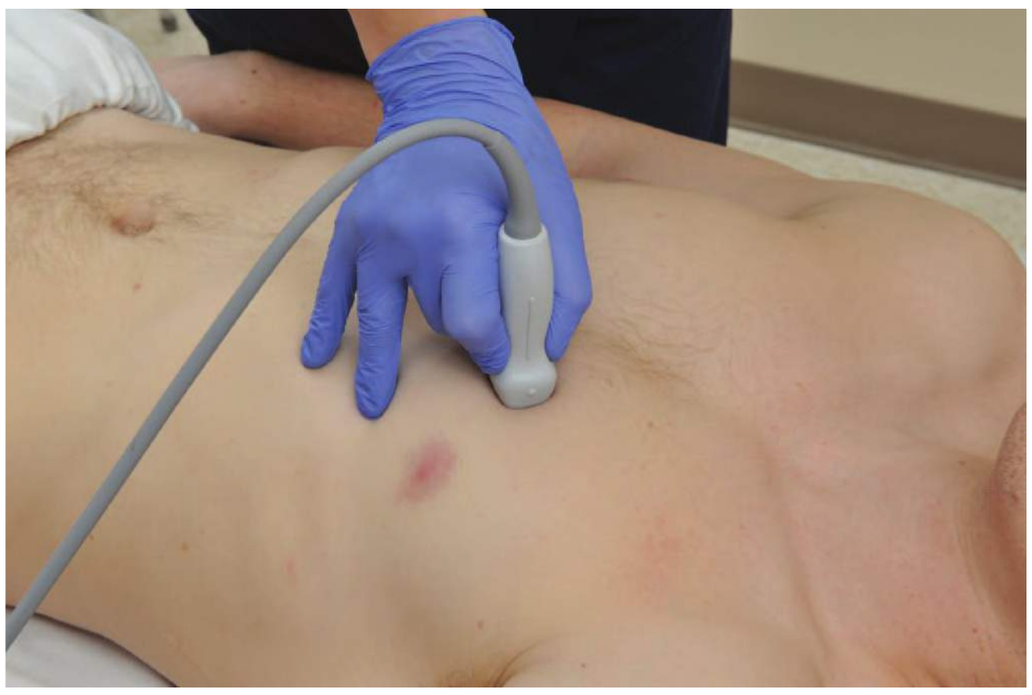


FIGURE 24.38 ■ **Parasternal Short-Axis View.** Place the transducer in the fourth or fifth left parasternal intercostal space in a line connecting the left clavicle/shoulder and the right hip, with the transducer rotated 90 degrees clockwise from the parasternal long-axis view. (Photo contributor: Lawrence B. Stack, MD.)

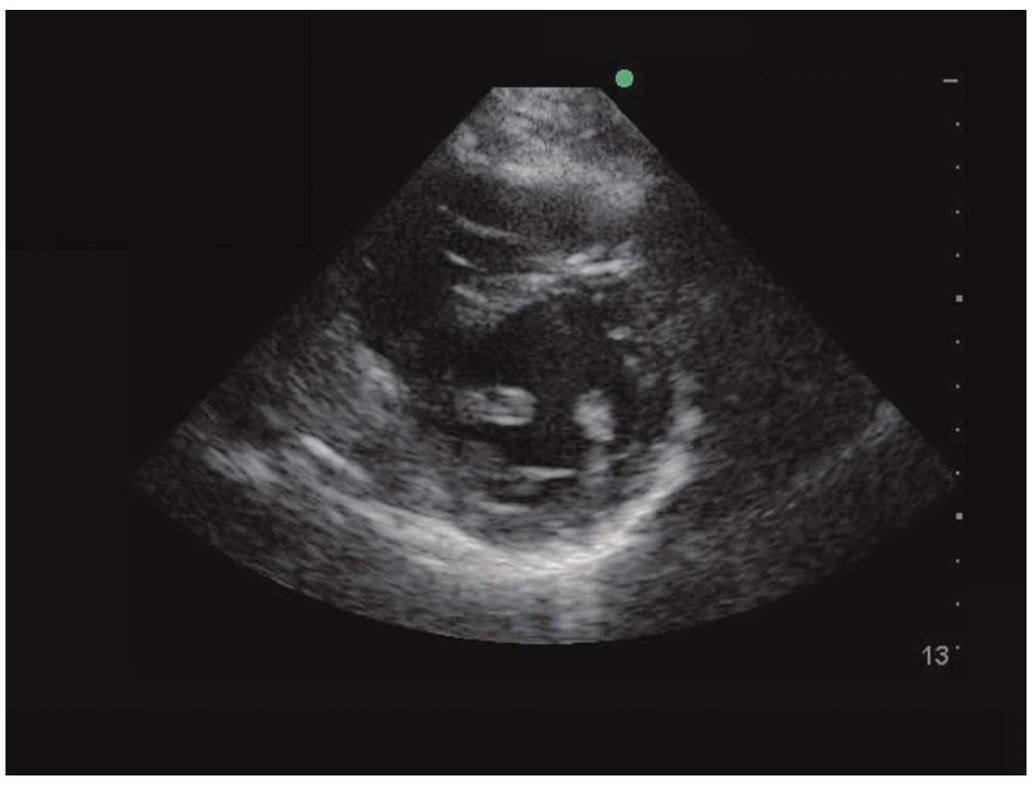
- Identify the left ventricle (circular), right ventricle (crescent-shaped), and surrounding pericardium (Fig. 24.39).

Abnormal Findings

- Pericardial effusion: Anechoic (black) region noted between the bright pericardium and the walls of the heart (Fig. 24.37).
- Dilated right ventricle: The right ventricle is normally a crescent-shaped structure. A rounded, dilated right ventricle that deforms the rounded left ventricle (creating an "OD" sign) suggests elevated right-sided pressures, as seen with pulmonary emboli and severe pulmonary hypertension (Fig. 24.40).

Pearl

1. The standard parasternal short-axis view is obtained with the image plane at the level of the papillary muscles. Visualization of the papillary muscles should ensure a true transverse section through the left ventricle, and provides a prime location for the evaluation of left ventricular contraction and motion.



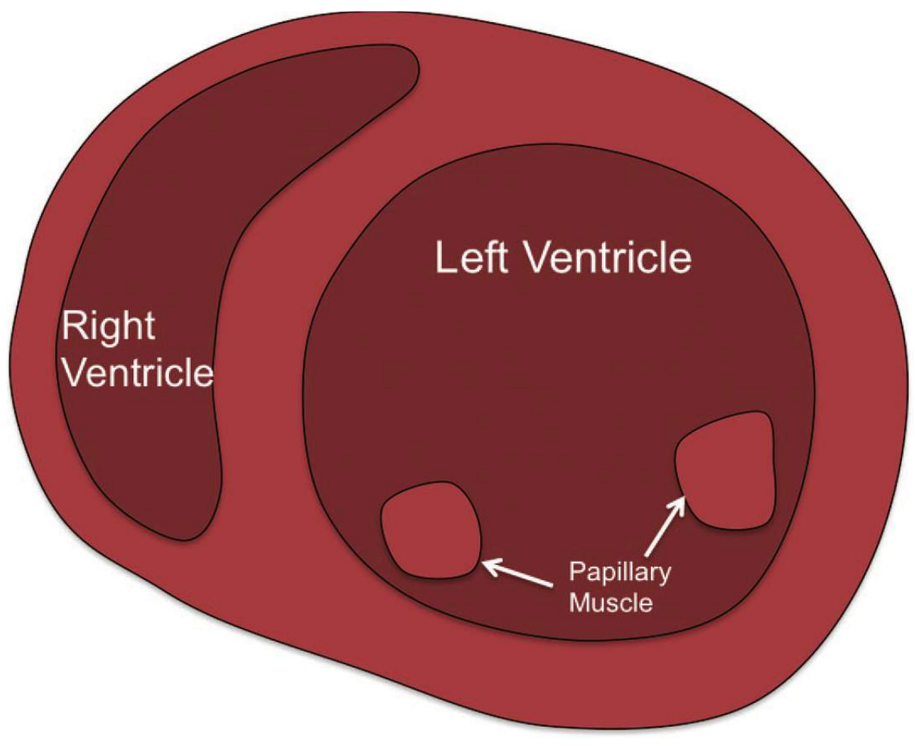
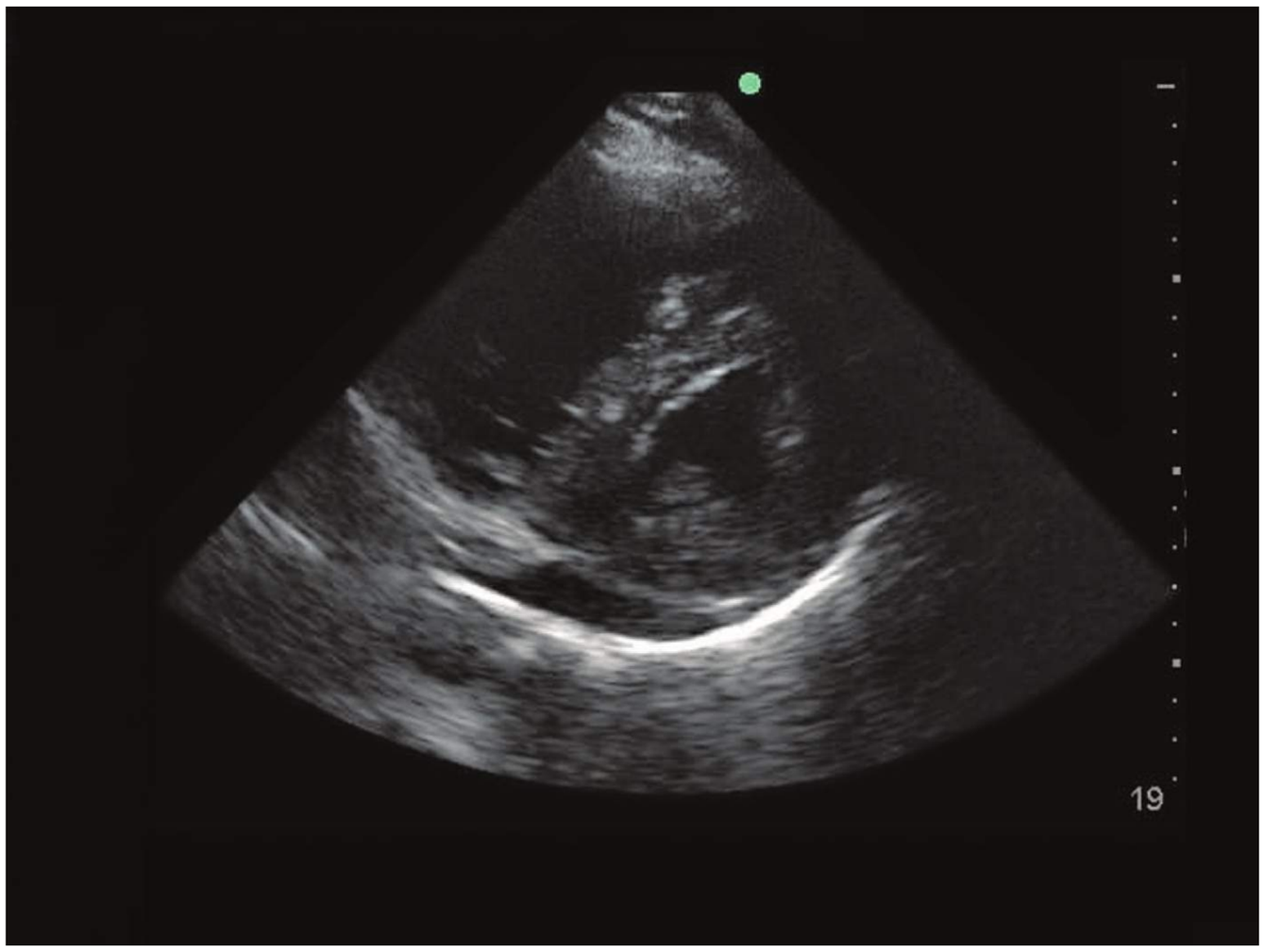


FIGURE 24.39 ■ **Parasternal Short Axis.** The left ventricle (circular) and right ventricle (crescent-shaped) and surrounding pericardium are seen. The papillary muscles are seen in cross-section. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)



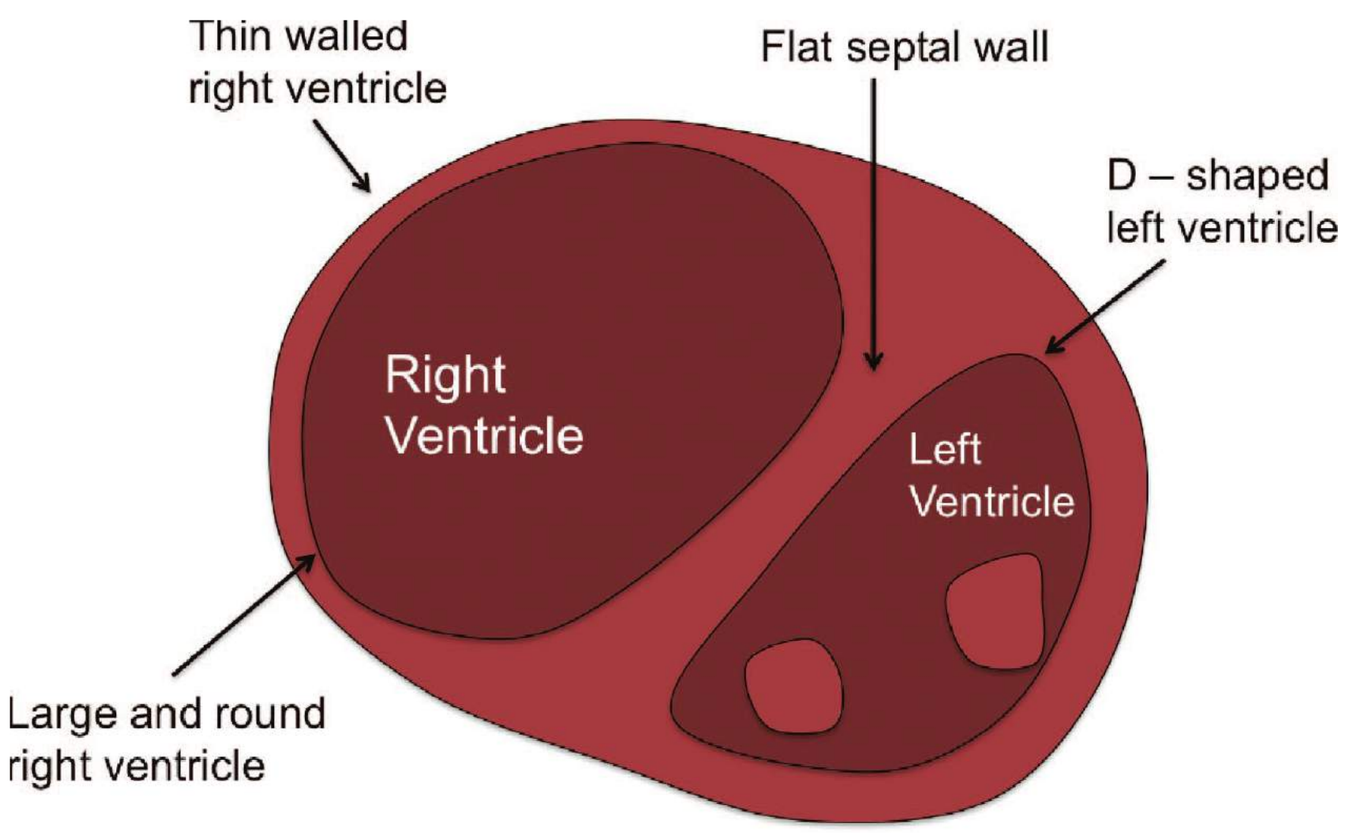


FIGURE 24.40 ■ **Parasternal Short Axis.** A rounded, dilated right ventricle that deforms the rounded left ventricle (creating an "OD" sign) suggests elevated right-sided pressures. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Technique

- The transducer indicator is directed toward the left axilla or directly posterior to the patient.
- The transducer is placed over the cardiac apex or the point of maximal intensity with the beam directed toward the right clavicle/shoulder in a plane coronal to the heart (Fig. 24.41).
- Identify the left ventricle, right ventricle, left atrium, right atrium, tricuspid valve, mitral valve, and surrounding pericardium (Fig. 24.42).

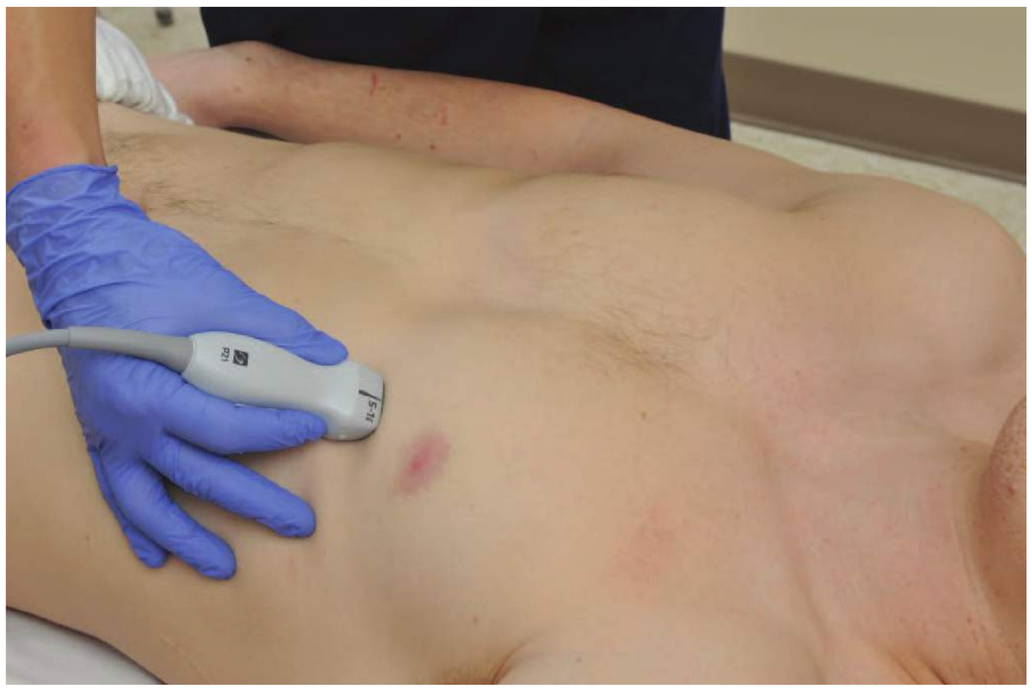


FIGURE 24.41 ■ **Apical Four-Chamber View.** The transducer is placed over the cardiac apex with the beam directed toward the right clavicle/shoulder in a plane coronal to the heart. (Photo contributor: Lawrence B. Stack, MD.)

Abnormal Findings

- Pericardial effusion: Anechoic (black) region noted between the hyperechoic pericardium and the walls of the heart (see Figs. 24.37 and 24.43).
- Dilated right atria/ventricle: If the right ventricle is equal to or larger than the left ventricle in size, or there is poor contractility of the right heart in comparison with the left, it may suggest elevated right-sided pressures as seen with pulmonary emboli and severe pulmonary hypertension (Fig. 24.44).
- Dilated cardiomyopathy: As cardiac remodeling occurs in states of dilated cardiomyopathy, the left ventricle begins to take on more of a dilated balloon-like shape rather than its normal more streamlined bullet-like shape (Fig 24.45).

Pearls

1. See "Pearls for the FAST Examination, Subxiphoid-Cardiac," item 4, above.
2. It is critical to realize the variance in the resting position of the heart. There can be significant differences in acoustic windows from patient to patient with the four-chamber apical view.

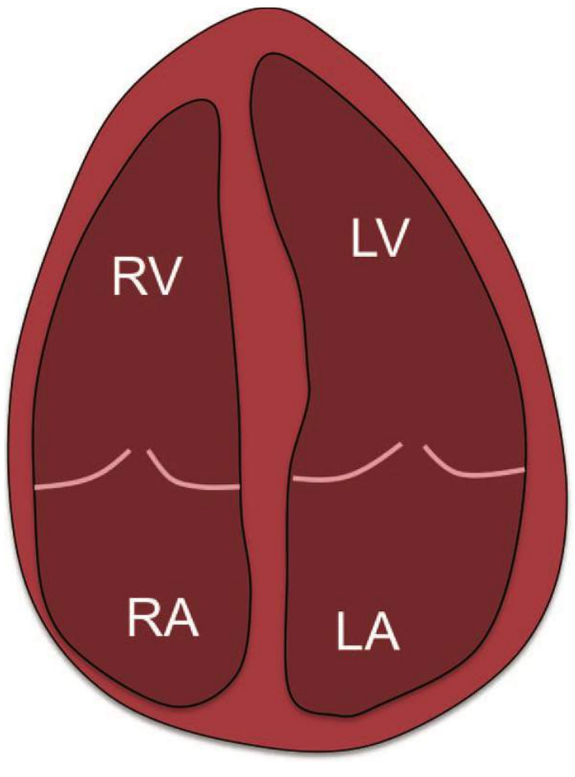


FIGURE 24.42 ■ **Apical Four-Chamber View.** The left ventricle, right ventricle, left atrium, right atrium, tricuspid, and mitral valves are seen. Ultrasound contributor: Stephen J. Leech, MD, RDMS; illustration courtesy of Robinson M. Ferre, MD.)

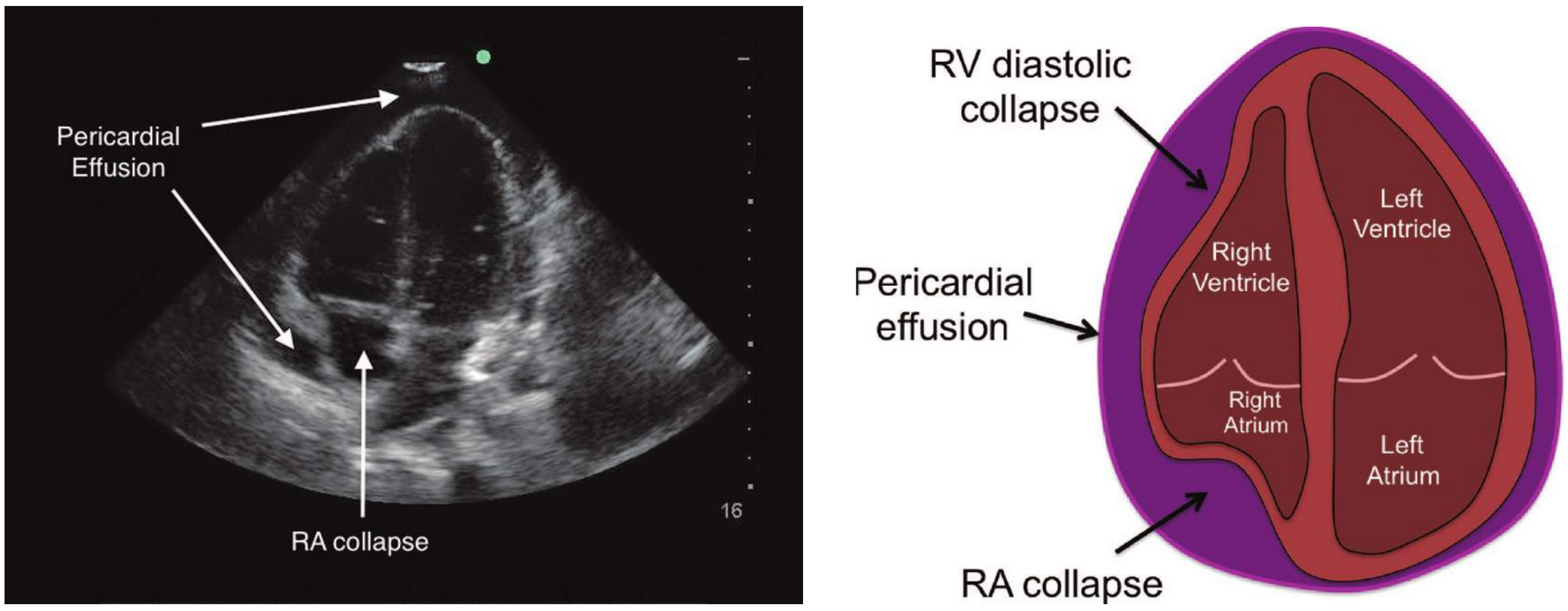


FIGURE 24.43 ■ **Apical Four-Chamber View.** A pericardial effusion appears as an anechoic (black) region between the hyperechoic pericardium and the walls of the heart. When tamponade physiology is present, right atrial and right ventricular collapse may be seen. Right atrial collapse is seen in the ultrasound image. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

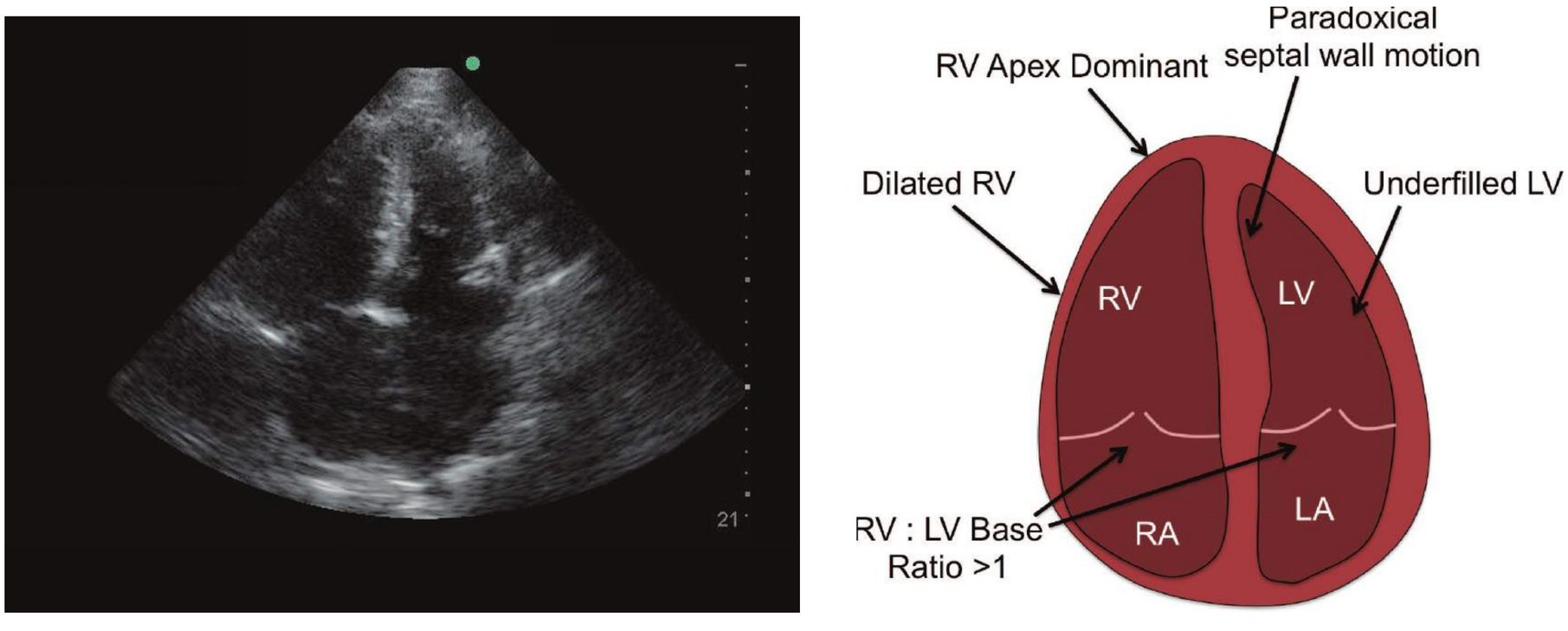


FIGURE 24.44 ■ **Apical Four-Chamber View.** A right ventricle that is equal in size or larger than the left ventricle suggests elevated right-sided pressures. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

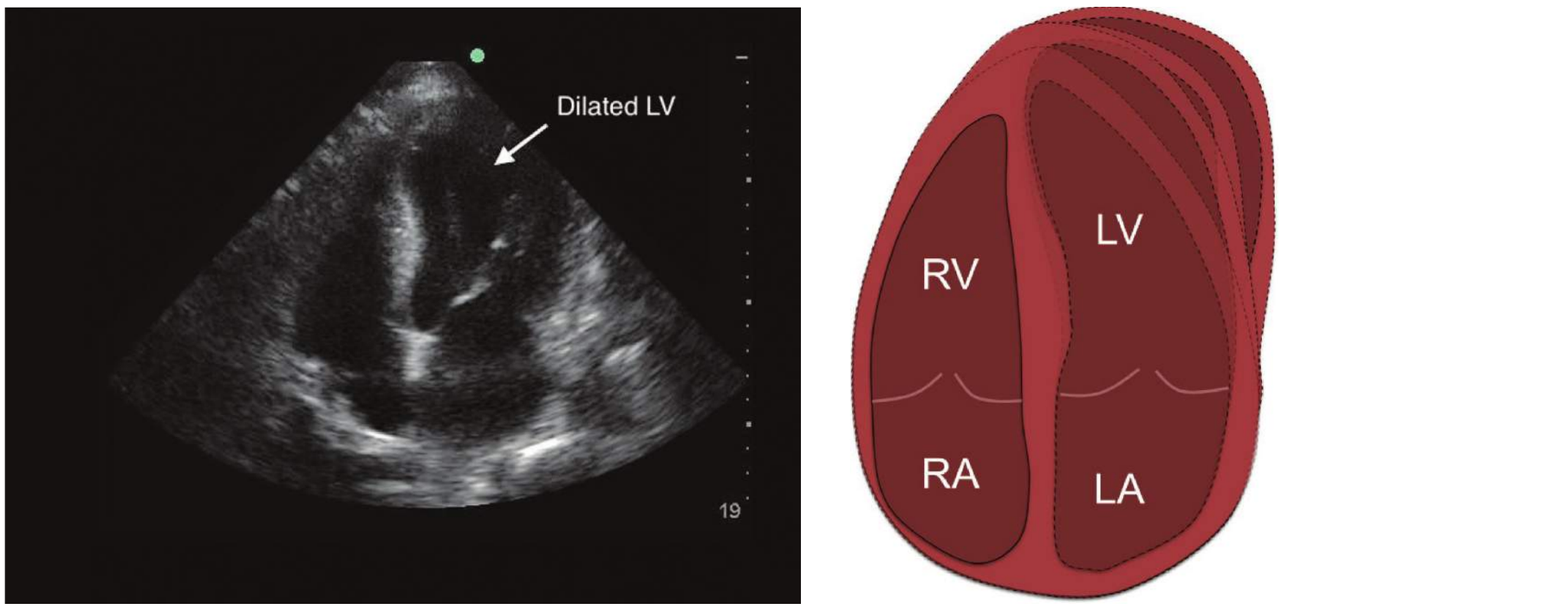


FIGURE 24.45 ■ **Apical Four-Chamber View.** As cardiac remodeling occurs in states of dilated cardiomyopathy, the left ventricle begins to take on more of a dilated balloon-like shape rather than its normal streamlined bullet-like shape. The diagram shows the progression of remodeling. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

Technique

- The transducer indicator is directed toward the patient's feet.
- The transducer is placed subxiphoid in a midline plane and tilted toward the patient's right to identify the IVC as it enters the right atrium (Fig. 24.46).
- Identify the liver, IVC, right atrium, and right hepatic vein (Fig. 24.47).
- Measure the IVC 2 to 3 cm distal to the entry point to the right atrium. Measure the IVC through the respiratory cycle at end inspiration and expiration (Fig. 24.48).

Abnormal Findings

- IVC collapse: The IVC should partially collapse during the normal respiratory cycle in nonventilated patients. A totally collapsed IVC is indicative of low central venous pressure (CVP). More than 50% collapse is less specific but also indicative of low CVP (Fig. 24.49).
- IVC size: The size of the IVC may be measured throughout the respiratory cycle. In general, an IVC that measures greater than 2 cm or greater with poor respiratory variation is indicative of elevated CVPs due to volume overload, RV failure, and/or tamponade physiology (Fig. 24.50A,B).
- IVC distensibility: In patients receiving positive pressure ventilation, the IVC will distend rather than collapse during inspiration. Measurements of IVC distensibility in ventilated patients can be used to calculate volume responsiveness to intravenous fluid challenge.

Pearls

1. Consider using M-mode through the IVC to aid in measurement for patients with high respiratory rates.
2. Some patients require a low anterior intercostal view to image the IVC as it enters the right atrium.

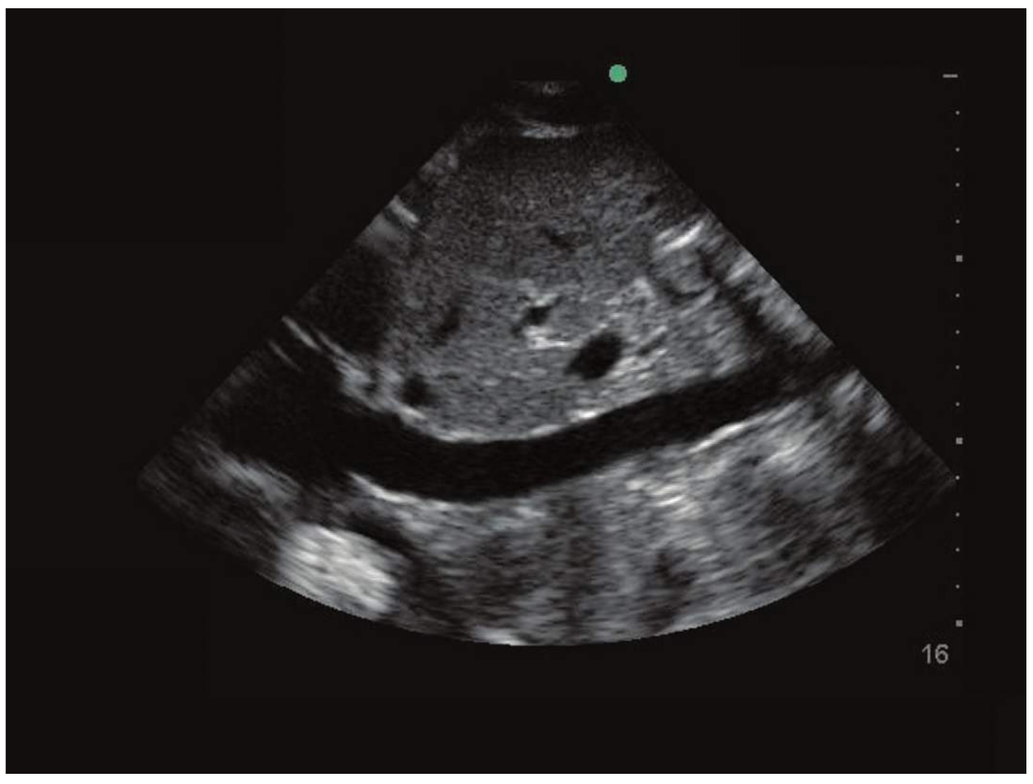


FIGURE 24.47 ■ **IVC View.** The liver is seen with the IVC entering the right atrium on the left. Probe marker (on right of the screen) is directed inferiorly here. (Ultrasound contributor: Jeremy S. Boyd, MD.)

FIGURE 24.46 ■ **IVC View.** The transducer is placed subxiphoid in a midline plane and tilted toward the patient's right to identify the IVC as it enters the right atrium. (Photo contributor: Lawrence B. Stack, MD.)

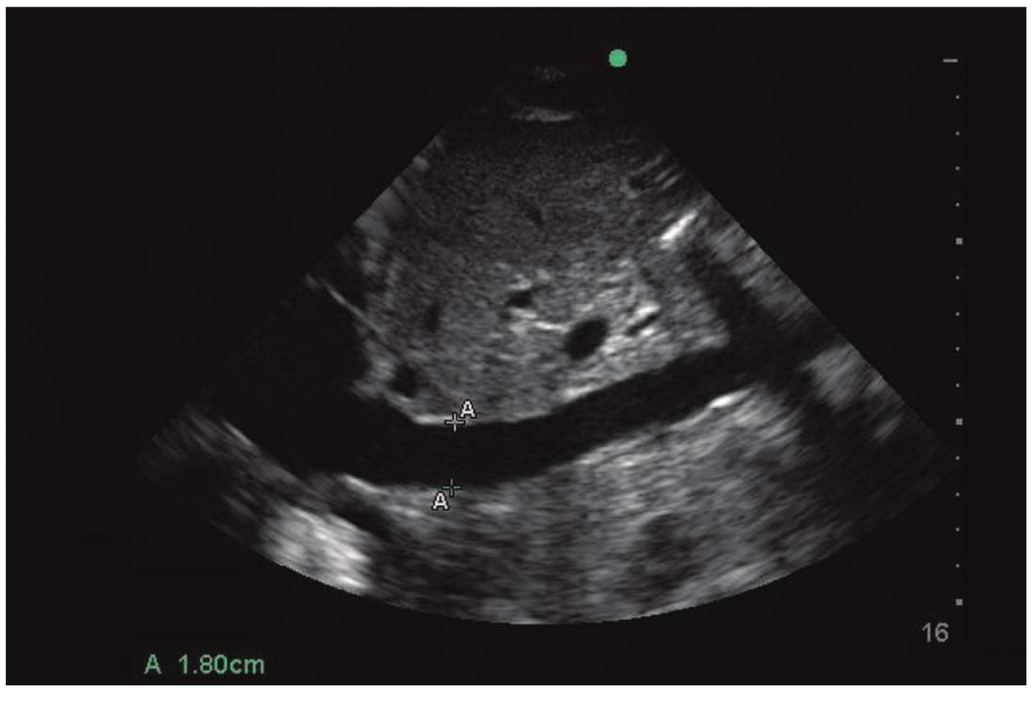


FIGURE 24.48 ■ **IVC View.** Measure the IVC 2-3 cm distal to the entry point to the RA. 1.5-2.5 cm is considered a normal IVC diameter. Greater than 2.5 cm is considered an enlarged or "plethoric" IVC. (Ultrasound contributor: Jeremy S. Boyd, MD.)

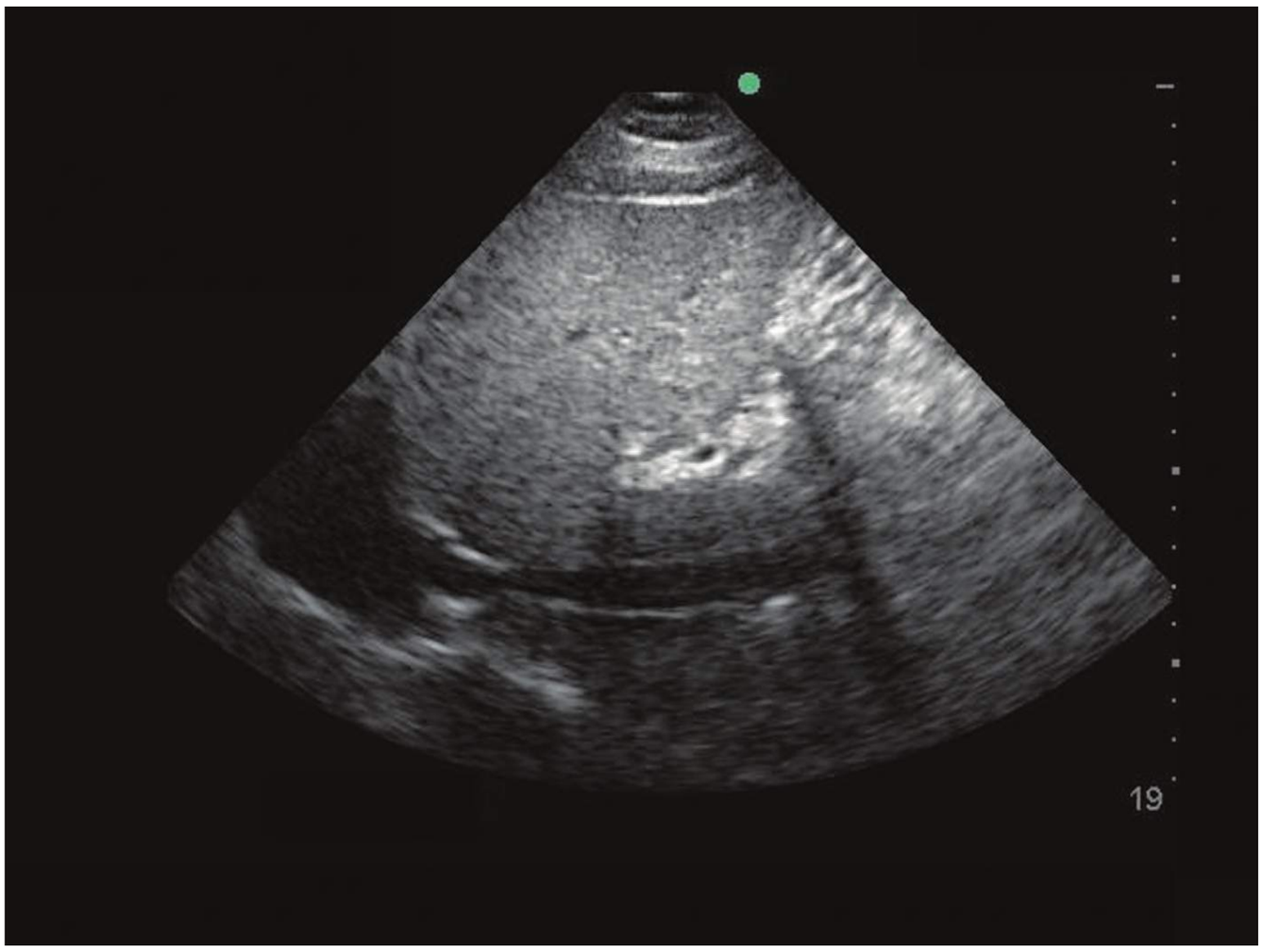


FIGURE 24.49 ■ **IVC View.** A totally collapsed IVC is indicative of low CVP. (Ultrasound contributor: Jeremy S. Boyd, MD.)

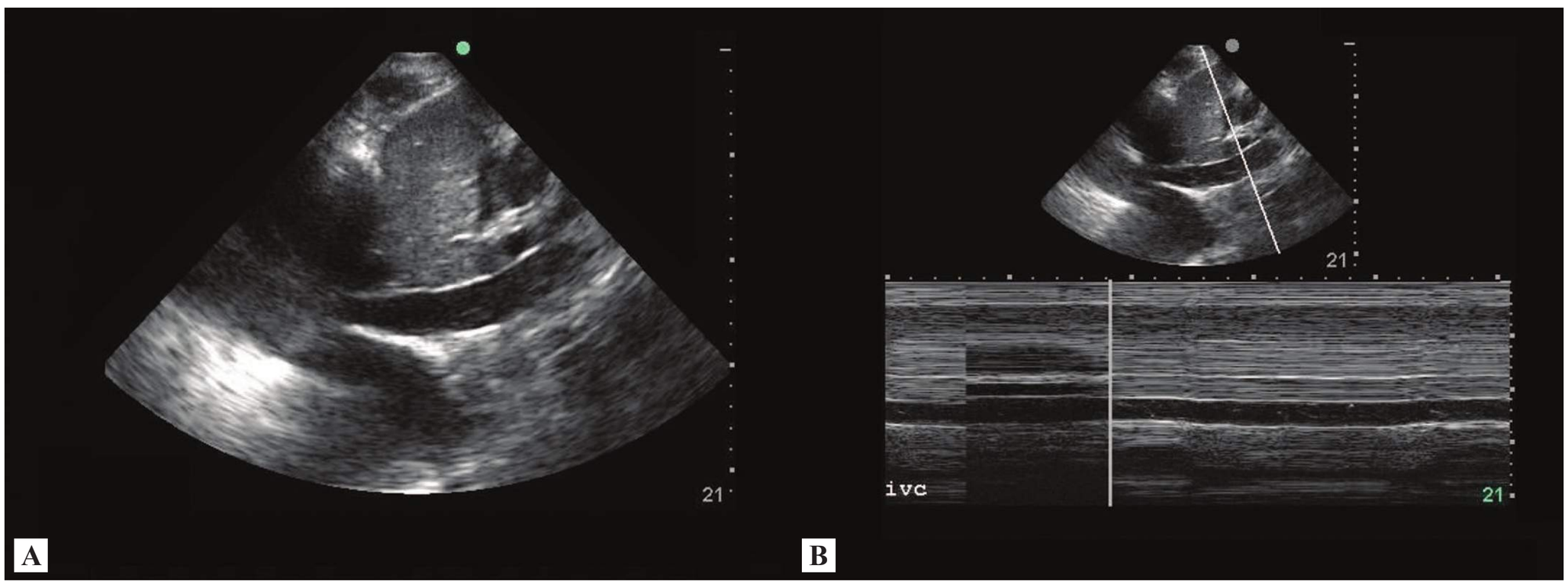


FIGURE 24.50A, B ■ **IVC View.** An IVC with poor respiratory variation (demonstrated in M-mode) is indicative of elevated central venous pressures due to volume overload, right ventricular failure, and/or tamponade physiology. (Ultrasound contributor: Jim Fiechtl, MD.)

Clinical Summary

Aerated lungs often interfere with sonographic visualization of the organs deep to them, such as often occurs in cardiac and biliary exams. This is due to the scattering effect of gas molecules on the ultrasound beam. For this reason, ultrasound of the lungs themselves was long thought to be of no diagnostic use. However, in recent years researchers and clinicians have discovered that lung ultrasound can be used to assess for pathologic conditions that occur at the interface of the lung and the chest wall such as pneumothorax, pulmonary edema, and pneumonia. The evaluation of the pleura and the finding of lung sliding to evaluate for pneumothorax was described earlier in the section on trauma ultrasound. Contemporary point-of-care lung ultrasound often evaluates for the presence of alveolar interstitial syndrome which occurs with the collection of fluid within the interlobular and intralobular lymphatics of the lung. This occurs in various pathologic states including cardiogenic pulmonary edema, acute respiratory distress syndrome, pneumonia, and diffuse parenchymal lung disease. Lung ultrasound can also effectively be used to visualize lung consolidation and pleural effusions.

Indications

- Shortness of breath and acute dyspnea
- Hypoxia
- Pleuritic chest pain
- Congestive heart failure
- Chest trauma

Required Views

- For comprehensive evaluation, current consensus recommendations include scanning the thorax in eight separate zones including the costophrenic angles of each chest (Fig. 24.51). Each hemithorax is divided into four different zones as shown in the figure.
- A more focused lung exam can be performed depending on the pathology suspected. For example, if assessing for a pneumothorax following a subclavian central venous line, anterior views along the midclavicular line would be sufficient.

Recommended Transducers

- Convex array (anterior and costophrenic angles)
- Phased array (costophrenic angles; poor for assessing lung sliding)
- Linear array (anterior chest only)

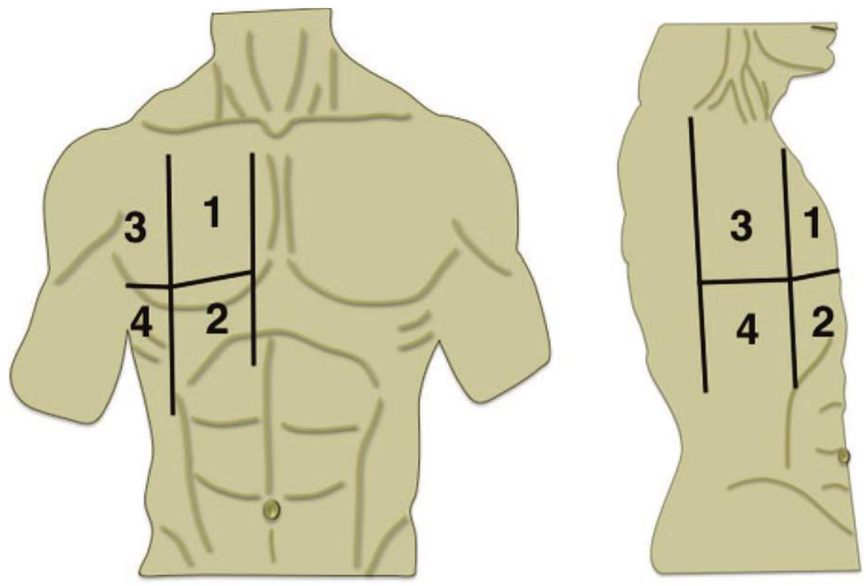


FIGURE 24.51 ■ **Lung Zones.** Current consensus recommendations include scanning the thorax in eight separate zones including the costophrenic angles of each chest. (Illustration courtesy of Robinson M. Ferre, MD.)

Patient Position

- The patient is traditionally imaged in a supine position, which is necessary if assessing for a pneumothorax.
- If assessing for conditions other than pneumothorax, a supine, semi-recumbent, or upright position may be used.

Technique

- To assess for alveolar interstitial syndrome or consolidation: With the indicator pointed toward the patient's head, each of the four zones of each hemithorax should be imaged. The depth on the screen should be between 13 and 15 cm deep. Each zone should be imaged thoroughly for evidence of A-lines, B-lines, or consolidation.
- To assess for pleural fluid: The technique for evaluating costophrenic angles for pleural effusion is identical to that employed in the E-FAST when evaluating for hemothorax.
- To assess for a pneumothorax: The technique for evaluating costophrenic angles for pleural effusion is identical to that employed in the E-FAST when evaluating for hemothorax.

Findings

- A-lines: Normal aerated lung is indicated by the presence of "A-lines" and lung sliding. A-lines are echogenic horizontal lines deep and parallel to the pleural line that occur at regular intervals and do not move with respiration or lung sliding. (Fig. 24.52)
- B-Lines: The presence of "B-lines" indicates alveolar interstitial syndrome. B-lines are defined as laser-like

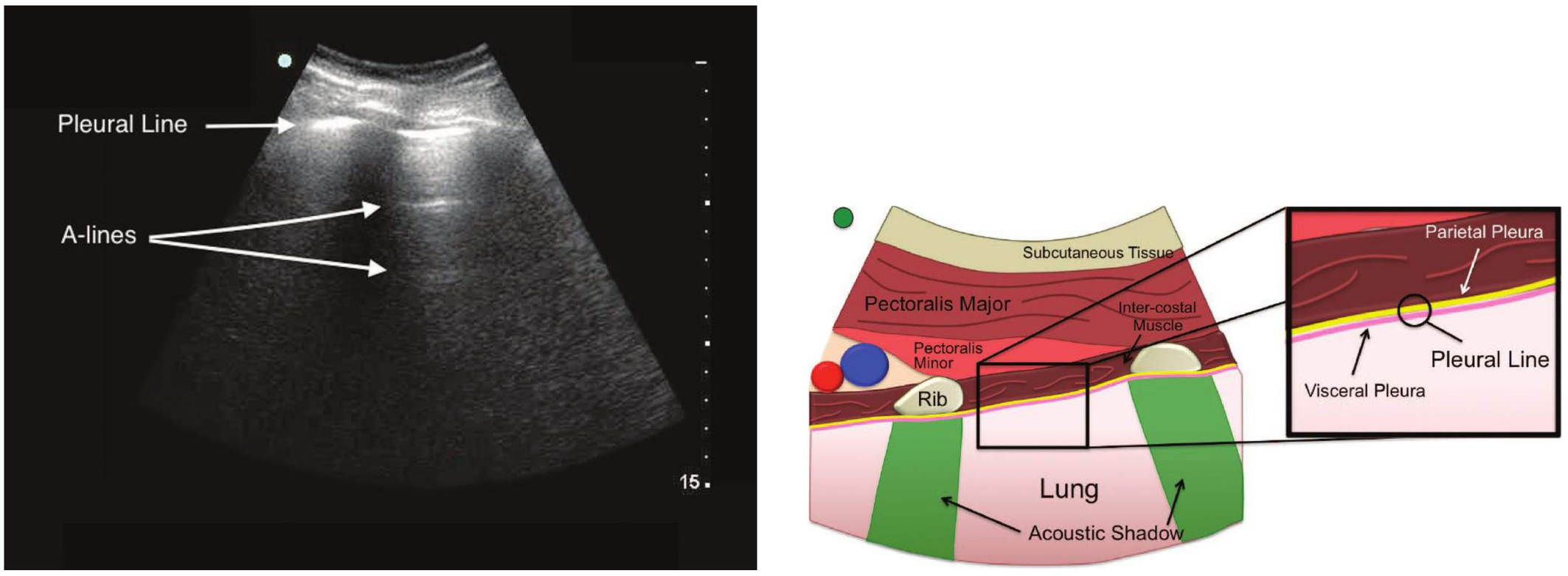


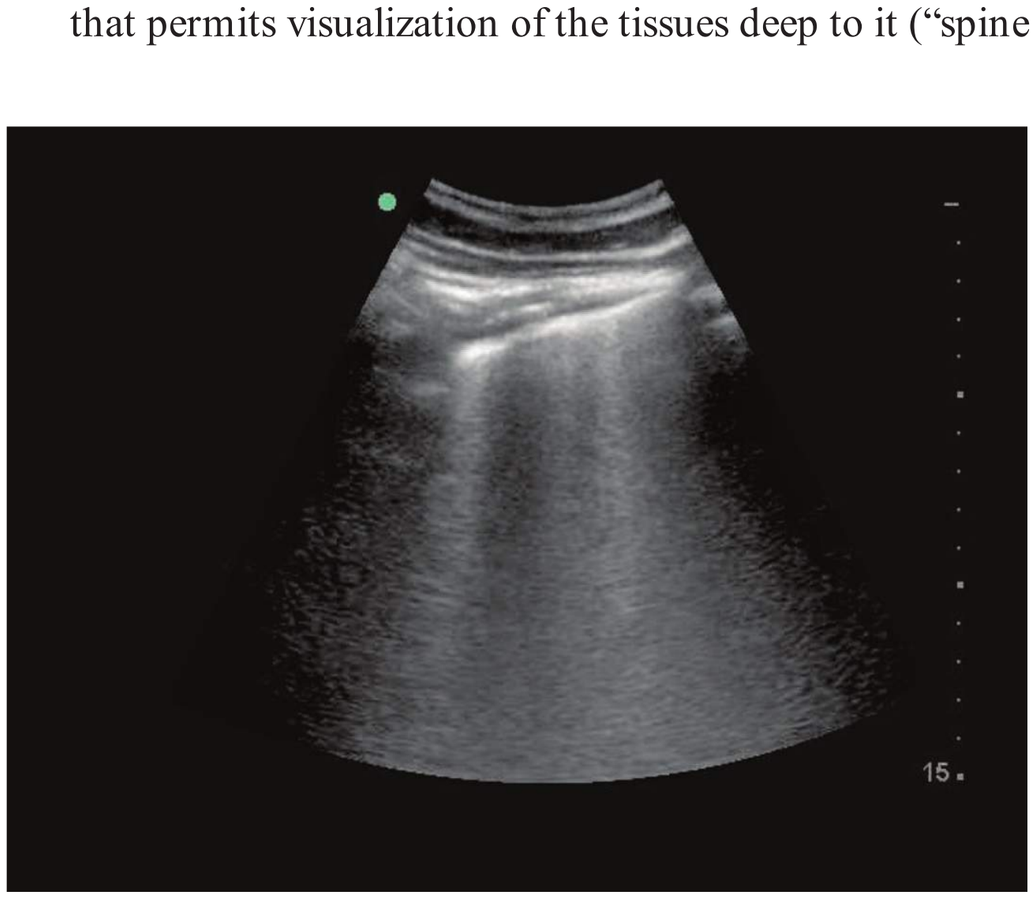
FIGURE 24.52 ■ **Lung Ultrasound: A-Lines.** Normal aerated lung is indicated by the presence of "A-lines" and lung sliding. A-lines are echogenic horizontal lines deep and parallel to the pleural line that occur at regular intervals and do not move with respiration. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

artifacts that begin from the pleural line and extend to the bottom of the screen without fading and move in conjunction with respiration or lung sliding (Fig. 24.53).

- Consolidation: Consolidation of lung tissue appears as a subpleural area of tissue-like echotexture. It may have comet-tail artifacts extending from the far-field border, a pattern called the "shred sign." Air and/or fluid bronchograms may also be noted within the consolidation (Fig. 24.54).
- Pleural effusion: A hypoechoic area located in the diaphragmatic recess superior/cephalad to the diaphragm that permits visualization of the tissues deep to it ("spine sign") is a pleural effusion. Consolidated lung can often be seen moving within the effusion in synchrony with the patient's respirations (Fig. 24.55).

Pearls

- The distribution of B-lines, whether diffuse, focal, unilateral, or bilateral, in conjunction with the clinical exam and history can help to determine the etiology of the patient's symptoms. Cardiogenic pulmonary edema will be found diffusely and bilaterally, whereas a focal pneumonia or



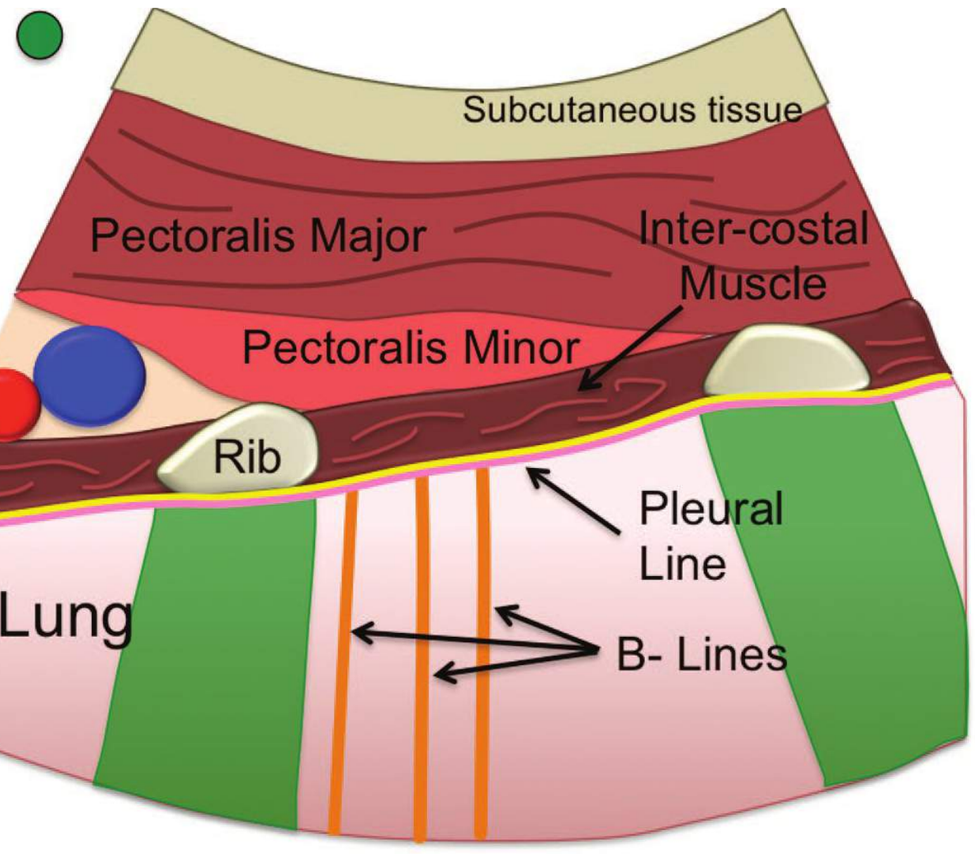


FIGURE 24.53 ■ **Lung Ultrasound: B-Lines.** The presence of "B-lines" indicates alveolar interstitial syndrome. B-lines are defined as laser-like artifacts that begin from the pleural line and extend to the bottom of the screen without fading and move in conjunction with respiration. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jordan Rupp, MD.)

pulmonary contusion may only present within a focal region of the ultrasound lung exam.

- In patients with cardiogenic pulmonary edema, the number and frequency of B-lines correlate with the severity of edema and can also be used to monitor the response to therapy.
- When performing an E-FAST exam, the linear probe is frequently used as it is most sensitive for lung sliding and visualization of the pleura. When using a different probe to evaluate the lungs, adjust the depth to a shallow setting to ensure adequate evaluation of the pleura for lung sliding. The remainder of the exam should be performed with a depth of at least 13 cm when assessing for B-lines and consolidation.

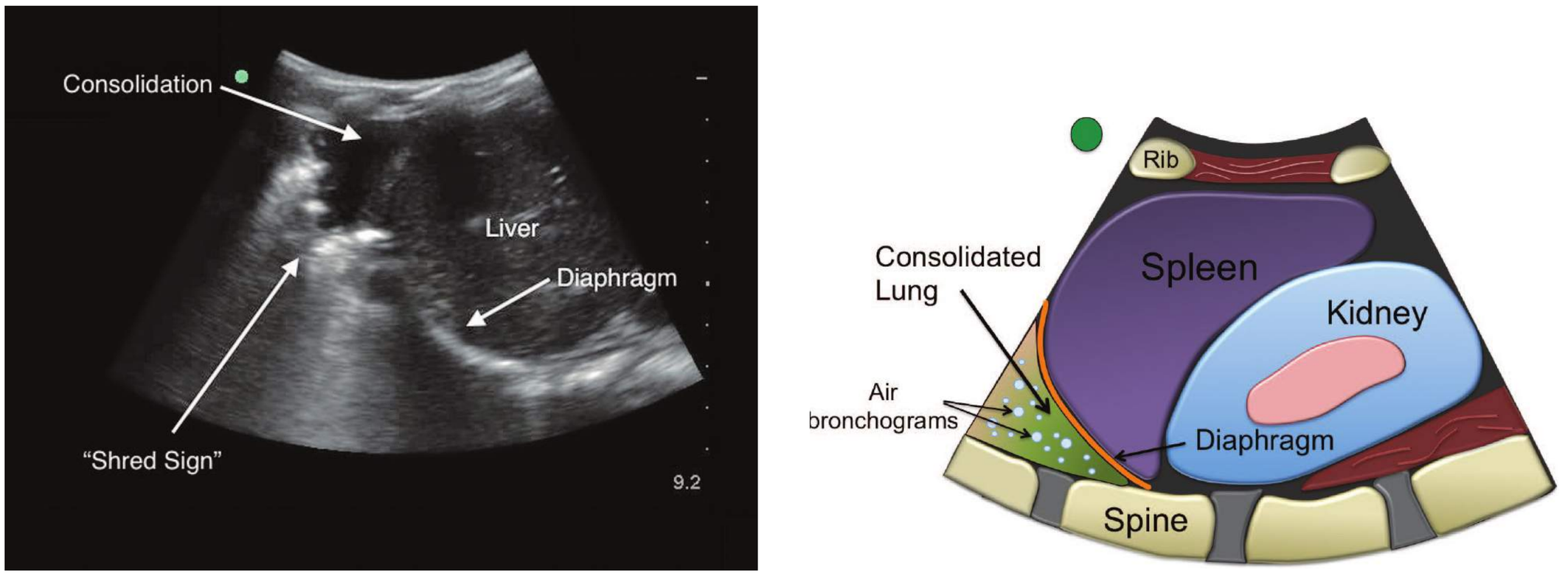


FIGURE 24.54 ■ **Lung Ultrasound**. Consolidation of lung tissue appears as a subpleural area of tissue-like echotexture. It may have comet-tail artifacts extending from the far field border, a pattern called the "shred sign," seen in this image of a pneumonia. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

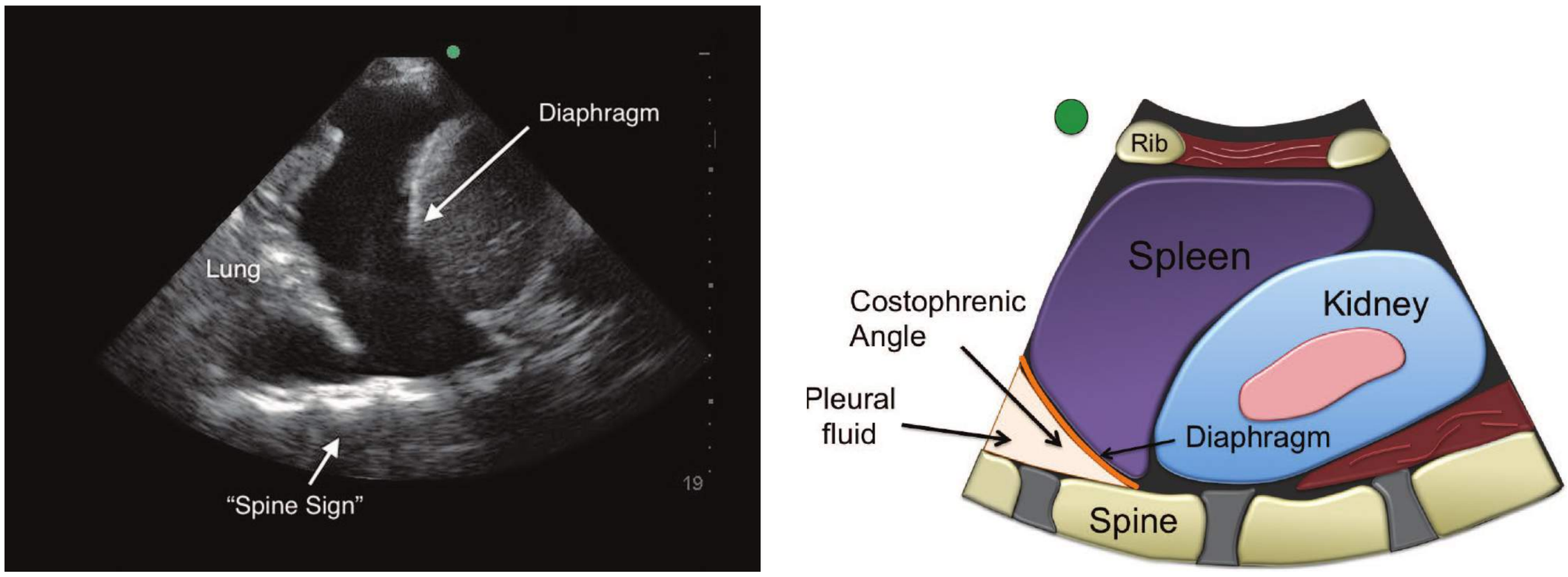


FIGURE 24.55 ■ **Lung Ultrasound.** A pleural effusion is seen as a hypoechoic area located in the diaphragmatic recess superior/cephalad to the diaphragm with the spine sign seen extending beyond the diaphragm into the thorax. Consolidated lung is seen within the effusion. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

ABDOMINAL AORTA ULTRASOUND

Clinical Summary

EUS of the abdominal aorta is used to diagnose or exclude an abdominal aortic aneurysm (AAA). Though the sensitivity of abdominal ultrasound for the detection of aortic dissection is limited, the presence of an intra-aortic flap is diagnostic for aortic dissection.

Indications

- Abdominal, back, or flank pain
- Pulsatile abdominal mass
- Undifferentiated hypotension

When a patient is unstable, there is no bedside test superior to an EUS of the abdominal aorta to diagnose an AAA. Early diagnosis can improve patient survival. An abdominal aortic measurement greater than 3 cm in diameter is abnormal and diagnostic of an AAA. While an AAA may rupture at any size, the risk of rupture is much greater starting with measurements greater than 5 cm. AAAs occur as both fusiform (more common) and saccular types. It is essential to image the aorta in both sagittal and transverse planes.

Views for Emergency Department Abdominal Aortic Ultrasound

1. Transverse view
2. Sagittal view

Equipment: Recommended Transducer for Abdominal Aortic Ultrasound

- Convex array

Patient Position

The patient is supine.

Techniques

Transverse View

- Place the transducer in the epigastrium with the transducer indicator oriented to the patient's right (Fig. 24.56).
- Identify the acoustic shadow caused by the vertebral body. The abdominal aorta is the circular, pulsating, anechoic structure anterior and slightly to the patient's left.
- Identify and measure the aorta at the proximal (at or above the origin of the superior mesenteric artery [SMA]), middle (near the renal arteries), and distal (immediately proximal to the bifurcation) in the anteroposterior plane (Figs. 24.57 and 24.58).
- Identify the aorta in relation to the IVC, SMA, and splenic vein.
- Move down the abdominal aorta to the bifurcation (approximately the level of the umbilicus) (Fig. 24.59).

Sagittal View

- Place the transducer in the epigastrium with the transducer indicator oriented toward the patient's head. Move down the abdominal aorta to the bifurcation (Fig. 24.60).
- Identify the liver, aorta, IVC, celiac trunk, and SMA (Fig. 24.61).

FIGURE 24.56 ■ **Abdominal Aorta Transverse View.** Place the transducer in the epigastrium with the transducer indicator oriented to the patient's right. (Photo contributor: Lawrence B. Stack, MD.)

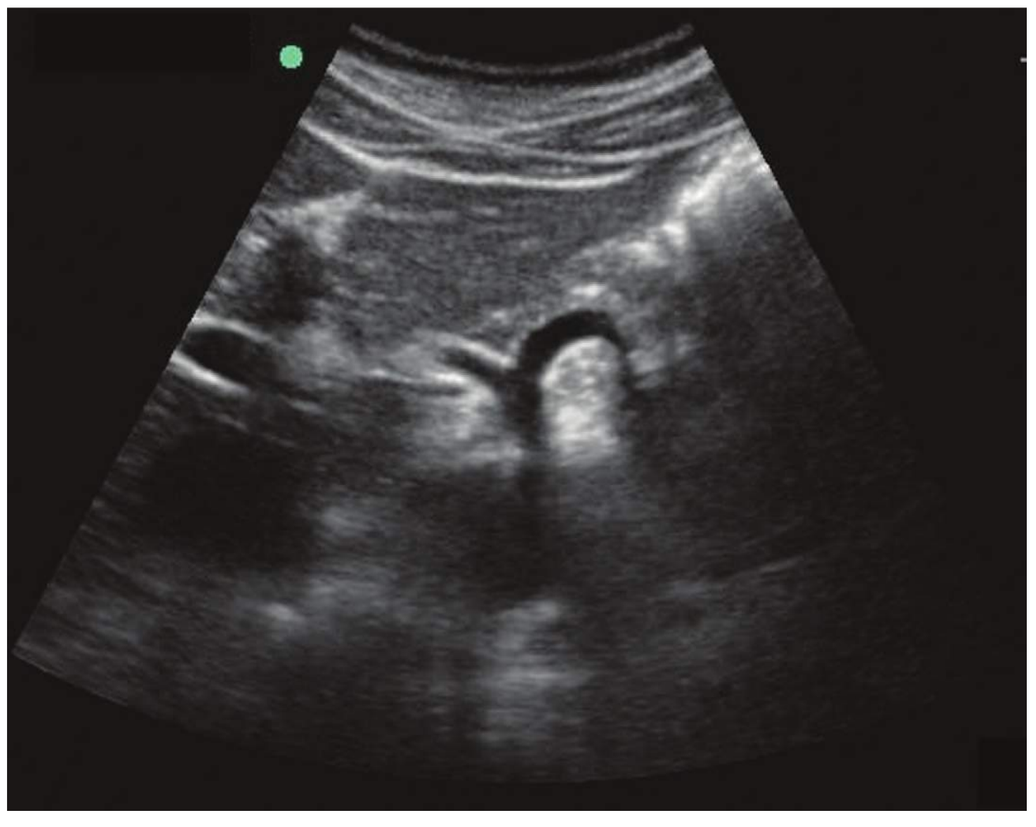


FIGURE 24.57 ■ **Abdominal Aorta: Transverse View.** Short-axis view of the proximal aorta showing the celiac artery and its branches. (Ultrasound contributor: Jeremy Boyd, MD.)

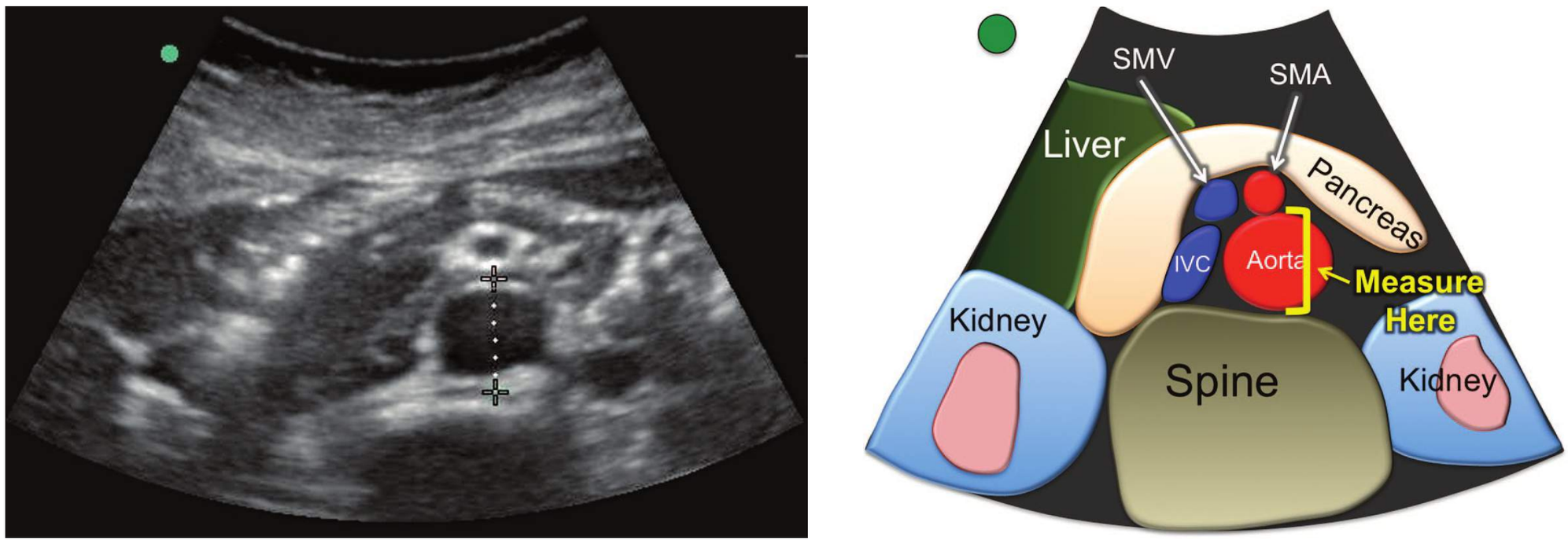


FIGURE 24.58 ■ **Abdominal Aorta: Transverse View.** The proximal aorta, superior mesenteric artery (SMA), and the surrounding anatomy are often compared to a mantle clock. (Ultrasound contributor: Eric Wu, MD; illustration courtesy of Robinson M. Ferre, MD.)

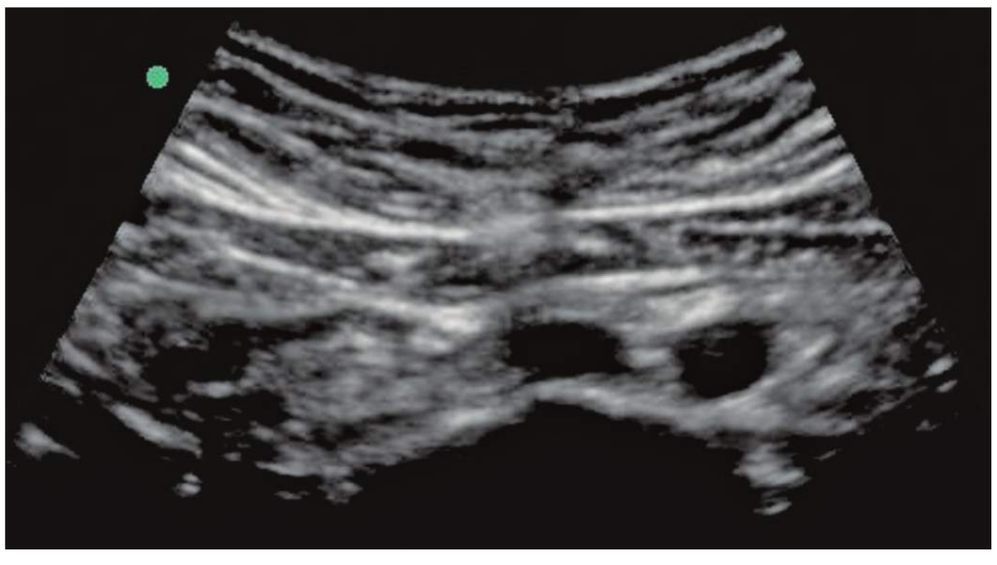
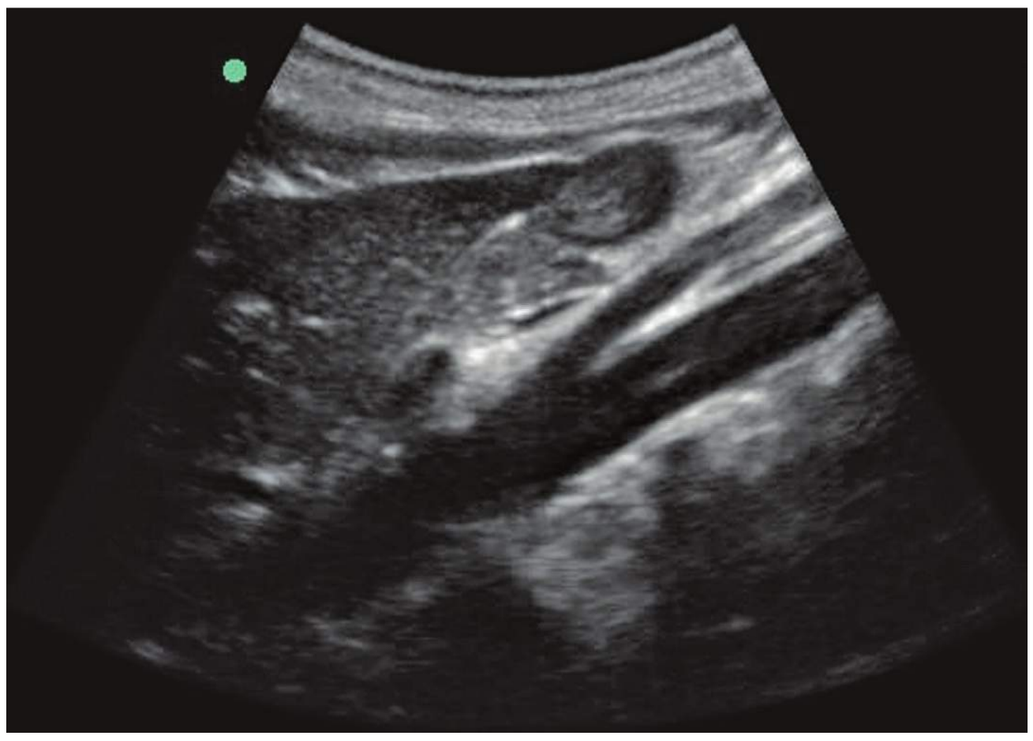


FIGURE 24.59 ■ **Abdominal Aorta: Transverse View.** Still image of the left and right iliac arteries just distal to the bifurcation of the aorta. (Ultrasound contributor: Frances Cheng, MD.)

FIGURE 24.60 ■ **Abdominal Aorta Sagittal View.** Place the transducer in the epigastrium with the transducer indicator oriented toward the patient's head. (Photo contributor: Lawrence B. Stack, MD.)



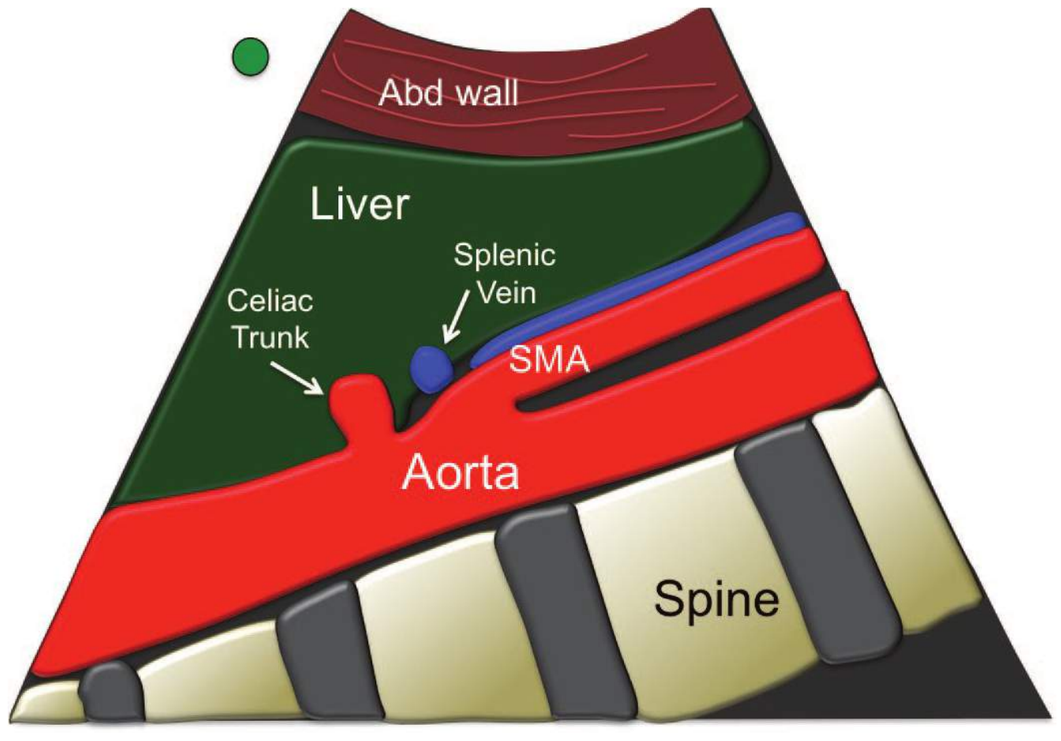


FIGURE 24.61 ■ **Abdominal Aorta.** Sagittal view, proximal aorta. (Ultrasound contributor: Jeremy Boyd, MD, RDMS; illustration courtesy of Robinson M. Ferre, MD.)

Abnormal Findings

- AAA: The abdominal aorta is aneurysmal if at any point it measures more than 3 cm. The aorta can also be aneurysmal if a distal segment measures more than 50% of a proximal segment (Fig. 24.62).
- Aortic dissection: The echogenic line of an aortic "flap" can be seen inside the lumen of the abdominal aorta when present (often moving with pulsations) (Fig. 24.63).

Pearls

1. Starting the exam in the transverse view will help orient the scanner to the anatomy.
2. Measure the entire diameter (outer wall to outer wall) of the aorta. Measuring the lumen is grossly inaccurate and will underestimate the size of an AAA when a clot is present (Figs. 24.64 and 24.65). Include measurements of the proximal, mid, and distal aorta.
3. If a significant amount of bowel gas is present, sit the patient at 45 degrees and apply constant gentle pressure.
4. The IVC may be confused for the aorta, especially in sagittal views. The "sniff test"—having the patient abruptly sniff—will cause the IVC to collapse as a result of the negative pressure transmitted to the venous system during this maneuver.
5. If spectral or color flow Doppler is available, it may be used to discriminate between the highly pulsatile flow of the aorta and the low amplitude of the IVC.
6. In patients with an aortic dissection, an intimal flap can be visible as a linear structure within the aorta. While this finding is highly specific, it is insensitive and its absence cannot be used to rule out a dissection (Fig. 24.63).

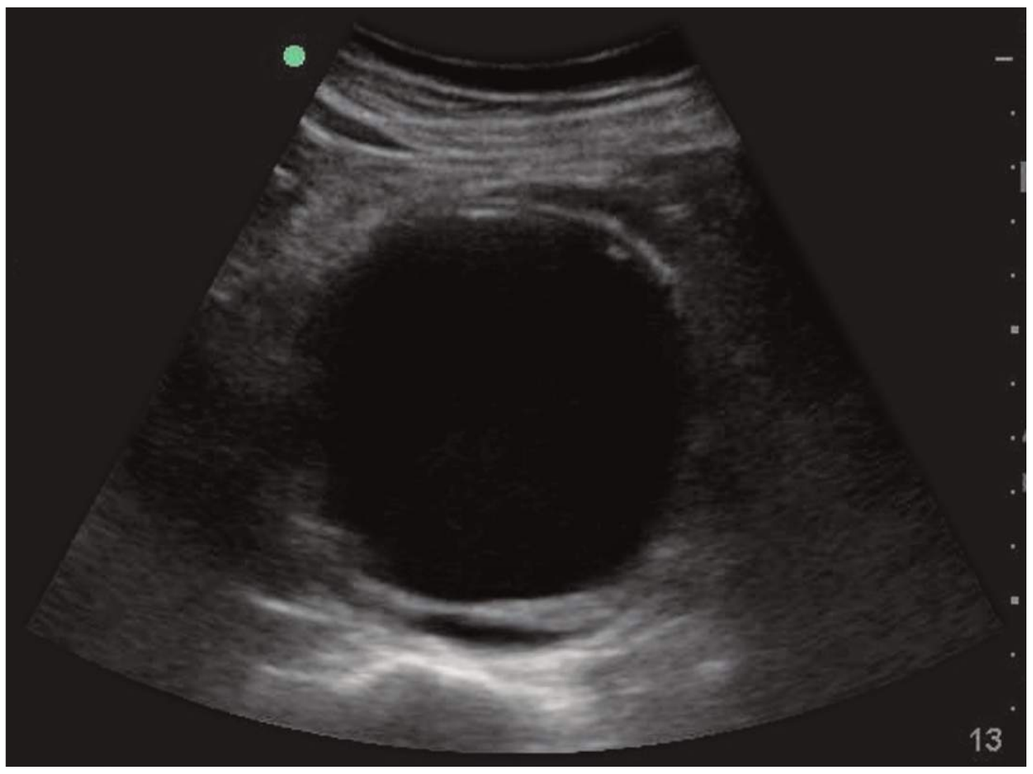


FIGURE 24.62 ■ **Abdominal Aorta: Transverse view, AAA.** Large proximal abdominal aortic aneurysm. (Ultrasound contributor: Lara Philips, MD.)

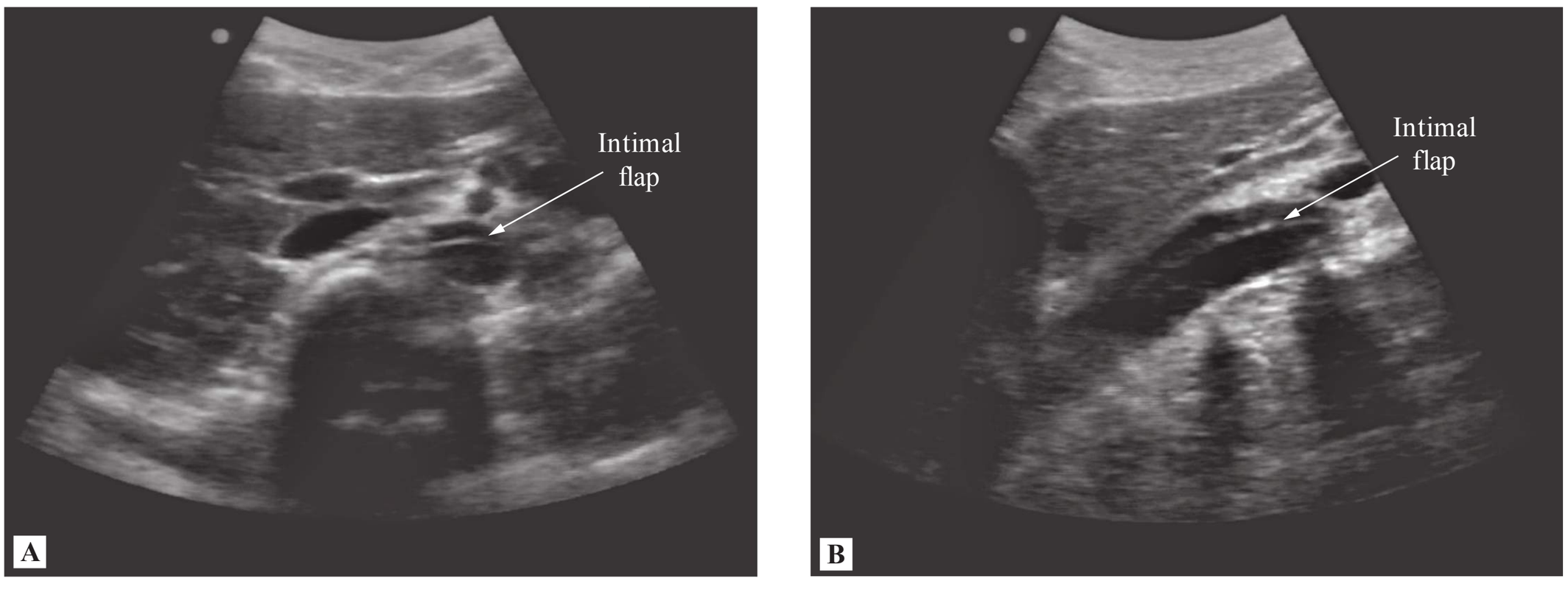


FIGURE 24.63 ■ **Abdominal Aorta: Dissection.** An aortic dissection flap is seen. (Ultrasound contributor: Paul R. Sierzenski, MD, RDMS.)

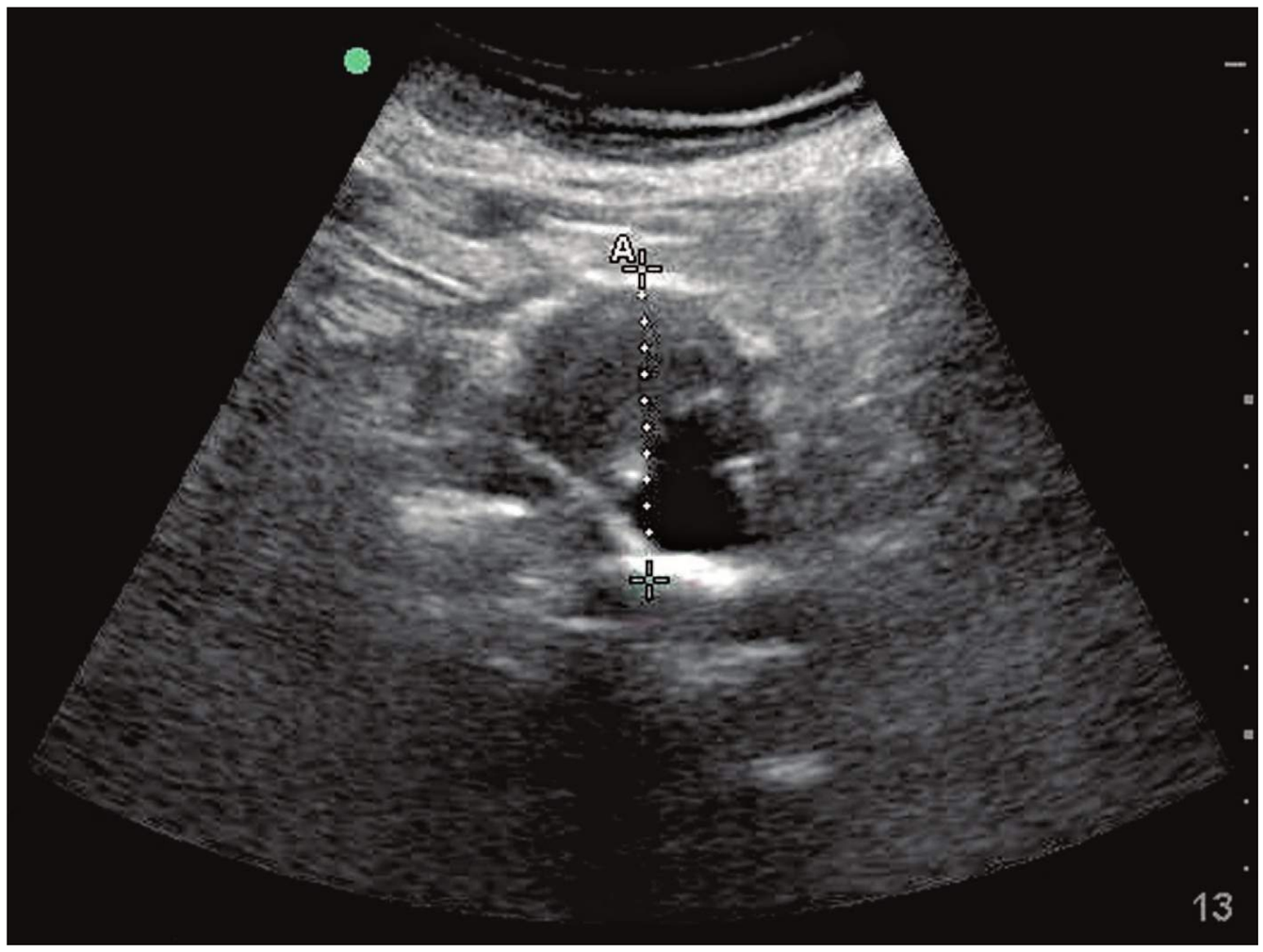


FIGURE 24.64 ■ **Abdominal Aorta: Transverse View, AAA.** Mid-aorta showing an abdominal aortic aneurysm with clot partially occluding the vessel. (Ultrasound contributor: Louis Frazier, MD.)

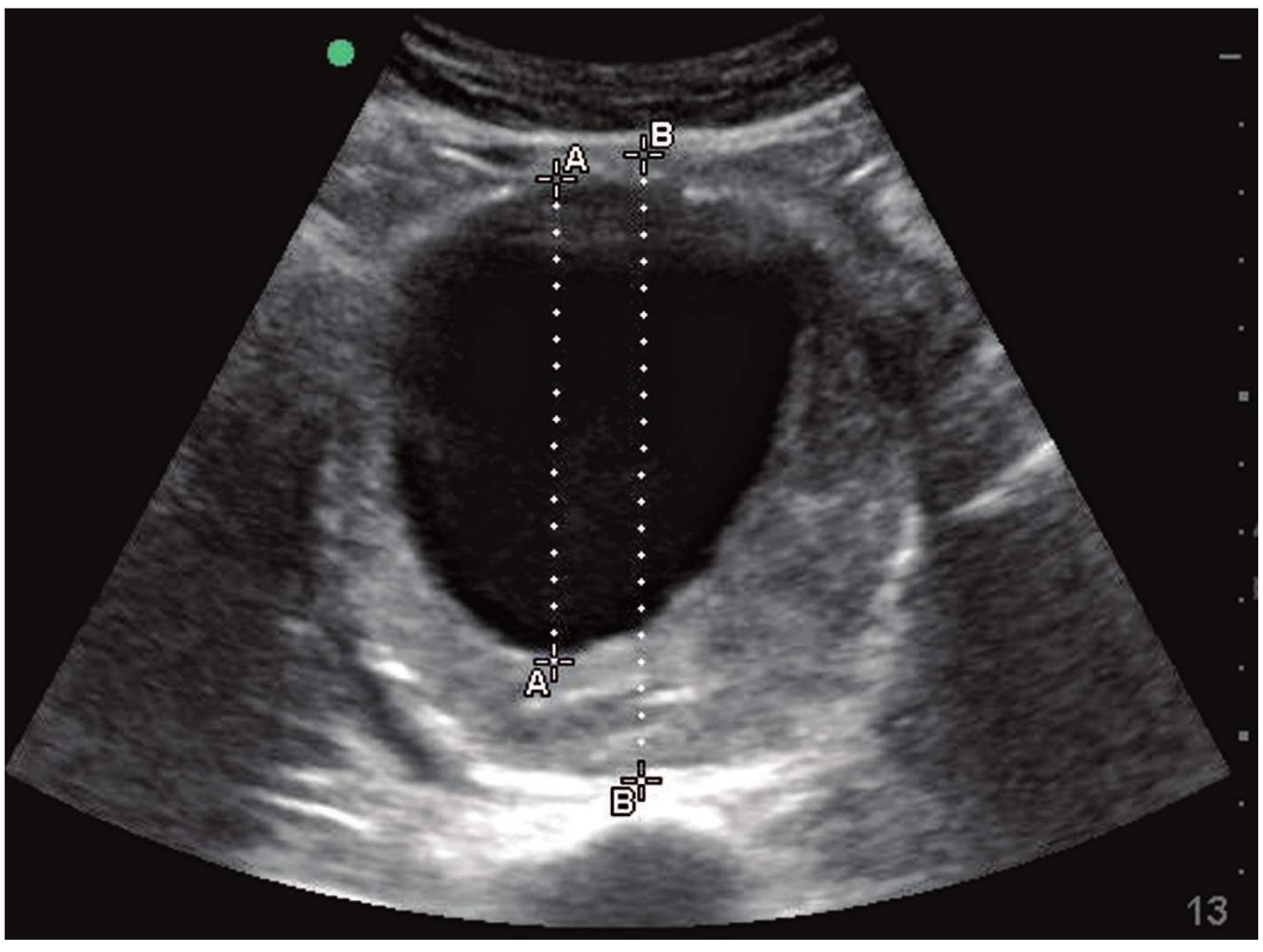


FIGURE 24.65 ■ **Abdominal Aorta: Transverse View, AAA.** Distal abdominal aortic aneurysm with clot filling a portion of the vessel. (Ultrasound contributor: Christopher Noel, MD.)

DEEP VENOUS THROMBOSIS ULTRASOUND

Clinical Summary

The presence of a deep venous thrombosis (DVT) is part of the differential diagnosis of a variety of signs and symptoms. Although clinical scoring algorithms have been developed to gauge risk, no clinical findings are conclusive of this condition and imaging is necessary to confirm the diagnosis. Ultrasound is a sensitive, noninvasive imaging modality that can be performed rapidly and essentially without contraindication. Radiology-performed studies are not always readily available emergently, thus an emergency physician properly trained in bedside ultrasonography can help guide and hasten care by performing a limited serial compression examination of the proximal leg veins. This exam has been shown to be highly sensitive for diagnosis of DVT in symptomatic ED patients when performed by properly trained individuals.

Indications

- Extremity swelling, pain, or erythema
- Dyspnea
- Chest pain

Required Views for Lower Extremity DVT Ultrasound

- Common femoral vein (CFV) (Fig. 24.66)
- Superficial femoral vein (SFV) (Fig. 24.67)
- Popliteal vein (PV) (Fig. 24.68)

Equipment: Recommended Transducers for DVT Ultrasound

- Linear
- Convex

Patient Preparation

- Ideally, the patient is recumbent with the head of the bed elevated 20 to 40 degrees for lower extremity imaging.
- External rotation of the patient's hip with partial knee flexion will aid imaging, especially the popliteal vessels. If tolerated, having the patient lie prone may also aid in imaging the popliteal vasculature.

FIGURE 24.66 ■ **DVT Ultrasound, CFV View.** The transducer should be oriented in a transverse plane just distal to the inguinal ligament. The vein is gently but firmly compressed until the walls completely collapse. (Illustration courtesy of Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Robinson M. Ferre, MD.)

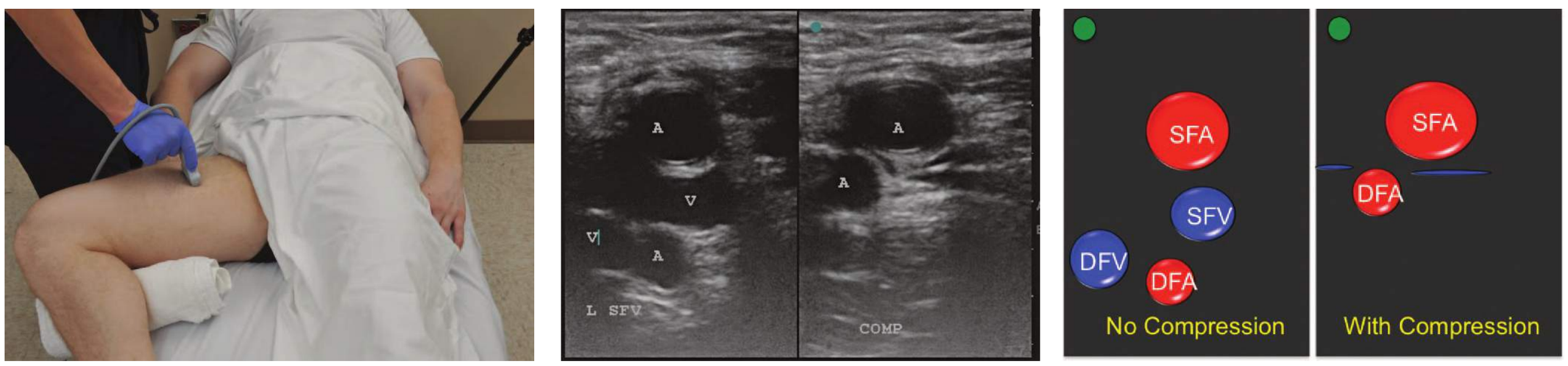


FIGURE 24.67 ■ **DVT Ultrasound, SFV View.** The transducer should be oriented in a transverse plane. The vein(s) is/are gently but firmly compressed until the walls completely collapse. (Illustration courtesy of Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Robinson, M. Ferre, MD.)

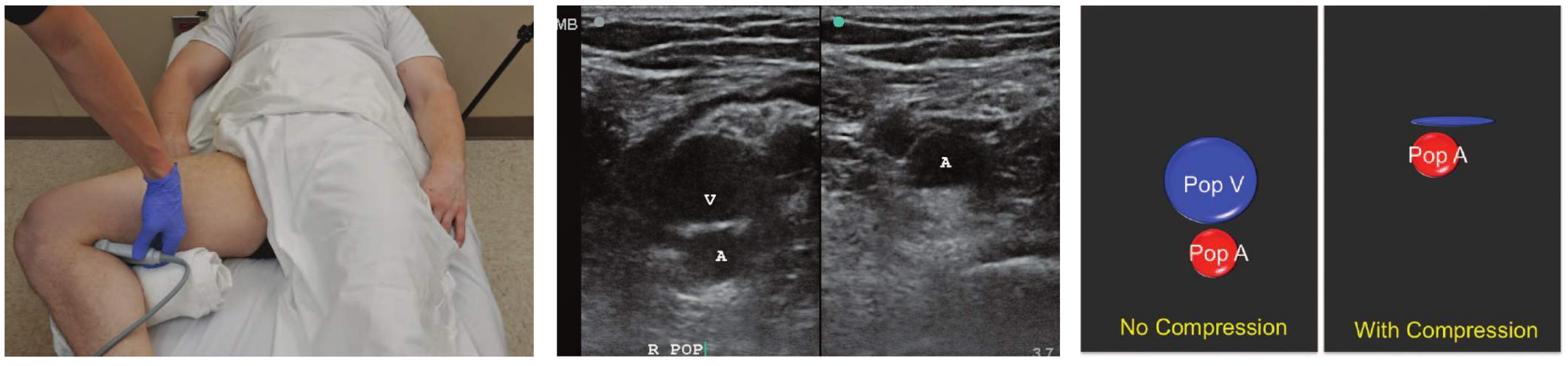


FIGURE 24.68 ■ **DVT Ultrasound, Popliteal View.** The transducer should be oriented in a transverse plane behind the popliteal fossa. The vein is gently but firmly compressed until the walls completely collapse. The popliteal vein is usually found superficial to the popliteal artery, ie, "pop on top." (Illustration courtesy of Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Suzanne Bryce, MD.)

Technique

Lower Extremity

- The transducer should be oriented in a transverse plane through the course of the vein.
- Imaging begins at the proximal CFV moving distally to the PV. Carefully identify the CFV, SFV, and PV (Fig. 24.69).
- The vein is gently but firmly compressed until the walls completely collapse. This process is repeated every 1 to 2 cm throughout each site.
- Color or spectral Doppler can be used at the CFV and PV to document phasicity with respiration or augmentation.

Abnormal Findings

- Inability to compress the vein suggests DVT. This may be acute, chronic, or due to technical limitations (eg, unable to apply enough pressure) (Fig. 24.70).
- Poor or absent phasicity suggests occlusion at a location proximal to transducer.
- Poor or absent augmentation suggests occlusion at a location distal to transducer.

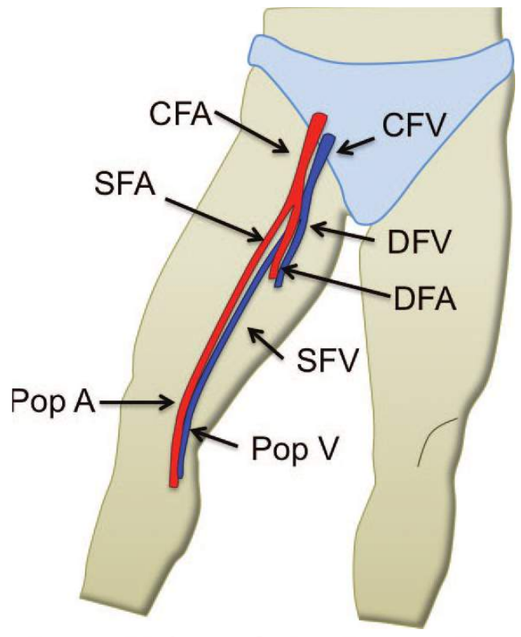


FIGURE 24.69 ■ **Deep Veins of the Lower Extremity.** Diagram illustrating the anatomy of the deep venous system and accompanying arterial system of the lower extremity. Note that the superficial femoral vein (SFV) is actually considered a deep vein of the extremity, despite its name. (Illustration courtesy of Robinson M. Ferre, MD.)

Pearls

1. The CFV is often located more proximally than new sonographers expect. Start the exam adjacent to the inguinal ligament.
2. Thoroughly evaluate the CFV at the confluence of the saphenous vein.
3. Ensure that the vascular structure is a deep vein. Deep veins are paired with arteries. Veins are easily compressible and arteries are not. Color or spectral Doppler should demonstrate characteristic arterial or venous waveforms.
4. If unable to adequately compress vessels, apply pressure from under the extremity toward the transducer. If the artery begins to distort its shape, adequate pressure has been applied.
5. Echogenic material within the vein may be an artifact and not a DVT. Visualize any abnormality in more than one plane and optimize settings to exclude artifacts.

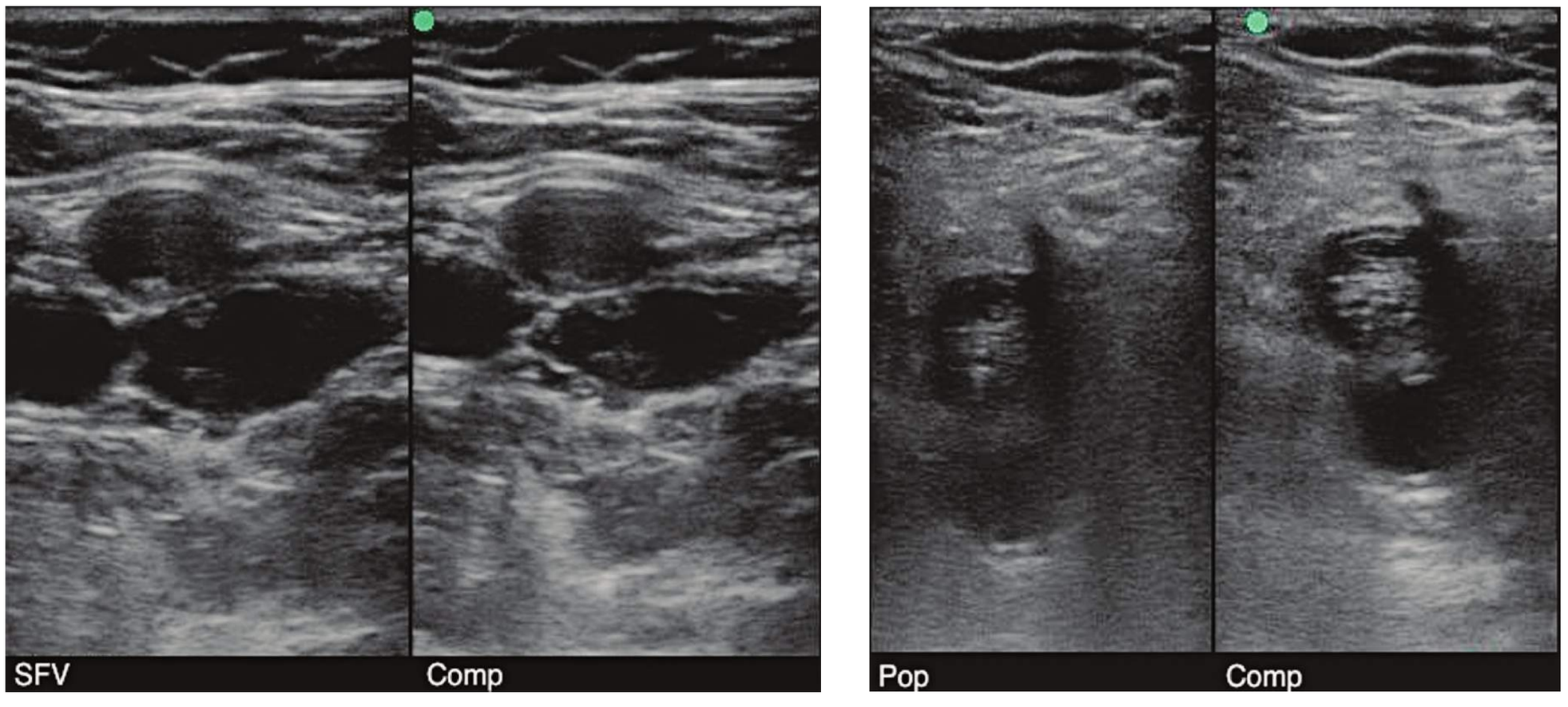


FIGURE 24.70 ■ **DVT Ultrasound.** A deep venous thrombosis is seen as a hyperechoic clot within the SFV (left) and popliteal vein (right) preventing compression. (Ultrasound contributor: Robinson M. Ferre, MD.)

Clinical Summary

As ultrasound is integrated into the bedside evaluation of many emergent symptoms and disease states, multiple protocols have emerged for the rapid assessment of critically ill patients in shock and cardiac arrest states. By combining many of the exams described previously in this chapter, one can quickly assess for the cause of a patient's hypotension or cardiac arrest, helping to sort through what is often an extensive differential diagnosis. A common approach is termed the "RUSH" protocol—Rapid Ultrasound for Shock and Hypotension. Though there are distinct protocols proposed by different authors, they combine a sonographic assessment of a patient's gross cardiac function, intravascular volume status, and identification of pathologic conditions (such as AAA or pneumothorax) that threaten a patient's hemodynamic stability. One group of authors promotes the use of the heuristic "Pump, Tank, and Pipes" as the mental framework for this evaluation, while another uses the mnemonic "HI-MAP" (heart, IVC, Morison [and other FAST views], aorta, and pulmonary/pneumothorax) to outline the protocol. Regardless of which specific protocol one uses for evaluation of the hypotensive patient, when combined with a history and physical exam, bedside ultrasound can be used to guide resuscitative efforts and tailor them to the type of shock encountered.

In addition, when confronted with a patient in cardiac arrest, bedside ultrasound can be used during CPR to help guide therapies and decision making. The protocols described previously can be used to search for the cause of a patient's arrest. Additionally, the subxiphoid and apical four-chamber windows can even be used during compressions or rapidly during pulse checks to determine the native function of the heart and distinguish between true PEA or "electromechanical dissociation" (where there is an electrical rhythm conducted but no cardiac activity) and "pseudo-PEA" where the heart's contractions are so poor that they do not produce palpable pulsations. One can sometimes even distinguish between asystole and fine ventricular fibrillation on the monitor by looking closely at the mitral valve for motion during a pulse check. Alternatively, some emergency physicians are utilizing transesophageal ultrasound probes for use in cardiac arrest—obviating the need to interrupt compressions and allowing for assessment of cardiac compressions during CPR.

Indications

- Unexplained hypotension or shock
- Cardiac arrest

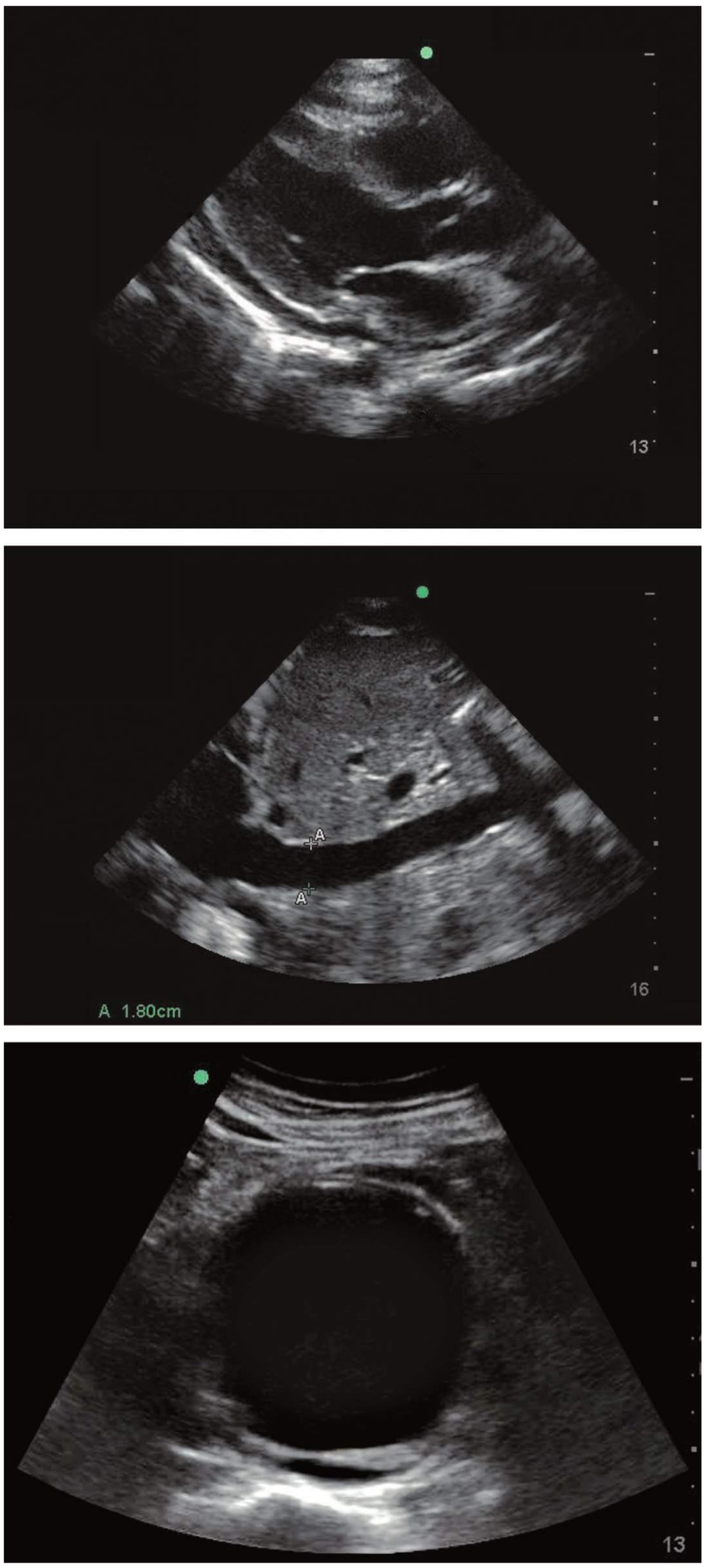


FIGURE 24.71 ■ **Ultrasound-Guided Resuscitation**. Representative images of anatomical areas assessed during ultrasound-guided resuscitation. The heart ("pump"), IVC + FAST ("tank"), and great vessels ("pipes") are assessed confluently to evaluate for the etiology of hypotension and shock. (Ultrasound contributors: Jeremy S. Boyd, MD and Lara Philips, MD.)

Once the patient has been evaluated, findings on ultrasound may be correlated with one of the types of shock identified below. The table below lists representative findings within each shock state. Note that each finding listed below is neither sufficient nor required for the diagnosis of different types of shock, and is merely suggestive. Of course, the clinical context of each patient's presentation must be taken into account in any resuscitation.

Pearls

- The subxiphoid and apical four-chamber views can be obtained during CPR without interrupting chest compressions.
- Parasternal cardiac views might be assessed during a pulse check, but should be evaluated as rapidly as possible so as to minimize chest compressions.
- When obtaining parasternal views during an arrest, have a towel ready to wipe off any gel afterward to avoid interfering with chest compressions.
- Tailor the resuscitation exam to the patient's presenting symptoms. If there is trauma, start with an E-FAST exam. If the patient presents with acute dyspnea, consider starting with cardiac or lung ultrasound first.
- In asystole, there is no spontaneous cardiac activity seen on cardiac ultrasound. Blood can often be seen swirling within the motionless ventricles when assessing after compressions.

TABLE 24.2 ■ APPROACH TO ULTRASOUND-GUIDED RESUSCITATION

	Views	Questions
"Pump"	Focused cardiac assessment: • Parasternal long • Parasternal short • Apical four-chamber • Subxiphoid	• Is there a pericardial effusion? • What is the gross left ventricular function? • Are there signs of right heart strain?
"Tank"	Volume status and integrity: • IVC • E-FAST exam	• Is the IVC collapsed or plethoric? • Is there hemoperitoneum? • Is there a hemothorax or pneumothorax? • Is there pulmonary edema?
"Pipes"	Vascular pathology: • Aorta • DVT	• Is there an AAA? • Is there an aortic dissection? • Is there a DVT?

TABLE 24.3 ■ SONOGRAPHIC FINDINGS IN VARIOUS SHOCK STATES

	Hypovolemic/ Hemorrhagic Shock	Cardiogenic Shock	Obstructive Shock	Distributive Shock
Pump	Hyperdynamic heart	Hypocontractile heart Dilated cardiomyopathy	Pericardial effusion Right heart strain McConnell sign[a] Hyperdynamic heart	Hyperdynamic heart
Tank	Collapsed IVC Hemoperitoneum Hemothorax	Distended IVC Pulmonary edema Pleural effusions	Distended IVC Absent lung sliding/lung point (pneumothorax)	Normal/collapsed IVC Pleural fluids (empyema) Focal B-lines (pneumonia)
Pipes	AAA Aortic dissection	Normal	DVT	Normal

Modified and adapted from Weingart et al. Rapid Ultrasound for Shock and Hypotension (RUSH-HIMAPP). 2009. **http://emedhome.com/**; Perera P, Mailhot T, Riley D, Mandavia D. "The RUSH exam: Rapid Ultrasound in SHock in the evaluation of the critically ill. Emerg Med Clin North Am. 2010;28(1):29–56; and Seif D, Perera P, Mailhot T, Riley D, Mandavia D. Bedside ultrasound in resuscitation and the rapid ultrasound in shock protocol. Crit Care Res Pract. 2012; 2012:503254.

[a]McConnell sign is a finding seen on focused cardiac ultrasound in the apical four-chamber view. It is characterized by RV free wall hypokinesis with preserved apical contractility. It is a sign of acute RV strain, whether due to massive pulmonary embolism or RV infarct.

Clinical Summary

Biliary ultrasound can be very rewarding. A rapid, focused exam can help determine if gallstones or gallbladder pathology is the etiology of a patient's presenting symptoms. In turn, bedside ultrasound can expedite therapy or expedite the pursuit of an alternative diagnosis. While the sonographic identification of gallstones may seem straightforward, the sonographic findings for cholecystitis are often subtle.

Indications

- Right-upper-quadrant pain
- Jaundice
- Epigastric pain

Views for Emergency Department Biliary Ultrasound

- Sagittal view of the gallbladder
- Transverse view of the gallbladder

Equipment: Recommended Transducers for Gallbladder Ultrasound

- Convex array
- Phased array

Patient Positioning

- Supine or left lateral decubitus position

Techniques

Sagittal View

- Initially, the transducer is placed in the subxiphoid region with the indicator directed toward the patient's head in a sagittal view and swept below the right costal margin to approximately the midclavicular line. The transducer is then rotated (often clockwise) to obtain a view of the gallbladder in its longest axis (Fig. 24.72).
- Scan through the gallbladder completely from medial to lateral borders including careful evaluation of the gallbladder neck. A thorough search for gallstones should be made, particularly at the neck where small symptomatic gallstones may be seen.
- Identify the liver, gallbladder, portal vein, hepatic artery, main lobar fissure, and the common hepatic duct (Figs. 24.73 and 24.75).

FIGURE 24.72 ■ **Gallbladder: Sagittal View.** Probe is placed just under the costal margin and swept across the costal margin to locate the gallbladder. The transducer is then rotated (often clockwise) to obtain a view of the gallbladder in its longest axis. (Photo contributor: Lawrence B. Stack, MD.)

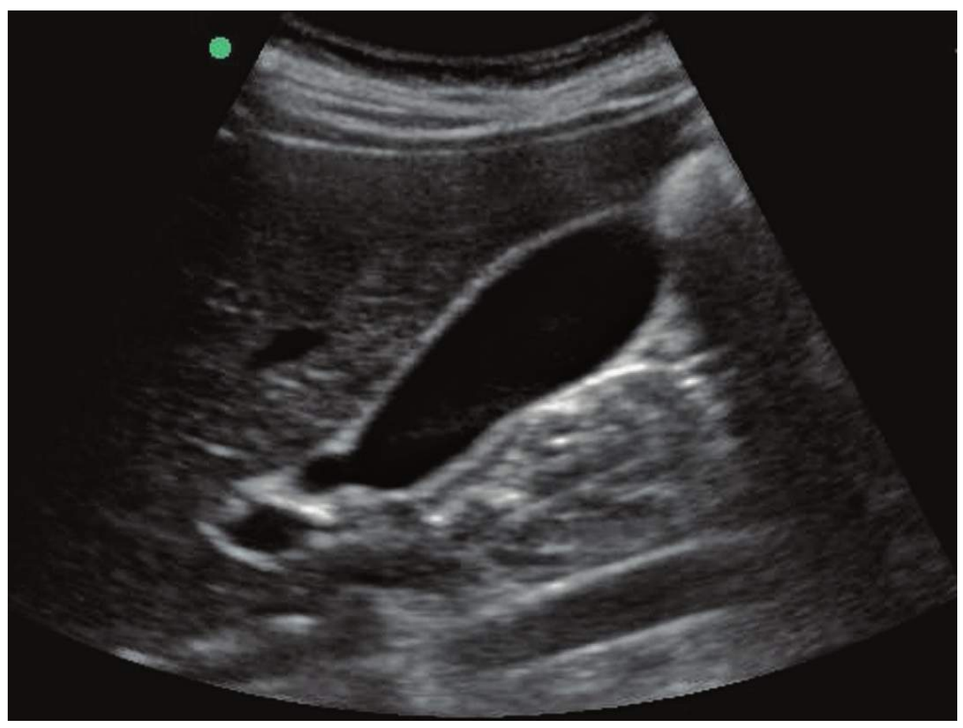


FIGURE 24.73 ■ **Gallbladder: Sagittal View.** Long-axis view of gallbladder often reveals an "exclamation mark" sign made of the gallbladder and the portal triad. (Ultrasound contributor: Robinson M. Ferre, MD.)

FIGURE 24.74 ■ **Gallbladder: Transverse.** Rotate the probe 90 degrees counterclockwise to bring the gallbladder into short axis. (Photo contributor: Lawrence B. Stack, MD.)

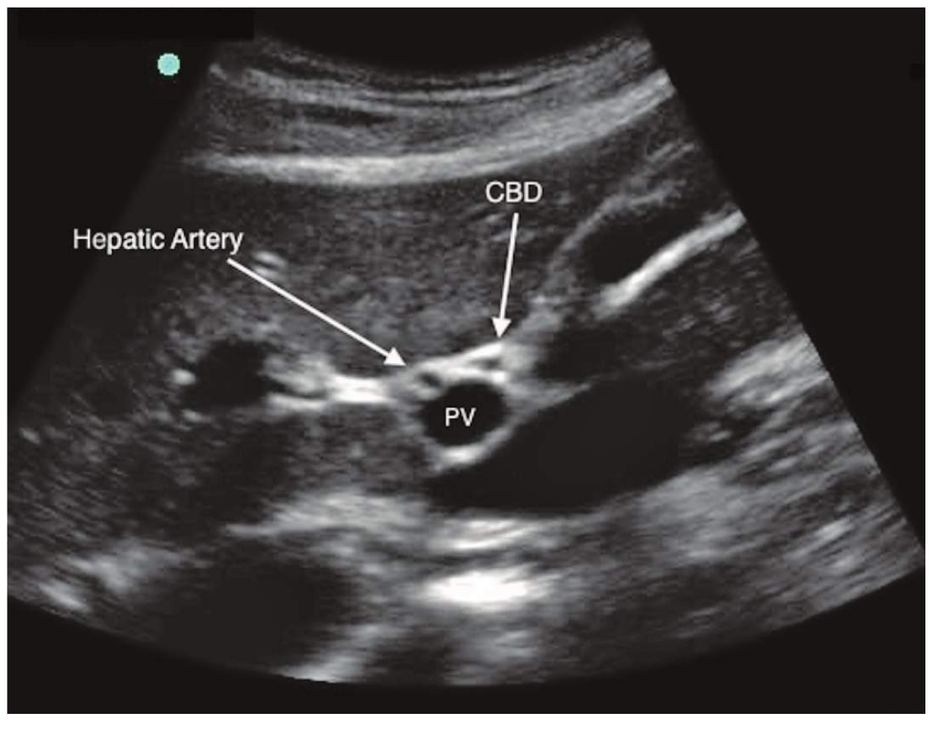


FIGURE 24.75 ■ **Gallbladder: Portal Triad.** The portal vein (PV), hepatic artery, and common bile duct (CBD) are readily seen in the transverse view. (Ultrasound contributor: Shannon Snyder, MD, RDMS.)

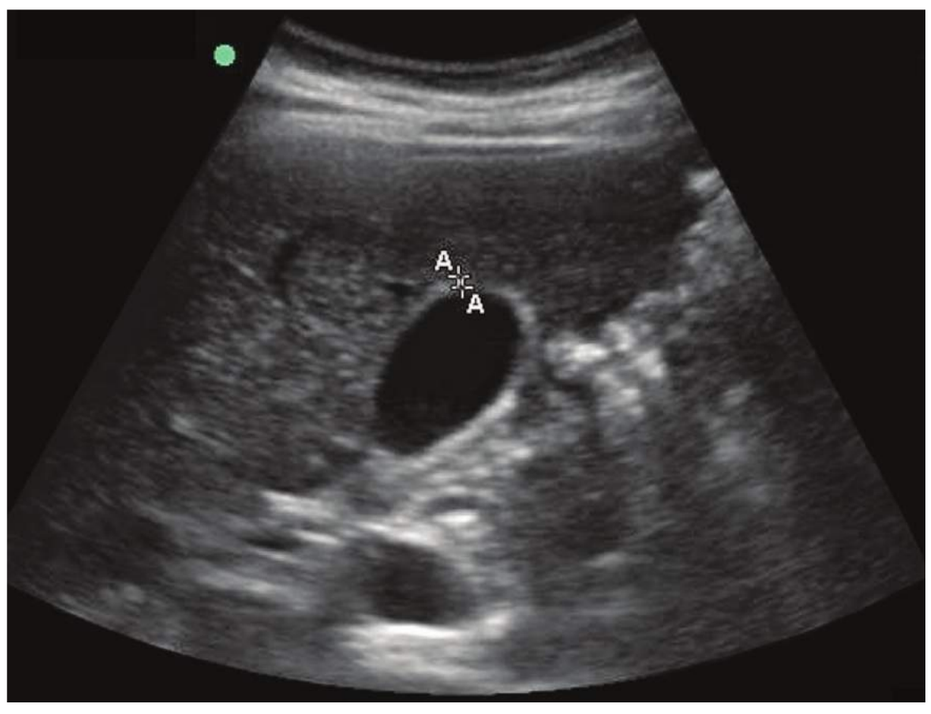


FIGURE 24.76 ■ **Gallbladder: Transverse.** Short-axis view of the gallbladder measuring the anterior wall thickness. (Ultrasound contributor: Robinson M. Ferre, MD.)

Transverse View

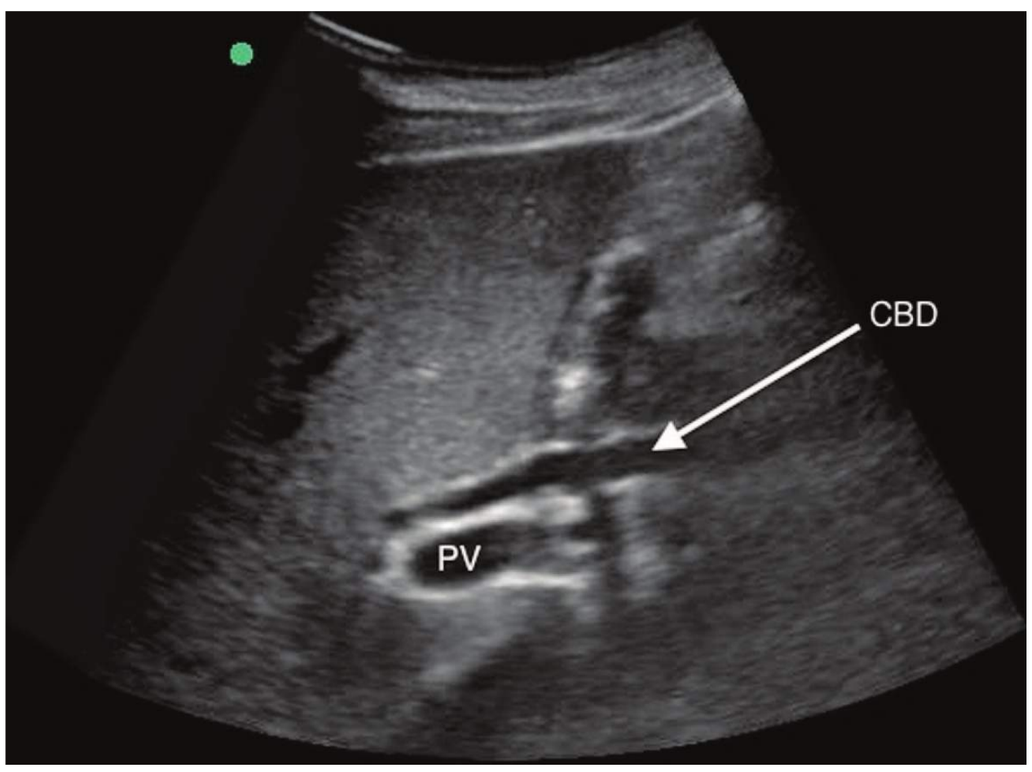
- After identifying the gallbladder in the sagittal position, rotate the transducer 90 degrees counterclockwise toward the patient's right (Fig. 24.74).
- Identify the liver, gallbladder, IVC, and common bile duct (CBD). Measure the anterior wall of the gallbladder for thickness (Fig. 24.76).

Additional Views

- If indicated, find the CBD. This can be done in two ways. First, identify the common hepatic duct at the level of the porta hepatis, and trace it down beyond the border of the liver.
- Measure the thickness of the CBD when visible from inside wall to inside wall (Fig. 24.77). Color flow Doppler will help delineate the CBD (no flow) from the hepatic artery (Fig. 24.78).

Abnormal Findings

- Gallstones: Hyperechoic (bright) oval to round structure(s) within the gallbladder, often with accoustic shadowing (Fig. 24.79A,B). Gallstones should be mobile unless impacted in the neck of the gallbladder (Fig. 24.80). Rolling the patient will allow assessment of gallstone mobility.



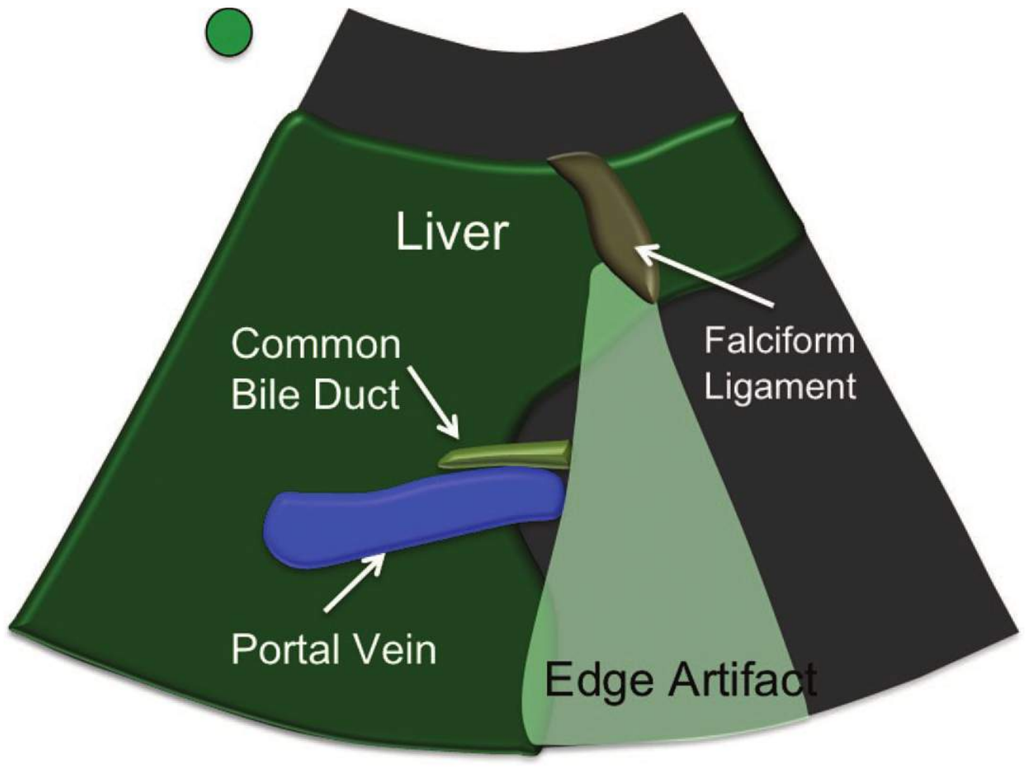


FIGURE 24.77 ■ **Gallbladder: Common Bile Duct.** Long-axis view of the portal vein (PV) and dilated common bile duct (CBD). The CBD is measured from inside wall to inside wall. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

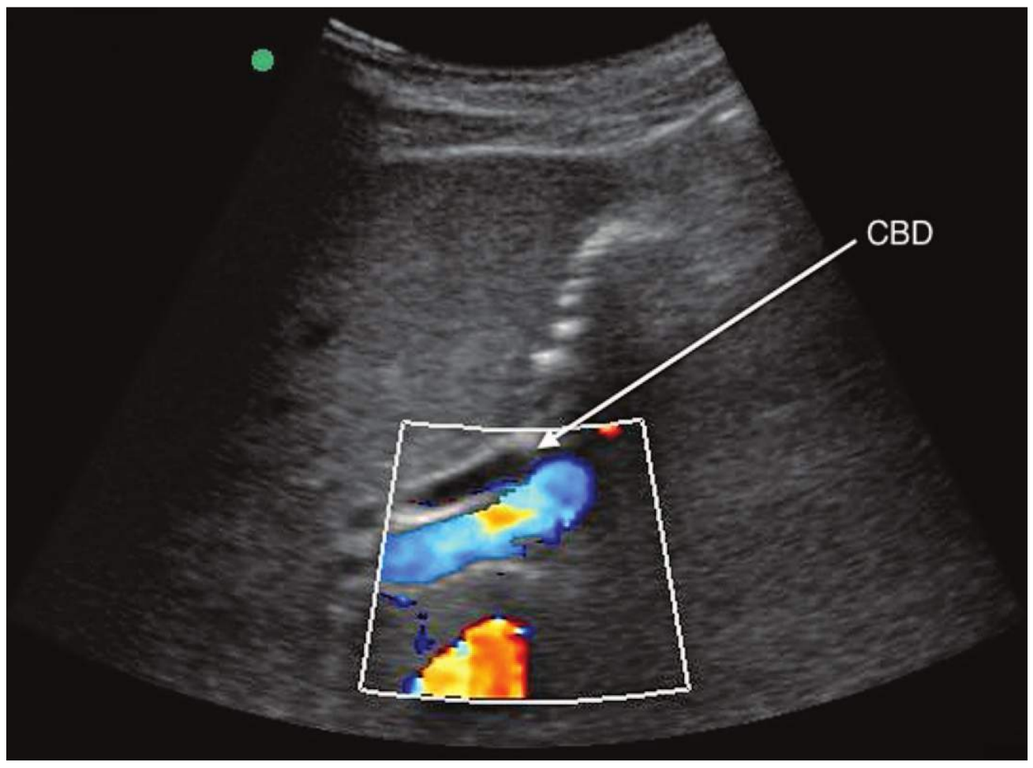
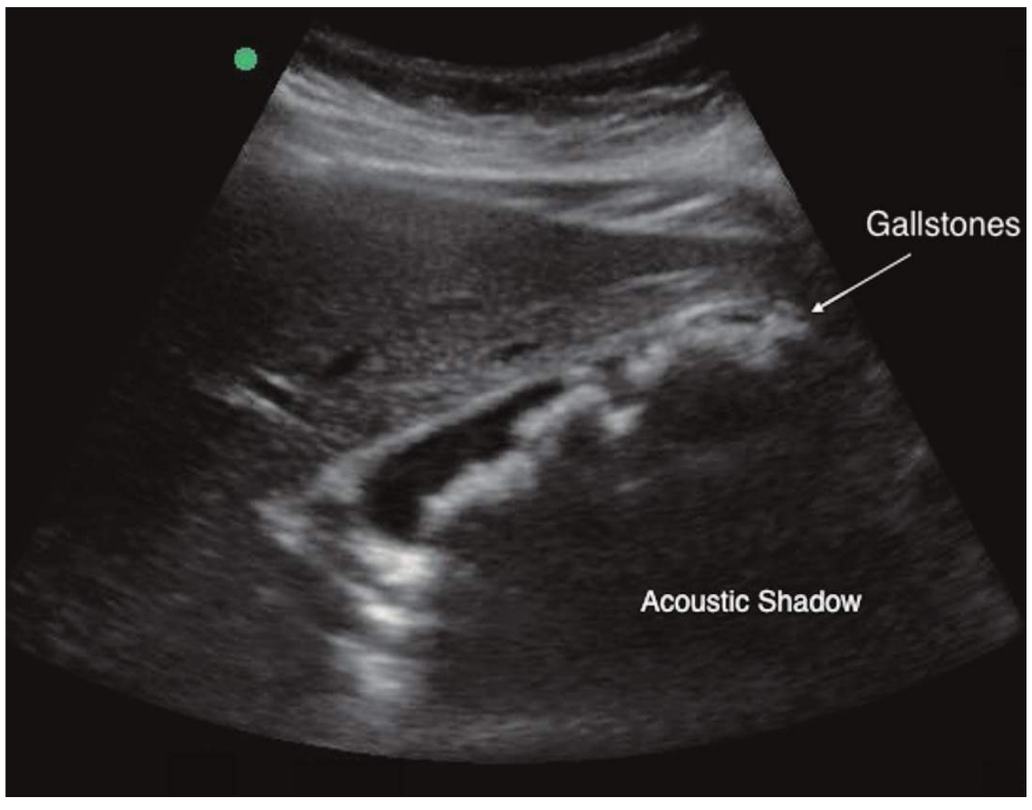


FIGURE 24.78 ■ **Gallbladdder: Common Bile Duct, Color Flow Doppler.** Long-axis view of the portal vein (color flow) and dilated common bile duct (CBD) without color flow. (Ultrasound contributor: Max Palatnik, MD.)

- Pericholecystic fluid: An anechoic (dark) stripe that borders the outer gallbladder wall and the liver. It will be visible in two views (Fig. 24.81A,B).
- Thickened gallbladder wall: A gallbladder wall that measures greater than 3 mm is considered abnormal (Fig. 24.82).
- Sonographic Murphy sign: When direct visualized compression of the gallbladder by the ultrasound probe reproduces the patient's symptoms of abdominal pain and this pain is not reproduced by compression with the ultrasound probe elsewhere in the surrounding area.
- Dilated CBD: A CBD with an internal diameter greater than 6 mm is dilated; however, documented measurements up to 8 to 9 mm can be normal in the elderly. One rule of thumb is that the CBD can measure up to 6 mm up to age 60 and thereafter an increase of 1 mm per decade of life represents the normal range.

Pearls

1. Other positions—including prone, right lateral decubitus, semi-erect, and standing—may be helpful in scanning the gallbladder and are patient specific.
2. Having the patient hold their breath after deep inspiration may help the gallbladder descend into view below the ribcage.
3. Always measure the anterior wall of the gallbladder. The thickness of the posterior wall often appears falsely thickened by posterior acoustic enhancement.
4. Small gallstones (1-3 mm) may not be large enough to cause acoustic shadowing. Increasing the frequency and ensuring that your focal zone is on the area of interest will increase the likelihood that shadowing will be seen.
5. The CBD lies just anterior to the portal vein, a structure with markedly hyperechoic walls compared to the hepatic veins. Placing the patient in the left lateral decubitus position may help visualize the CBD.



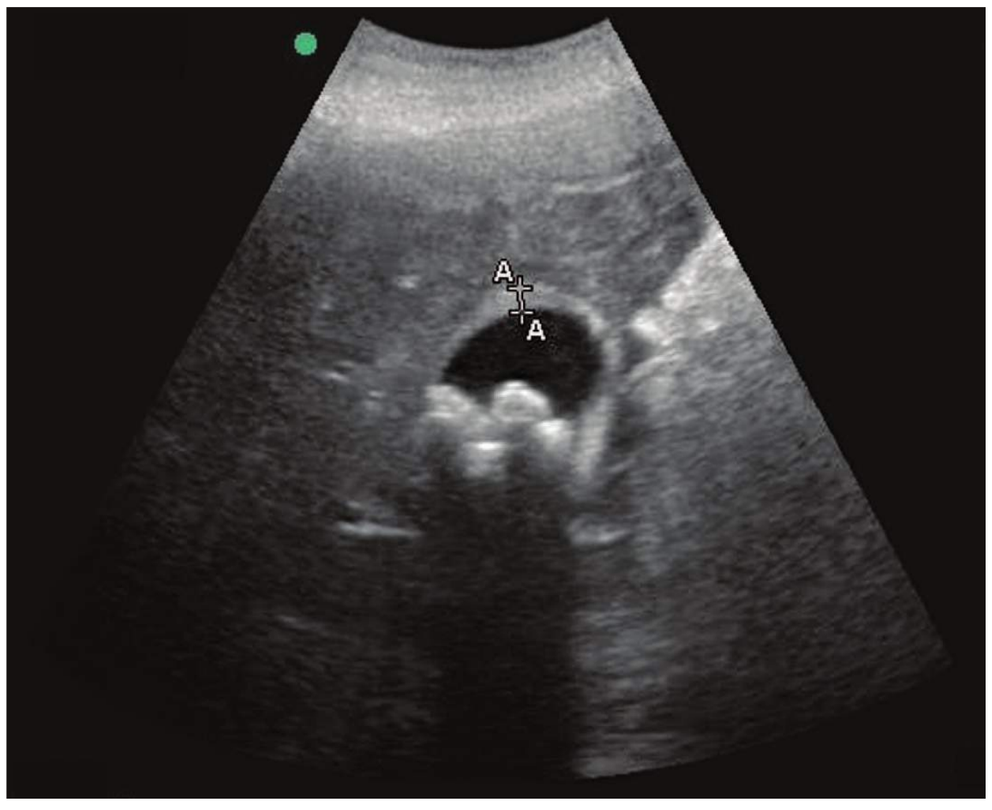


FIGURE 24.79A, B ■ **Gallbladder: Gallstones.** Long- and short-axis views of the gallbladder with a large number of gallstones casting an acoustic shadow posterior to the calcified stones. (Ultrasound contributor: Shannon Snyder, MD, RDMS.)

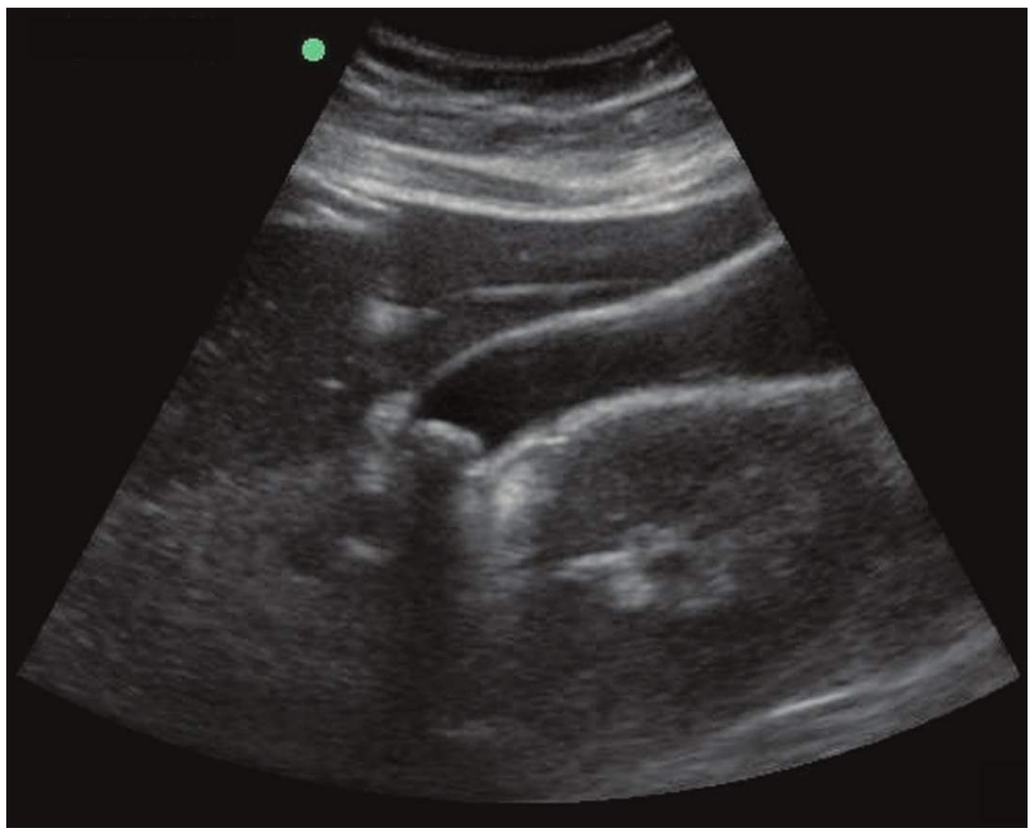


FIGURE 24.80 ■ **Gallbladder: Sagittal View, Gallstone.** Long-axis view of the gallbladder with a single gallstone lodged in the gallbladder neck. (Ultrasound contributor: Robinson M. Ferre, MD.)

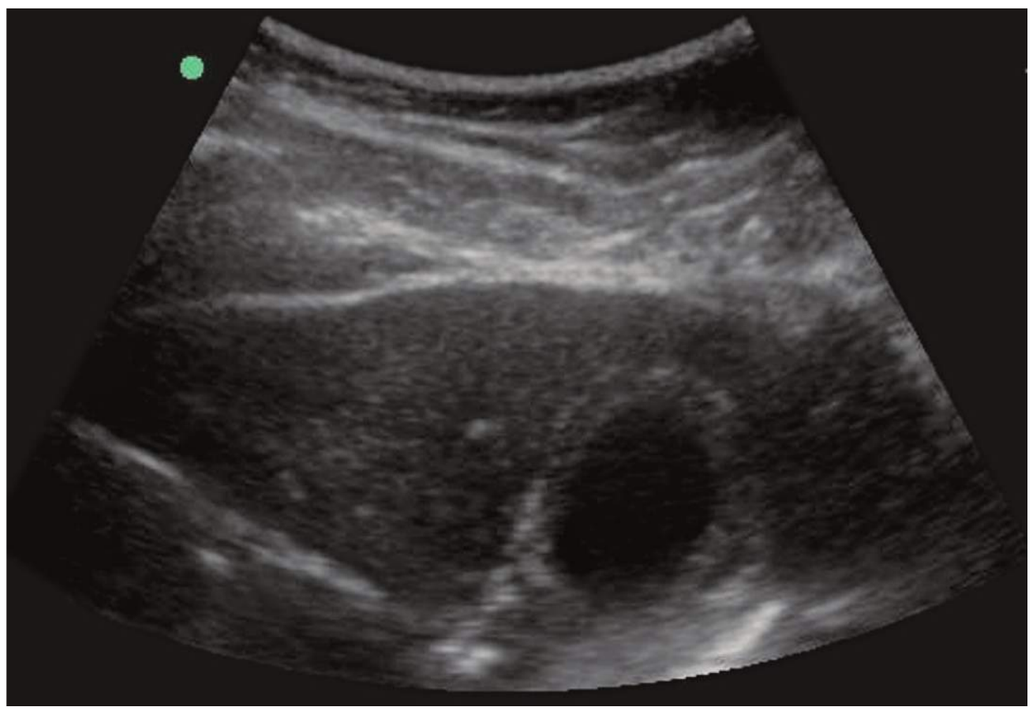
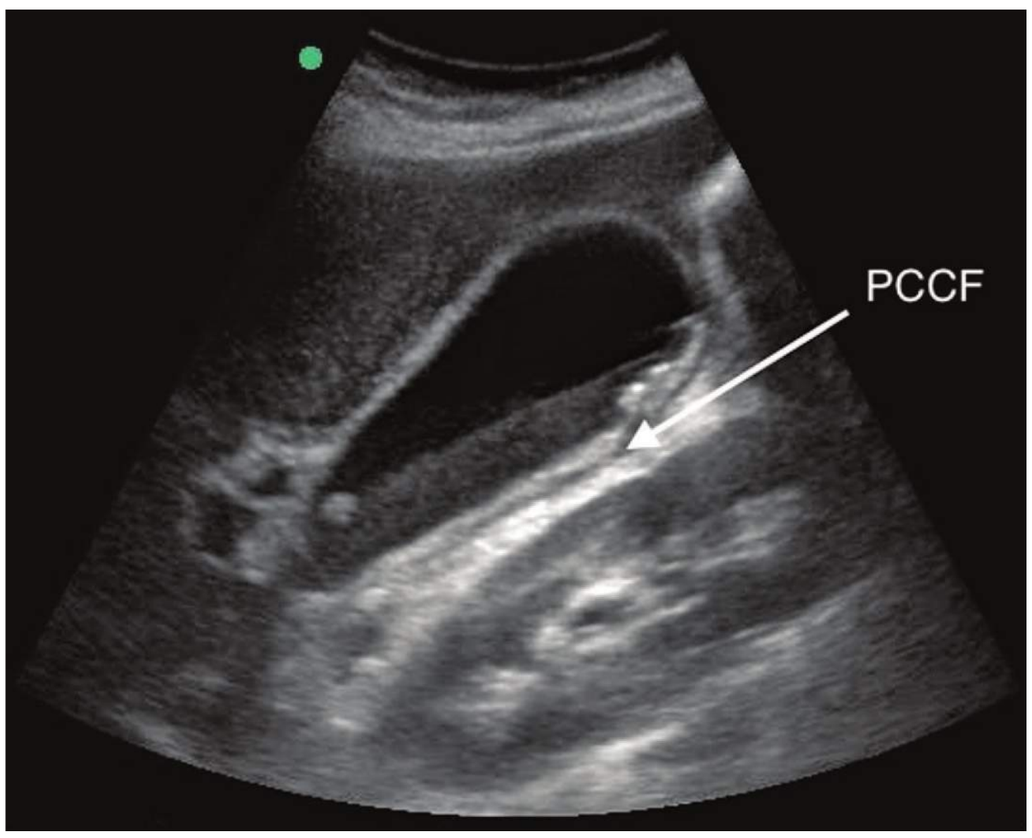


FIGURE 24.82 ■ **Gallbladder: Transverse View, Cholecystitis.** Short-axis view of the gallbladder with thickening of the wall. (Ultrasound contributor: Jordan Rupp, MD.)



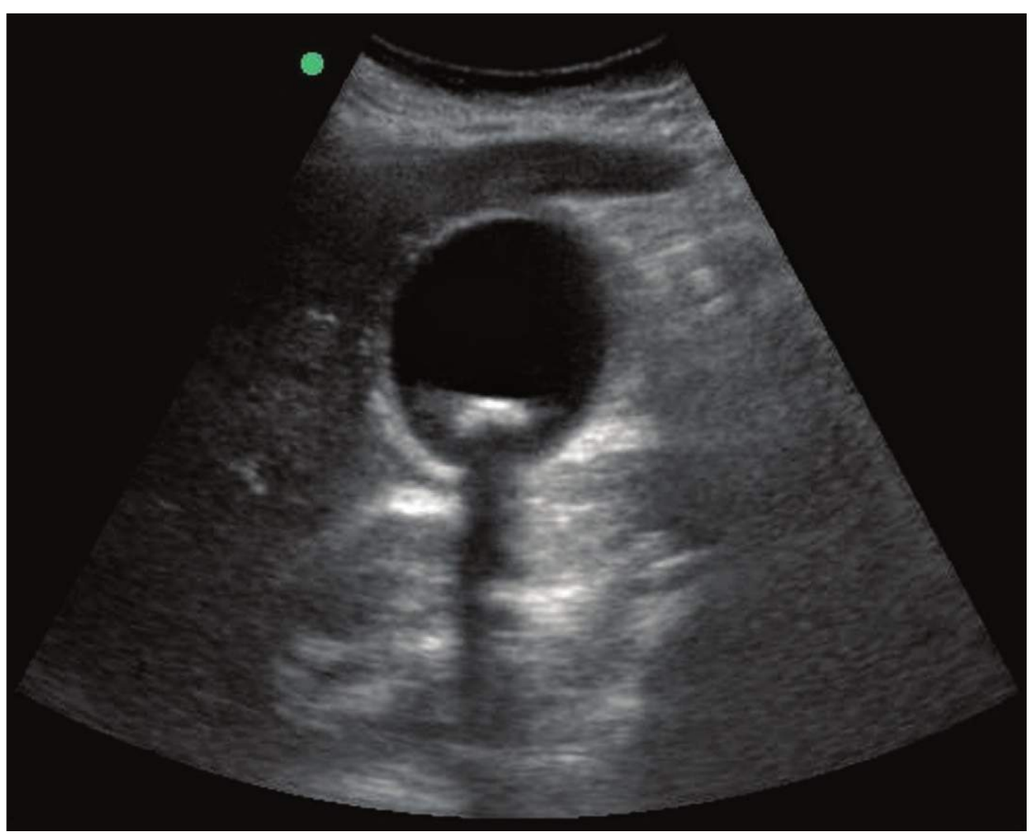


FIGURE 24.81A, B ■ **Gallbladder.** Gallbladder sludge, gallstone, and pericholecystic fluid (PCCF). Long- and short-axis view of the gallbladder showing a gallstone and biliary sludge. There is also a thin stripe of PCCF. (Ultrasound contributor: Jeremy Boyd, MD.)

Clinical Summary

Ultrasound of the kidneys and bladder can yield helpful diagnostic information for patients presenting with symptoms of urinary retention, decreased urinary output, or abdominal/flank pain consistent with renal colic. Obstructive uropathy due to kidney stones is the principal pathology identified with emergency physician–performed renal ultrasound. Bladder volume is easily calculated by viewing and measuring the bladder in two planes. In focused renal ultrasound, the goal is not to identify renal or ureteral calculi, rather the kidneys are evaluated for hydronephrosis. The presence of hydronephrosis in the patient with renal colic is presumed to be a direct result of ureteral obstruction. There are no accurate means of determining the degree of obstruction by the presence of hydronephrosis.

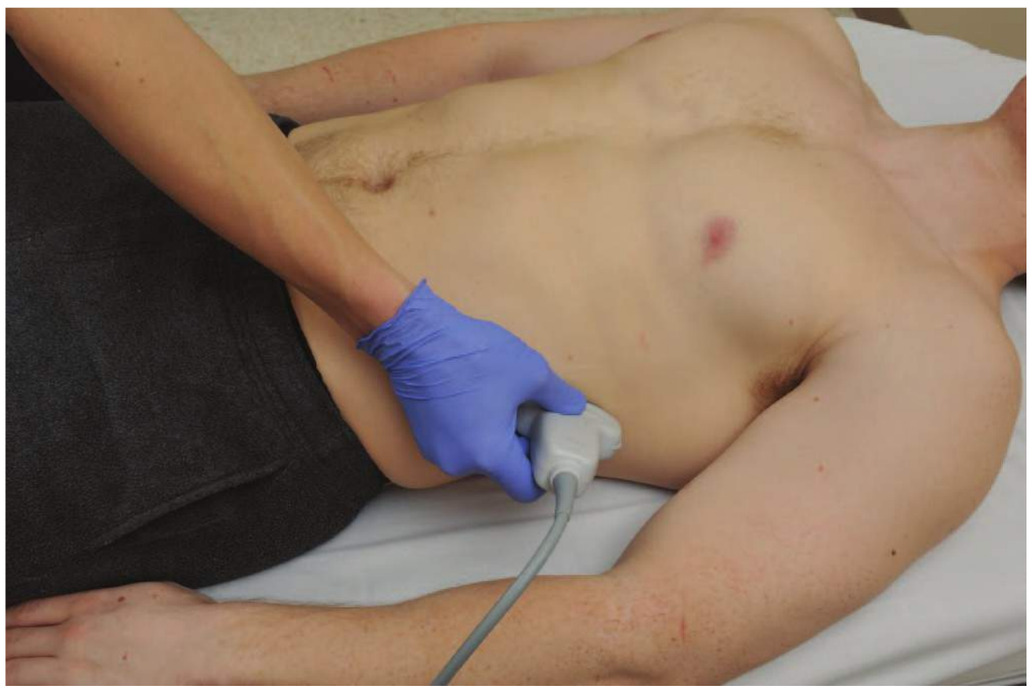


FIGURE 24.83 ■ **Renal Ultrasound.** The transducer is directed as a coronal section through the body in the mid to posterior axillary lines. (Photo contributor: Lawrence B. Stack, MD.)

Indications

- Flank/abdominal pain
- Renal colic
- Hematuria
- Urinary retention
- Decreased urinary output

The recommended sonographic approach to the kidney is similar to that for the RUQ and LUQ windows in the E-FAST examination previously discussed, with the probe angled toward the retroperitoneum for better views of the kidneys. The coronal view allows the sonographer to visualize the right or left kidney from the superior to inferior poles. Transverse or axial views of the entire kidney should be obtained as well. Renal EUS is most easily interpreted when comparative images are obtained between the right and left kidneys. The bladder view is also nearly identical to that of the E-FAST exam described earlier. However, rather than focusing on the potential spaces of the peritoneum, attention is focused on the bladder and its contents.

Views for Emergency Department Renal and Bladder Ultrasound

1. Coronal or sagittal kidney view
2. Transverse kidney view
3. Sagittal bladder view
4. Transverse bladder view

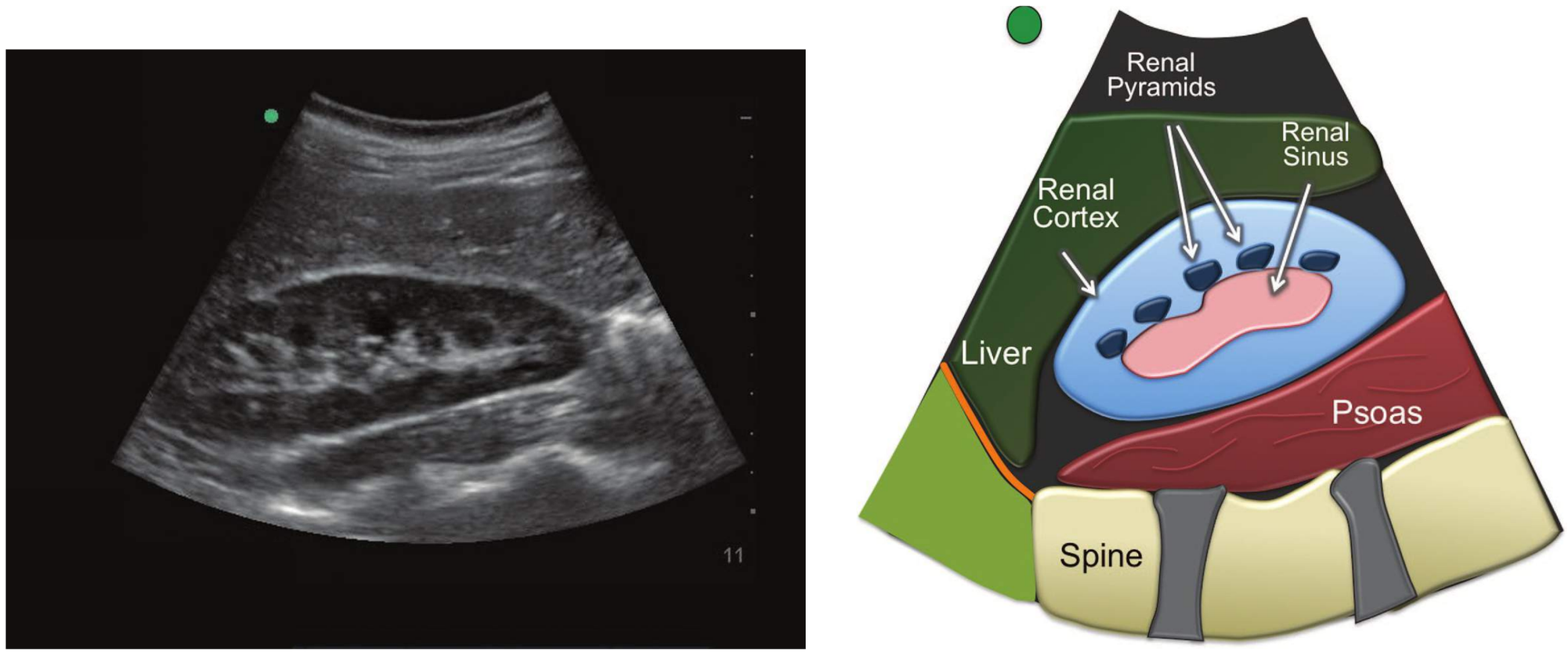


FIGURE 24.84 ■ **Renal Ultrasound, Right Coronal View.** The liver, right kidney, renal cortex (with pyramids), and central renal sinus are seen. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)

Equipment: Recommended Transducers for Renal Ultrasound

- Convex array
- Phased array

Patient Position

- The patient is supine.
- Alternatively, the patient may be placed in a prone position with the probe placed in the midscapular line and the transducer directed toward the abdomen.

Technique

Kidney: Right and Left Coronal Views

- The transducer indicator is oriented toward the patient's head.
- The transducer is directed as a coronal section through the body in the mid to posterior axillary lines (Fig. 24.83). Begin scanning between the 9th and 11th ribs on the right and the 8th and 11th ribs on the left.
- Identify the liver, right kidney, renal cortex (with pyramids), and central renal sinus (Fig. 24.84).
- Identify the spleen, left kidney, renal cortex (with pyramids), and central renal sinus.

Kidney: Right and Left Transverse Views

- Obtain the coronal view as directed above, but rotate the transducer 90 degrees counterclockwise to visualize the renal parenchyma and central renal sinus in a cross-sectional view (Fig. 24.85).

Bladder: Sagittal View

- With the indicator oriented toward the patient's head, the transducer is placed just above the symphysis pubis and is directed into the pelvis (see Fig. 24.17).
- Identify the bladder (triangular in this view when fully distended), uterus (pear-shaped if present), prostate, seminal vesicles, and rectum (see Figs. 24.18 and 24.19).

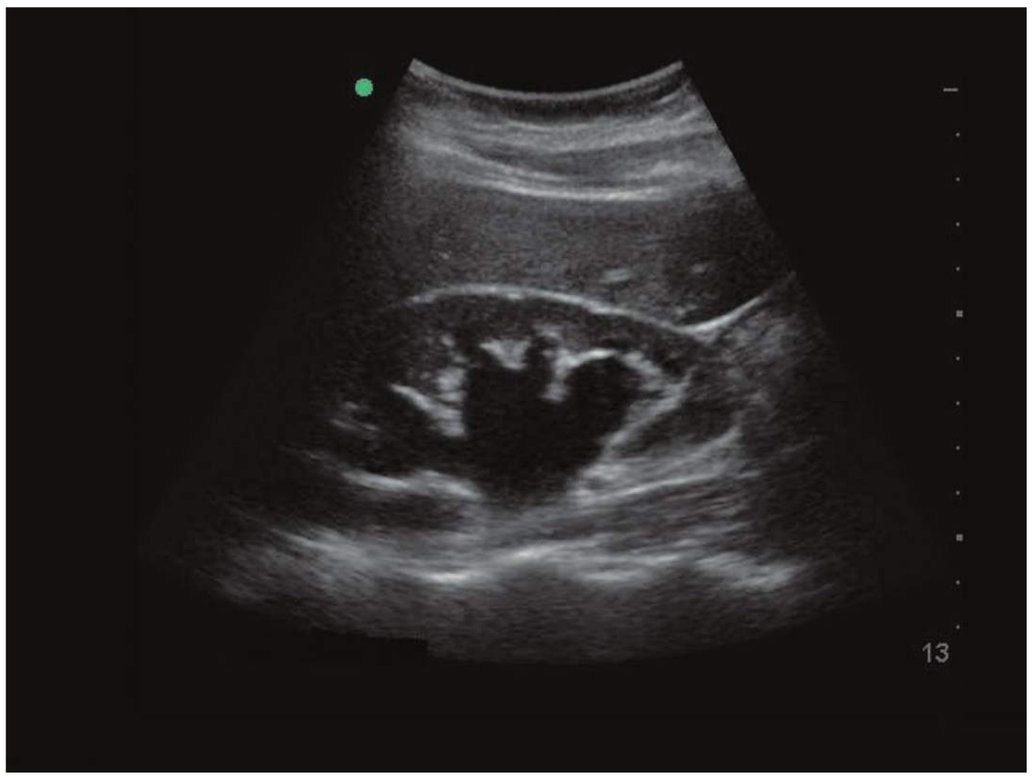
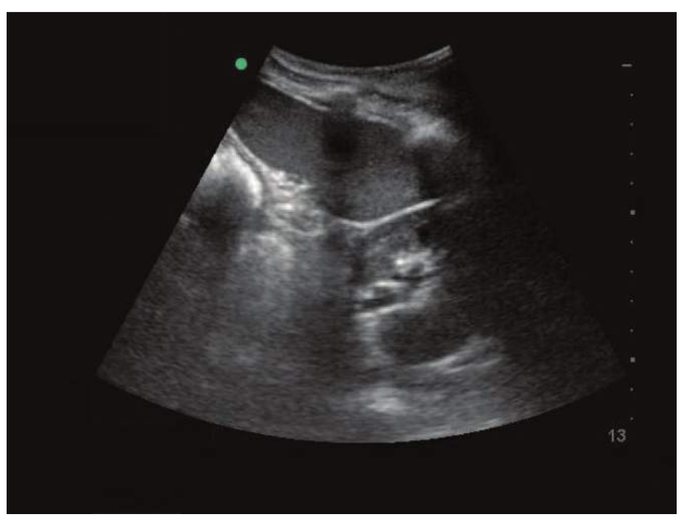


FIGURE 24.86 ■ **Renal Ultrasound: Hydronephrosis.** Moderate hydronephrosis is seen as dilatation of the bright renal sinus and secondary calyces by anechoic (black) fluid. (Ultrasound contributor: Shannon Snyder, MD, RDMS.)



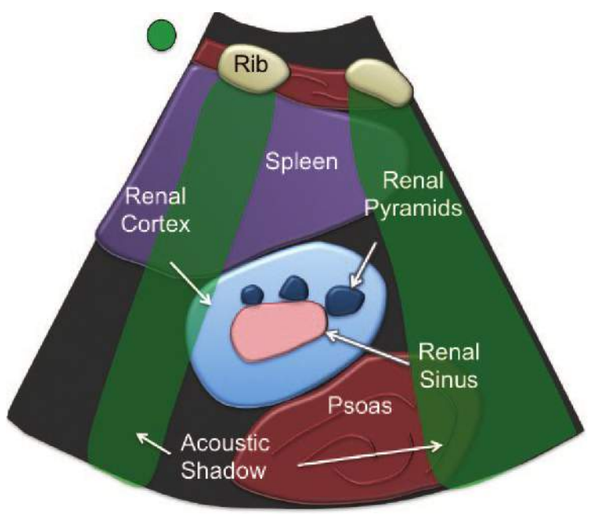


FIGURE 24.85 ■ **Renal Ultrasound, Left Transverse View.** Rotate the transducer 90 degrees counterclockwise to visualize the renal parenchyma and central renal sinus in a cross-sectional view. (Illustration courtesy of Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)

Bladder: Transverse View

- With the indicator oriented toward the patient's right, the transducer is placed about 1 to 2 cm above the symphysis pubis, with the transducer angled into the peritoneum (see Fig. 24.20).
- Identify the bladder (rectangular in this view when fully distended), uterus (oval hyperechoic structure if present), prostate, seminal vesicles, and rectum (see Fig. 24.21).

Findings

- Hydronephrosis: Dilatation of the bright renal sinus with black, anechoic fluid (Fig. 24.86). Hydronephrosis is graded based on its extension into the secondary calyces and whether there is accompanying cortical thinning (Fig. 24.87).
- Renal calculi: Bright hyperechoic oval/round structures within the cortex or renal sinus (acoustic shadowing is often present). With color flow Doppler, uroliths will often demonstrate twinkling artifact deep to their reflective echogenic surface (Fig. 24.88).
- Urinary retention: By calculating the volume of the bladder, one may determine the presence of urinary retention. Greater than 50 to 100 mL of retained postvoid urine is diagnostic.

Pearls

1. Rib shadows will be evident with the coronal view and transverse views. Have the patient hold his or her breath in inspiration and move the transducer a rib space higher or lower to visualize the kidney from the superior to the inferior pole.
2. The presence of a ureteral jet confirms the passage of urine into the bladder from a specific side. Its absence is sensitive, but not specific for complete ureteral obstruction (Fig. 24.89).
3. When evaluating for renal colic, stones may frequently be seen on the bladder views when they are located at the ureterovesicular junction (UVJ). (Fig. 24.90).

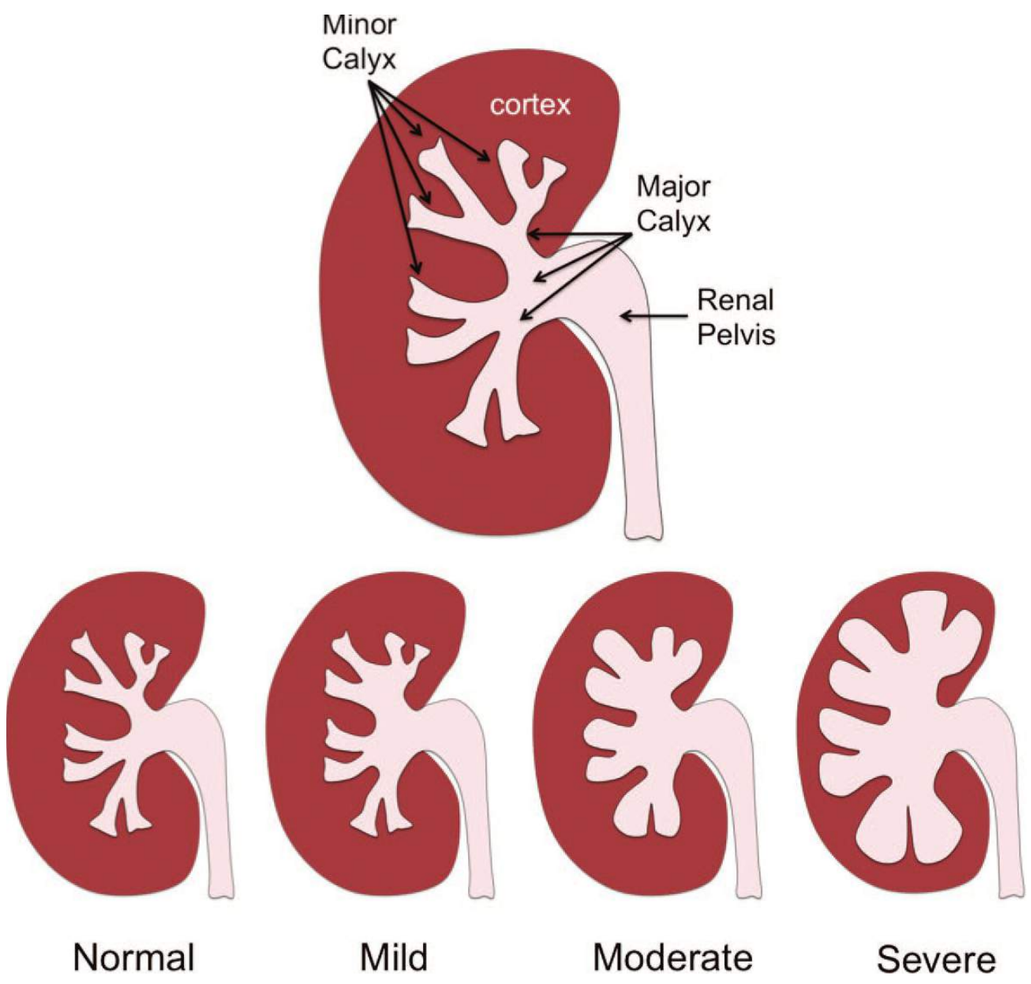


FIGURE 24.87 ■ **Hydronephrosis Grading.** Hydronephrosis is graded based on its extension into the secondary calyces and whether there is accompanying cortical thinning. Mild hydronephrosis does not involve the secondary calyces, moderate does, and severe hydronephrosis is accompanied by cortical thinning. (Illustration courtesy of Robinson M. Ferre, MD.)

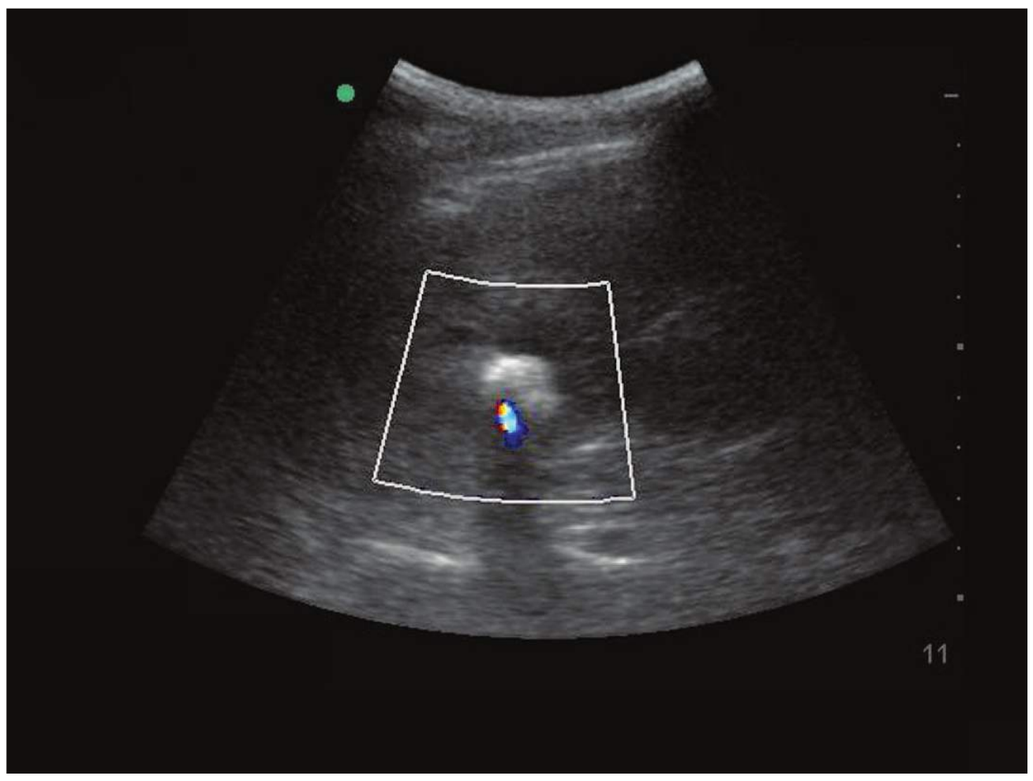


FIGURE 24.88 ■ **Renal Calculi, Twinkle Artifact.** With color flow doppler, uroliths will often demonstrate twinkling artifact deep to their reflective echogenic surface. (Ultrasound contributor: Jeremy S. Boyd, MD.)

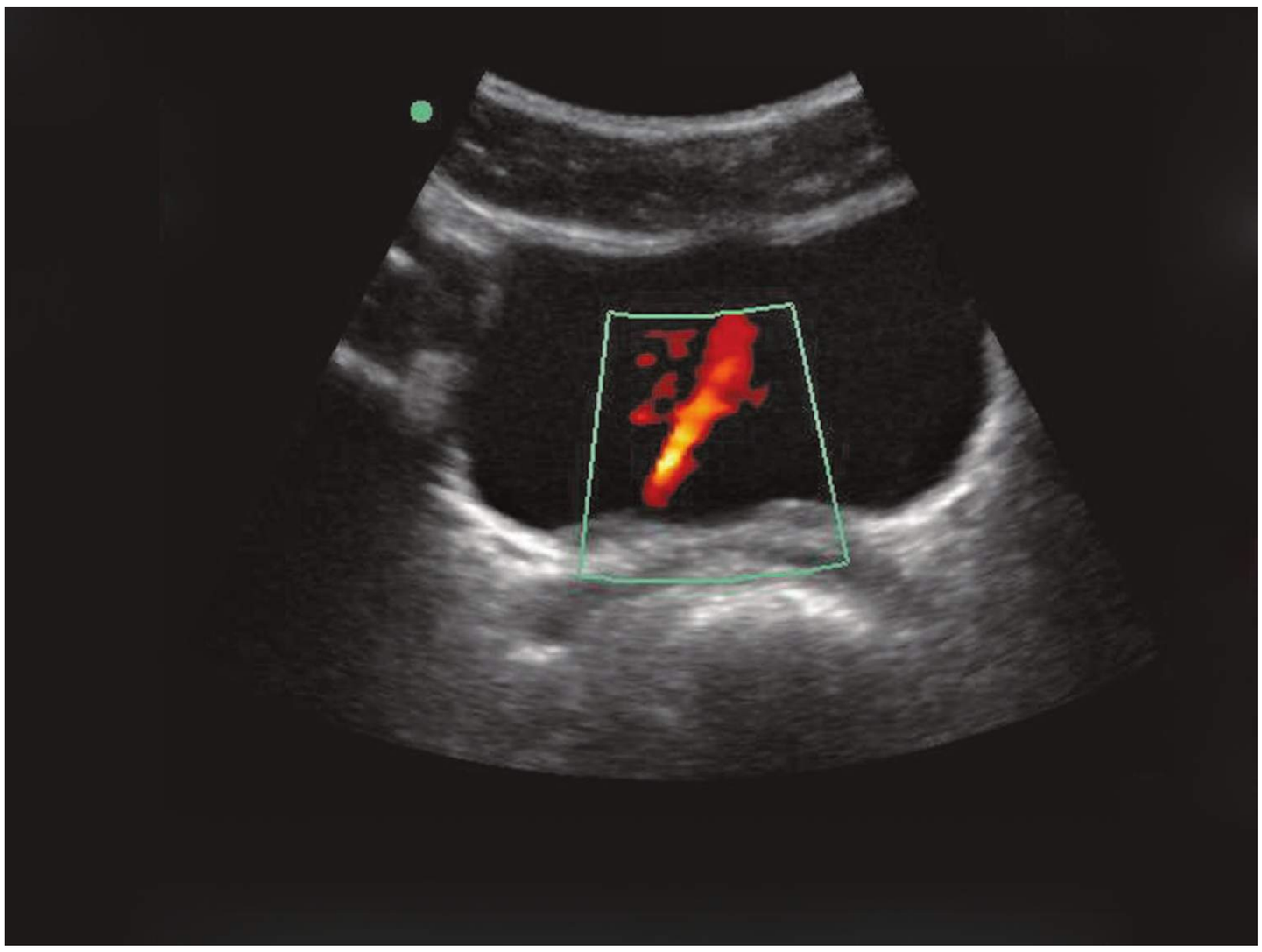


FIGURE 24.89 ■ **Ureteral Jet.** The presence of a ureteral jet confirms the passage of urine into the bladder from a specific ureter. (Ultrasound contributor: Jeremy S. Boyd, MD.)

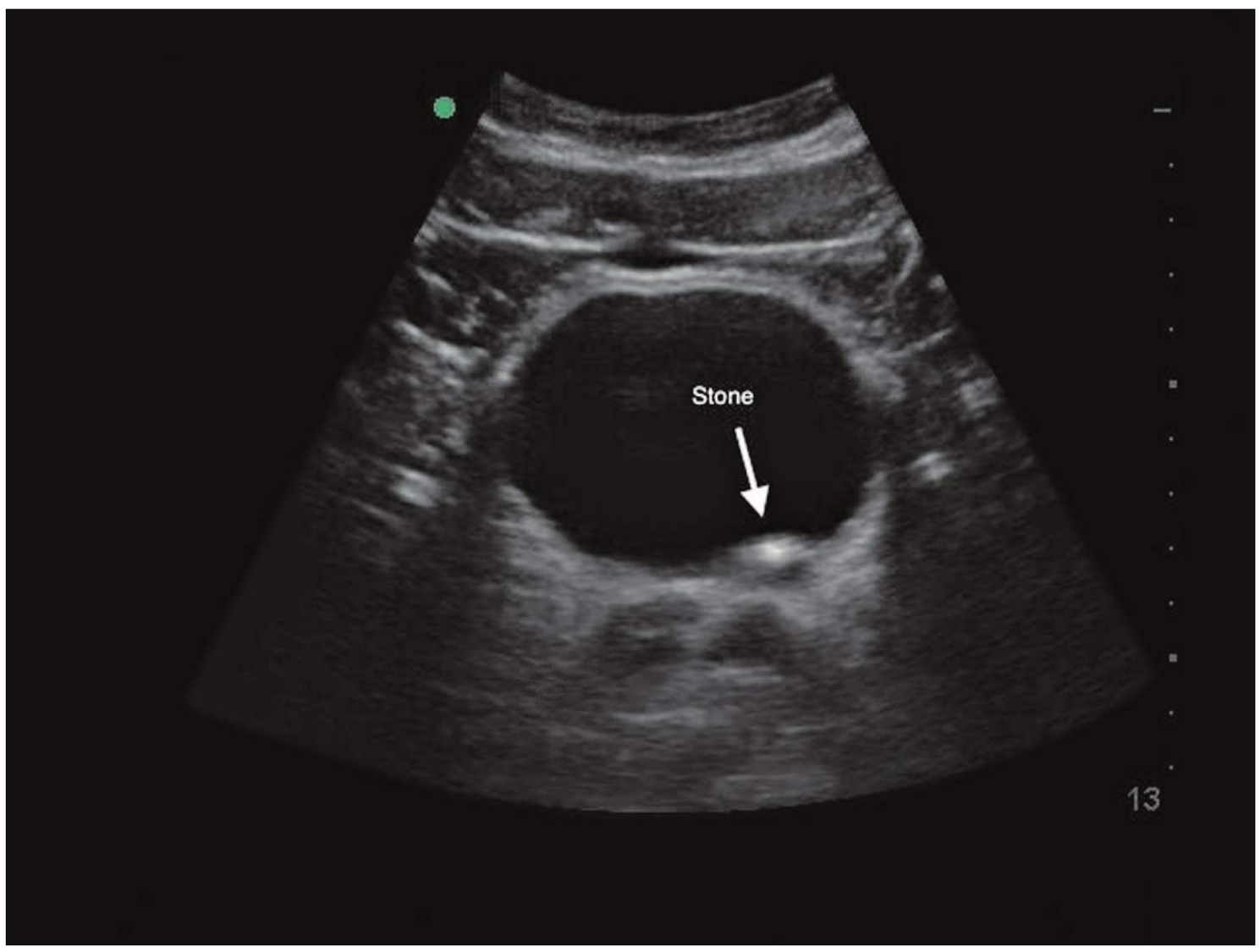


FIGURE 24.90 ■ **Uterovesicular Junction (UVJ) Stone.** Stones may frequently be seen on the bladder views when they are located at the UVJ. (Ultrasound contributor: Robinson M. Ferre, MD.)

Clinical Summary

Ultrasound is frequently used to evaluate the patient presenting with pelvic pain and/or vaginal bleeding, and may aid in the diagnosis of numerous forms of pathology such as urinary retention, molar pregnancies, and ovarian torsion. Pelvic EUS is most commonly used in the evaluation of pregnant patients who present with signs concerning for ectopic pregnancy or miscarriage. The primary goal of pelvic EUS is to evaluate for the presence of an intrauterine pregnancy (IUP) and assess for risk factors that are concerning for an ectopic pregnancy. Ultrasound of pregnant patients is accomplished with two different scanning techniques: transabdominal and transvaginal.

Pregnant patients presenting with abdominal pain or vaginal bleeding during the first trimester must be evaluated for the presence of an ectopic pregnancy. This is commonly accomplished in the ED setting by identifying an IUP in a patient who is at low risk for a heterotopic pregnancy. Patients not receiving infertility treatments with evidence of an IUP can be safely discharged from the ED.

Indications

- Pelvic/abdominal pain
- Vaginal bleeding
- Suspected pregnancy

Required Views for the Emergency Department Pelvic Ultrasound

- Sagittal view
- Transverse view

Equipment: Recommended Transducers for Pelvic Sonography

- Convex array (transabdominal)
- Endocavitary probe (transvaginal)

Patient Positioning

- The patient is supine for transabdominal views.
- The patient is supine and preferably in the lithotomy position for transvaginal views.

Techniques

Transabdominal Sonography: Sagittal View

- With the indicator pointed toward the patient's head, place transducer superior to the symphysis pubis (Fig. 24.91).
- Identify the bladder, uterus, rectum, ovaries, and the rectouterine pouches (pouch of Douglas). Identify an IUP if present (Fig. 24.92).

Transabdominal Sonography: Transverse View

- From the sagittal view, rotate the transducer 90 degrees counterclockwise so the transducer indicator points to the patient's right. The transducer should be superior to symphysis pubis (Fig. 24.93).

FIGURE 24.91 ■ **Transabdominal Sagittal View.** With the indicator pointed toward the patient's head, place the transducer superior to the symphysis pubis. (Photo contributor: Lawrence B. Stack, MD.)

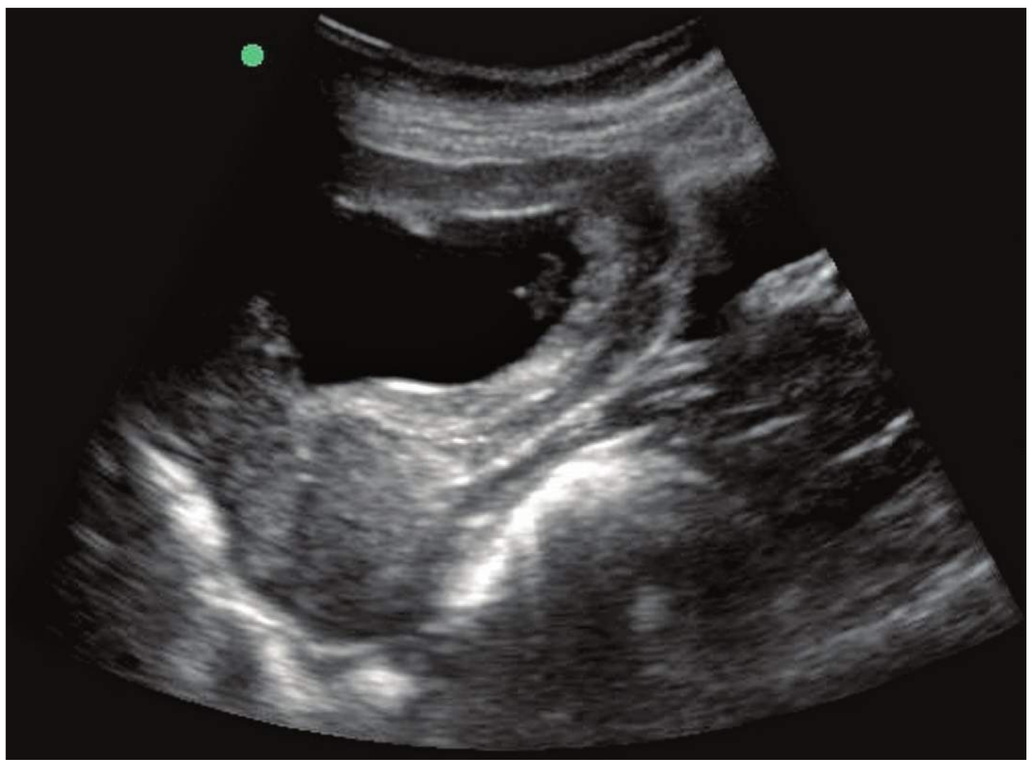


FIGURE 24.92 ■ **Transabdominal Sagittal View.** Gravid uterus showing cervix and vagina posterior to the bladder. (Ultrasound contributor: Robinson M. Ferre, MD.)

FIGURE 24.93 ■ **Transabdominal Transverse View.** From the sagittal view, rotate the transducer 90 degrees counterclockwise so the transducer indicator points to the patient's right. (Photo contributor: Lawrence B. Stack, MD.)

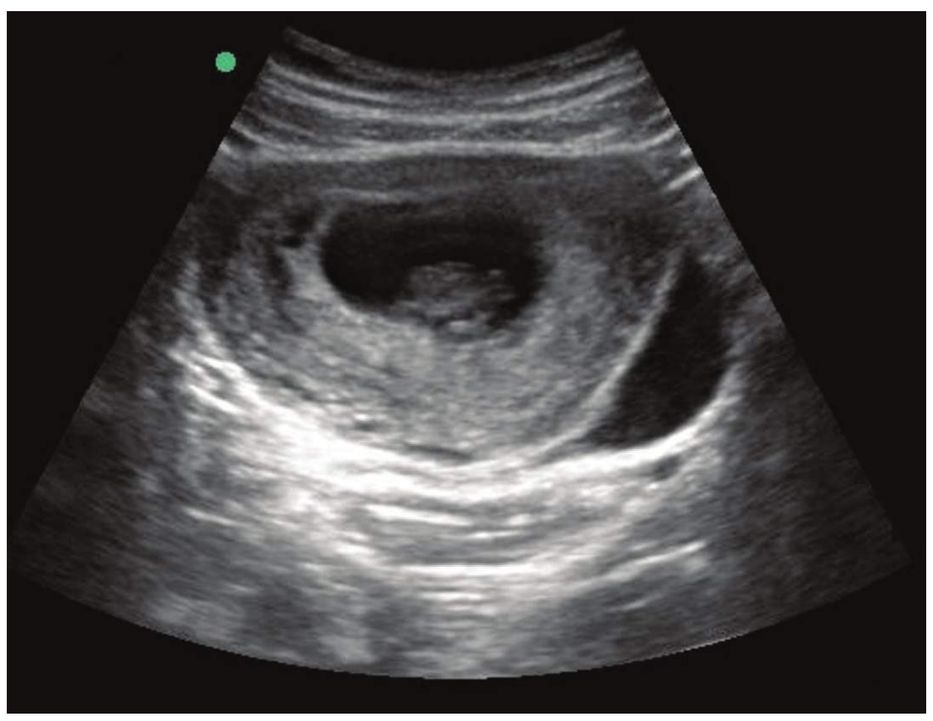


FIGURE 24.94 ■ **Transabdominal Transverse View: Uterus.** First trimester pregnancy showing uterus with gestational sac and fetal pole. Some of the urinary bladder is seen deep and lateral (right on the image) to the uterus. (Ultrasound contributor: Robinson M. Ferre, MD.)

- Identify the bladder, uterus, rectum, ovaries, and the vesicouterine and rectouterine (pouch of Douglas) areas (Fig. 24.94).

Transvaginal Sonography: Sagittal View

- With a condom or commercially available endocavitary probe cover covering the transducer, insert the transducer with the marker directed toward the ceiling. The transducer should be inserted until it rests in the anterior fornix of the vagina (Fig. 24.95).
- Identify the uterus, rectum, ovaries, and rectouterine pouch (Fig. 24.96,). Identify an IUP if present.

Coronal View

- From the sagittal view, rotate the transducer counterclockwise 90 degrees so that the indicator is directed toward the patient's right (Fig. 24.97).
- Identify the uterus, rectum, ovaries, and rectouterine pouch (Fig. 24.98).

Abnormal Findings

- Free intraperitoneal fluid: Anechoic (black) bands of fluid located in the rectouterine pouch (pouch of Douglas).
- Ectopic pregnancy: Most commonly, an ectopic pregnancy is identified as an adnexal mass with an associated empty uterus (Figs. 10.39 and 24.99). At times, an extrauterine gestational sac with or without a fetal pole may be seen. Rarely, a pseudogestational sac (an anechoic fluid

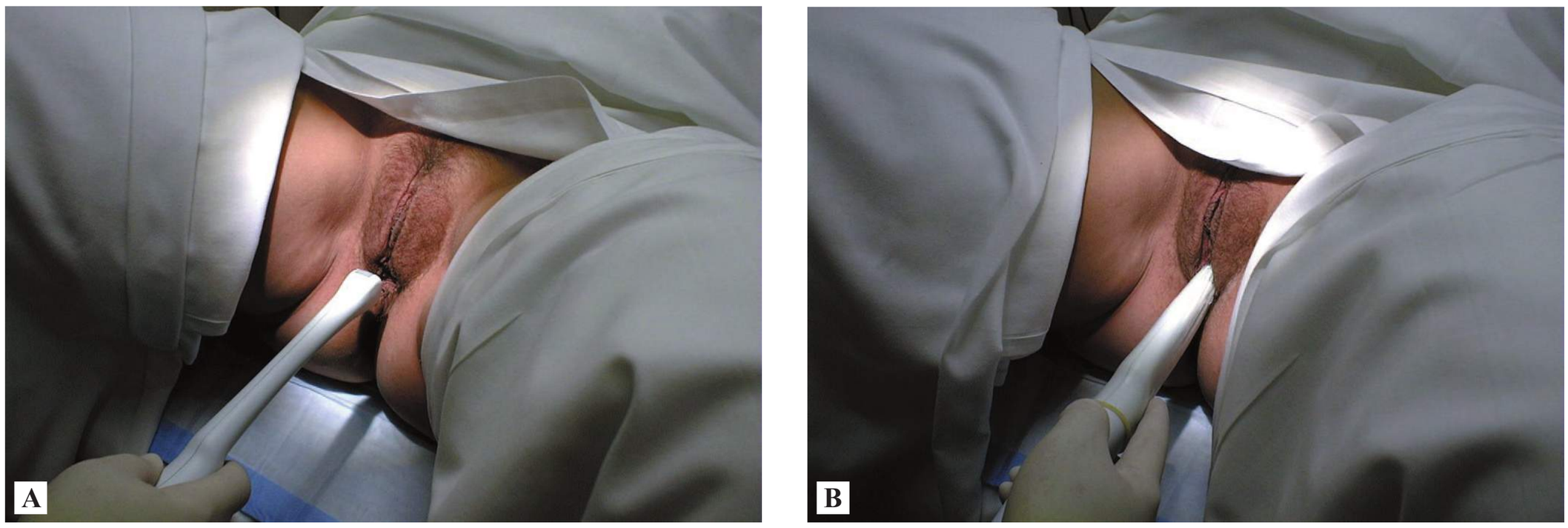


FIGURE 24.95 ■ **Transvaginal Sagittal View.** The transducer is directed toward the anterior fornix in a line through the umbilicus (**A**); it is placed into the vagina (**B**). The probe is advanced gradually. (Photo contributor: Michael J. Lambert, MD.)

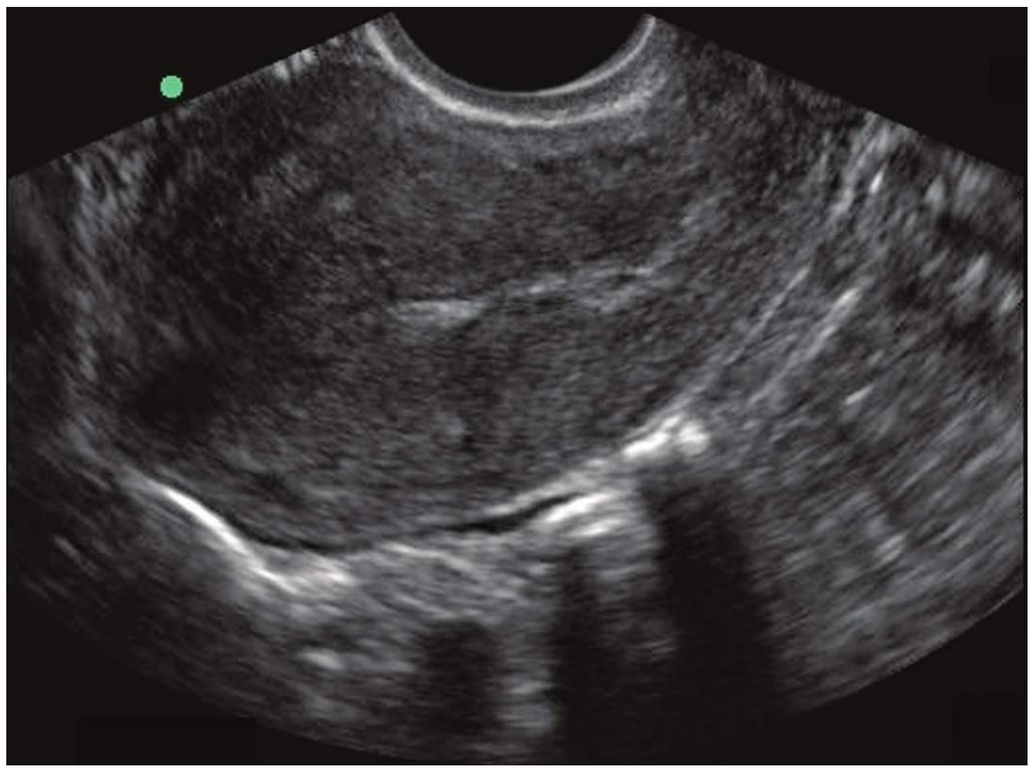


FIGURE 24.96 ■ **Transvaginal: Sagittal View.** The uterus with endometrial stripe. (Ultrasound contributor: Robinson M. Ferre, MD.)

collection less than 1 cm in diameter within the uterus) may mimic an early gestational sac.

- IUP: The first evidence of an IUP is a gestational sac with two thick, concentric echogenic ring within the body or fundus of the uterus. Because this may be confused with a pseudogastional sac, for EUS, the first definitive evidence of an IUP is a gestational sac with a visualized yolk sac and/or fetal pole. A live IUP has both a fetal pole and cardiac activity. (See Figs. 10.37 and 10.38.)
- Abnormal IUP: A fetal pole greater than 7 mm without cardiac activity is diagnostic of a fetal demise. A gestational sac greater than 16 mm without a fetal pole and an enlarged yolk sac greater than 7 mm are concerning for failed IUP, but are not diagnostic. A mean gestational sac diameter of 25 mm or more without a fetal pole is diagnostic of failed IUP. (See Figs. 10.45 and 10.46.)
- Interstitial or cervical ectopic pregnancy: An IUP must be within the body or fundus of the uterus. A gestational sac that is within the cornua or cervix of the uterus, although technically within the uterus, is still considered an ectopic pregnancy. A gestational sac within 5 to 7 mm of the border of the myometrium is concerning for interstitial ectopic pregnancy (see Figs. 10.41 and 10.42).

Pearls

1. A full bladder improves visualization of structures posterior to the bladder in the transabdominal approach. An empty bladder is necessary for transvaginal ultrasound.
2. A small amount of free fluid found in the pouch of Douglas may be physiologic.
3. The bladder is a good landmark to find initially for orientation to the anatomy when scanning transabdominally.
4. When scanning transvaginally in the sagittal view, identify the endometrial stripe from the fundus of the uterus to the cervix. This is accomplished by tilting the probe (anteriorly to posteriorly) while maintaining a sagittal plane of the uterus.

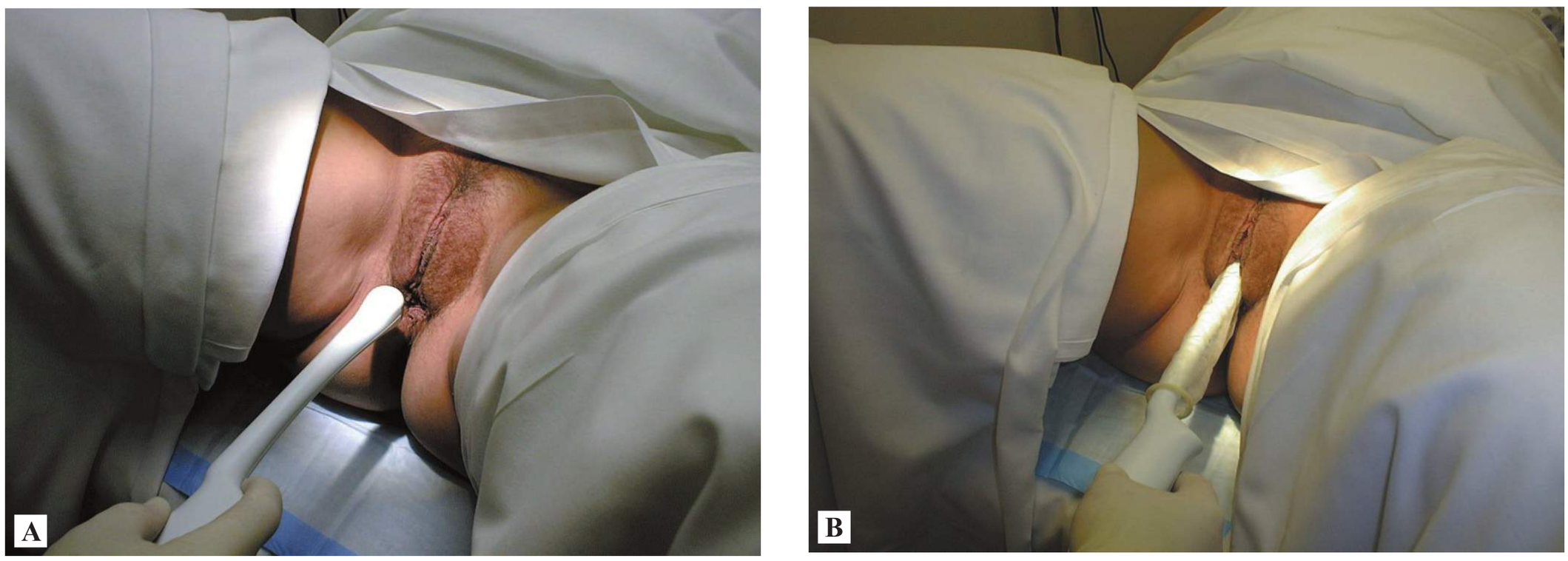


FIGURE 24.97 ■ **Transvaginal: Coronal View.** From the sagittal view, the transducer is rotated counterclockwise 90 degrees (**A**) and directed toward the posterior fornix in a line through the umbilicus (**B**). (Photo contributor: Michael J. Lambert, MD.)

5. Return to the fundus of the uterus in a sagittal view and slowly evaluate the right and then left borders of the uterus in the longitudinal axis.
6. Turn the transducer counterclockwise 90 degrees to enter the coronal plane. The uterus usually appears as an oval. Scan posteriorly to the cervix and then superiorly to the fundus of the uterus to exclude the presence of a bicornuate uterus.
7. If a pregnancy or intrauterine sac is identified with fetal cardiac activity, M-mode, not Doppler, should be used for documentation of fetal heart rate.
8. Evaluate both adnexa/ovaries individually by scanning each side in both coronal and sagittal planes. The ovaries ideally will be located anteromedially to the external iliac vessels. This is often only a guide to their location, and a methodical approach is often required to visualize both ovaries.
9. If the uterus is difficult to identify on TVS, withdraw the transducer slightly. A common error is insertion of the transducer too far, thus bypassing the uterus and imaging only bowel.

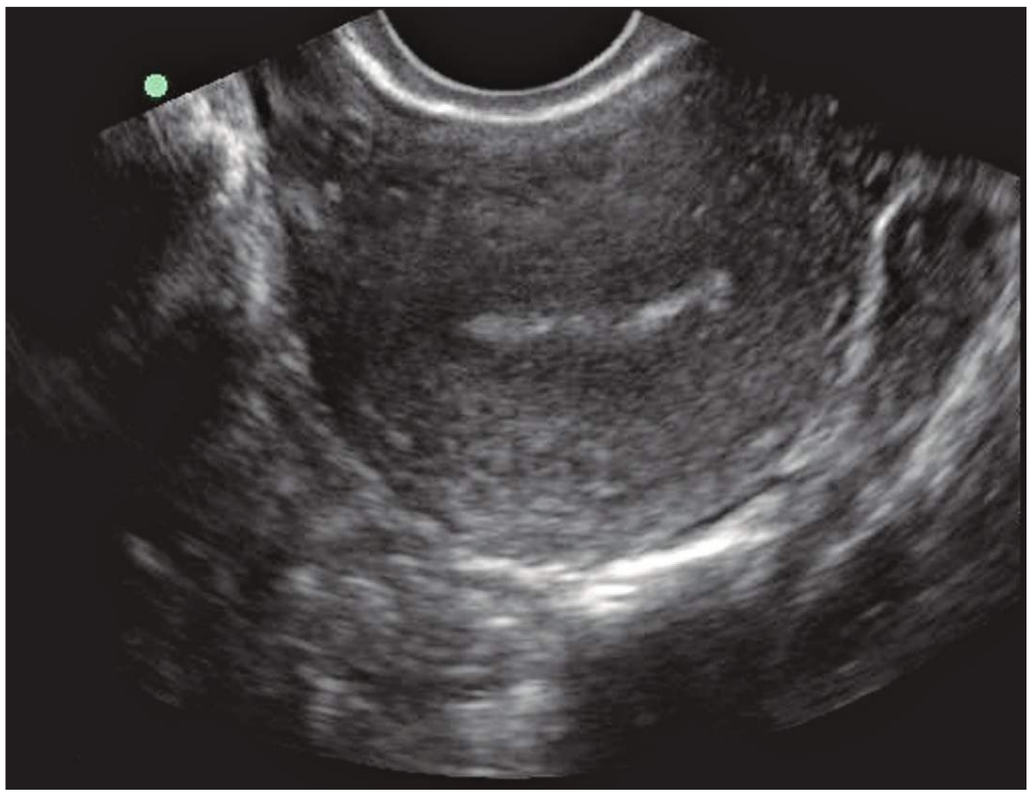


FIGURE 24.98 ■ **Transvaginal: Coronal.** The uterus with endometrial stripe. Left ovary visualized adjacent to the uterus on the right of the image. (Ultrasound contributor: Robinson M. Ferre, MD.)

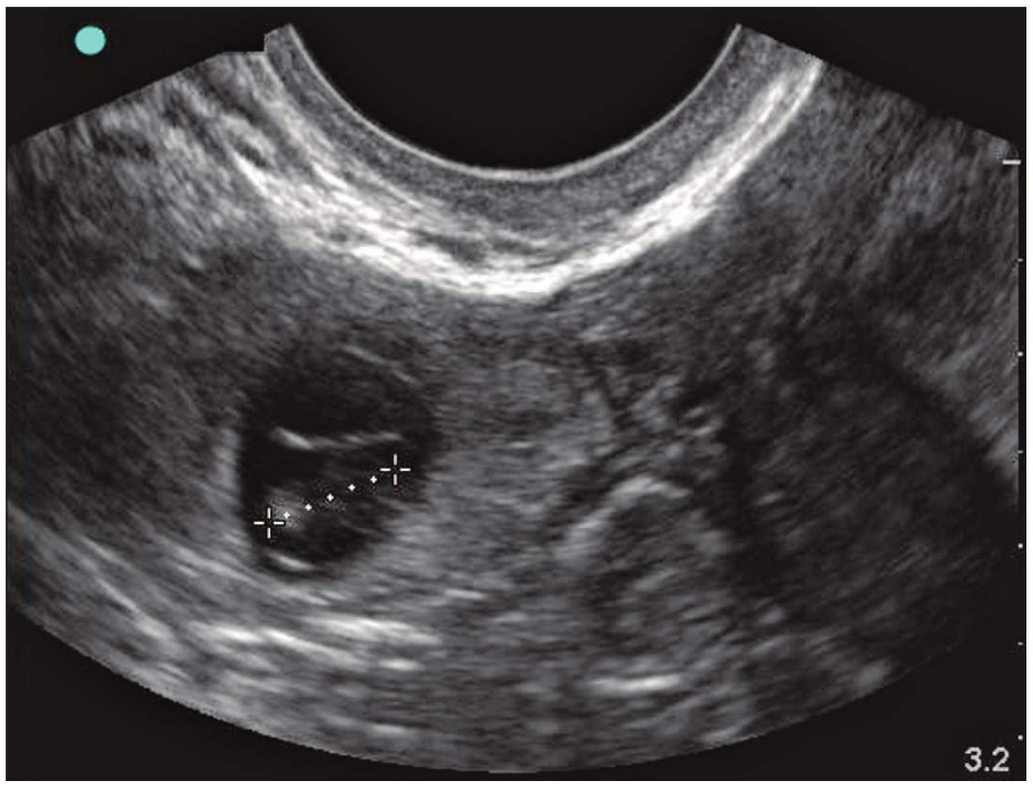


FIGURE 24.99 ■ **Transvaginal: Right Adnexa.** Ectopic pregnancy located in the right adnexa. (Ultrasound contributor: Robinson M. Ferre, MD.)

Clinical Summary

Vascular access is a necessity for evaluation and treatment of many patients in the ED. Traditionally, knowledge of common venous anatomy in experienced hands was the standard method of obtaining venous access. Factors such as obesity, prior access, and volume depletion can increase the difficulty of the conventional approach. This increased difficulty not only leads to failure to obtain access, but also increases the risk of complications.

Ultrasound-guided vascular access reduces the variables associated with the traditional landmark-based approach for central venous access and has become the standard of care. Additionally, ultrasound is useful for both difficult peripheral venous and arterial access.

Indications

- Need for venous access
- Obscured landmarks (obesity, trauma, etc)
- Cardiac arrest

Equipment

- Linear
- Convex

Patient preparation

- Position and prepare the patient as per convention for access and use a sterile sheath for the transducer.
- For central venous access, the patient is supine with the access site in a dependent position.

Technique

There are two main approaches to all venous access.

Short-Axis Approach

- In a transverse view, identify the structures of interest:
  - Vein
  - Corresponding artery
  - Others (nerve, trachea, etc)
- Confirm the vein as the collapsible vascular structure, and trace the vessel to ensure there are no anatomic variants that may disrupt your procedure.
- In the short-axis approach, maintain your transverse view and position the vein in the center of the image.
- Introduce the needle immediately distal to the transducer's edge at approximately 45-degree angle to the patient's skin and perpendicular to the long axis of the probe (Fig. 24.100).
- Maintaining the probe's orientation to the vessel, slide the probe away from the puncture site to maintain visualization of the tip of your needle by observing:
  - The needle tip
  - Tissue movement
  - Ring-down artifact from the needle
- Follow the tip of the needle until it contacts the anterior surface of the vein.
- Continue to advance until a venous flash is seen and the needle has reached the intended depth. If no flash is seen at the appropriate depth in the correct path, slowly withdraw the needle and observe for venous return.
- Continue catheter placement as per common practice.

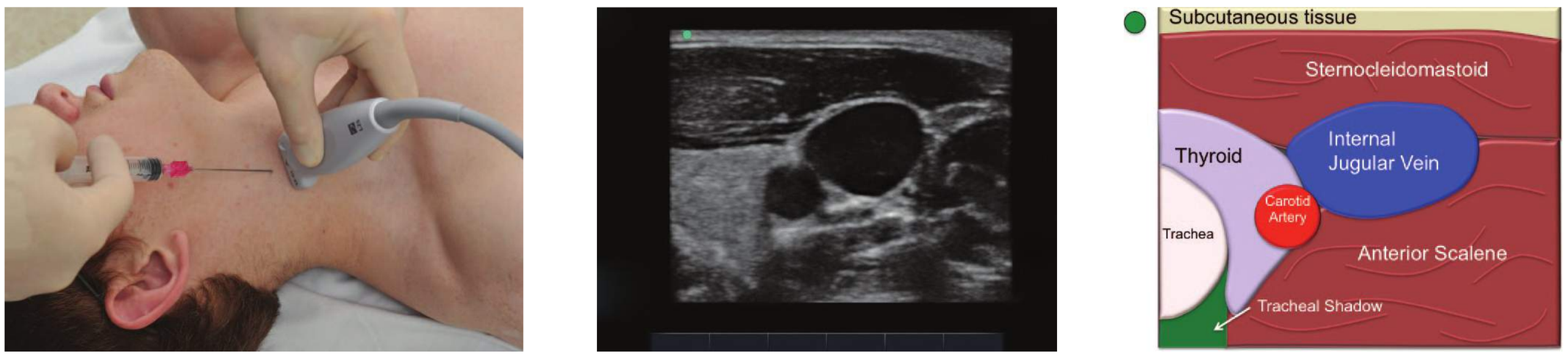


FIGURE 24.100 ■ **Internal Jugular Vein Cannulation, Short-Axis Approach.** The internal jugular vein and carotid artery are identified in cross-section. Introduce the needle immediately distal to the transducer's edge at approximately a 45-degree angle to the patient's skin and perpendicular to the long axis of the probe. (Photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

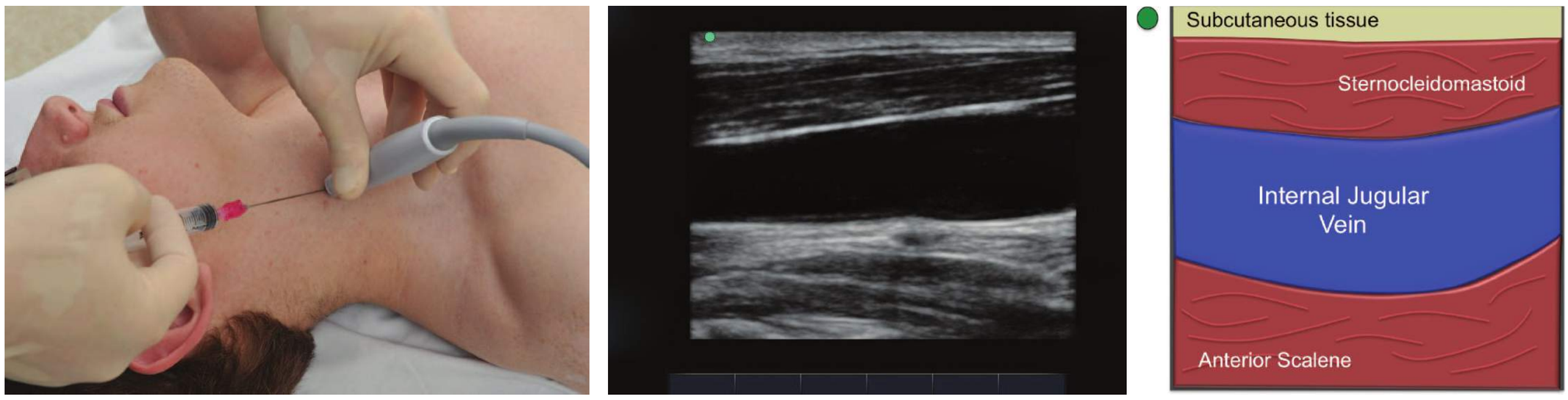


FIGURE 24.101 ■ **Internal Jugular Vein Cannulation, Long-Axis Approach.** The internal jugular vein is identified in its long axis. Introduce the needle immediately distal to the transducer's edge at approximately a 45-degree angle to the patient's skin and parallel to the long axis of the probe. (Photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

Long-Axis Approach

- In a transverse view, identify the structures of interest and confirm the vein as the collapsible vascular structure as above.
- Once the vein of interest is identified and centered on the screen, rotate the probe so that the transducer is in the same axis as the vein.
- Introduce the needle at the distal edge of the transducer.
- Direct the needle along the long axis of the probe careful to keep the needle completely in-line with the probe and ultrasound beam. (Fig. 24.101)
- Identify the path of the advancing needle as indicated by observing:
  - The needle tip
  - Tissue movement
  - Ring-down artifact from the needle
- Follow the tip of the needle until it contacts the anterior surface of the vein.
- Continue to advance until a venous flash is seen or the needle has reached the intended depth.
- If no flash is seen at the appropriate depth in the correct path, slowly withdraw the needle and observe for venous return.
- Continue catheter placement as per common practice.

Central Venous Access

Special considerations for central access:

- Typical sites include the internal jugular vein (Figs. 24.100 and 24.101) and the femoral vein, but the subclavian/axillary vein can also be accessed with ultrasound guidance (Fig. 24.102).
- Be careful to maintain sterile precautions including a sterile transducer cover.

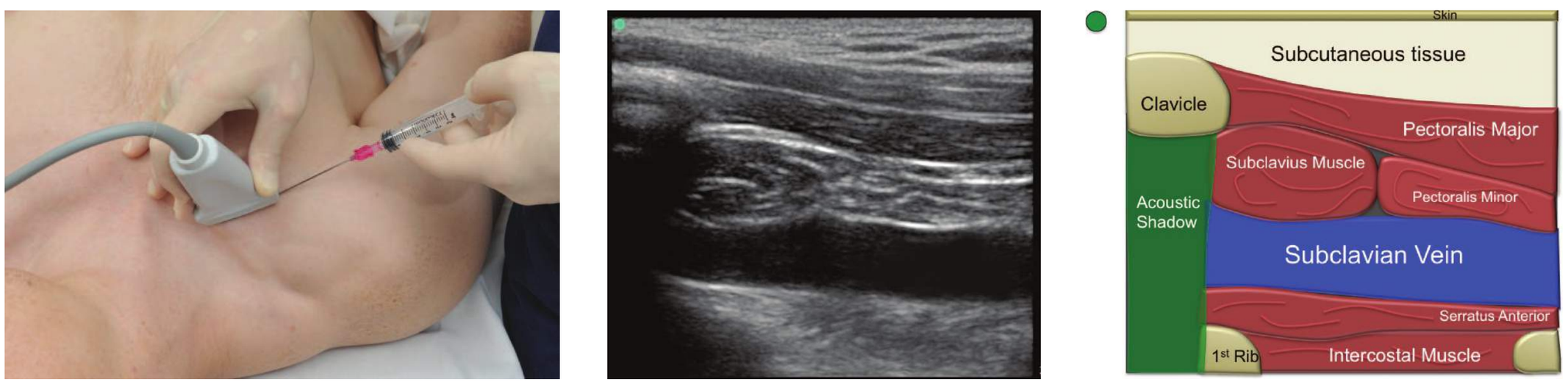


FIGURE 24.102 ■ **Subclavian Vein Cannulation, Long-Axis Approach.** The subclavian vein is identified in its long axis with the proximal edge of the transducer overlying the clavicle. Introduce the needle immediately distal to the transducer's edge at approximately a 30- to 45-degree angle to the patient's skin and parallel to the long axis of the probe. The needle should be visualized throughout its entire course. (Illustration courtesy of Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD.)

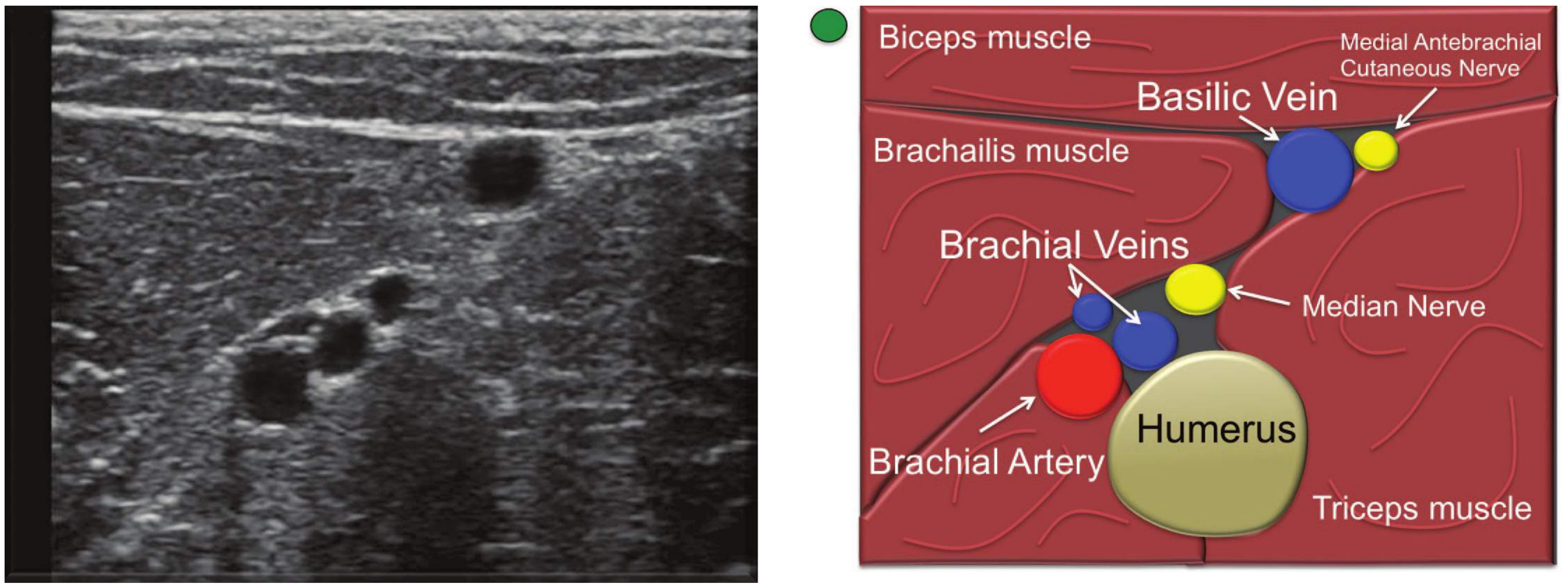


FIGURE 24.103 ■ **Deep Brachial Veins.** Transverse anatomy of the deep veins of the upper arm. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Steven Downie, MD.)

Peripheral Venous Access

Special considerations for peripheral access:

- Apply a tourniquet if needed.
- Ensure that both the needle tip and the plastic catheter are within the vein before advancing the catheter overlying the needle. This often requires advancing the needle just beyond the depth where an initial flash is obtained.
- The veins of the upper arm (basilic, deep brachial, and cephalic) are good targets for ultrasound-guided peripheral venous access (Figs. 24.103 and 24.104).

Pearls

1. Ultrasound identifies the relevant anatomy, but care must be used to follow the typical procedures for venous access.
2. When using a sterile sheath (or glove), use conductive media both inside and outside the sheath. The gel outside the sheath should also be sterile.
3. Venous access may be performed by a single provider or with an assistant.
4. Having the patient cough or perform the Valsalva maneuver may distend the internal jugular vein to facilitate venous access.

FIGURE 24.104 ■ **Peripheral IV Insertion.** Approaches to ultrasound-guided peripheral venous access of the upper extremity: short-axis (**A**), long-axis (**B**), and "backscratcher" approach (**C**). The backscratcher approach facilitates easy access to the more medial basilic vein while allowing improved patient comfort. It is easily performed from the head of the bed. (Photo contributor: Lawrence B. Stack, MD.)

PROCEDURAL GUIDANCE: THORACENTESIS/PARACENTESIS

Clinical Summary

Ultrasound is a source of valuable information that can improve the safety of a wide variety of procedures. The same principles of needle localization and guidance apply. Thoracentesis and paracentesis may be performed using these dynamic "real-time" needle imaging techniques. Alternatively, a static approach may be employed by identifying the location of pathologic fluid and a site for needle insertion. The procedure is then performed in the usual fashion without use of the transducer.

Indications

- Need for thoracentesis or paracentesis.
- Pleural effusion or ascites

Equipment

- Convex
- Linear

Patient Preparation

- For paracentesis, supine position or supine with the head of bed slightly elevated.
- For thoracentesis, the seated upright position is preferred. Lateral decubitus positioning or recumbent position with the ipsilateral arm raised behind the head may also be used.

Technique

Paracentesis

- After positioning patient, ultrasound the abdomen to identify the largest and safest pocket of ascites (Fig. 24.105). The patient may be repositioned to further improve the target.
- After optimizing the image and the patient's positioning, the proceduralist may ultrasound throughout the paracentesis (dynamic technique) or mark the ideal target on the patient and then move forward with the procedure in the usual fashion (static technique).
- The same sterile techniques should be applied with use of ultrasound as per common practice including a sterile probe cover and sterile gel if dynamic imaging is used.
- Continue the procedure as per common practice.

Thoracentesis

- After positioning patient, ultrasound the pleural space posteriorly to identify the largest and safest pocket of pleural fluid (Fig. 24.106). The transducer should be held in the long or sagittal plane with the indicator aimed superiorly. The patient may be repositioned to further improve access.

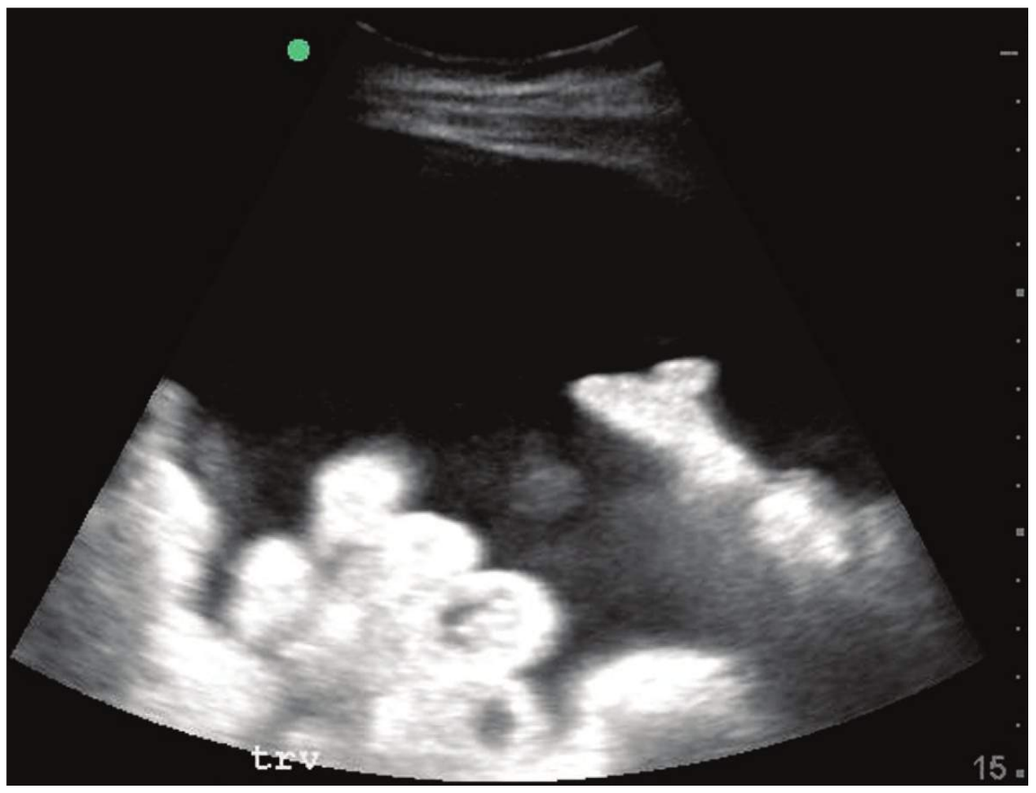


FIGURE 24.105 ■ **Paracentesis: Abdomen, Transverse.** Inferior abdomen with significant ascites. (Ultrasound contributor: Eric Wu, MD.)

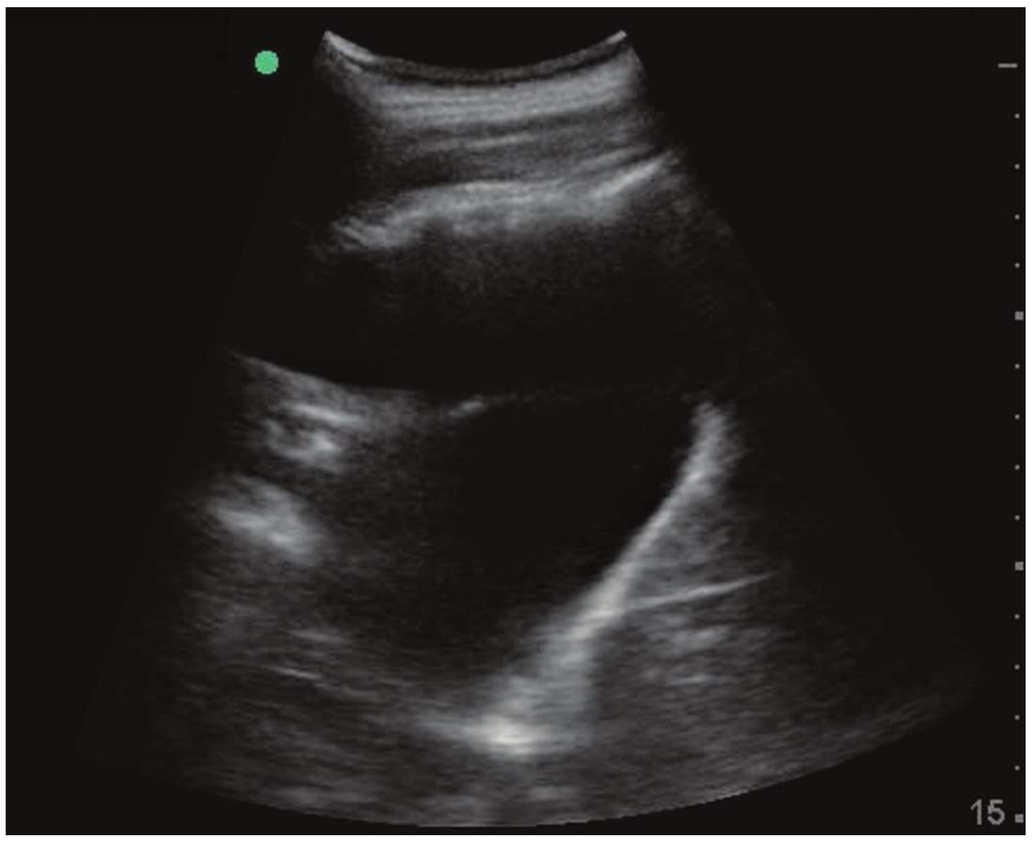


FIGURE 24.106 ■ **Thoracentesis: Right Thorax, Sagittal View.** Large pleural effusion identified prior to being drained via thoracentesis. (Ultrasound contributor: Craig Sheedy, MD.)

- After optimizing the image and the patient's positioning, the proceduralist may ultrasound throughout the paracentesis (dynamic technique) or mark the ideal target on the patient and then move forward with the procedure in the usual fashion (static technique).
- When choosing a procedure site using a static technique, locate a site that would be safe throughout the respiratory cycle and avoid diaphragmatic injury. The diaphragm and subdiaphragmatic contents can be easily visualized by ultrasound during normal respiration.
- The same sterile techniques should be applied with use of ultrasound as per common practice including a sterile probe cover and sterile gel if dynamic imaging is used.
- Continue the procedure as per common practice.

Pearls

1. When performing procedures with dynamic ultrasound guidance, keeping the needle tip in view is crucial to procedure safety.
2. Ultrasound identifies the relevant anatomy, but care must be used to follow the typical procedures for paracentesis/thoracentesis (eg, inserting needle immediately superior to the rib to avoid the intercostal neurovascular bundle).
3. Color flow Doppler imaging should be used to identify abdominal or thoracic wall vessels and prevent vascular injury.

OCULAR ULTRASOUND

Clinical Summary

Emergency physician–performed ocular ultrasound is a rapid and reliable way to evaluate the anterior and posterior chambers of the eye for a variety of emergent conditions, including lens dislocation, intraocular foreign body, and retinal and posterior vitreous detachment. It is particularly useful in the setting of ocular trauma when a direct fundoscopic examination is needed, but difficult or impossible to obtain.

Indications

- Visual loss
- Ocular trauma

Equipment: Recommended Transducers for Ocular Ultrasound

- High-frequency linear array
- It is important to select the "ophthalmic exam setting" or confirm and then adjust the energy output levels of your ultrasound system that are approved for ocular ultrasound.

Patient Positioning

- With the patient lying supine, place a liberal amount of gel on top of the patient's closed eyelid. The orientation marker should be directed toward the patient's right for transverse images and cephalad for sagittal images.
- The probe should exert little to no pressure on the patient's eye. This is accomplished by stabilizing your fingers on the bridge of the nose (when imaging the right eye) or the zygomatic arch (for the left eye) (Fig. 24.107A,B).

Techniques

Transverse and Sagittal Views

- With the indicator pointing toward the patient's right, identify the anterior chamber, lens, posterior chamber, retina, optic nerve, and retrobulbar space (Fig. 24.108).
- The probe should be fanned back and forth to ensure that the entire retina is visualized.
- The overall gain should be increased to assess for evidence of hemorrhage or vitreous detachment in the posterior chamber.
- If assessing for elevated intracranial pressure, the optic nerve sheath diameter (ONSD) should be measured 3 mm from the retina (Fig. 24.109). If measuring the ONSD, it is essential to ensure that the plane of the ultrasound beam is not tangential to the optic nerve.
- The eye should be imaged in both the sagittal and transverse planes to increase the accuracy of detecting pathology, particularly retinal detachment.

Abnormal Findings

- Retinal detachment: A retinal detachment is the separation of the neurosensory retina from the retinal pigment epithelium. The neurosensory retina is a dense neurovascular structure that is highly reflective of ultrasound and will appear as a bright echogenic line on top of the remaining choroid and retinal pigment epithelium (Fig. 24.110). An acute retinal detachment will appear to "wave" if the patient is asked to move their eyes side to side. An older retinal detachment is more rigid and will often lack this mobility.

FIGURE 24.107A, B ■ **Ocular Ultrasound.** Place a liberal amount of gel on top of the patient's closed eyelid. The orientation marker should be directed toward the patient's right for transverse images and cephalad for sagittal images. The probe should exert little to no pressure on the patient's eye. (Photo contributor: Lawrence B. Stack, MD.)

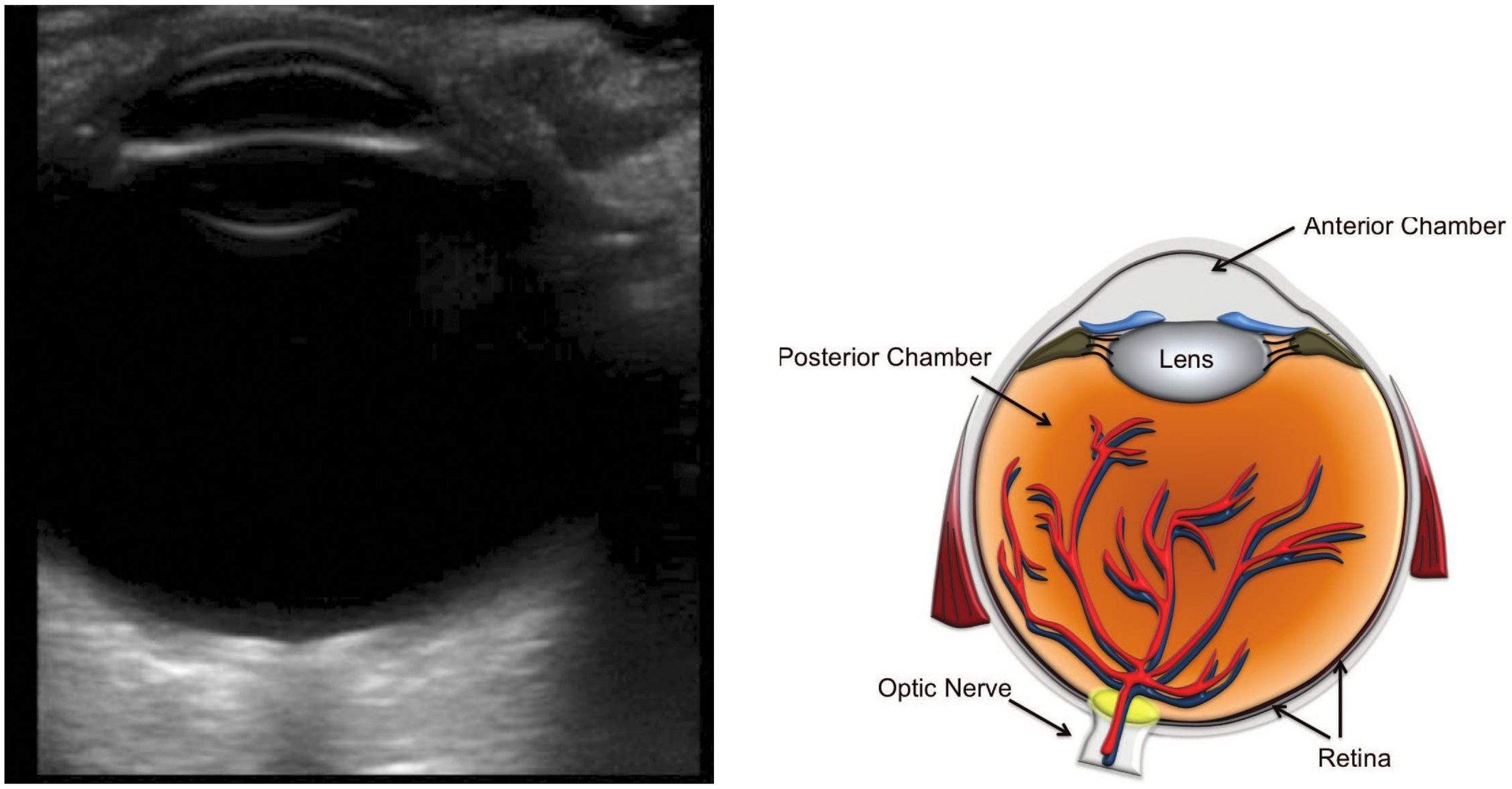


FIGURE 24.108 ■ **Ocular Ultrasound.** Normal eye anatomy. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

- Vitreous detachment: The normally anechoic vitreous will demonstrate various echoes that are usually only seen with increasing the gain. These small, filament-like threads of the detached vitreous body will "swirl" around with eye movements (Fig. 24.111).
- Vitreous hemorrhage: Hyperechoic areas in the vitreous or anterior chamber may represent hemorrhage. It is usually necessary to increase the gain to see posterior vitreous hemorrhage (Fig. 24.112).
- Dilated optic nerve sheath: As intracranial pressure increases, this pressure is transmitted to spinal fluid and along the optic nerve sheath. An ONSD greater than 4.5 mm in children and 5 mm in adults is a sensitive, but not specific, finding for elevated intracranial pressure. The average of three repeated measurements increases accuracy.
- Dislocated lens: The lens is normally positioned in a central location, immediately under the iris. The anterior convex portion of the normal lens is not seen with

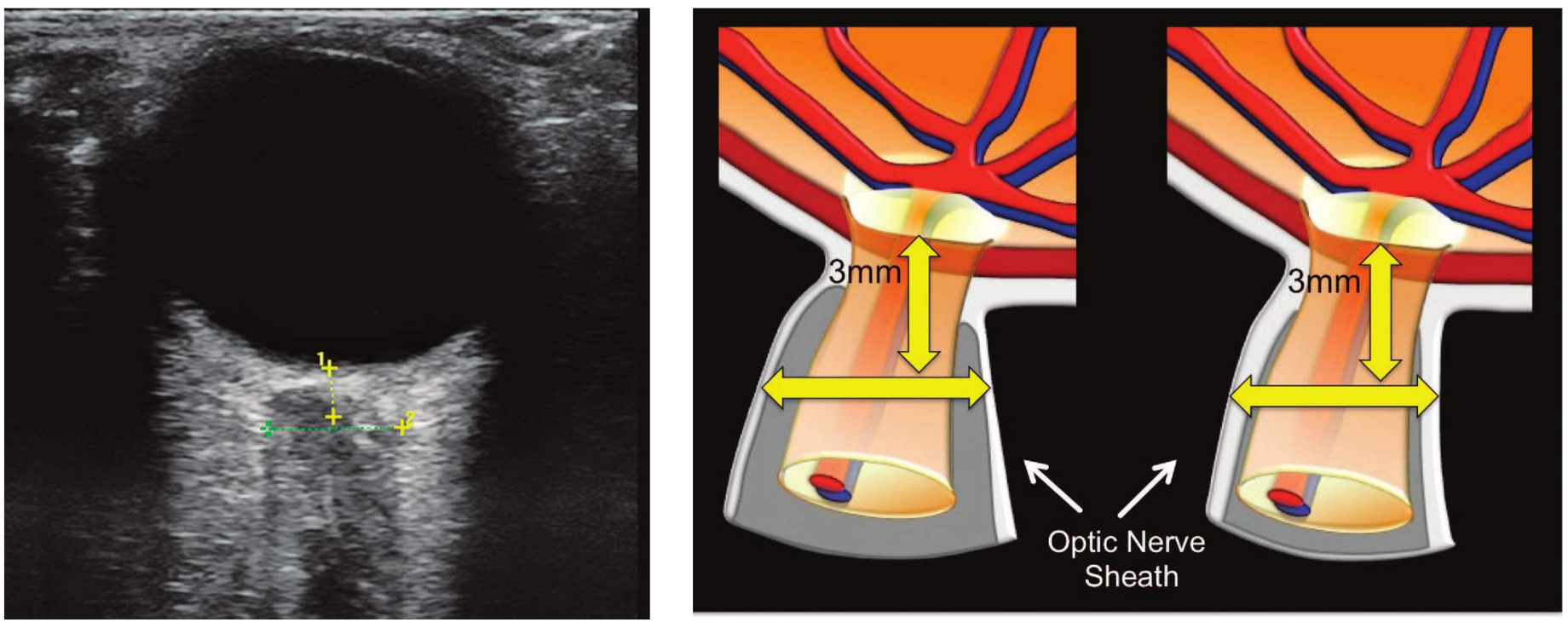


FIGURE 24.109 ■ **Ocular Ultrasound.** The ONSD is measured 3 mm from the retina. The diagram shows representations of a dilated ONSD (left) next to a normal one (right). (Illustration courtesy of Robinson M. Ferre, MD.)

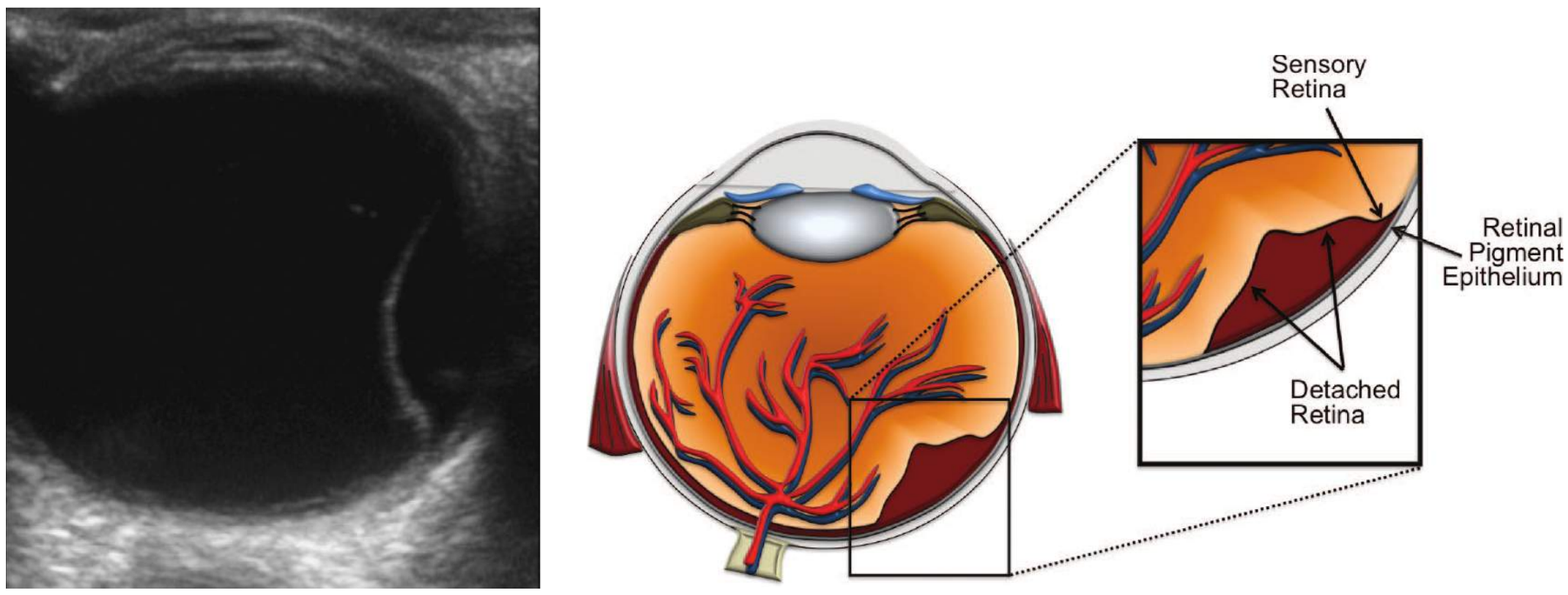


FIGURE 24.110 ■ **Ocular Ultrasound: Retinal Detachment.** A retinal detachment is the separation of the neurosensory retina from the retinal pigment epithelium. (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)

ultrasound, but the posterior concave portion is easily seen. A dislocated lens will be absent from its normal position and a diligent search will reveal its location somewhere in the posterior chamber (Fig. 24.113). A lens subluxation is much more subtle and is visualized as instability of the lens with eye movement.

Pearls

1. Confirmation of an abnormality can often be made by using the unaffected eye as the control. Using multiple planes to image a structure will improve accuracy.
2. Ocular ultrasound often necessitates increasing gain settings to visualize pathologic findings, particularly in the posterior chamber. Overgain the image, then gradually decrease the gain. This will allow small areas of vitreous hemorrhage or the lacy appearance of a vitreous detachment to be readily identified.
3. Both retinal and vitreous detachments are tethered to their origin and can be seen moving when the eye is moved by the patient medially or laterally. Because the retinal is continuous with the optic nerve, a retinal detachment will never appear to cross over the optic nerve head.
4. If it is important to keep the eye free of ultrasound gel, a small Tegaderm™ can be placed over the closed eyelid and the gel can then be placed on top of the Tegaderm™.

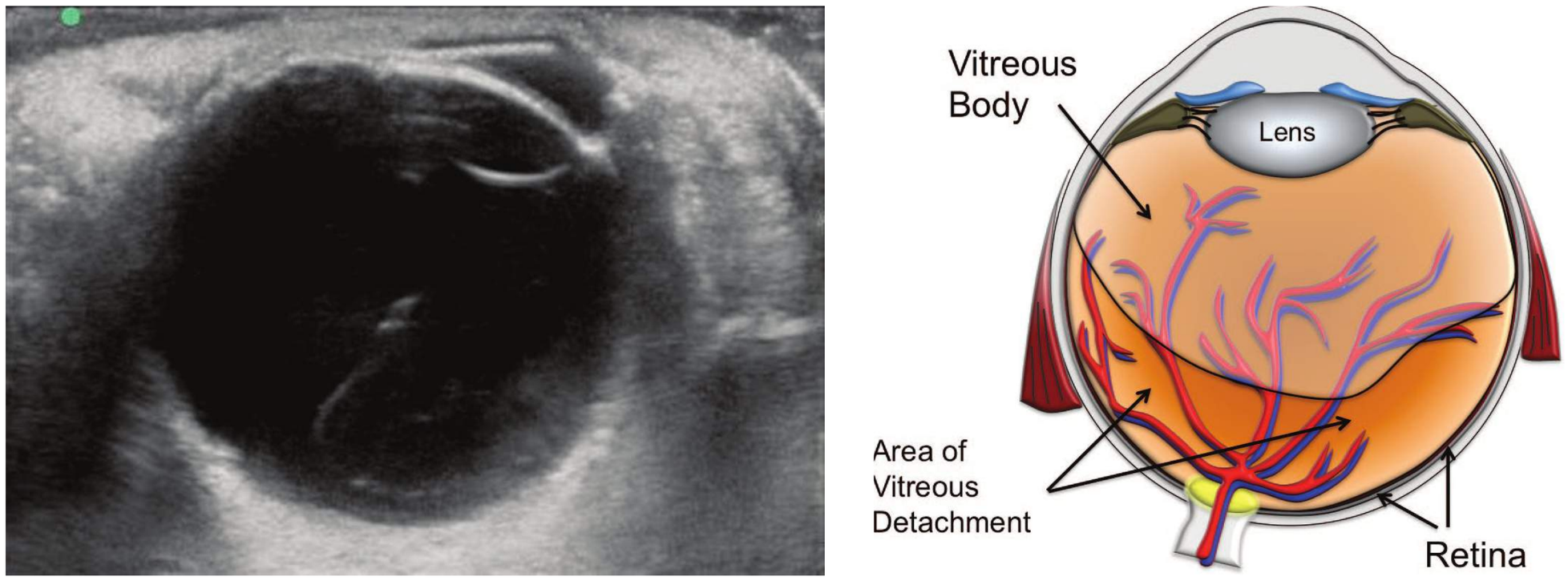
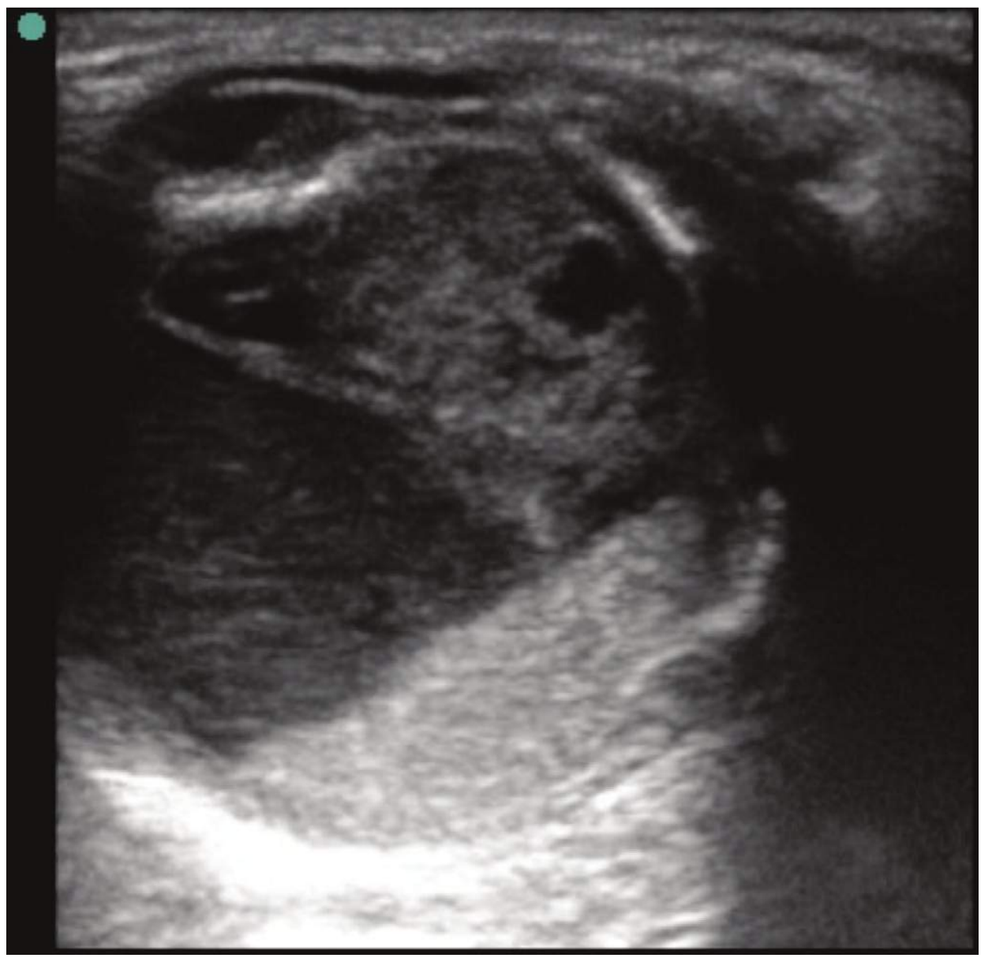


FIGURE 24.111 ■ **Ocular Ultrasound: Vitreous Detachment.** The normally anechoic vitreous will demonstrate various echoes that are usually only seen with increasing the gain. (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)



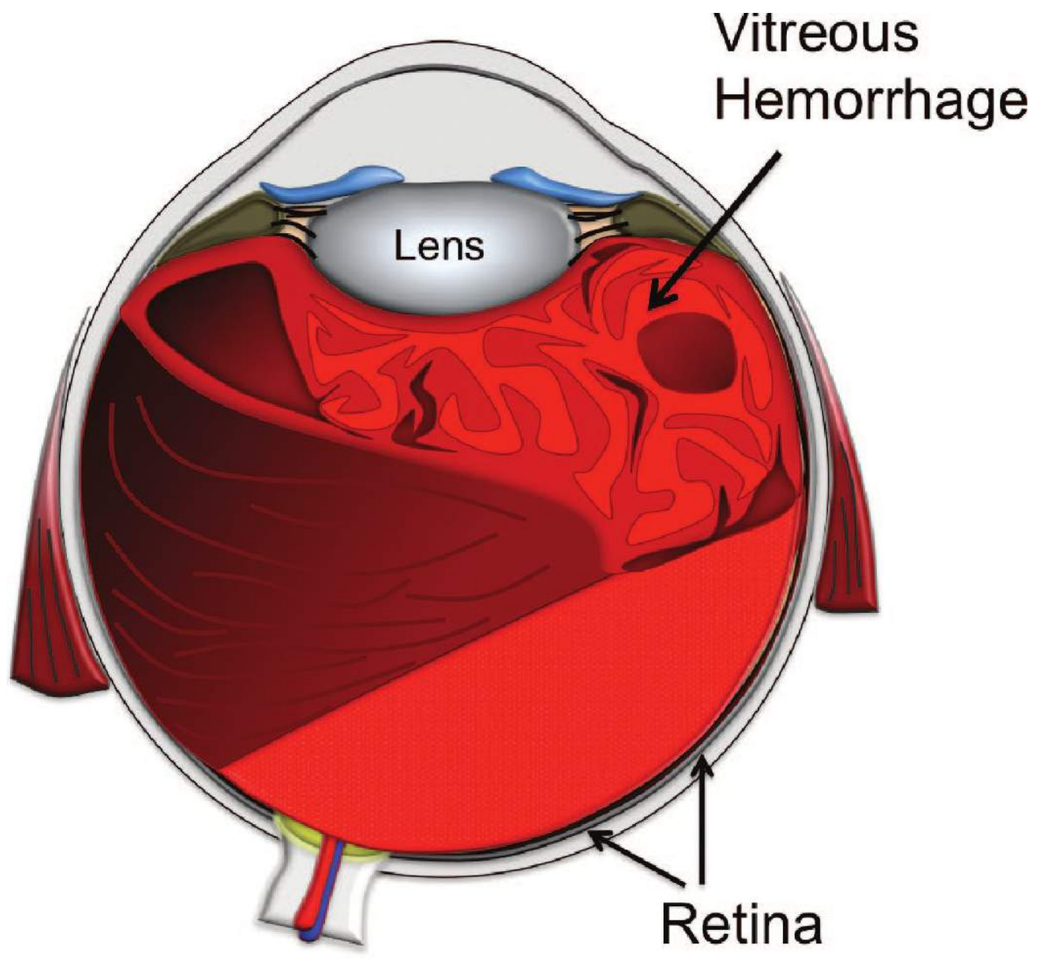
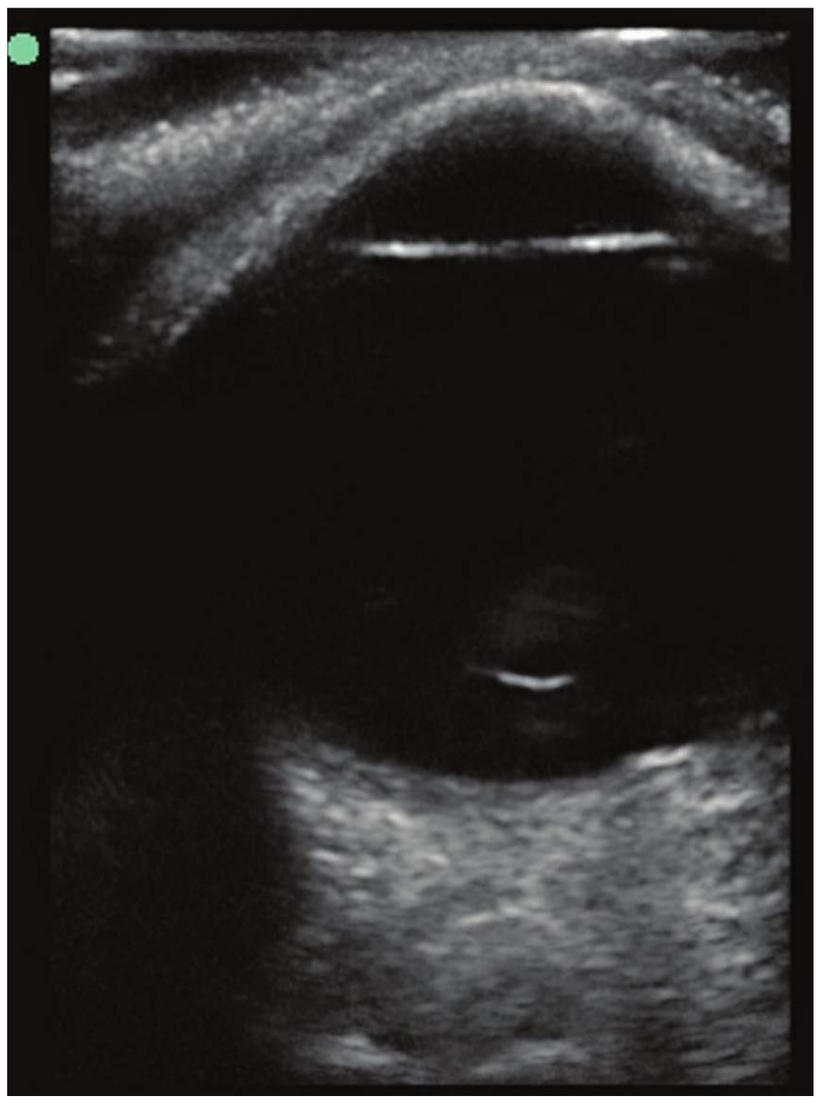


FIGURE 24.112 ■ **Ocular Ultrasound: Vitreous Hemorrhage.** Hyperechoic areas in the vitreous or anterior chamber may represent hemorrhage. It is usually necessary to increase the gain to see posterior vitreous hemorrhage. (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)



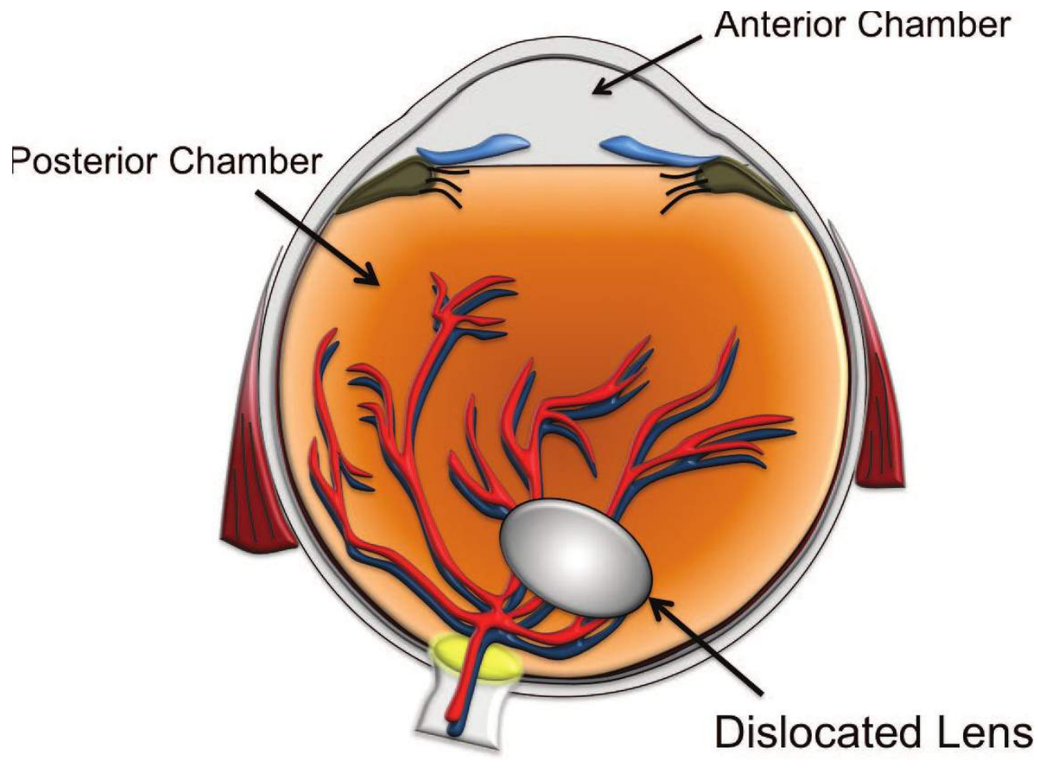


FIGURE 24.113 ■ **Ocular Ultrasound: Lens Dislocation.** A dislocated lens will be absent from its normal position and a diligent search will reveal its location somewhere in the posterior chamber. (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)

SOFT TISSUE ULTRASOUND

Clinical Summary

Soft tissue ultrasound has been shown to increase diagnostic accuracy, speed disposition, and reduce complications of procedures.

Indications

- Suspected fluid collection (abscess, hematoma, etc)
- Localization of subcutaneous foreign body
- Guidance for procedures

Equipment

- High-frequency linear array

Patient Positioning

- Place the patient in a position of comfort that allows maximum exposure of the area of interest.

Technique

- Image the area of interest by gently sweeping the probe across the skin. This should be done in two perpendicular planes. If possible, orient the planes along conventional axes of the body (ie, transverse and longitudinal).
- Identify the dermis, subcutaneous tissue, tendon, muscle, ligament, bone, and relevant neurovascular structures.
- Waterbath evaluation technique (WET): Distal extremities can be placed in a tub of water covering the area of interest and the water can be used as a conductive media for the ultrasound (Fig. 24.114). Ultrasound waves travel well through water, permitting a standoff approach where the probe need not actually touch the skin. This is better tolerated by the patient as no contact with the painful area is necessary and this technique facilitates imaging of extremely superficial structures.
- Apply superficial marking to overlying skin if necessary.

Abnormal Findings

- Edema/cellulitis: Increased interstitial fluid appears similar on ultrasound regardless of the cause (ie, cellulitis, lower extremity–dependent edema, etc). Initially, increased echogenicity and loss of definition of the tissue is noted in the subcutaneous fat. This obscures deeper structures such as muscle and bone. As the condition progresses, fluid is noted to accumulate diffusely in a reticular pattern, creating a cobblestone appearance (Fig. 24.115).
- Abscess: An abscess appears as a focal fluid accumulation, often with a significant amount of increased echogenicity in the surrounding tissues. The fluid may appear anechoic or have mixed complex echoes (Fig. 24.116). If drainage will be attempted, the depth and location of the abscess, any associated tracts, and other important and relevant adjacent structures should be noted and marked if appropriate (vessels, nerves, etc).

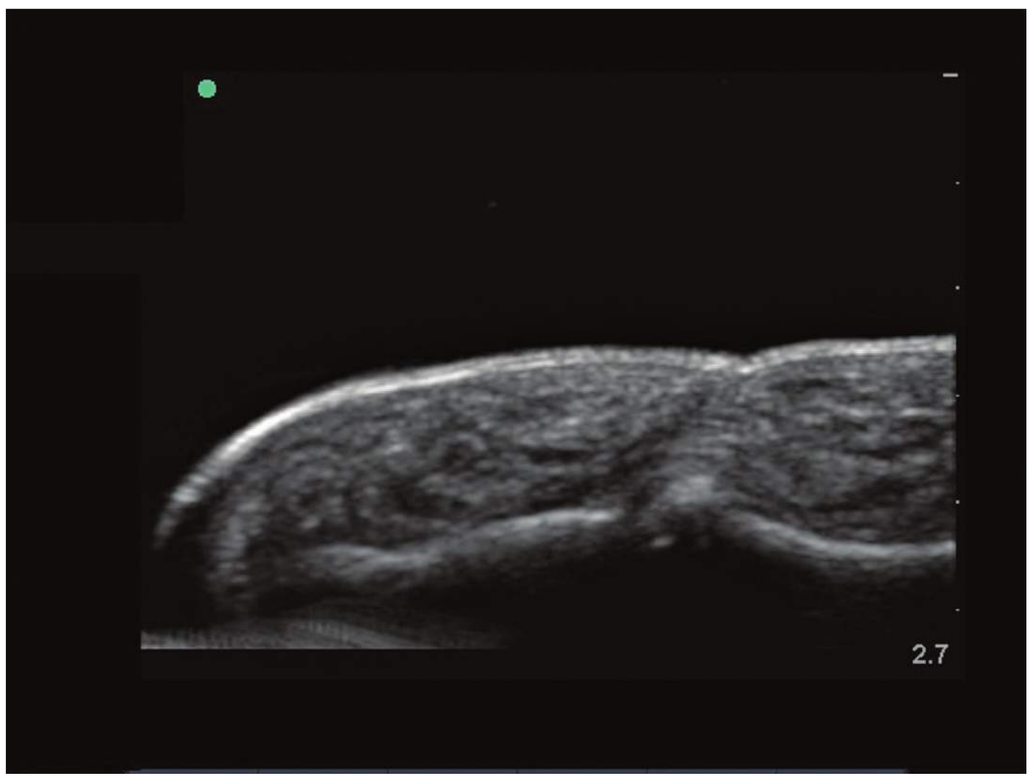


FIGURE 24.114 ■ **Waterbath Evaluation of the Extremity.** Distal extremities can be placed in a tub of water covering the area of interest and the water can be used as a conductive media for the ultrasound. The ultrasound image shows the volar aspect of the distal phalanx of the middle finger obtained using the waterbath technique. (Photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Jordan Rupp, MD.)

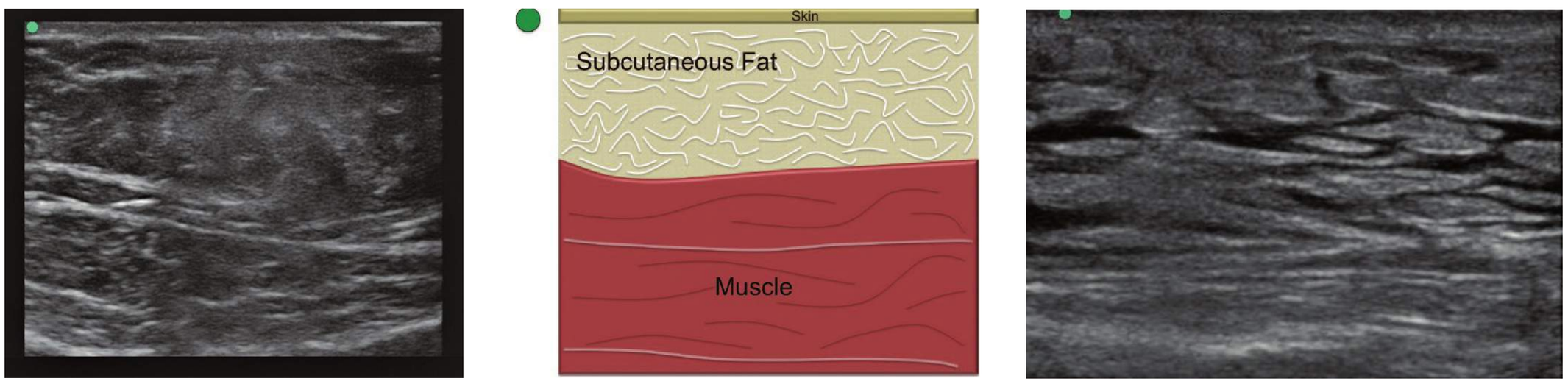


FIGURE 24.115 ■ **Soft Tissue Ultrasound: Cellulitis.** Initially, increased echogenicity and loss of definition of the tissue are noted in the subcutaneous fat (left). As the condition progresses, fluid is noted to accumulate diffusely in a reticular pattern creating a cobblestone appearance (right). (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)

- Joint effusion: Ultrasound can readily identify fluid accumulation within joints. A distended joint capsule will be visualized with anechoic or heterogeneous echogenic fluid (Fig. 24.117). Thickened synovial lining may be noted as well. Drainage can be assisted either statically, locating and marking area of maximal effusion, or dynamically, using ultrasound to guide the needle throughout the entire procedure.
- Subcutaneous foreign body: Most foreign bodies are visible using ultrasound. The foreign body's appearance depends on the shape, size, and composition of the object. Careful, slow passes should be made over the likely location, noting any abnormal object or defect in tissue (Fig. 24.118). EUS can assist in removal by either a static approach, marking the skin overlying the location, or dynamic technique guiding removal during real-time ultrasound. Care should be taken to identify any structures damaged by the foreign body or that may complicate removal.

Pearls

1. Always scan the area of interest in two planes. This will help to identify the exact location of the abnormality and exclude artifacts.
2. If needed, use the surrounding, unaffected tissue or other extremity as a control.
3. To optimize the image, reduce the depth to the appropriate level and increase the frequency of the probe. The dermis and other extremely superficial structures (<0.5 cm) may not be well visualized due to near-field artifact. Consider the use of a standoff pad or water bath if feasible.
4. For procedures requiring sterile precautions, a sterile sheath should be used to cover the transducer for dynamically guided procedures. Many sterile ultrasound sheaths are commercially available, although, less preferably, a sterile glove or transparent adherent dressing (eg, Tegaderm™) can be used. Sterile ultrasound gel or another sterile liquid (eg, Betadine or chlorhexidine) should be used as a conductive medium.

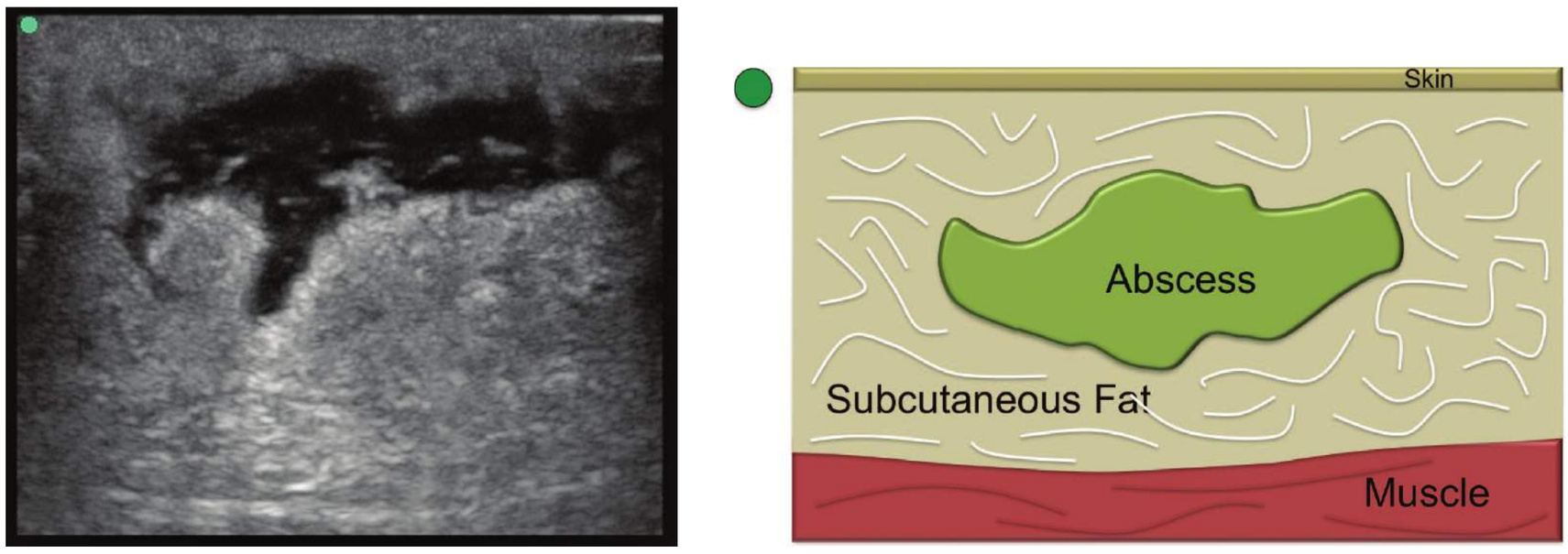


FIGURE 24.116 ■ **Soft Tissue Ultrasound: Abscess.** An abscess appears as a focal fluid accumulation, often with a significant amount of increased echogenicity of the surrounding tissues. The fluid may appear anechoic or have mixed complex echoes. (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)

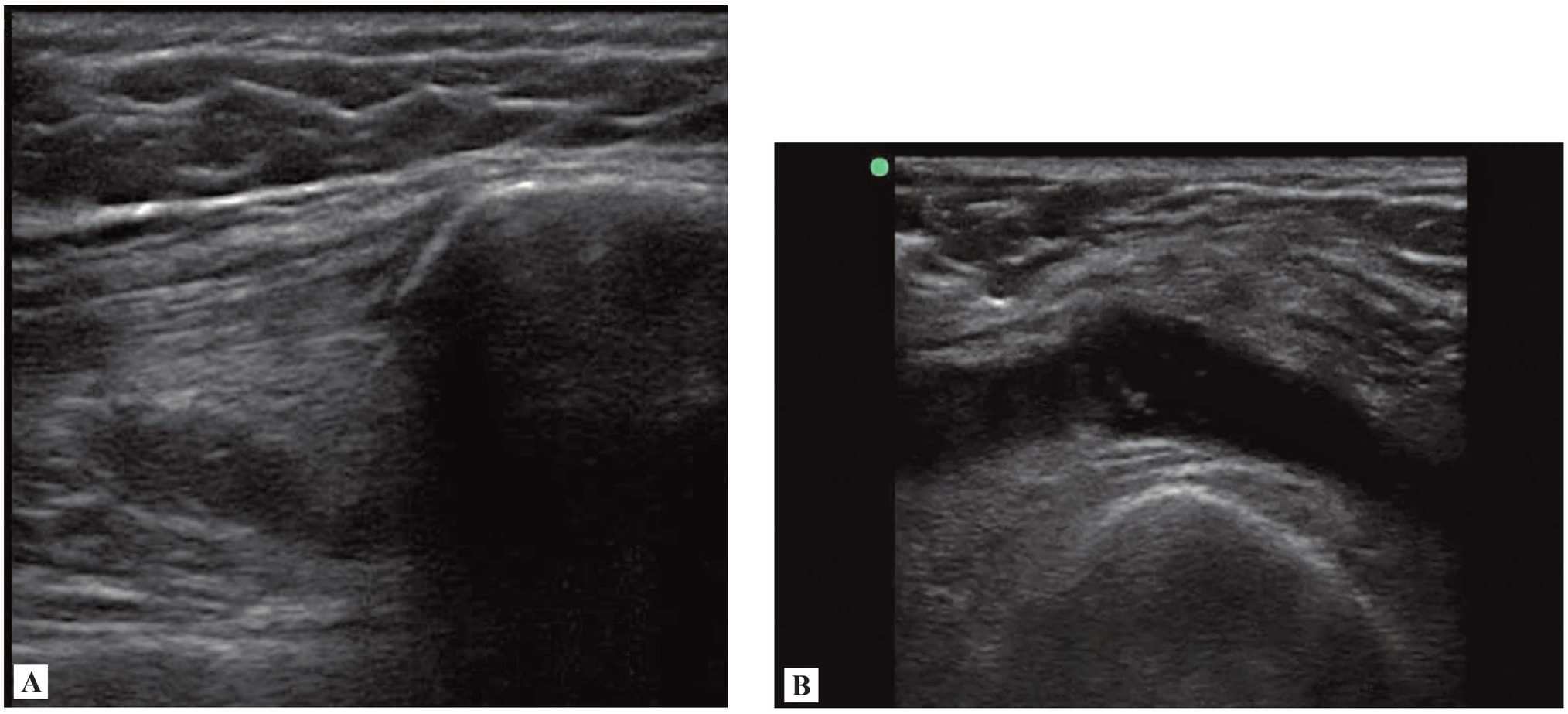


FIGURE 24.117 ■ **Soft Tissue Ultrasound: Joint Effusion.** A knee effusion is seen in sagittal (**A**) and transverse (**B**) views. (Ultrasound contributor: Robinson M. Ferre, MD.)

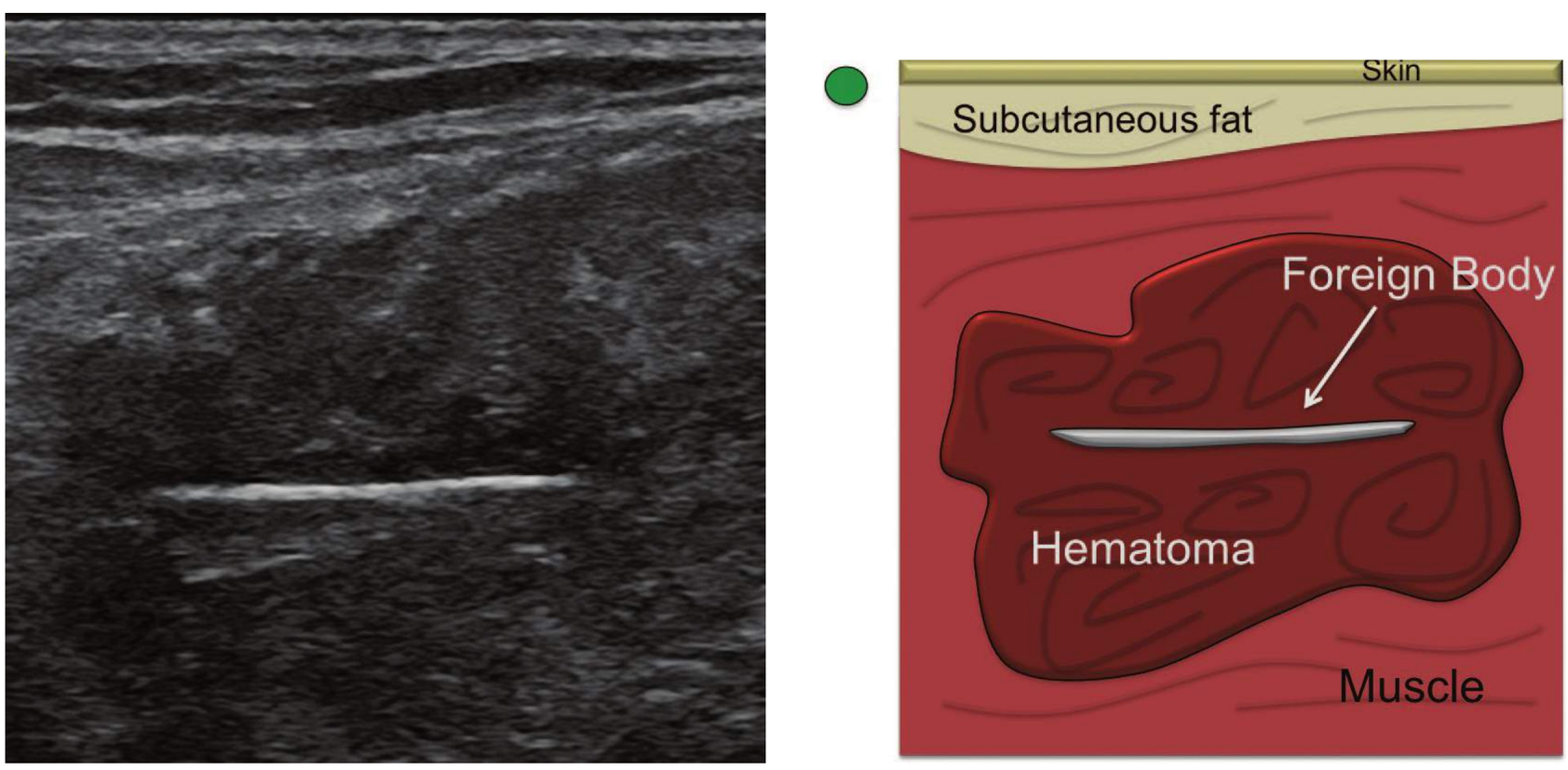


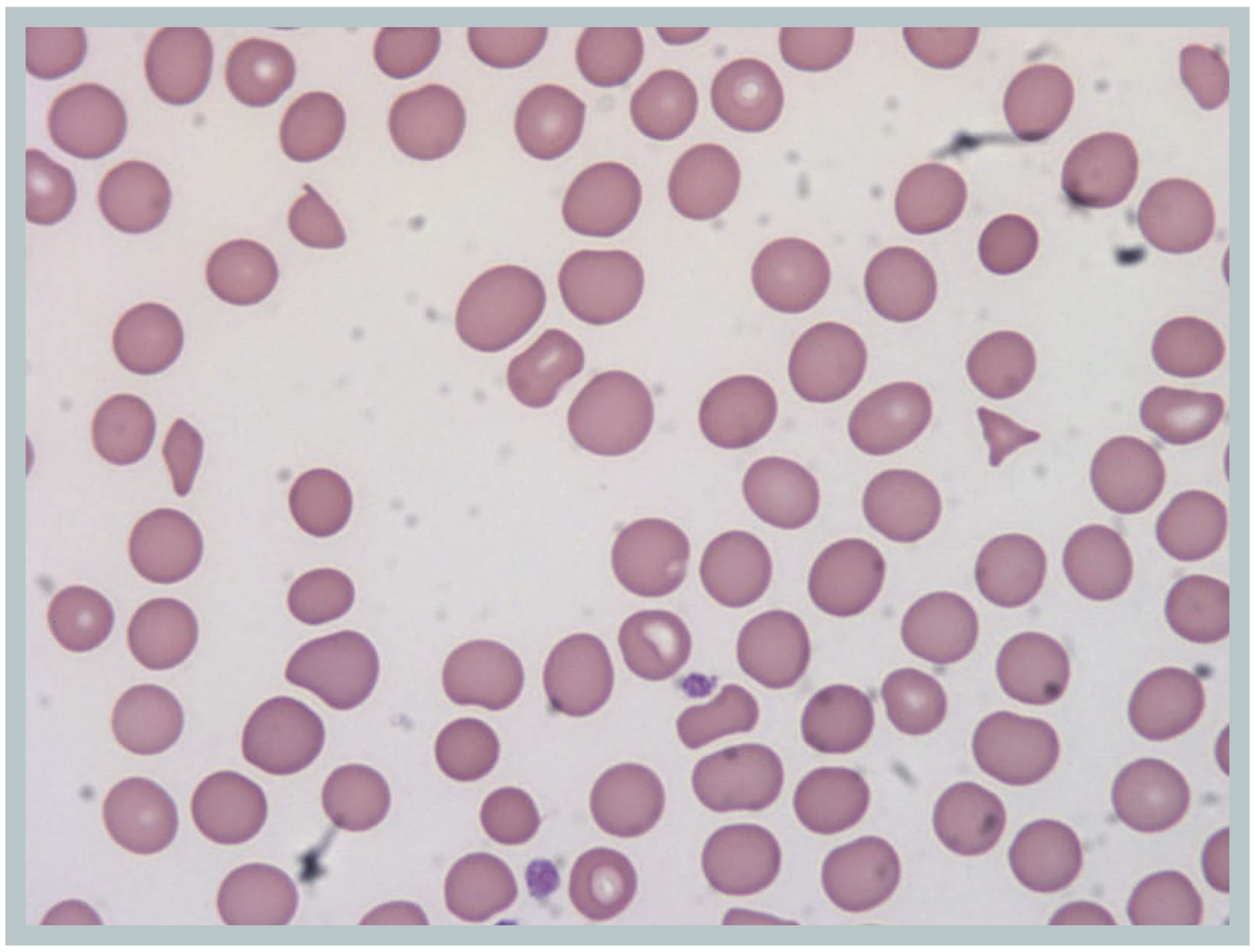
FIGURE 24.118 ■ **Soft Tissue Ultrasound: Foreign Body.** An echogenic, linear foreign body is visualized in the superficial soft tissues with surrounding hematoma. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

Chapter 25

MICROSCOPIC FINDINGS

Brett Bechtel

Scan QR code or go to www.ematlas.com for video related to chapter



Schistocytes. Several fragmented red blood cells are seen in this smear in a patient with microangiopathic hemolytic anemia. (Photo contributor: James P. Elrod, MD, PhD.)

The author wishes to thank Diane M. Birnbaumer for her contributions to prior editions.

Uses

To evaluate for the presence of cells, casts, and crystals.

Materials

Freshly collected urine specimen, centrifuge, graduated centrifuge tubes, glass microscope slide, and coverslip.

Method

1. Pour 10 mL of freshly collected urine into a graduated centrifuge tube.
2. Centrifuge at ×400 to ×450 gravity for 5 minutes.
3. Decant 9 mL of supernatant, leaving 1 mL in the tube.
4. Resuspend the centrifuged pellet in the remaining 1 mL of urine by stirring with a pipet.
5. Place one drop of resuspended urine on a glass microscope slide.
6. Overlay with a coverslip.
7. Examine initially using scanning ×10 power, emphasizing the periphery of the coverslip, since urinary elements tend to gather at the edges.
8. Switch to ×40 power to focus on specific urinary elements such as cells, casts, and crystals. Use ×100 power as needed for specific identification.

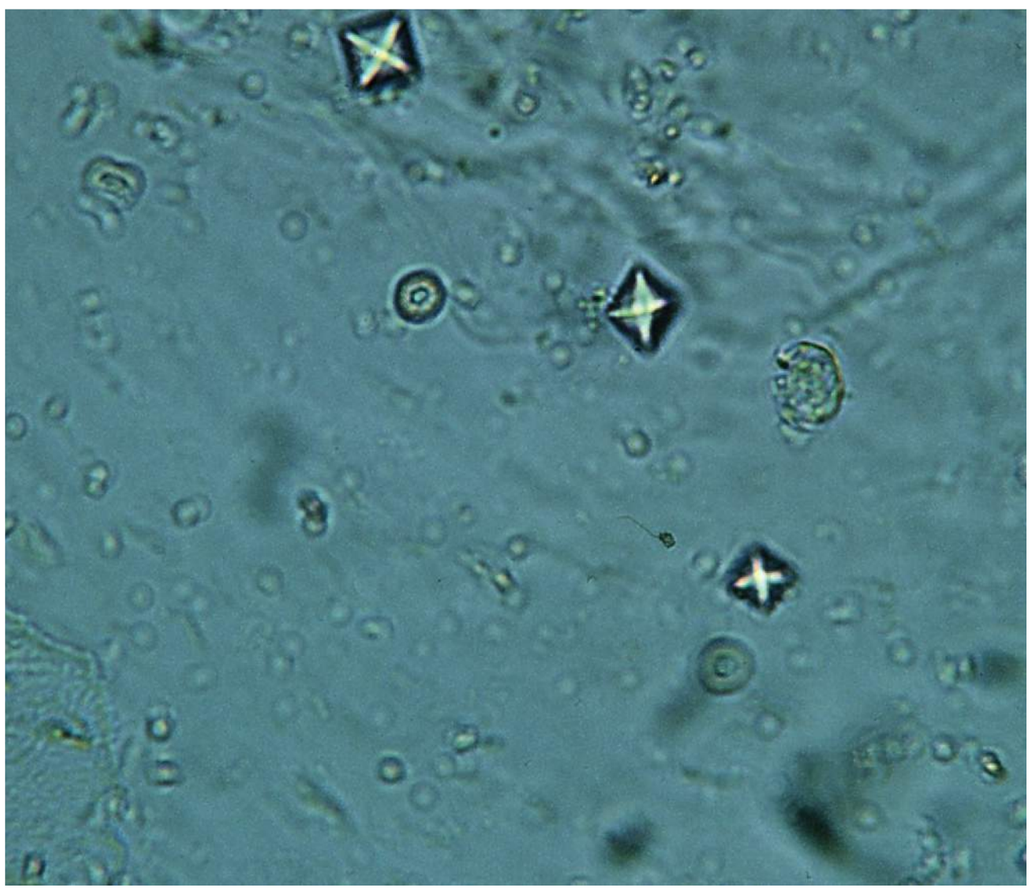


FIGURE 25.1 ■ **Calcium Oxalate Crystals.** Calcium oxalate crystals come in two shapes. The classically described octahedral, or envelope-shaped, crystals are made of calcium oxalate dihydrate. Calcium oxalate monohydrate crystals are needle-shaped. They are seen in acid or neutral urine. They may be found in the urine of patients with ethylene glycol ingestion. In addition, the urine of patients with ethylene glycol ingestion may also fluoresce under a Wood lamp. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 4th ed. Philadelphia: F.A. Davis Company; 2001, p. 97.)

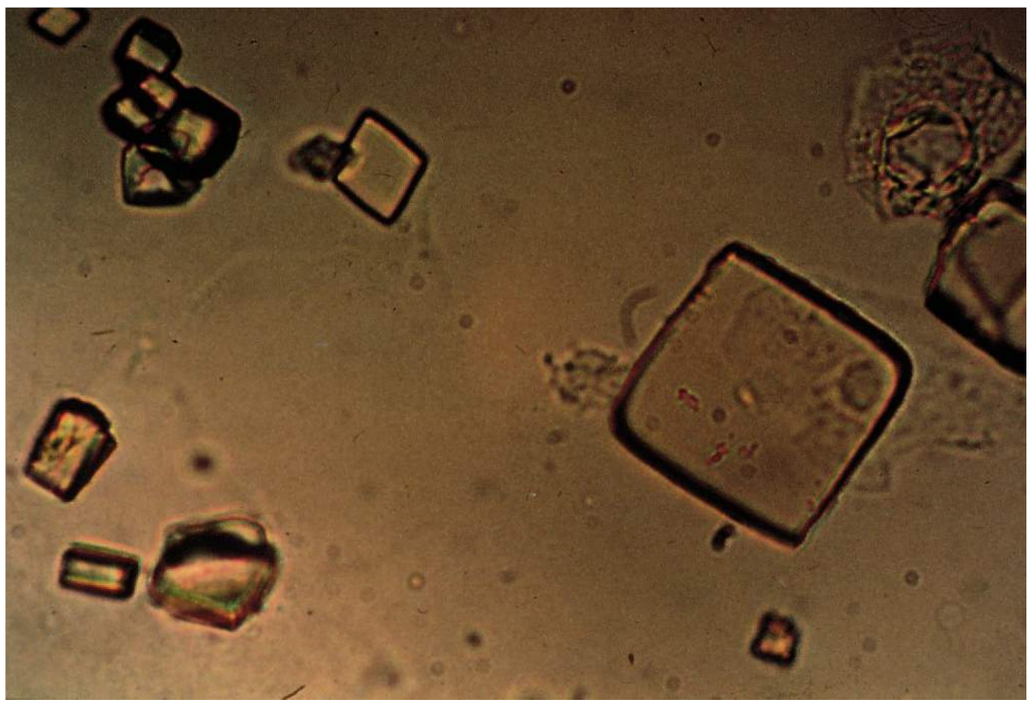


FIGURE 25.2 ■ **Uric Acid Crystals.** Uric acid crystals often have a yellow hue and a variety of sizes and shapes. They are found in acidic urine. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 114.)

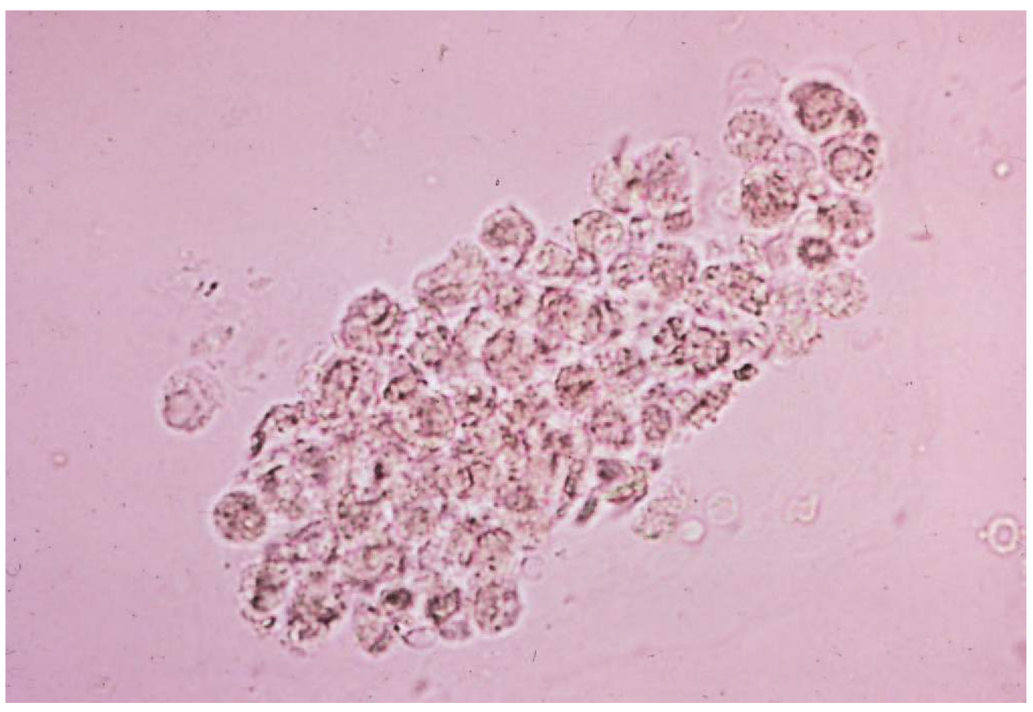


FIGURE 25.3 ■ **White Blood Cell Casts.** Usually two to three cells in width, white blood cell casts are indicative of upper urinary tract infection such as pyelonephritis. (Photo contributor: The American Society of Clinical Pathologists. © American Society of Clinical Pathologists.)

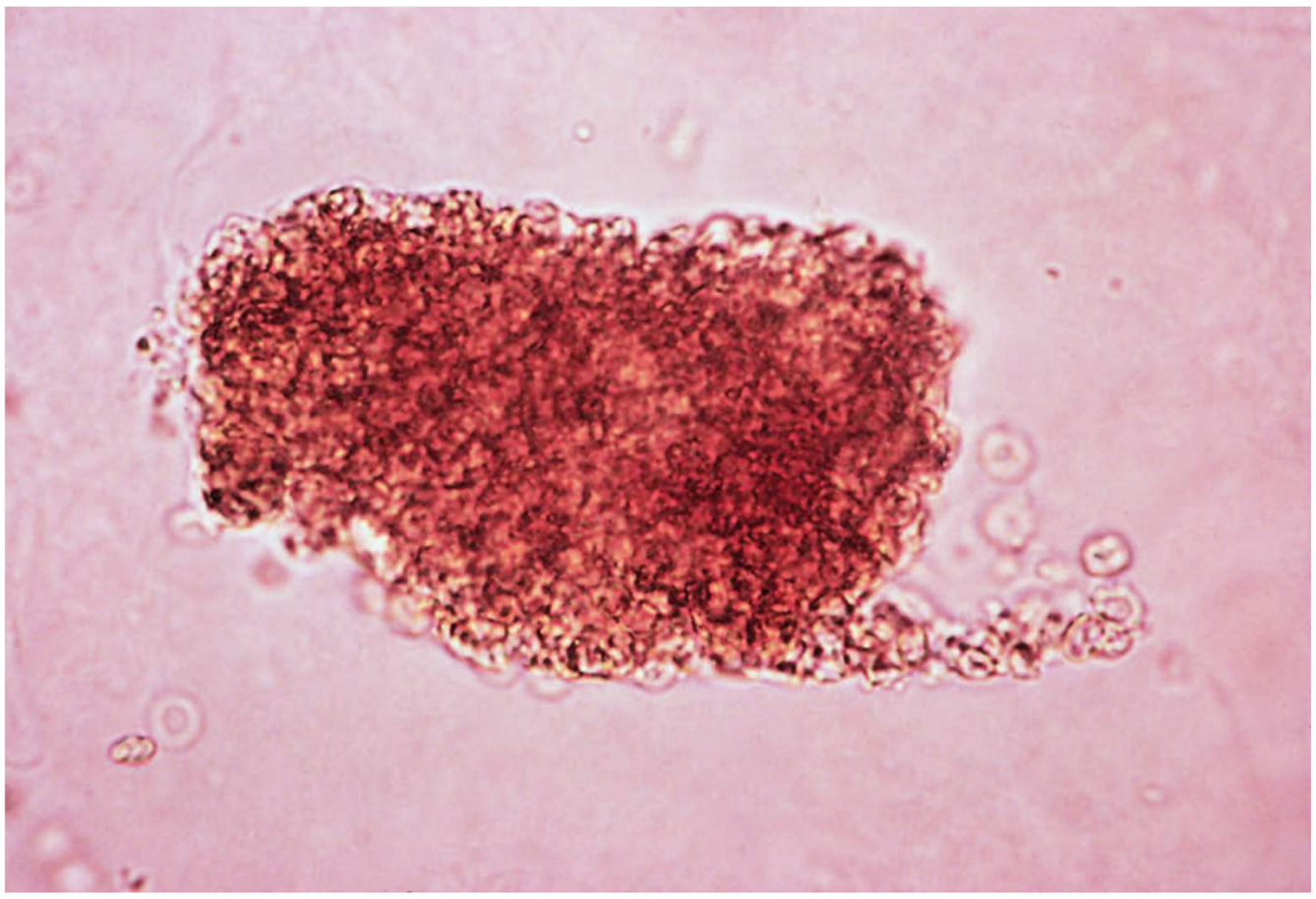


FIGURE 25.4 ■ **Red Blood Cell Casts.** Red blood cell casts range from 3 to 10 cells in width and are seen in glomerulonephritis. (Photo contributor: The American Society of Clinical Pathologists. © American Society of Clinical Pathologists.)

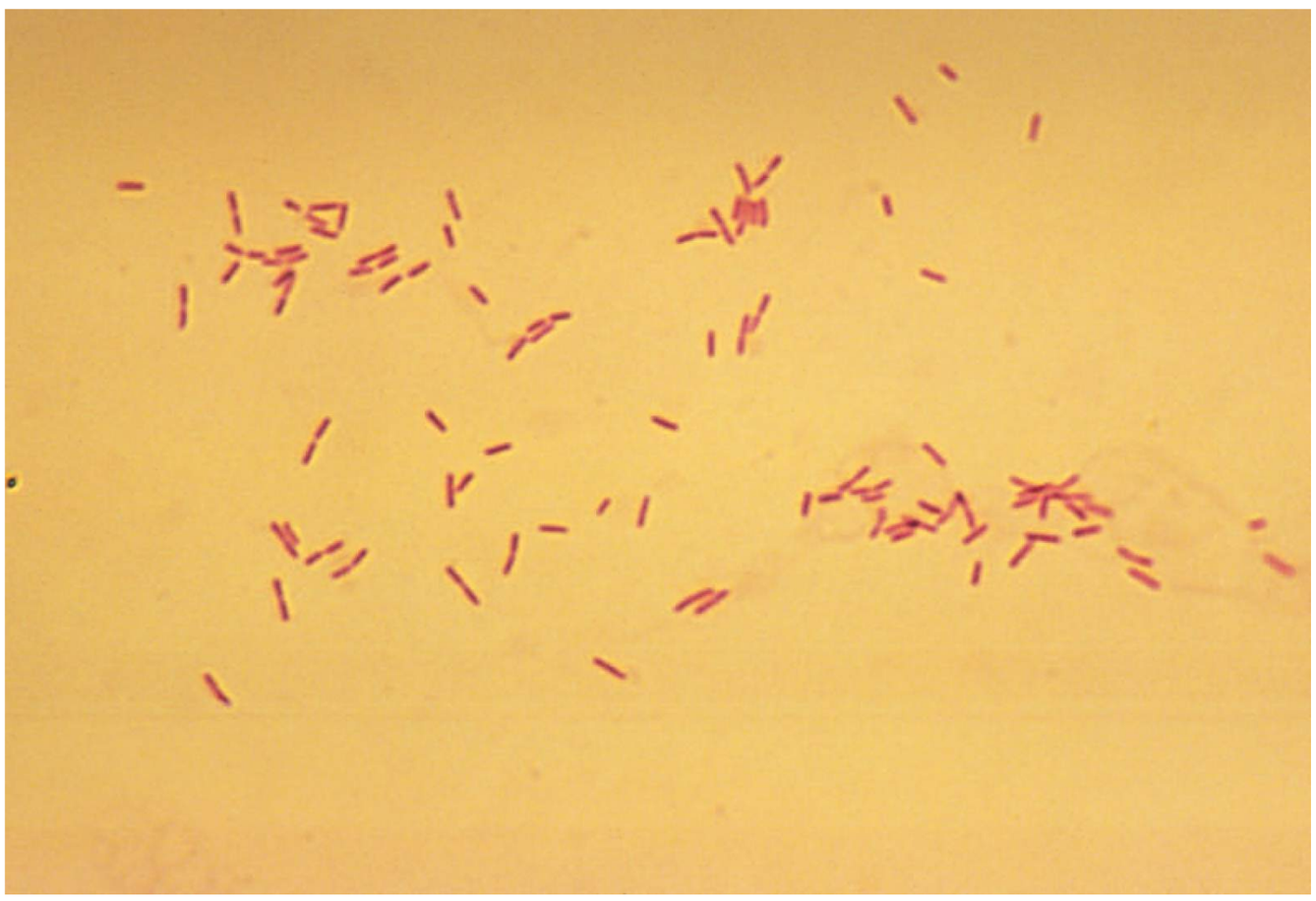


FIGURE 25.5 ■ **Bacteria.** Bacteria are often seen in urine specimens and either can be consistent with infection or may result from local contamination from surrounding skin during specimen collection. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc., Nutley, NJ.)

SYNOVIAL FLUID ANALYSIS FOR CRYSTALS

Uses

To determine the presence of uric acid crystals (in patients with gout) or calcium pyrophosphate crystals (in patients with pseudogout) in joint fluid.

Materials

Freshly collected joint fluid, glass microscope slide, coverslip, and polarizer.

Method

1. To prevent interference from polarizing artifacts, clean the slide and coverslip with alcohol prior to using them.
2. Using freshly collected unspun joint fluid, place a drop of joint fluid on the glass microscope slide.
3. Overlay coverslip.
4. View the slide using the polarizer.
5. Scan at ×10 power; ×100 power is needed to see intracellular crystals.

FIGURE 25.6B ■ **Compensated Polarized Uric Acid Crystals (×500).** Once crystals are found with a direct polarizing light, identification is made by using a compensated polarized light. The yellow crystal is aligned parallel to the slow vibration component of the compensator (negatively birefringent). The blue crystal is perpendicular (crossed urate blue). (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 217.)

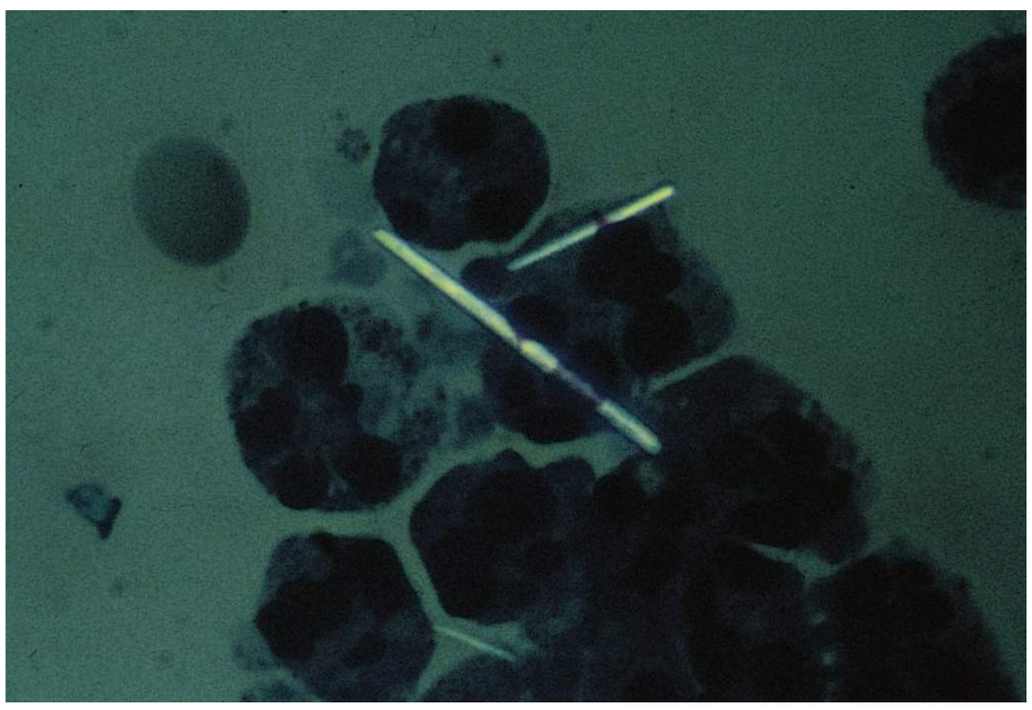


FIGURE 25.6A ■ **Polarized Uric Acid Crystals (×500).** Intracellular needle-like uric acid crystals are seen within the polymorphonuclear cells from the joint fluid in a patient with gout, using a direct polarizing light. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 216.)

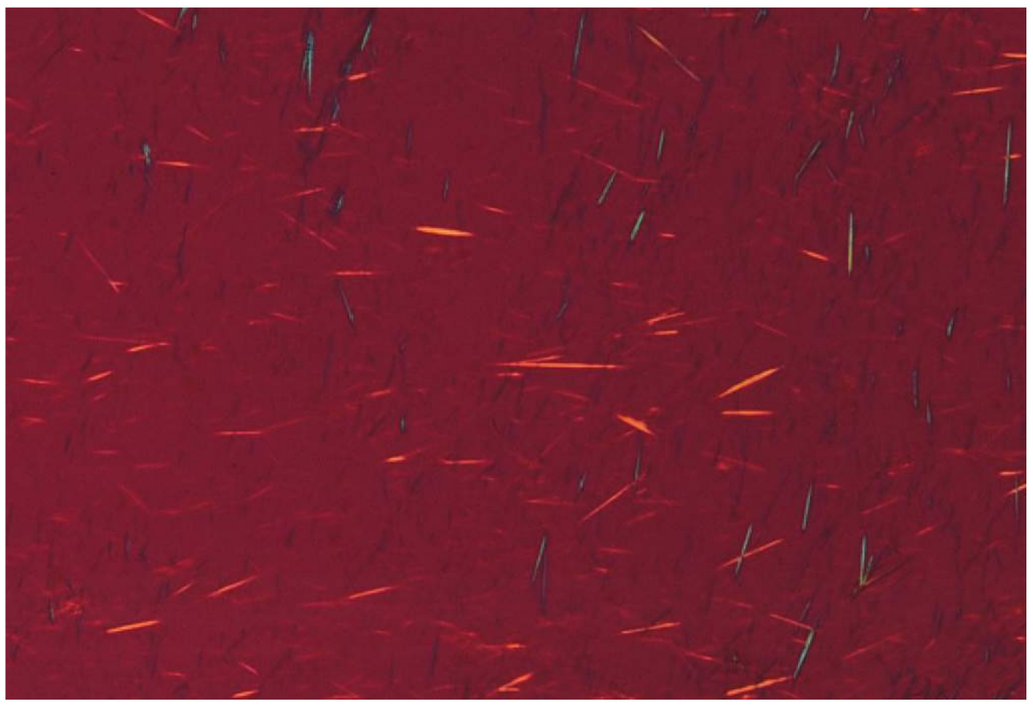


FIGURE 25.6C ■ **Extracellular Uric Acid Crystals (×100).** Extracellular uric acid crystals are seen under compensated polarized light. Notice the change of color with crystal alignment. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 216.)

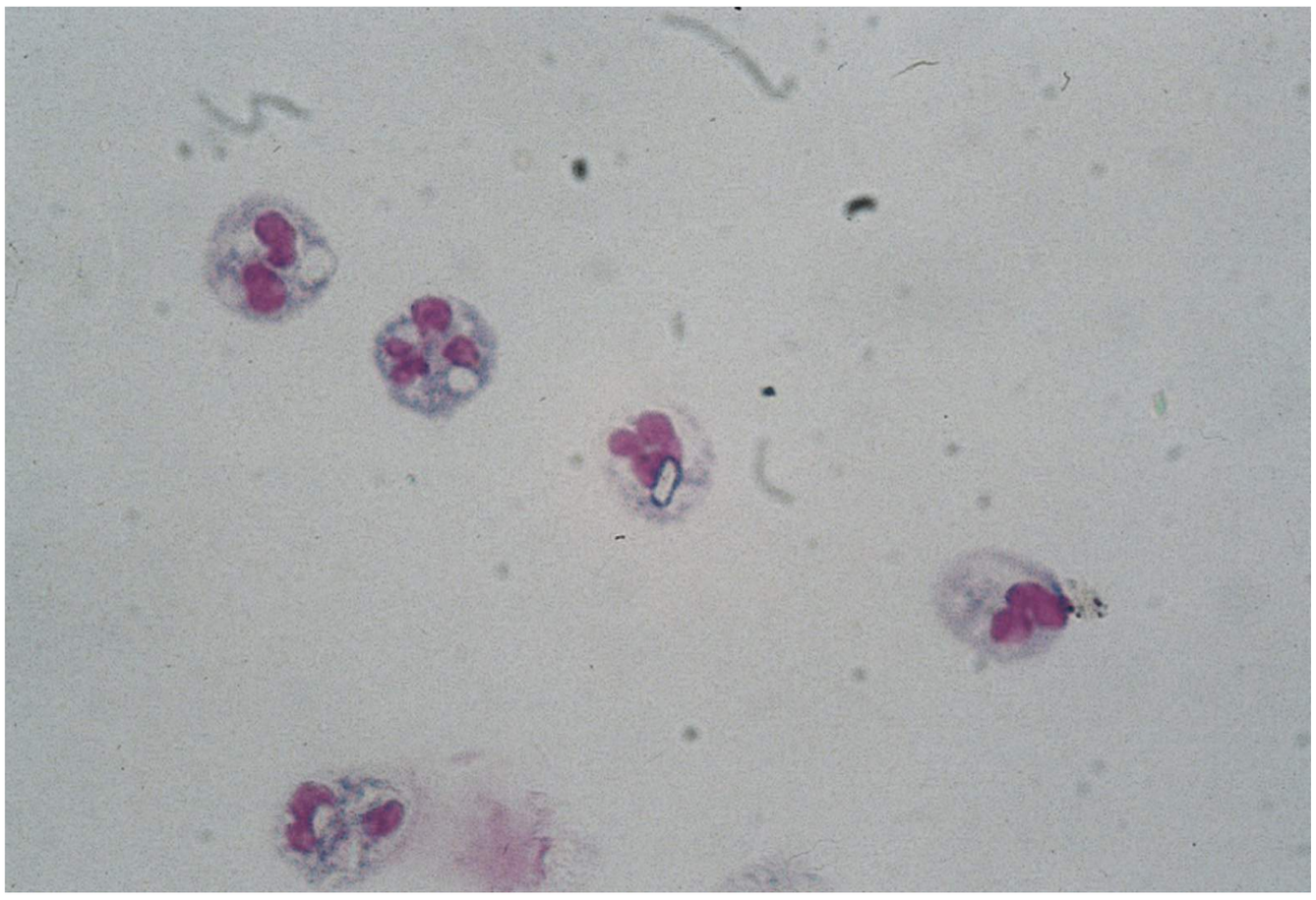


FIGURE 25.7A ■ **Polarized Calcium Pyrophosphate Crystals (×1000).** Intracellular rhomboid crystals in the joint of a patient with pseudogout. They may also appear as rods. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 216.)

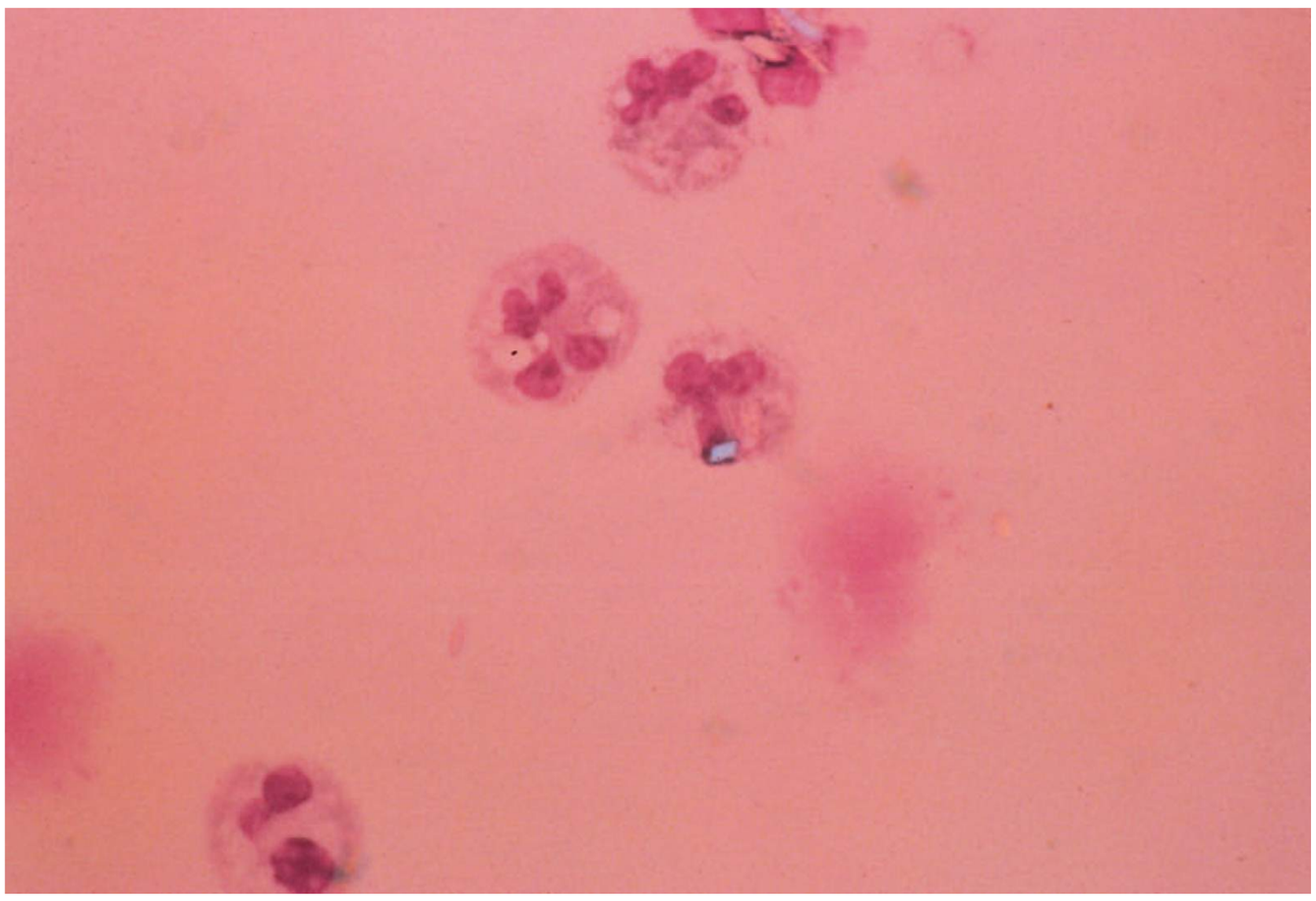


FIGURE 25.7B ■ **Compensated Polarized Calcium Pyrophosphate Crystals (×1000).** The blue calcium pyrophosphate crystal is aligned parallel to the slow vibration component of the compensator (positively birefringent). (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 217.)

GRAM STAIN

Uses

The first step in the identification of a predominant bacterial organism in a specimen. Classifies an organism by its cell wall's ability to retain crystal violet dye during solvent treatment. Morphology of the organism is also identified.

Materials

Freshly collected specimen to be examined, glass microscope slide, crystal violet, Gram iodine, acetone-alcohol (acetone, 30 mL, and 95% alcohol, 70 mL), safranin, and Bunsen burner.

Method

1. Put specimen on dry, clean glass microscope slide and allow to air dry.
2. Heat-fix specimen by gently passing over flame.
3. Cover specimen with crystal violet for 1 minute.
4. Rinse off completely with water; do not blot.
5. Cover specimen with Gram iodine for 1 minute.
6. Rinse off completely with water; do not blot.
7. Decolorize for 30 seconds with gentle agitation in acetone-alcohol.
8. Rinse off completely with water; do not blot.
9. Cover with safranin for 10 to 20 seconds.
10. Rinse off completely with water and let air dry.

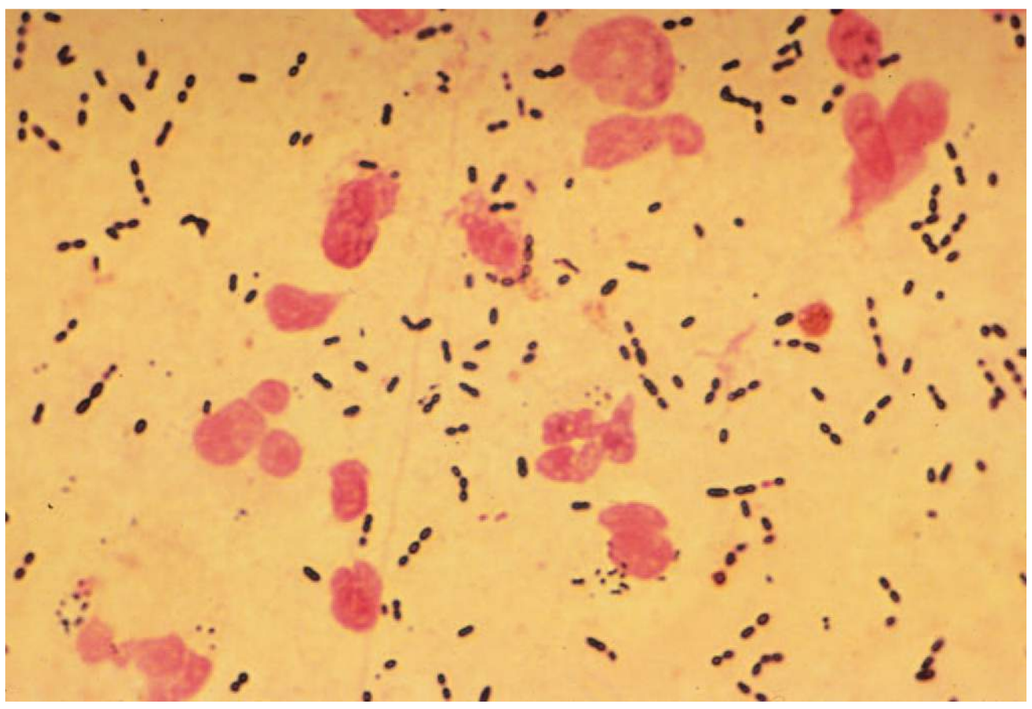


FIGURE 25.8 ■ **Gram Stain**—Streptococcus pneumoniae. Gram-positive, kidney-shaped diplococci of S pneumoniae. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc. Nutley, NJ.)

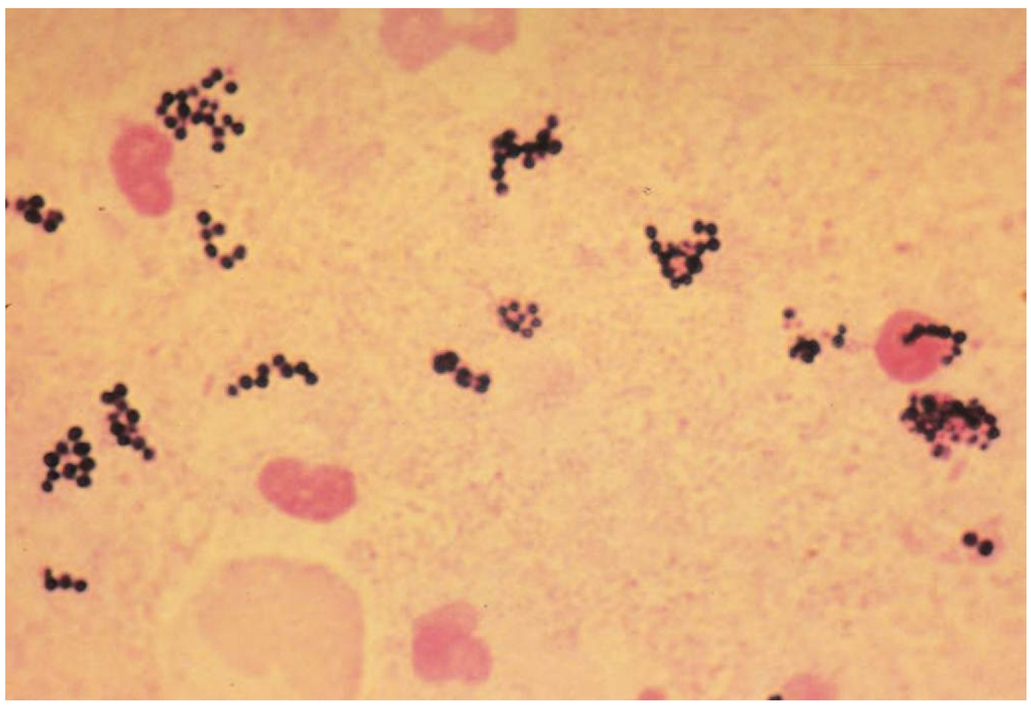


FIGURE 25.9 ■ **Gram Stain**—Staphylococcus aureus. Small clusters of gram-positive cocci seen in S aureus infection. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc. Nutley, NJ.)

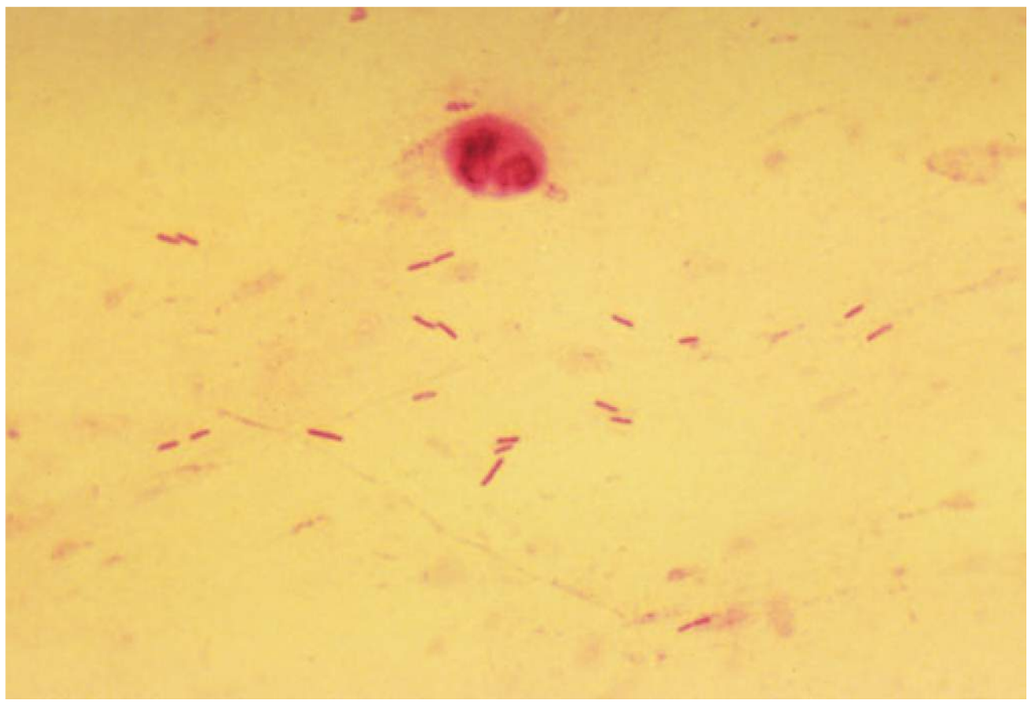


FIGURE 25.10 ■ **Gram Stain—Gram-Negative Rods.** Gram-negative rods of Pseudomonas aeruginosa. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc. Nutley, NJ.)

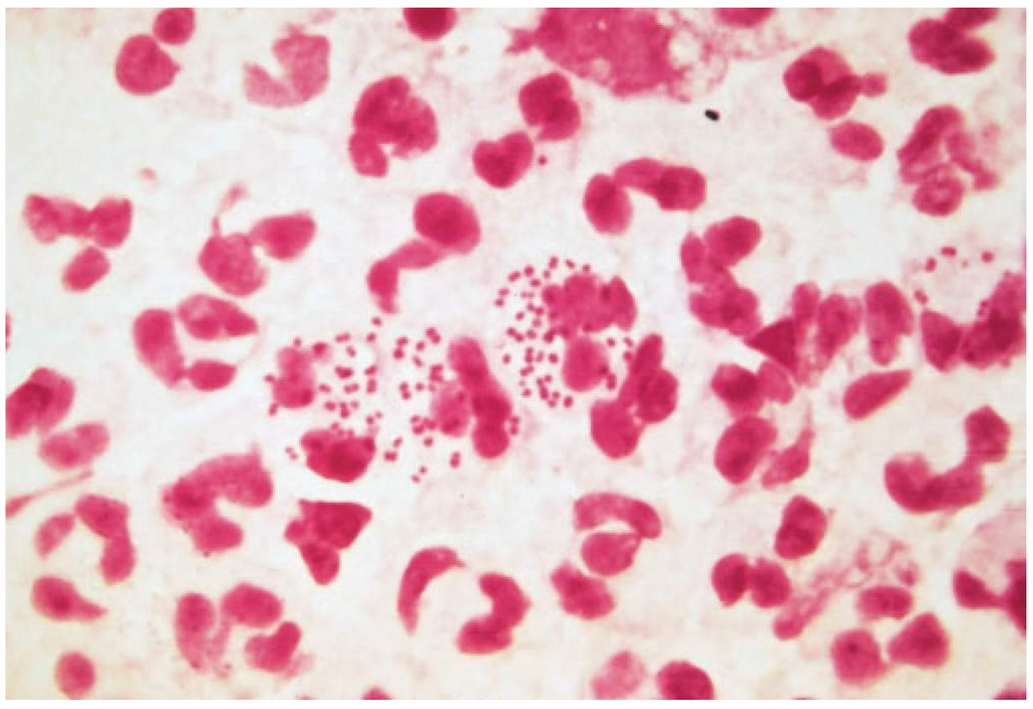


FIGURE 25.11 ■ **Gram Stain**—Neisseria gonorrhoeae. Multiple gram-negative, intracellular diplococci from a patient with N gonorrhoeae. (Reproduced with permission from Morse SA, Holmes KK, Ballard RC, Moreland AA (eds). Atlas of Sexually Transmitted Diseases and AIDS. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)

Uses

To evaluate a lesion (chancre, mucous patche, condyloma lata, skin rash) for the presence of Treponema pallidum.

Materials

Compound microscope with dark-field condenser (dark-field microscope), glass microscope slide, coverslip, and physiologic saline.

Method for Obtaining and Viewing the Specimen

1. From chancre or condyloma lata:
   a. Gently abrade the lesion with a dry gauze.
   b. Dab away any bleeding.
   c. Touch slide to exudative fluid in base of lesion.
   d. Overlay coverslip and view immediately under dark-field microscope using ×40 and ×100 objectives.
2. From mucous patch:
   a. Touch slide to mucous patch.
   b. Overlay coverslip and view immediately under dark-field microscope using ×40 and ×100 objectives.
3. From skin lesion:
   a. Gently scrape surface of skin lesion with edge of a number 15 scalpel blade.
   b. Dab away any bleeding.
   c. Touch slide to exudative fluid rising from skin lesion.
   d. Overlay coverslip and view immediately under dark-field microscope using ×40 and ×100 objectives.

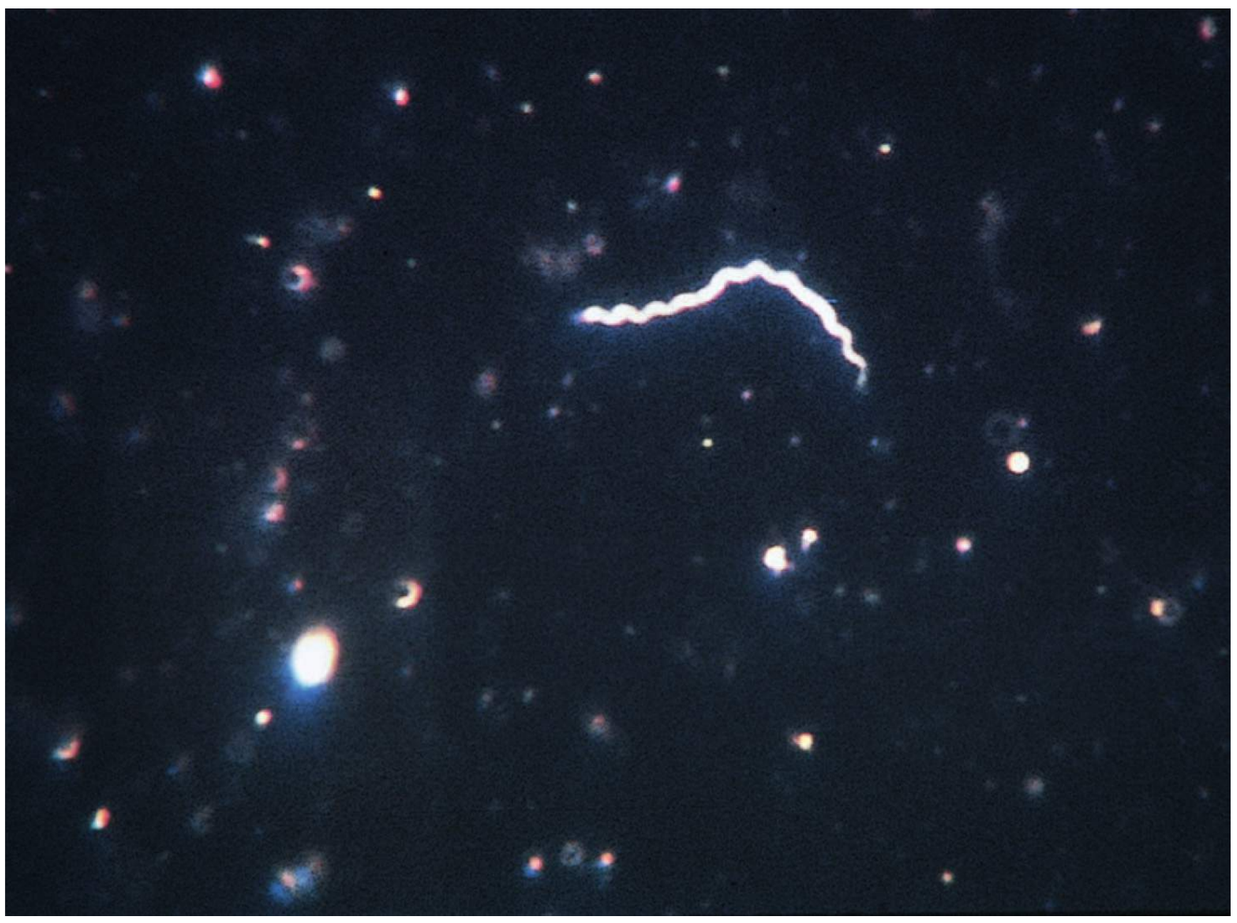


FIGURE 25.12 ■ **Dark-Field Microscopy.** Examined under a dark-field microscope at ×40 or ×100 power, spirochetes appear as motile, bright corkscrews against a black background. (Reproduced with permission from Morse SA, Holmes KK, Ballard RC, Moreland AA (eds). Atlas of Sexually Transmitted Diseases and AIDS. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)

VAGINAL FLUID WET MOUNT

Uses

To examine for clue cells, Trichomonas, and sperm.

Materials

Aqueous sodium chloride, glass microscope slide, and coverslip.

Method

1. Place a drop of saline onto the middle of the glass slide. (Alternative method: Place several drops of saline in a small glass test tube and place the swab in the tube. The swab can then be wiped onto a slide at a later time.)
2. Mix a small amount of vaginal fluid to be examined into the saline drop.
3. Overlay a coverslip.
4. Examine directly through microscope at ×40 and ×100 (oil immersion).

FIGURE 25.13 ■ **Clue Cells.** "Glitter cell" or "clue cell": Epithelial cell covered with adherent bacteria in a wet mount of a vaginal specimen from a patient with Gardnerella vaginalis (also known as nonspecific vaginitis or bacterial vaginosis). Note the refractile appearance, indistinct borders, and ragged edges of the epithelial clue cell. (Photo contributor: Curatek Pharmaceuticals.)

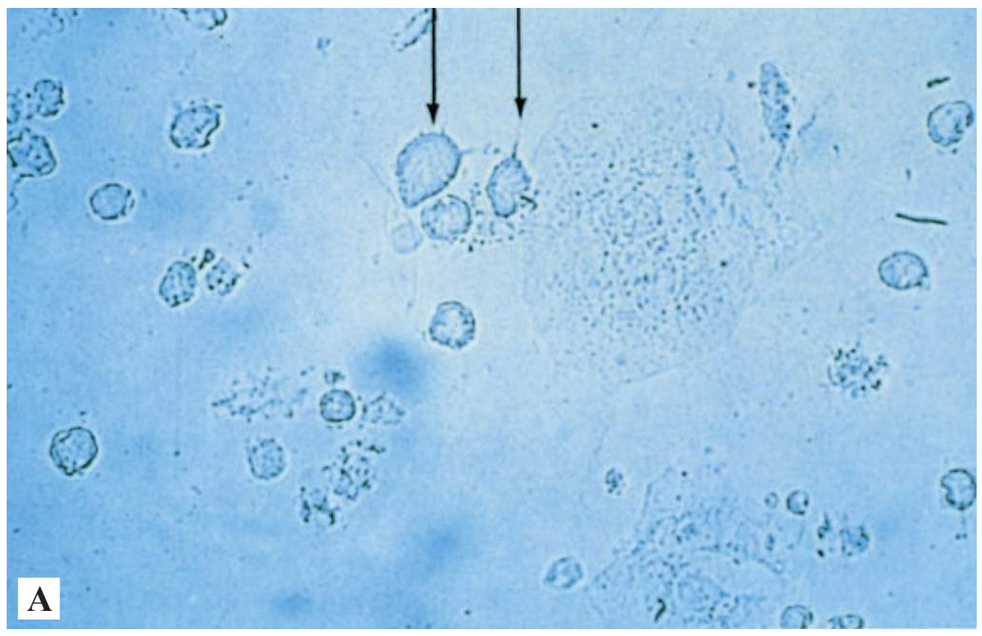


FIGURE 25.14 ■ Trichomonas. Saline wet mount demonstrating oval-bodied, flagellated trichomonads. They are similar in size to leukocytes and can be distinguished from them by their motility and presence of flagella. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)

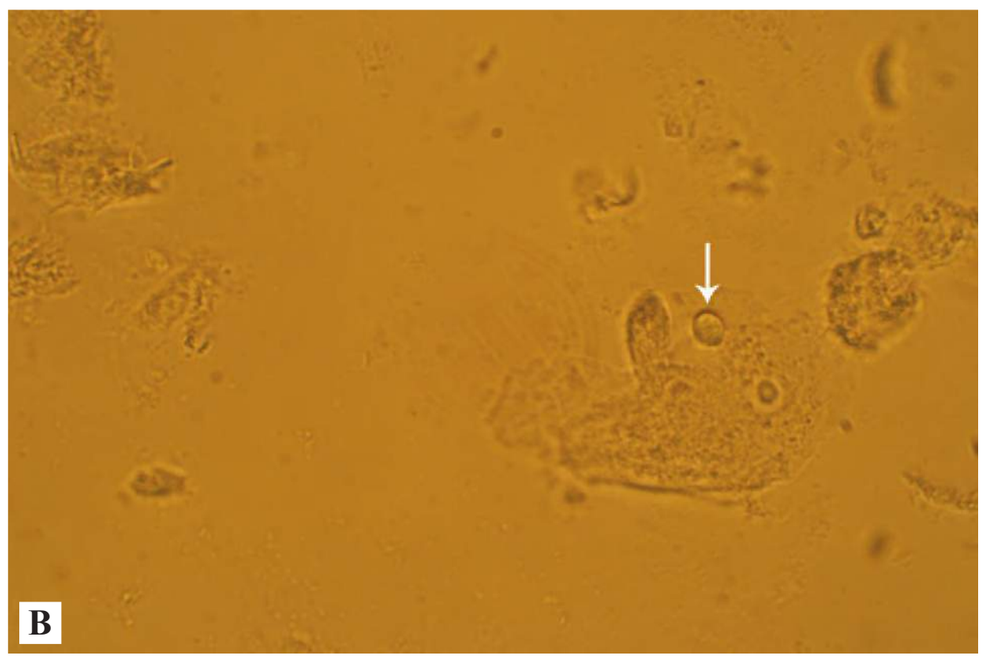


FIGURE 25.14 ■ Trichomonas. Saline wet mount demonstrating a single trichomonad. Its flagella are not easily seen in this photograph. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 25.15 ■ **Spermatozoa.** Spermatozoa may be motile or immotile. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 102.)

POTASSIUM HYDROXIDE MOUNT

Uses

To examine for yeast and fungus.

Materials

Aqueous potassium hydroxide (KOH) 10%, glass microscope slide, and coverslip.

Method

1. Place a drop of KOH onto the middle of the glass slide.
2. Suspend a small amount of vaginal fluid into the drop of KOH.
3. Overlay a coverslip.
4. Let sit at room temperature for 30 minutes; as an alternative, gently heat the slide over a Bunsen burner but do not boil.
5. Examine under microscope for hyphae and spores.

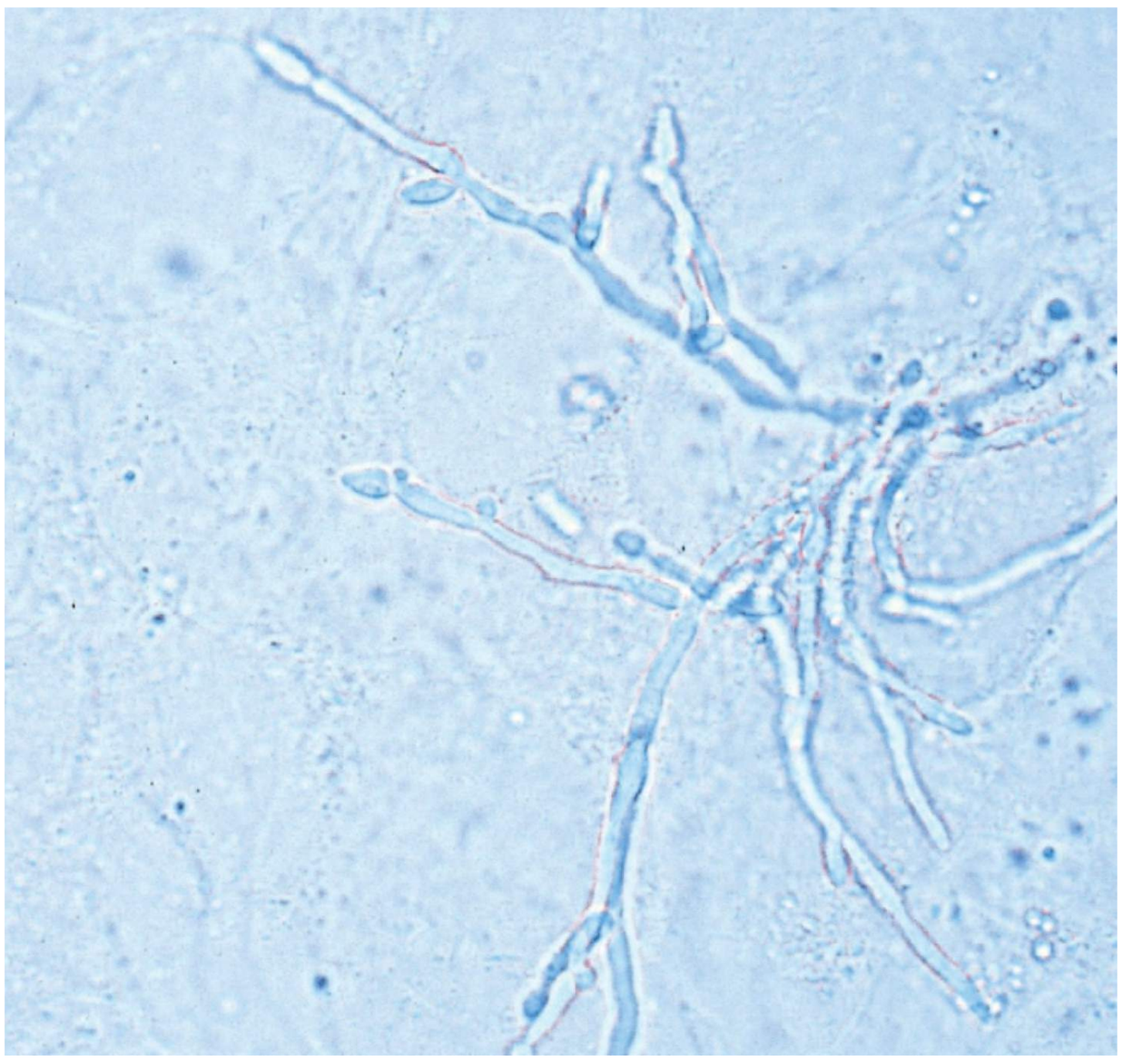


FIGURE 25.16 ■ Candida albicans. Potassium hydroxide preparation of vaginal secretions from a patient with vaginal candidiasis due to C albicans. Note the pseudohyphae characteristic of this organism. (Photo contributor: David A. Eschenbach, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)

STOOL EXAMINATION FOR FECAL LEUKOCYTES

Uses

To evaluate a patient's stool sample for the presence of fecal leukocytes.

Materials

Freshly collected liquid stool specimen, glass microscope slide, coverslip, and methylene blue.

Method

1. Place a drop of liquid stool onto the glass slide.
2. Add two drops of methylene blue to the stool specimen.
3. Mix thoroughly.
4. Overlay with a coverslip.
5. Place the edge of a piece of filter paper adjacent to the coverslip to absorb any excess methylene blue.
6. Examine using ×10 objective to scan specimen and ×40 and ×100 to identify specific leukocytes.

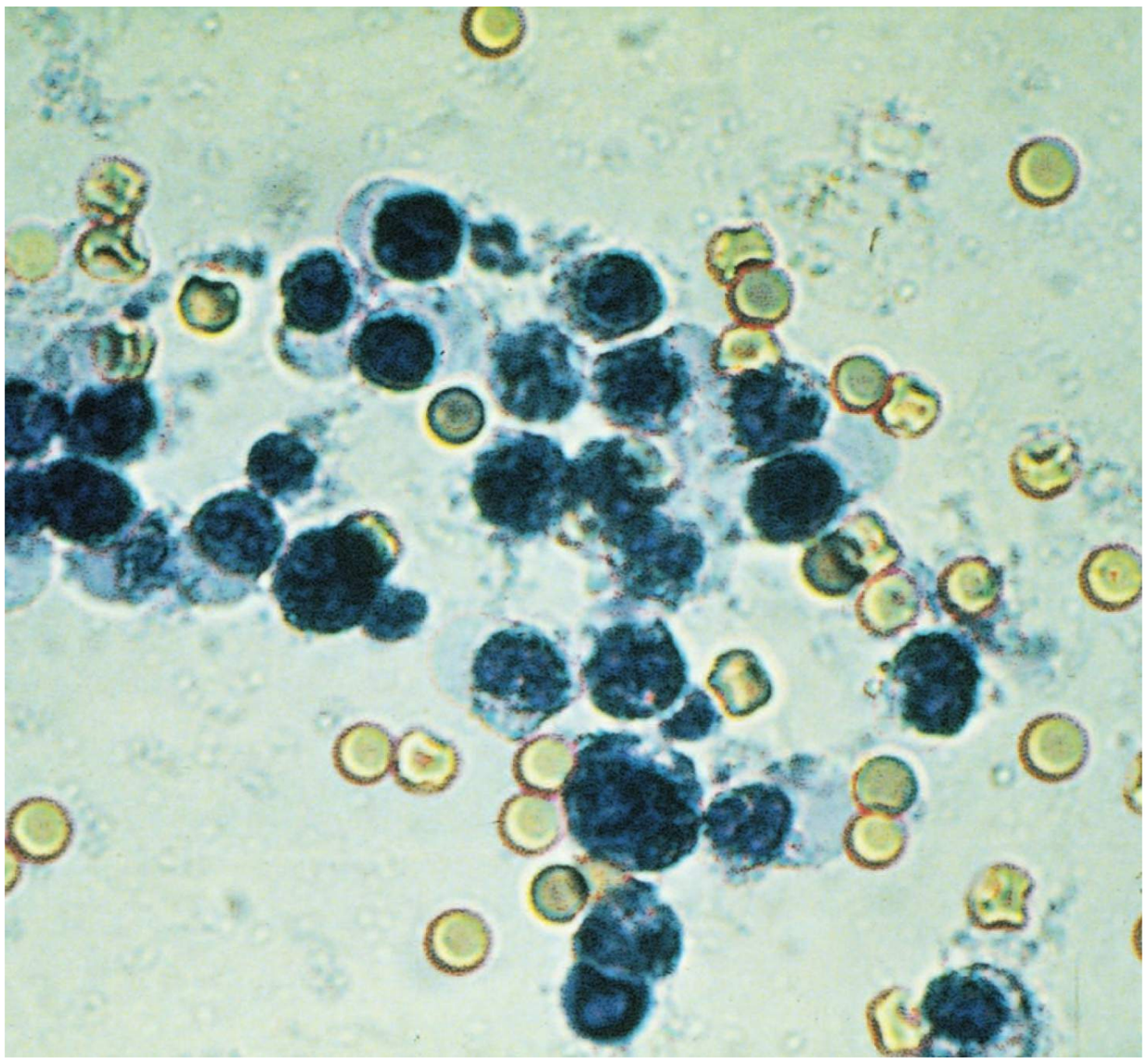


FIGURE 25.17 ■ **Fecal Leukocytes.** Multiple white cells in the stool specimen from a patient with bacterial diarrhea. (Photo contributor: Herbert L. DuPont, MD.)

SKIN SCRAPING FOR DERMATOSES AND INFESTATIONS

Uses

To determine fungal dermatoses or skin infestations.

Materials

Fresh skin scraping, glass microscope slide, coverslip, and 10% potassium hydroxide or mineral oil.

Method

1. Specimen collection:
   a. Gently scrape skin lesion with edge of a number 15 scalpel.
2. Slide preparation:
   a. Pediculosis may be seen grossly clinging to individual hair or under low power. Live nits may fluoresce with a Wood lamp.
   b. For scabies, place a drop of KOH or mineral oil onto the slide.
   c. Suspend a small amount of the scraping onto the drop.
   d. Overlay a coverslip.
   e. Let sit at room temperature for 30 minutes; as an alternative, gently heat the slide over a Bunsen burner but do not boil.
   f. Examine under microscope for hyphae, spores, or infestations.

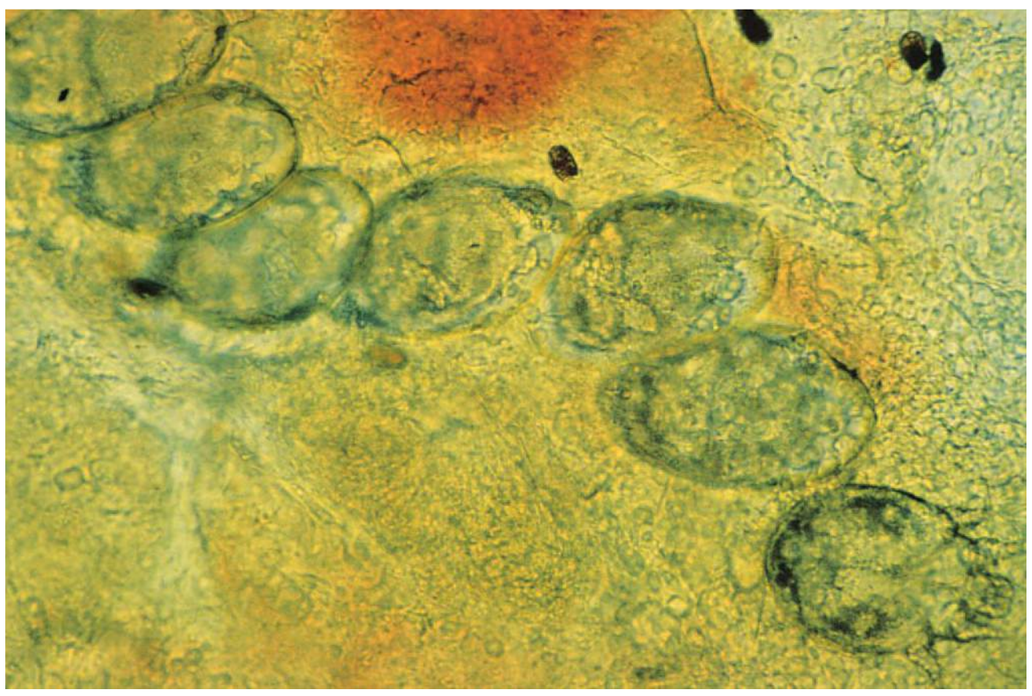


FIGURE 25.18 ■ **Scabies.** Skin scraping from a patient with scabies. Note the intact mite at the lower right of the photograph, and the ova and fecal pellets. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)

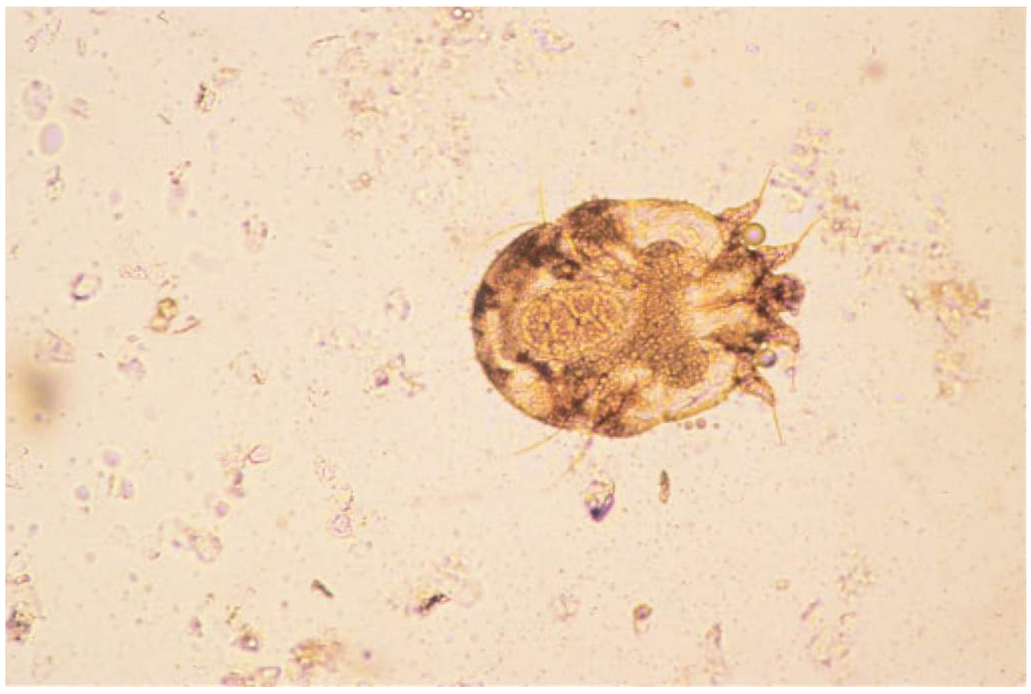


FIGURE 25.19 ■ **Scabies.** Adult female scabies mite. (Reproduced, with permission, from Morse SA, Ballard RC, Holmes KK, Moreland AA (eds). Atlas of Sexually Transmitted Diseases and AIDS. 3rd ed. Philadelphia: Mosby; 2003. Copyright Elsevier.)

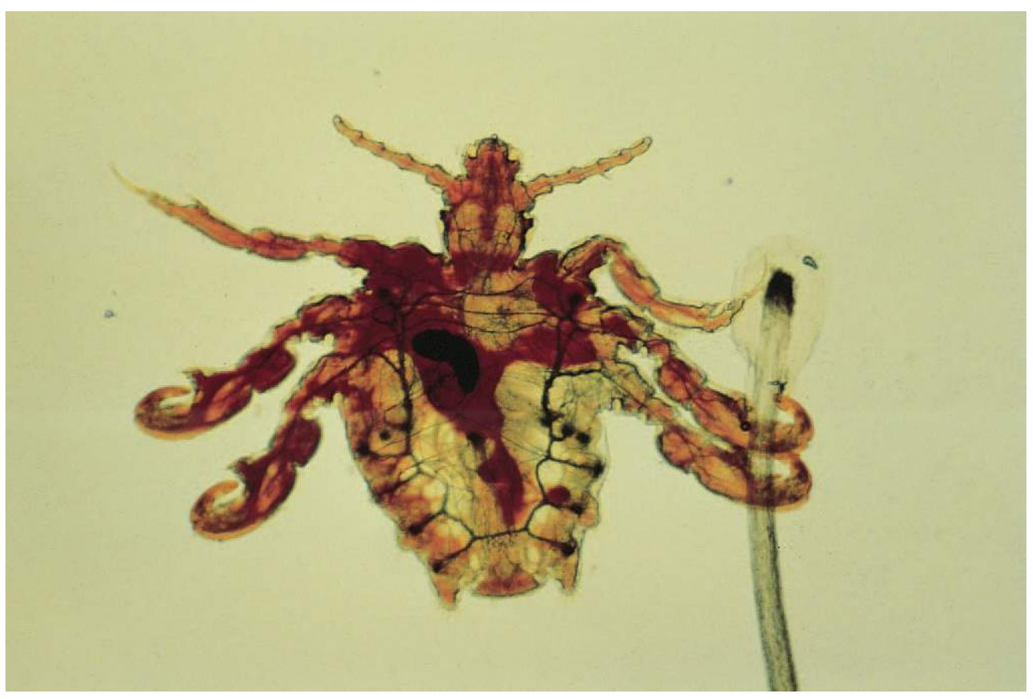


FIGURE 25.20 ■ **Pediculosis.** Phthirus pubis, the crab louse. Note the short body and claw-like legs, which are ideally suited for clinging to the hair shaft. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)

FIGURE 25.21 ■ **Pediculosis.** Phthirus corporis, the body louse. Note the elongated body. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)

CEREBROSPINAL FLUID EXAMINATION: INDIA INK PREPARATION

Uses

To examine cerebrospinal fluid for organisms with capsules, particularly Cryptococcus neoformans.

Materials

India Ink, glass microscope slide, and coverslip.

Method

1. Lightly centrifuge cerebrospinal fluid to concentrate cells at bottom of tube (1-2 minutes).
2. Pour off excess fluid (retain if further testing may be necessary).
3. Take a drop from the bottom of the centrifuge tube and place it in the middle of a glass microscope slide.
4. Place a drop of India Ink into the specimen drop; gently mix.
5. Overlay a coverslip.
6. Examine at ×10 to screen specimen, use ×40 objective to confirm findings.

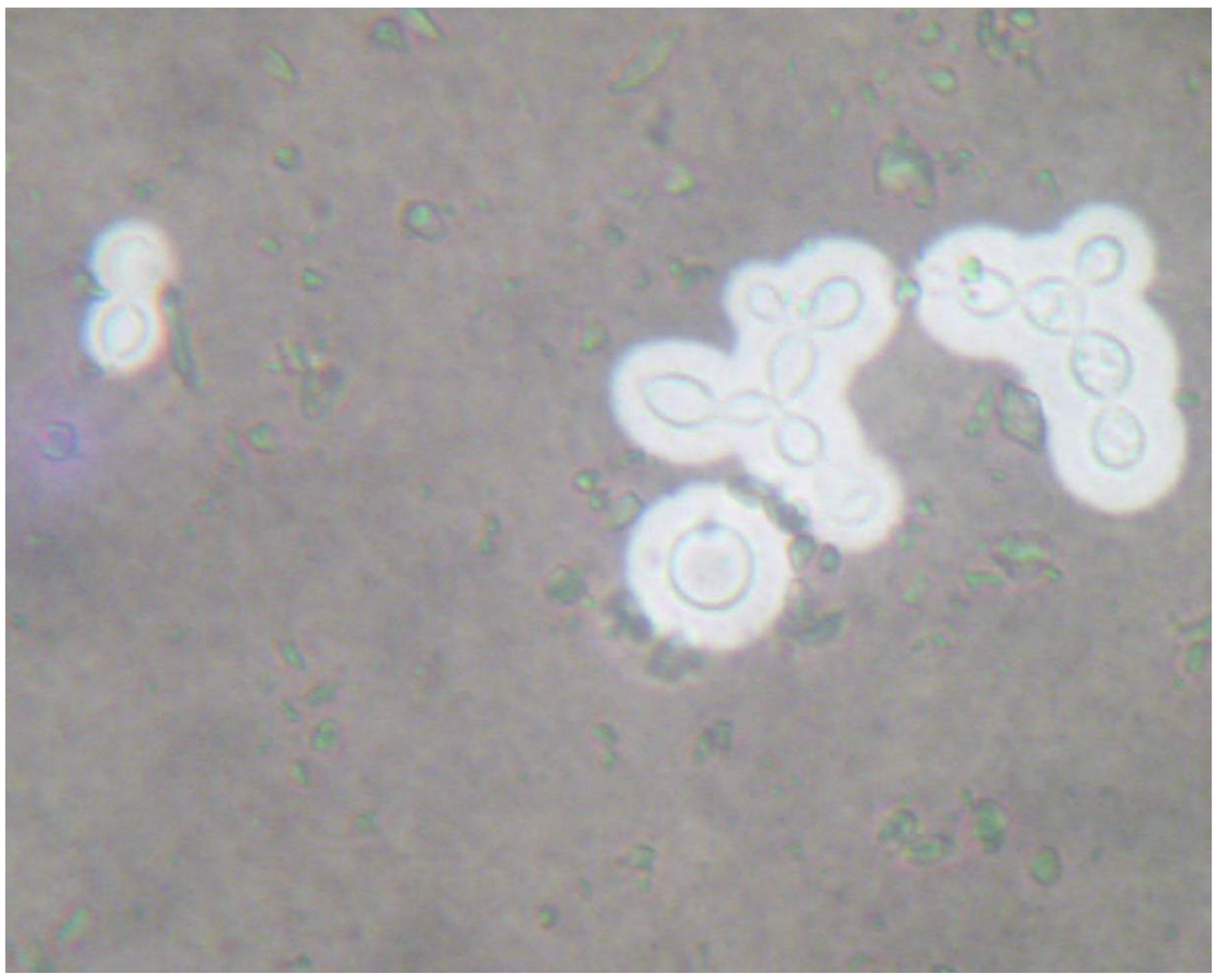


FIGURE 25.22 ■ **India Ink Preparation.** Budding yeast with prominent capsule on India Ink preparation from a patient with C neoformans meningitis. (Photo contributor: Seth Wright, MD.)

WRIGHT STAIN—THIN SMEAR FOR MALARIA

Uses

To evaluate for the presence of ring trophozoites.

Materials

Air-dried blood smear, Coplin jar of Wright stain, slide rack, pH 7.2 buffer, and blotting paper.

Method

1. Place a drop of blood on the middle of a slide.
2. Hold another slide evenly on top of the slide at a 45-degree angle and drag the slide over the drop of blood to the opposite edge to spread the blood evenly.
3. Allow the blood to dry for 5 to 10 minutes.
4. Stain air-dried smears in a closed Coplin jar of Wright stain for 5 minutes.
5. Place the slide on a rack.
6. Rinse and treat with pH 7.2 buffer primed with 1 mL Wright stain per 400 mL for 3 minutes.
7. Rinse in pH 7.2 buffer for 20 seconds.
8. Blot dry and mount on microscope at ×100 (oil immersion).

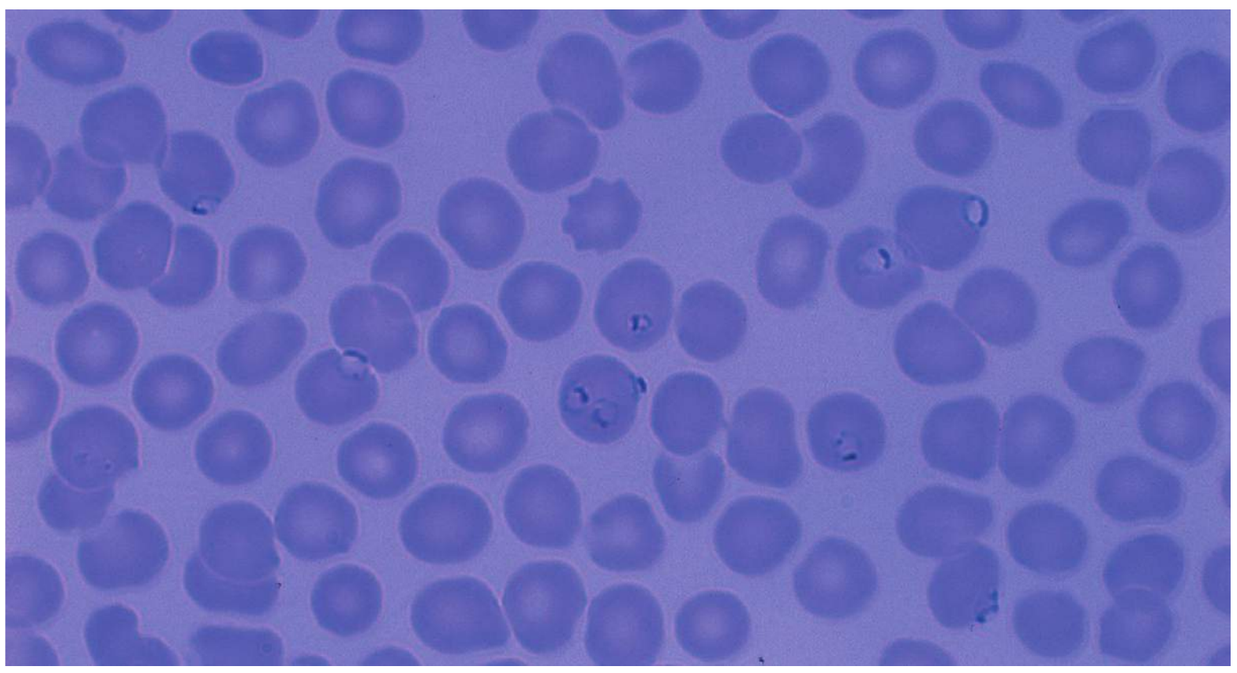


FIGURE 25.23 ■ Plasmodium falciparum **Thin Film.** Ring forms (trophozoites) of P falciparum are seen on the Wright stain thin film in a patient with intermittent fever who had recently traveled to Africa. (Photo contributor: James P. Elrod, MD, PhD.)

FERNING PATTERN FOR AMNIOTIC FLUID

Uses

To distinguish between amniotic fluid due to membrane rupture and normal vaginal secretions in patients beyond the 20th week in preganacy who present with spontaneous vaginal fluid passage. Characteristic arborization, or typical ferning pattern, confirms amniotic fluid and spontaneous membrane rupture.

Materials

Sterile speculum, vaginal fluid, microscope slide, microscope, and serile swab or pipette.

Method

1. Place patient in dorsal lithotomy position.
2. Do not use lubricants or cleaning agents.
3. Place sterile speculum into vaginal vault.
4. Obtain sample of vaginal secretions from posterior vaginal pool using a pipette or steril swab.
5. Place a drop of vaginal fluid on a microscope slide.
6. Spread the specimen evenly so that a thin smear is formed.
7. Allow the fluid to air dry for 5 to 10 minutes. Do not apply heat.
8. Examine slide under low power (10×) for ferning pattern.

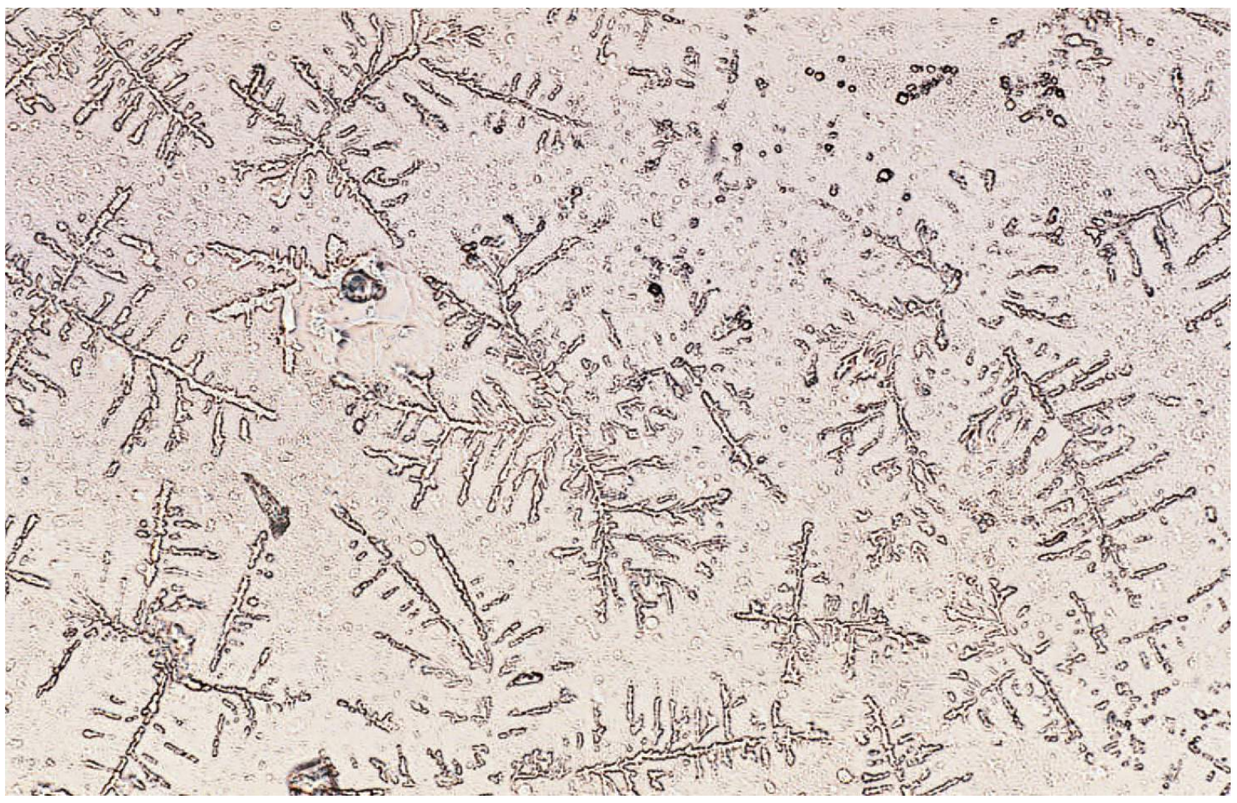


FIGURE 25.24 ■ **Ferning Pattern.** The arborization pattern found when a drop of amniotic fluid is allowed to air dry on a microscope slide, known as ferning. (Photo contributor: Robert Buckley, MD.)

PERIPHERAL BLOOD SMEAR FOR SCHISTOCYTES AND BLAST CELLS

Uses

To detect the presence of schistocytes or blast cells in a peripheral blood smear. Schistocytes are fragmented red blood cells due to shearing forces in microarterioles lined or meshed with fibrin strands. They are found in patients with disseminated intravascular coagulation, throbotic thrombocytopenic purpura/haemolytic uremic syndrome, microangiopathic haemolytic anemia, uremia, and carcinoma. Turbulent blood flow due to congestive heart failure, artificial heart valves, or vavlular stenosis may cause schistocyte formation. Greater than 1% of forms or greater than two schistocytes per high-powered field suggest schistocytosis.

Peripheral blood smear blasts are circulating immature white blood cells. When elevated white blood cell count and circulating blasts are seen in addition to anemia and thrombocytopenia, acute leukemia is suspected. As leukemia blast cells build up in the bone marrow, they allow less room for production of healthy white blood cells, red blood cells, and platelets. Although the diagnosis of leukemia can usually be made from the peripheral smear, bone marrow examination (aspiration or needle biopsy) is routinely done for definitive diagnosis.

Materials

Two glass microscope slides, drop of blood, pipette, Wright stain, Giemsa stain, immersion oil, and microscope.

Method

Smear Preparation

1. Agitate sample well, by inversion of tube or mechanical rocker.
2. Place a 2- to 3-mm drop of whole blood ¼ in from the right edge of a 1 × 3 in slide using a wooden applicator stick.
3. Place the slide on a flat surface and hold securely.
4. Grasp a second slide (spreader slide) in the right hand between thumb and forefinger.
5. Place the spreader slide onto the lower slide in front of the blood drop, and pull the slide back until it touches the drop.
6. Allow the blood to spread by capillary action almost to the edges to fht lower slide.
7. Push the spreader slide forward at a 30-degree angle, using a rapid even motion. The weight of the spreader slide should be the only weight applied. The drop of blood must be spread within seconds or the cell distribution will be uneven.
8. Allow to air dry.

Wright Stain of Peripheral Smear

1. Completely cover peripheral smear with Wright stain using a pipette. The stain layer should be ⅛ in thick.
2. Wait for 2 minutes.
3. Cover with equal amount of Giemsa solution.
4. Blow gently on the slide to mix solutions.
5. Allow to stand for 4 minutes.
6. Wash slide for 30 seconds with distilled water.
7. Allow to air dry
8. View smear with oil emersion at high (100×) power.

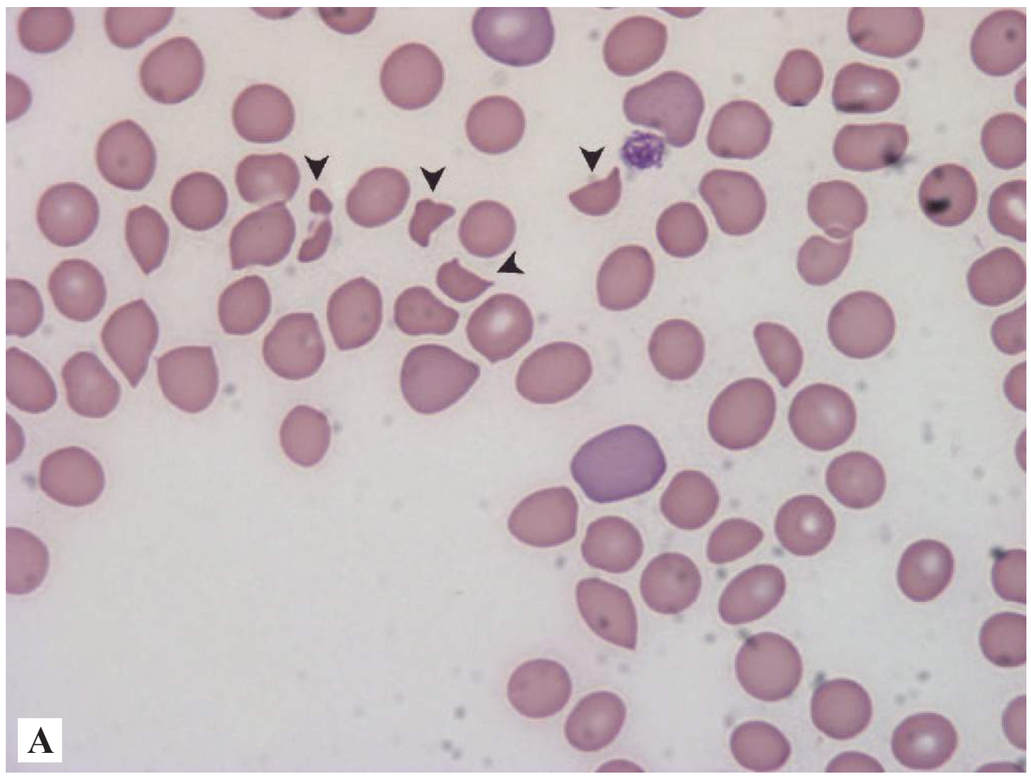


FIGURE 25.25A ■ **Schistocytes.** Multiple fragmented red blood cells (black arrowheads) seen on the peripheral smear at ×100 in a patient with microangiopathic hemolytic anemia. (Photo contributor: James P. Elrod, MD, PhD.)

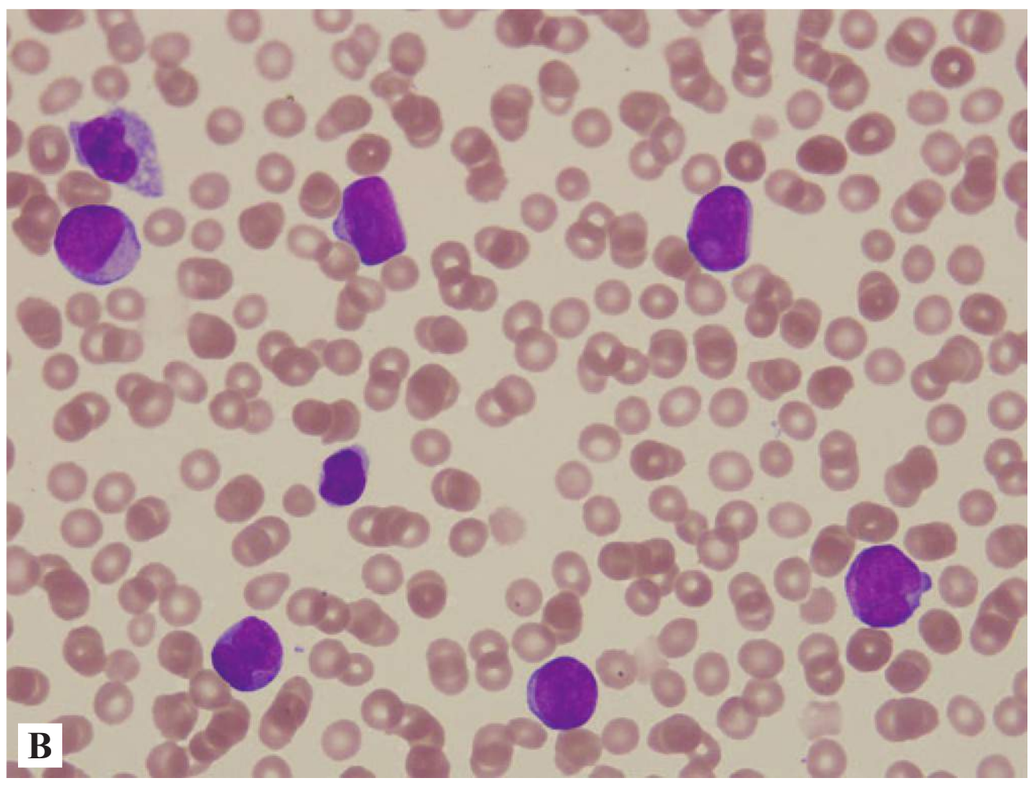


FIGURE 25.25B ■ **AML Blast Cells.** Wright stained peripheral blood smear, ×1000. Multiple blast cells seen in this smear from a patient with shortness of breath and a WBC of 105 K/uL, 80% blasts, HCT 20%, and PLT 75 K/uL. Flow cytometry confirms these are myeloid blasts conforming AML. (Photo contributor: Mary Ann Thompson Arildsen, MD, PhD.)

TAPE TEST FOR ENTEROBIAS VERMICULARIS EGGS

Uses

To detect the presence of eggs of Enterobias vermicularis in patients who present with nocturnal perianal pruritis or concern for pinworm infestation.

Materials

Microscope slide, clear transparent tape, tongue blade, microscope, or a cellulose-tape slide preparation.

Method

1. Affix ½ in of a 4-in piece of tape to the underside of a microscope slide.
2. Hold the slide against the tongue depressor 1 in from the end and lift the long end of the tape from the slide.
3. Loop tape over end of the depressor to expose gummed surface.
4. Hold tape and slide firmly against the tongue depressor.
5. Press gummed surfaces of the tape against several areas of the perianal area.
6. Affix long portion of the tape to the slide.
7. Smooth tape with cotton gauze.
8. View specimen under low (10×) power.

Note: Specimens are best obtained several hours after going to sleep or upon waking before a bowel movement or bath.

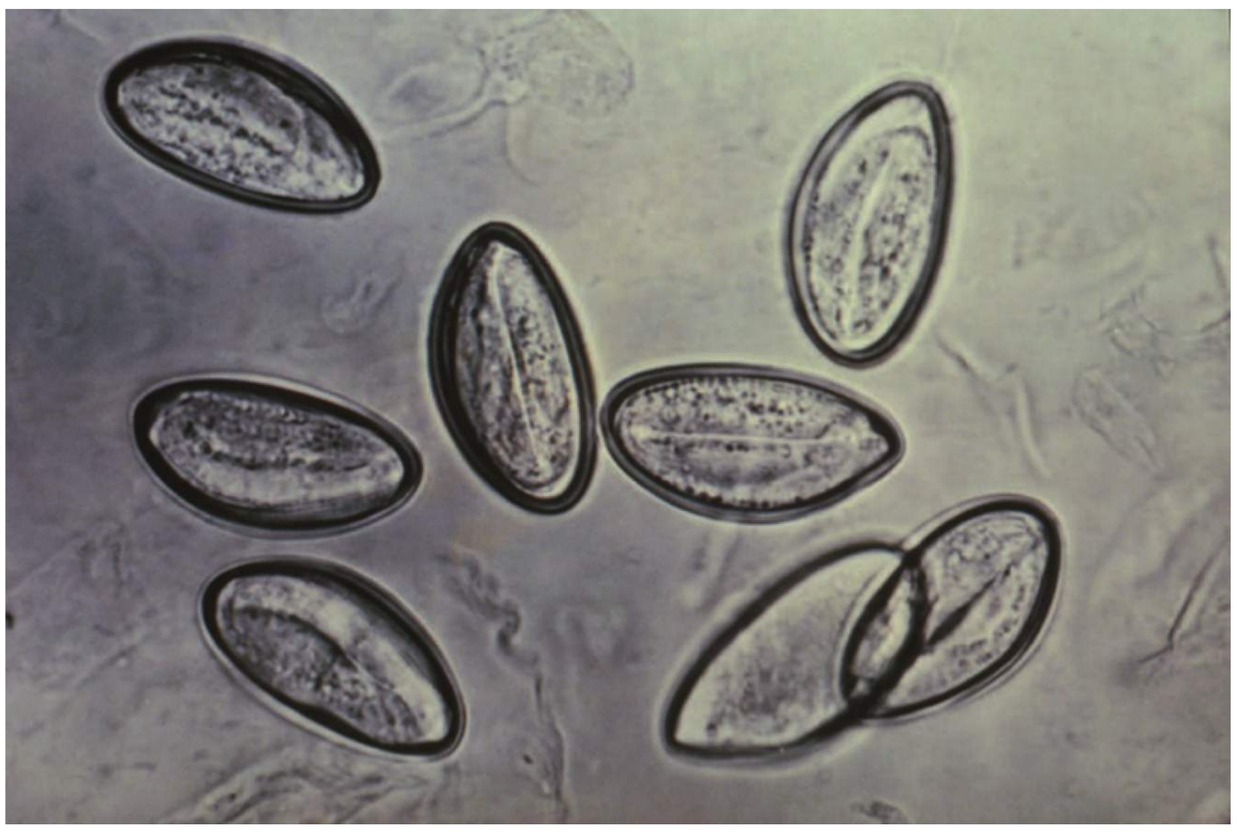


FIGURE 25.26 ■ Enterobius vermicularis. Characteristic appearance of the E vermicularis egg with contained larval form.

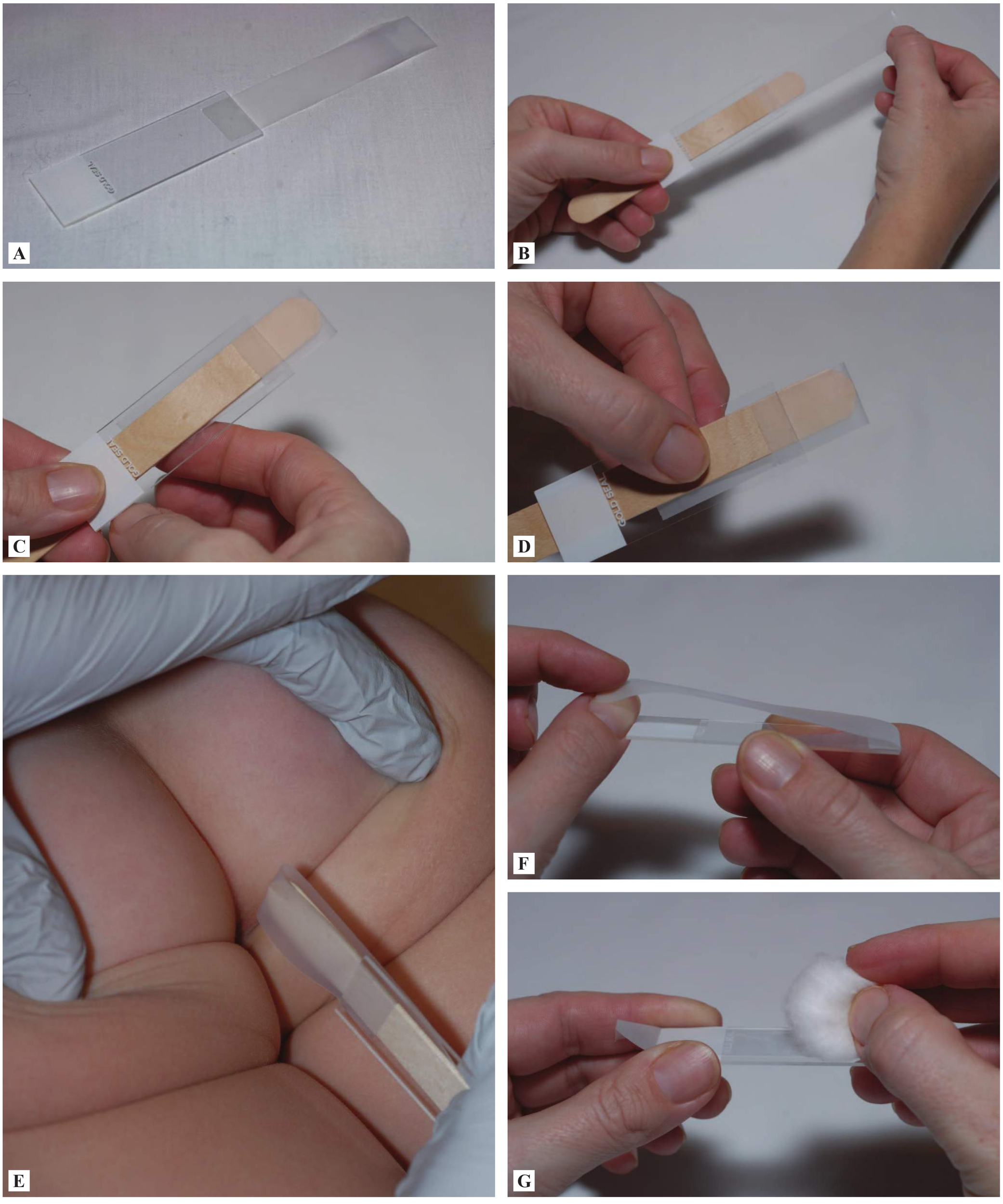


FIGURE 25.27 ■ **Cellophane Tape Preparation.** Steps for obtaining the eggs of E vermicularis. See text for details. (Photo contributor: Lawrence B. Stack, MD.)

TZANCK PREPARATION

Uses

To detect the multinucleated giant cells confirming the presence of a herpes infection.

Materials

Microscope, Bunsen burner, glass microscope slide, 5% methylene blue or Wright stain, or Geimsa stain, immersion oil, and sterile scalpel or hypdermic needle.

Method

1. Unroof a fresh, uncrusted vesicle with a sterile hypodermic needle or scalpel.
2. Scrape the floor of the vescicle with the scalpel and smear scrapings of the lesion onto a glass microscope slide.
3. Let air dry.
4. Fix specimen with absolute alcohol or gentle heat.
5. Stain with blue stain (5% methylene blue, Wright stain, Geimsa stain) for 5 seconds, rinse, and air dry.
6. View preparation through immersion oil at high power (40-50×).

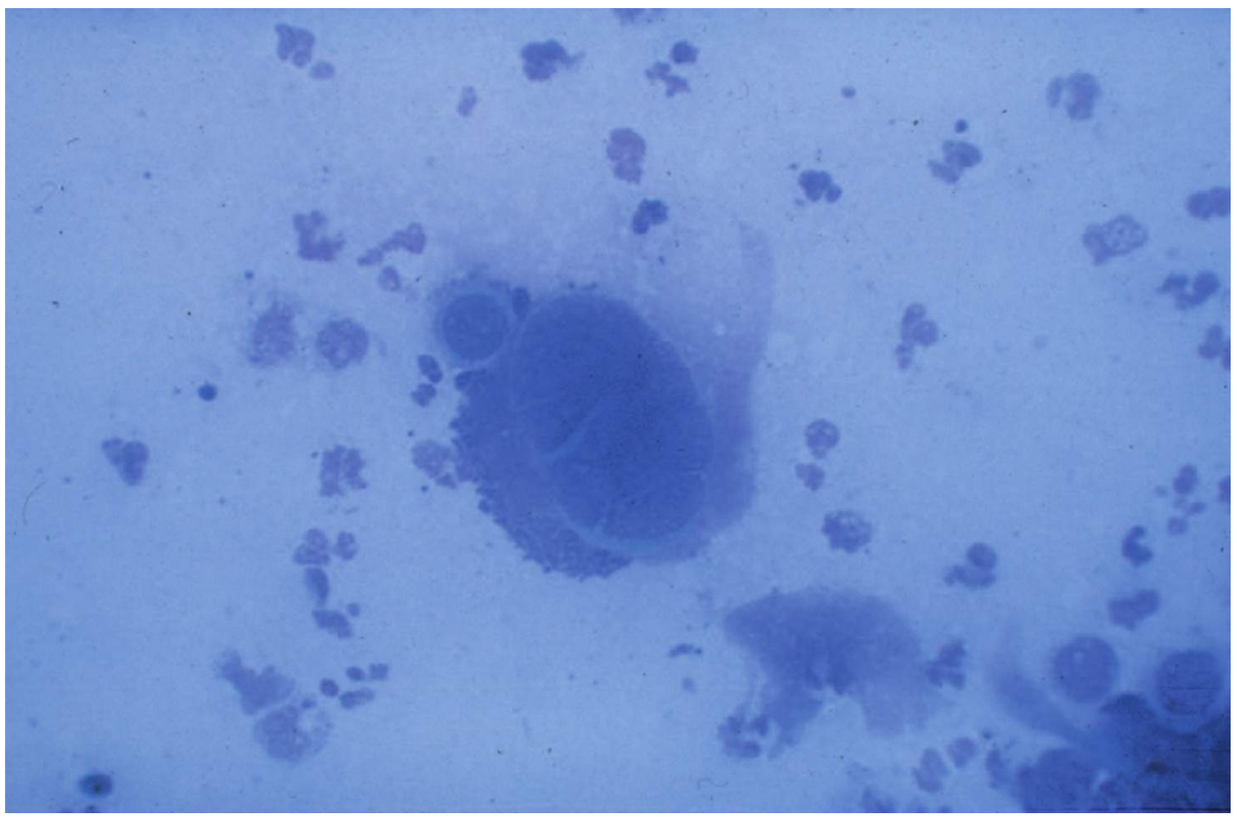


FIGURE 25.28 ■ **Tzanck Preparation.** A Tzanck preparation of both the roof and floor of a herpetic vesicle demonstrating a multinucleated giant cell. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brook Army Medical Center, San Antonio, TX.)

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